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The impact of compartmentalised housing on direct encephalomyocarditis virus (EMCV) transmission among pigs; insight from a model

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ABSTRACT

Although generally considered a rodent virus, pigs sometimes were suggested a potential reservoir host for encephalomyocarditis virus (EMCV), implying pig-to-pig transmission can cause major outbreaks in a pig population (basic reproduction ratio, $R_0 > 1$). An earlier experimental study on EMCV transmission among pigs was inconclusive in this respect ($R_0 \approx 1.24$; CI 0.4–4.4). In this study we used a simulation model to extrapolate the experimental results to commercial, compartmentalised pig housings and tested to what extend contacts between pigs in different pens needed to be reduced in order to prevent major outbreaks in a compartment following a single introduction. The final size of simulated outbreaks was measured and the probability to observe outbreaks that affected at least 50 or 80% of the pens was calculated. Simulation scenarios compare one homogeneously mixing compartment (no fence) to epidemiological theory and an increasing effect of fencing on the pig-to-pig transmission between pigs in neighbouring pens. For any $R_0 < 1.24$ the probability to observe outbreaks affecting more than 50% of the pens remained below 10% if compartmentalisation was introduced leaving per capita transmission rate unchanged. If fences also reduced contact transmission the probability to observe major outbreaks was below 50% for any $R_0 < 2.7$. Only for $R_0 > 4$, major outbreaks occurred with more than 50% chance even if only minimal contact between adjacent pens was allowed. In conclusion the results suggested that in a compartmentalised pig housing one single EMCV introduction is unlikely to cause a major outbreak by direct pig-to-pig transmission alone. Other mechanisms e.g. multiple introductions from a rodent reservoir may be required for large outbreaks to occur.

1. Introduction

Although encephalomyocarditis virus (EMCV) is generally considered a rodent virus it has been isolated from various other mammals as well (Tesh and Wallace, 1977). In the nineties encephalomyocarditis was diagnosed more frequently, which might reflect the emergence of EMCV in domestic pigs in Europe (Maurice et al., 2002). Infection with EMCV, an RNA virus (Minor et al., 1995), may result in acute myocarditis recognised by sudden death in young pigs up to 4 months old or reproductive failure in sows (Joo, 1999; Zimmerman, 1994; Acland, 1989; Gainer, 1967). Therewith EMCV outbreaks lead to undesirable consequences for pigs and farmer (Maurice et al., 2005; Koenen et al., 1999, 1996; Paschaleri Papadopoulou et al., 1994, 1990).

At current two routes of infection are suggested for the spread of EMCV within a pig farm. The first route is infection of pigs that ingest substances (e.g. feaces or carcasses) of infected rodents (Acland, 1989; Seaman et al., 1986; Littlejohns and Acland, 1975). The second route is direct horizontal or vertical pig-to-pig transmission (Foni et al., 1993; Koenen et al., 1999).

Domestic pigs have been suggested a potential reservoir host (Smith et al., 1992) which would imply that major EMCV outbreaks

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can occur in a pig population by pig-to-pig transmission alone. From an epidemiological point of view this means that an infectious pig in a population of susceptibles infects on average more than one other pig or in other words the basic reproduction ratio (R_0) exceeds one (Diekmann et al., 1990). This information is important because it would imply that the control of rodents (or any other potential non-pig transmission route) alone might not control EMCV infections on a pig farm. From experiments in pigs R_0 for EMCV infections was estimated at $R_0 = 1.24$, but the associated confidence interval (CI) was inconclusive with respect to the threshold value of 1 (95% CI 0.39–4.35; Maurice et al., 2002). The R_0-estimate derived from a field observation was also inconclusive ($R_0 = 1.36$; 95% CI: 0.93–2.23) and additionally is hampered by possible interference of other potential transmission routes (farmer, rodents) or multiple introductions in the stable (Kluivers et al., 2006).

