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A mathematical model of detection and dynamics of porcine transmissible gastroenteritis

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SUMMARY

Transmissible gastroenteritis (TGE) is a viral disease causing dehydration, diarrhoea and death in pigs. The disease is widespread in pig-producing areas of the world but does not occur in Australia. A mathematical model of TGE spread within a pig herd is proposed and calibrated by reference to published data. The model is then applied to two situations of special interest; first to estimate the delay before detection of TGE (6 to over 30 days) when infection is first introduced into a herd of domestic or feral pigs, and second the effect of the disease in a population of feral pigs (could become endemic if transmission is high).

INTRODUCTION

Transmissible gastroenteritis (TGE) is a contagious viral pig disease which causes high mortality of piglets [1] and has no effective treatment or vaccine [2]. As a result, it is of particular concern to pig producers where the disease occurs in north America, Europe and many parts of Asia [1]. TGE has not been eradicated from any country after it has become established, but it is currently not found in Australia and New Zealand. Effective control of TGE in Australia would be aided by early detection of any outbreaks.

Feral pigs are widespread in Australia, especially in the east and north [3]. The number of feral pigs is not known accurately [4]. Wild boar, the same species as feral pigs, has been reported in France as a host of TGE virus [5] though only 1 of 213 wild boar tested was apparently infected. Feral pigs in the USA had no detectable antibodies to TGE but did develop antibodies after experimental infection [6]. Contact between feral and domestic pigs in Australia occurs in and around small piggeries but is very unlikely in larger piggeries. Contact in and around the small piggeries may allow transmission of diseases, including those diseases spread by close contact, such as TGE.

The aim of this study was to predict whether TGE will establish in pig populations in Australia and the disease dynamics in each population should the disease enter Australia. On the basis of the results estimates are made of the likely

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delays to detection of the disease and hence start of disease control activities. Predictions are made, by mathematical modelling, of TGE establishment, dynamics and detection. Two different sorts of piggeries are examined; a breeding sow piggery with sale of older pigs as porkers or baconers, and a piggery with fattening pigs but no breeding. These types of piggeries represent a cross-section of the industry in Australia and are similar to the types of piggeries in other countries in which TGE has occurred. Piggeries were assumed to be of average size in Australia, that is with 328 pigs as of 1988 [7]. The role of feral pigs as alternate hosts of TGE is also assessed as Australia is different from Britain and north America in having large, widespread populations of feral pigs [8].

MODELLING

Model structure

Deterministic models are used here as they mix theory and data with good agreement for many diseases [9, 10]. A herd of pigs is assumed to consist of susceptible (X), latent (infected but not infectious L), infectious (Y) and immune (Z) individuals. Pigs change from X to L to Y to Z and maybe back to X, at certain *per capita* rates, as described in many models of infectious diseases [9–12]. The changes in the number of hosts in each segment or compartment of the herd can be represented by a series of differential equations.

$$dX/dt = -\beta XY + a(X+L+Y) - (b+c)X + wZ + i \quad (1)$$

$$dL/dt = \beta XY - (b+\sigma+c)L \quad (2)$$

$$dY/dt = \sigma L - (b+\alpha+\nu+c)Y \quad (3)$$

$$dZ/dt = aZ + \nu Y - (b+w+c)Z \quad (4)$$

where the *per capita* birth rate is a, the death rate is b, the transmission coefficient is β, the inverse of the latent period is σ, the mortality rate from TGE is α, the recovery rate is ν and the rate of loss of immunity is w. Purchases of pigs enter the population at a rate, i, and all purchases are assumed to be susceptible. Pigs are culled at a *per capita* rate c. The basic model does not include age classes of pigs. The within-herd model assumes homogeneous mixing of susceptible (X) and infectious (Y) pigs so that the number of new infections in a time interval produced by a given number of infectious pigs increases directly proportional to the number of susceptible pigs without limit.

All pigs born to susceptible, latently infected sows and infectious sows are assumed to be susceptible, and all piglets born to immune sows are assumed to be immune [2], though immunity is lost following weaning [13] with the loss of maternally derived antibodies. The modelling requires several assumptions about the disease in pigs. The population of pigs in Australia is assumed to be susceptible initially. It is assumed that there is no density-dependent mortality in domestic pigs as feed would not be a limiting factor for domestic pigs. The fattening herds are operated as all-in and all-out herds. There is no evidence of a true carrier state [14], and the role of carrier pigs was difficult to assess [2] so no carrier pigs have been included in the model. It is assumed that vertical transmission does not

occur. The model is of a single species and hence any possible role of other species, such as birds, is not considered.