Since the point estimate of R_0 is close to one, a very large animal experiment would be necessary to have sufficient power to conclude on the value of R_0, which was considered both unethical (mortality of pigs) and expensive. We applied a simulation model to evaluate pig-to-pig EMCV spread after a single introduction in a compartmentalised setting for the reported range of R_0-values (Maurice et al., 2002). We tested what effect fences between pens should have on direct contacts among pigs in adjacent pens in order to prevent major outbreaks in a compartment. The total per-pig frequency of making effective contact with other pigs in the compartment was kept constant (Bouma et al., 1995), to allow comparison of scenarios regarding different effect of the fences.

2. Materials and methods

A stochastic individual-based (pigs), time-discrete (days) and spatially explicit (configurations of pens) simulation model was developed in C++.

2.1. Modelling unit

The infection process was modelled on the level of individual pigs. An imaginary finishing pig compartment was created with two rows of 12 pens with 12 pigs each in total containing 288 pigs, following a commonly applied Dutch set up (Fig. 1). The compartment was treated as either a) one pen, e.g. to mimic random mixing of all pigs, or b) 24 separated pens, e.g. to simulate the spatio-temporal spread of the infection throughout the compartment.

2.2. Modelling pig-to-pig transmission

Assuming that EMCV could only be transmitted by direct pig-to-pig contact (Smith et al., 1992; Foni et al., 1993) and virus re-activation in pigs recovered from the disease does not occur frequently under standard rearing conditions, transmission of infection was modelled according to the standard SEIR-approach, using the states Susceptible (S), Incubating/Exposed (E), Infectious (I) and Removed (R) (De Jong and Kimman, 1994; Becker, 1989; Anderson and May, 1979). With I counting the number of infectious individuals, transmission was modelled using $\left(\beta \frac{I}{N}\right)$ as force of infection with β being a transmission rate and N the number of mixing pigs. Thus the resulting probability of infection (P_{SI}) for a susceptible pig in a group of randomly mixing mates is given by:

$$P_{SI} = 1 - \exp\left(-\beta \frac{I}{N} \Delta t\right), \text{ with } \Delta t = 1 \text{ day}. \quad (1)$$

The infection of susceptible pigs followed a Bernoulli chance process using P_{SI} as event probability.

The value for R_0 is calculated of β, N and the probabilities for the possible lengths of the infectious period as:

$$R_0 = \beta \cdot T_{INF}, \quad (2)$$

with T_{INF} being the average length of the infectious period.

Stochastic simulation of the infection cycle was performed by updating the status of each individual on a daily basis using the processes given in Table 1. The length of incubation and infectious period (both maximal 4 days) was determined for each infected pig individually by Monte Carlo sampling from the respective empirical distributions (Billinis et al., 2004).

2.3. Theoretical validation of the pig-to-pig transmission model

The model was run treating the whole compartment as one unit of 288 mixing pigs, assuming different values for R_0, calculating the respective value of β and randomly infecting one pig. Depending on R_0, Fig. 2 shows the outcome of the simulations as the final number of infected pigs per model run. This output data reflect the distribution of final size of the outbreaks in the homogeneously mixing compartment. The greater the assumed R_0 the more models runs ended with nearly all 288 pigs infected (compare line graphs in Fig. 2).

In order to check model validity the final size distribution obtained from the simulations was compared with predictions of the analytical theory based on the branching process approximation (Diekmann and Heesterbeek, 2000).

The general equation for the probability of a small outbreak in the branching process approximation using a discrete probability density of the infectious period $f(\Delta T_i), i \in \{1,\ldots,n\}$ results from the generating function

$$g(z) = \Sigma_{i=1}^{n} f(\Delta T_i)\, e^{\beta \Delta T_i (z-1)}. \quad (3)$$