The disease will establish in a population if the number of susceptibles exceeds the threshold host number (K_T) [15]. The threshold is:

$$K_T = \frac{(b+\alpha+\nu+c)(b+\sigma+c)}{\beta\sigma} \tag{5}$$

The number of secondary cases of TGE per infectious pig (R_0) is the basic reproductive rate and is:

$$R_0 = \frac{X}{K_T} \tag{6}$$

If $R_0 = 0$ then the disease disappears when the infectious pig dies. If $0 < R_0 < 1$ the disease will gradually disappear. If $R_0 \geqslant 1$ the disease will establish. Hence R_0 is an estimate made on the first day of disease introduction of the expected number of secondary infections due to a single infectious pig.

The size and geographical distribution of a disease outbreak will be directly related to the time that elapses between introduction of the disease and its detection. It is expected that the longer the time until detection, the larger the outbreak. Hence early detection should assist disease control or eradication [16]. The duration of the time delay can be estimated from estimates of the number of cases expected to occur before the first case is detected (N), and a model of disease dynamics [17]. The number of disease cases before detection occurs was estimated from:

$$N = \frac{\log_{10}(1-P)}{\log_{10}(1-Q)} \tag{7}$$

where P is the probability of detecting with reasonable certainty at least one case and Q is the probability of detecting an individual case of TGE. The estimated number of cases is considered to be a maximum number. The time that would have elapsed was estimated from the number of cases and the dynamics of the disease predicted by the basic model.

In each simulation it is assumed that one infectious pig exists in the herd on day zero, and breeding herds and feral pig populations are modelled for 2 years and the fattening herd modelled for 140 days. In the breeding piggery it was assumed that pigs were retained for sale as either porkers or baconers. In the fattening piggery it was assumed that pigs were purchased as weaners and sold as a batch after 140 days with no culling until sale (an all-in all-out policy).

ESTIMATION OF PARAMETERS

Birth and natural death rates

Adult sows produce about 10 live piglets/litter and may produce 2–2·4 litters per year [18]. In breeding herds [19], adult sows are assumed to have an average of 20 piglets (10×2) per year, so the birth rate (a) was $20/365 = 0{\cdot}0548$/day. The birth rate (a) in the simulated average breeding piggery was assumed also to be 0·0548/day.

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A weighted average death rate in the absence of TGE was assumed to be 17% per year or $b = 0{\cdot}0005$/day ($=(-\ln(1-0{\cdot}17))/365$). The natural death rate of pigs in the average Australian piggery was assumed also to be 0·0005/day. The natural death rate of fattening pigs was assumed to be 16% over 140 days [20]. This is a rate (b) of 0·001/day ($=(-\ln(1-0{\cdot}16))/140$).

Latent period

The latent period between an individual being infected and being infectious was reported as 18 h–3 days [21], being within 18–24 h in many cases [22], 1–3 days [2] and 3·5 days in SPF piglets [23]. A mean of 2 days was used so the rate (σ) was $1/2 = 0{\cdot}5$/day.

Recovery rate

Infected pigs shed virus for about 2 weeks [2] or up to 3 weeks [24]. Assuming a mean duration of 2·5 weeks (17·5 days) the recovery rate (ν) was $1/17{\cdot}5 = 0{\cdot}057$/day.

Mortality rate

In the simulated breeding herd TGE-induced mortality was averaged across age classes. The disease-induced mortality in adults was assumed to be 10% and in piglets 95% in the first outbreak, so mean TGE-induced mortality for the herd was approximately 60%. The case mortality (cm) was converted to an instantaneous mortality (im) ($im = -\ln(1-cm)$), and then converted to a daily rate by dividing by the duration of the infectious period (17·5 days). The mortality rate (α) was then 0·0524/day. The death rate assumed for adults (10%) may be slightly high although this rate has been reported [25]. The effect on the mortality rate (α) is very small as α is heavily weighted by the very high piglet mortality. In the fattening pig herd, TGE-induced mortality was assumed to be 10% ($\alpha = 0{\cdot}0060$/day) over the infectious period (17·5 days).

Transmission coefficient

TGE virus is excreted in milk and faeces with the faeces–oral route considered the most important means of transmission [1]. The virus may also spread by aerosols although the importance of this means is unclear. Such spread has been considered to be important [26] and unimportant [2].

The estimation of the *per capita* transmission coefficient (β) is the most difficult part of any epidemiological model. An estimate of the transmission coefficient was obtained here by comparing predictions of the model with observed results from two herds; specifically two herds, S and M, described by another author [19]. Those herds were modelled as sufficient details of pig husbandry and death rates were described to permit simulation and the herds, each of 330 sows using outdoor housing, could be treated as replicates. Estimation assumed that disease transmission occurred between pigs. The original study [19] suggested birds may have been important in the introduction into one of the herds, herd S. The emphasis here is on what happened after disease introduction, so the potential role of birds is not considered.

The approach used was to fit, by trial and error, the predicted pattern of weekly piglet deaths to what has been reported [19]. As full results were not reported in the original studies the fit of the predicted to the observed could only be assessed by visual comparison. Ideally some statistical measure of goodness-of-fit would be used if full details of deaths were reported. The criteria used in the visual comparison were a sharp peak in deaths in weeks 3 and 4, and persistence of deaths over 2 years. Both were obvious in the observed results when survivors lost immunity (Fig. 1). The *per capita* transmission coefficient that gave the closest fit of the predicted to the observed was 0·0007 new infections per infectious pig per susceptible pig per day.

Culling and purchasing rates

Between 40 and 45% (mean 42·5%) of sows in the breeding piggery were culled per year and were replaced by susceptible sows [19]. Weaners were also culled. The culling percentages are similar to those reported in Australia [27]. In the disease model, pigs were culled from each segment of the population. The *per capita* culling (c) rate was assumed to be 0·0160/day and the purchasing rate (i) was $330 \times 0{\cdot}0160$ pigs/day, so culling and purchases balanced. In the simulated average breeding piggery the culling percentage was 41% [27] and the *per capita* rate of culling (c) was 0·0051/day and the purchasing rate balanced to it. It was assumed there was no culling ($c = 0$) or purchasing ($i = 0$) of pigs during the growth stages in the fattening piggery.

Loss of immunity

Few data are available on the duration of active immunity in pigs after oral infection with virulent TGE virus [28]. Pigs that have recovered are usually immune to subsequent challenge, but the age at initial infection and the severity of the challenge may greatly influence the completeness and duration of the immunity [2]. Young pigs that have recovered from TGE usually resist reinfection or challenge to virulent virus [29], though the statement was not substantiated.

It is assumed in the modelling that immune piglets lose immunity immediately after weaning [13] and that older pigs that have survived infection lose immunity after 3 months. The duration of 3 months was selected as the initial modelling (Fig. 1) showed agreement between observed and estimated deaths. In the average breeding piggery, the weighted *per capita* estimate of the rate of loss of immunity (w) was 0·0031/day. That corresponds to loss of immunity in adults after 3 months, adjusted for immune piglets born to immune sows. In the fattening piggery and the population of feral pigs, the *per capita* rate of loss of immunity was 0·0111/day. It is recognized that these estimates of the rate of loss of immunity are tentative and may be slight overestimates. However, the literature is not very specific and more research is obviously needed on this topic.

TGE in feral pigs

The dynamics of TGE in feral pigs were modelled assuming an introduction of 1 infectious pig into an isolated population of 328 pigs. Population parameters [30] were; birth rate (a) 0·0025/day, natural death rate (b) 0·00089/day, rate of increase (r) 0·0016/day, density dependent mortality 0·0000049/day. The latter