The probability of a small outbreak, z_∞, can be calculated as the minimum root of $g(z) = z$ (Diekmann and Heesterbeek, 2000). Using $\beta = R_0/T_{INF}$ from (2) and $T_{INF} = 3$ from Table 1 i.e. $f(\Delta T_i) = \{0.25; 0; 0.25; 0.5\}$, for any value of R_0 (3) unfolds into (Schmid, 2015)

$$z = 0.25e^{\frac{R_0}{3}(z-1)} + 0\, e^{\frac{R_0}{3}2(z-1)} + 0.25e^{\frac{R_0}{3}3(z-1)} + 0.5e^{\frac{R_0}{3}4(z-1)} \quad (4)$$

The simulations of EMCV outbreaks confirmed exactly the theoretically predicted probability to end-up in a major outbreak for R_0 values above 1.6. Below, the cut-off separating small and large outbreaks blurred, as shown in Fig. 2. In detail, for $R_0 = 3.0$ (4.0) one finds $z_\infty = 0.141$ (0.084) and thus theory implies that in 85.9% (91.6%) of all simulation runs with this R_0 value should end up in a large outbreak. Actually 85.8% (91.4%) of all model runs without pen structure and assuming $R_0 = 3.0$ (4.0) ended in large outbreaks affecting on average 271 (282) pigs ranging between 239 and 288 (265–288) pigs. The remainder 14.2% (8.6%) runs ended with 1–25 (1–7) infected pigs (see Fig. 2). Assuming $R_0 = 1.24$, theoretically 31.2% large outbreaks are predicted which would correspond to those 31.2% simulations finished after 37–204 pigs were infected. Finally, for $R_0 = 0.6$ no large outbreak would be predicted and maximum 50 pigs became infected with 99% of the simulated outbreaks affecting at most 15 pigs.

2.4. Modelling pig-to-pig virus transmission with subdivision in pens

The imaginary compartment was subdivided into 24 pens by introducing fences. Initially the fence (F) was considered only a physical barrier precluding contacts between pigs in non-adjacent pens and therewith avoiding random mixing of all pigs in the compartment (F = 0). Increasing the fence effect was assumed to reduce the contacts between pigs in adjacent pens up to a level where pigs

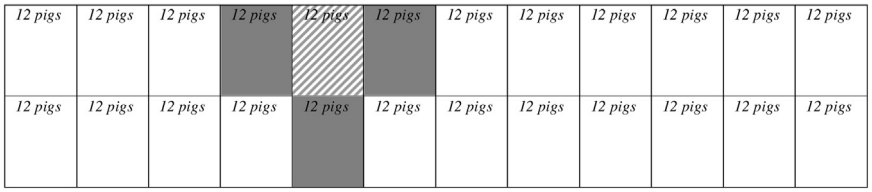

Fig. 1. Arrangement and contact structure of pens in pig housings. A single pen (▨) was considered in contact with 3 adjacent pens (■) whilst all 4 together provide the neighbourhood of the pen. For pens at the corners of the stable, only 2 contact pens are assumed, forming a neighbourhood of 3 pens.

Table 1
Parameters determining the simulation of EMCV spread in the model.

Process	Parameter	Source	Distribution/Range	Reference
Infection	P_{SI_total} depending on		Ber(P_{SI_total})	
S = > E	– R_0 (Reproduction ratio)	Data	(0.3–4.8) step 0.3	Maurice et al. (2002) (for F = 1)
	– F (Fence effect)	Not known	(0.0–0.9) step 0.1	–
IncubationE = > I	Length of incubation period (days)	Data	(25%, 25%, 75%, 100%) Cumulative frequency of transition after 1–4 days	Billinis et al. (1999)
Recovery I = > R	Length of infectious period (days)	Data	(25%, 25%, 50%, 100%) Cumulative frequency of transition after 1–4 days	Billinis et al. (1999)