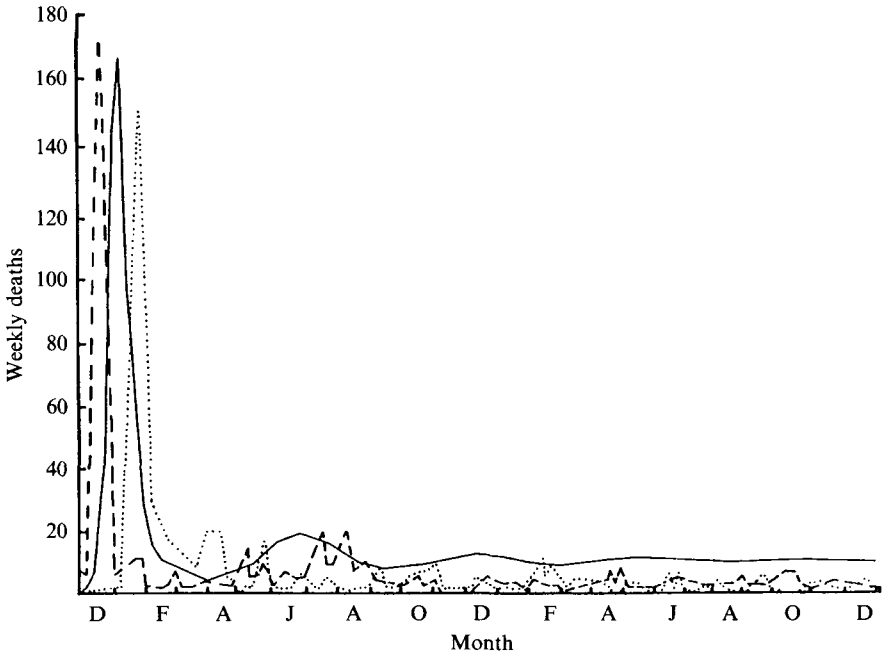

Fig. 1. Trends in deaths from transmissible gastroenteritis in two piggeries (dashed and dotted lines) [19] and the best-fit trend in deaths predicted (solid line) by a model described in the text. The model parameters are described in the text.

estimate corresponded to the change from the density model [30] to an abundance model here. Disease parameters were as for domestic pigs, except for the transmission coefficient.

The effects of varying the transmission coefficient and disease-induced mortality were investigated by sensitivity analysis. The transmission coefficient ($\beta = 0{\cdot}0007$/pig/day) and the mortality rate ($\alpha = 0{\cdot}0524$/day) used in the simulation of two herds [19] were used as starting points for the simulations and sensitivity analysis. Such a transmission coefficient from a domestic herd probably represents a higher rate than would occur in a feral pig population, so the lower transmission coefficient (0·00014/pig/day) is more realistic. A transmission coefficient of 0·00099/pig/day was estimated for classical swine fever in wild boar [31]. Classical swine fever is a highly contagious disease [1] so probably has a higher transmission coefficient than TGE in feral pigs.

RESULTS

Disease establishment

The estimates of threshold number of susceptible pigs, from equation 5, were 166 for the breeding herd, 92 for the fattening herd and 161 for feral pigs. The estimates of the basic reproductive rate (R_0), from equation 6, were 2 for the breeding herd, 4 for the fattening herd and 2 for feral pigs.

As the pigs in two herds [19] were kept outdoors, then the transmission coefficient of 0·0007/pig/day probably underestimates the rate of spread of TGE

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Table 1. *Sensitivity analysis of the expected maximum number of cases of TGE in pigs as estimated by equation 7 and the time until detection of the first case as estimated by numerical simulation of equations 1–4*

(The pig populations are the average breeding and fattening herds of 328 pigs and a population of 328 feral pigs.)

Reporting rate (Q)	Probability TGE is detected (P)	No. cases of TGE (N)	Days to detection		
			Breeding herd	Fattening herd	Feral pigs
0·10	0·95	28	> 30	29	> 30
	0·99	44	> 30	> 30	> 30
0·25	0·95	10	24	20	24
	0·99	16	30	24	29
0·50	0·95	4	15	14	15
	0·99	7	20	18	20
0·75	0·95	2	10	9	10
	0·99	3	13	12	12
0·90	0·95	1	6	6	6
	0·99	2	10	9	10

in a closed intensive piggery. If the transmission coefficient was five times higher (0·0035/pig/day), the threshold number in the breeding piggery is reduced to 33 susceptible pigs and the basic reproductive rate increased to 10. A similar change in the fattening piggery herd reduces the threshold number to 18 pigs and increases the basic reproductive rate to 20. The estimated transmission coefficient (0·0007/pig/day) may be too high for a population of feral pigs, so was reduced fivefold to 0·00014/pig/day. This reduced the basic reproductive rate to 0·4, in which case establishment should not occur, and increased the threshold number to 805 susceptible pigs.

Disease detection

The predicted maximum number of cases of TGE before initial detection was estimated using equation 7 and the predicted trends in infection (Table 1). With the highest chance of reporting an individual case ($Q = 0{\cdot}90$) and reasonable certainty that detection occurred ($P = 0{\cdot}99$) there was a delay of at least 9 days until detection in the breeding, fattening and feral herds. At low reporting rates, as is presumably more likely with feral pigs, detection occurred over 30 days after disease introduction in the breeding, fattening and feral herds. If the transmission coefficient was low (0·00014/pig/day), then it is highly likely that the disease will disappear before it is detected in feral pigs, because the disease persisted for less than 1 month under those conditions.

Feral pigs

The disease was predicted to disappear quickly from the feral pig herd if the transmission coefficient was very low and this was independent of the disease-induced mortality rate (Table 2). When the transmission coefficient was higher (0·0007/pig/day) the disease was predicted to persist over 2 years when disease-induced mortality was low.

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Table 2. *Sensitivity analysis of the predicted effects of TGE on a population of 328 feral pigs*

Transmission coefficient (/pig/day)	Disease mortality (/day)	Persistence (months)	Week of peak deaths	Peak deaths /day	Minimum no. pigs
0·00014	0·0128	< 1	1	< 1	328
	0·0524	< 1	1	< 1	328
	0·0920	< 1	1	< 1	328
0·0007	0·0128	> 24	9, 36, 62	1, < 1, < 1	200
	0·0524	> 24	10, 65	3, < 1	178
	0·0920	8	12	2	204

DISCUSSION

The mathematical modelling predicts that TGE is likely to establish in breeding and fattening piggeries of average size in Australia. The threshold number of susceptible pigs for TGE establishment is 90–160, depending on assumptions of disease transmission. To facilitate comparisons, the initial number of pigs in each piggery and in the population of feral pigs was assumed to be 328 pigs. This was the average number of pigs in piggeries in Australia in 1988 [7]. While this is the average herd size, about 20% of piggeries have more pigs and hence establishment is more likely in these. Repeated peaks in deaths were more frequent in large compared to smaller piggeries in England [24].

The effects of TGE control in Australia were not included in the modelling work, as it was assumed that control would be by slaughter, so there would be no pigs to include in the model. The modelling predicts that if the chance of detecting an individual case of TGE in a piggery is 0·90, then an outbreak will be detected within 1–2 weeks of virus introduction. If Australian pigs are susceptible to TGE, as evidence suggests they are [32], then the clinical signs may be more obvious than in countries where TGE is endemic and the outbreak may be detected more quickly. Any delay in detection, or incorrect diagnosis, may lead to an increased likelihood of movement of infected pigs between piggeries. Such movement may be after direct sale between piggeries or through markets. The latter are recognized as potential points of disease concentration [33].

Diagnosis of TGE is considered easier than for many other diseases [34]. However, because of the similarities of TGE to other causes of diarrhoea, diagnosis may be inaccurate [35]. The causes of diarrhoea in young piglets in the USA [36] and in Australia [1] have been described so misdiagnosis should not occur particularly as procedures for differential diagnosis of diarrhoea have been described [37]. The occurrence of misdiagnosis [35] was supported [24] as veterinarians in Britain often did not recognize signs of TGE. A severe form of TGE with high mortality was easily diagnosed by veterinarians in Quebec [38] although a milder (low mortality) form of TGE was difficult to differentiate from other forms of diarrhoea. In Singapore in 1985, 3 years after TGE was first diagnosed, it was clinically indistinguishable from severe *Escherichia coli* diarrhoea. Differential diagnosis could be made at post-mortem by examination [39].

TGE was often not diagnosed in finishing or adult pigs because of the mild signs.

such as reduction in appetite [2, 40]. A survey of pig diseases in Ohio estimated that a veterinarian was involved in only 20% of disease diagnoses, so incorrect diagnosis of diseases such as TGE must be expected [41]. A consequence of delayed diagnosis may be a need to trace movements of pigs, people and equipment onto and off infected piggeries. Tracing, and restrictions on further stock movements, could be expected as part of any TGE control or eradication efforts in Australia, since it is an exotic livestock disease.

The introduction of one infectious pig into an isolated population of susceptible feral pigs may result in endemic disease if the transmission rate or population size is high enough. The number of feral pigs in a population can exceed the estimated thresholds of 161 or 805; 1238 feral pigs in 120 km^2 of New South Wales wetlands [42], 180 feral pigs in 100 km^2 of Australian Capital Territory forest [43] and 1808 feral pigs in 295 km^2 of Northern Territory wetland [44]. The population size considered should be regarded not as the total over an extensive area but an in-contact population size within which homogeneous mixing can be assumed to occur. In the wetlands of New South Wales or the Northern Territory an infected feral pig could be in contact with over 161 other feral pigs in areas as low as 15 km^2 and 26 km^2 respectively, both areas being within potential home range sizes. Populations of feral pigs can be quickly and substantially reduced by shooting and poisoning [42–45] but whether those reductions would be sufficient to achieve TGE eradication is not known.

On the basis of the study piggeries most at risk of not correctly diagnosing a TGE infection appear to be those with older pigs such as fattening piggeries; where there is little or no veterinary involvement in disease diagnosis; and where endemic or sporadic diarrhoea associated with other pathogens occurs.

The piggeries most at risk of endemic TGE appear to be those with large numbers of susceptible pigs; continuous breeding of susceptible pigs; high numbers of purchases of pigs from other piggeries; and close contact between infected feral pigs and susceptible domestic pigs.

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