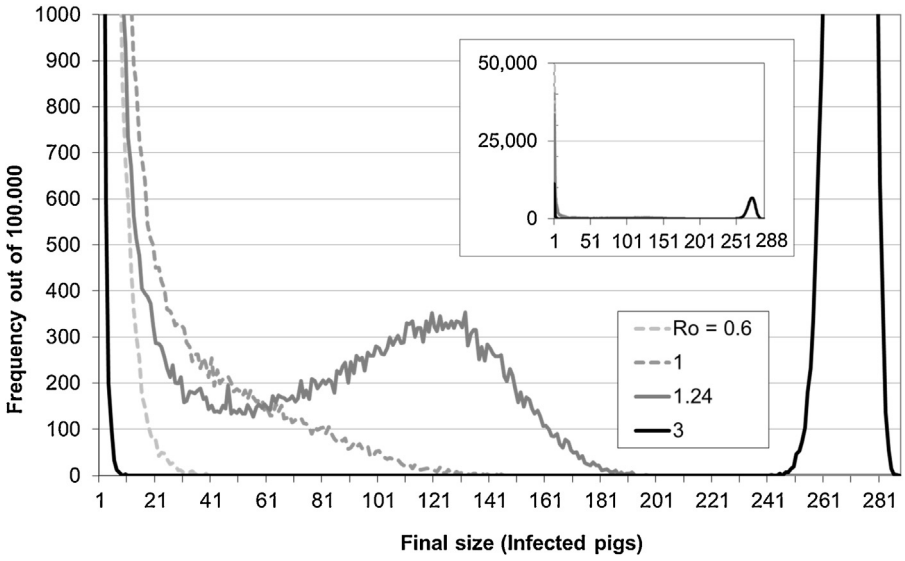

Fig. 2. The frequency distribution of possible final size values found out of 100.000 simulated EMCV-outbreaks in a group of 288 randomly mixing pigs. Different graphs reflect different values of R_0 around the theoretical threshold of 1. In agreement with theory the model shows only small outbreaks for $R_0 < 1$ (dotted lines). Increasing R_0 above 1 ascertains large outbreaks, more often affecting up to 288 pigs (NB: The insert shows the diagram up to scale of 50.000 repetitions while the main graph is cut at 1.000 corresponding to a maximum frequency of 1%).

within a pen could be considered fully separated (F = 1). Therefore, depending on the fence effect, an individual susceptible pig could be infected either by its infectious pen mates only (F = 1) or by its infectious neighbourhood mates (i.e. the "population" of its own pen mates and pigs out of the 3 (or 2) adjacent pens, F = 0). Assuming the per-pig contact rate and therewith it's potential to transmit virus as constant (i.e. β constant) (Bouma et al., 1995), the fence effect "distributes" these contacts between the within pen population and the neighbourhood population. In line with Klinkenberg et al. (2002) the resulting probability of infection for a susceptible pig (P_{SI_total}) is determined by:

$$P_{SI\_total} = 1 - \exp\left(-\beta\left[F\frac{I_{in}}{N_{in}} - (1-F)\frac{I_{all}}{N_{all}}\right]\right), \quad (5)$$

F = fence effect reducing contacts between pigs in adjacent pens (0–1),

I_{in} = number of infectious pen mates,

I_{all} = number of infectious mates in the neighbourhood including the central pen,

N_{in} = number of pigs within pen (12),

N_{all} = number of pigs in neighbourhood including the central pen (48 or 36).

P_{SI_total} was calculated from (5) for any individual according to the neighbourhood of the pig's pen. The Fig. 3a-d visualise by choosing hypothetically an extreme value of β=4/day the effect of varying the fence parameter F in the transmission model (5) showing P_{SI_total} for all combinations of infectious pen mates (left bottom axis, I_{in}) and infectious pigs in adjacent pens (right bottom axis, I_{out}). Due to the physical subdivision of a compartment into pens any pig has contact with 11 pen mates ($N_{in} - 1$) and at most 36 pigs from adjacent pens. If F = 0, the 48 pigs (N_{all}) form "one" group and P_{SI_total} changes with any further infectious animal in the group (Fig. 3a). On the contrary if maximal fence effect is assumed (i.e. F = 1, Fig. 3d) infectious pigs in adjacent pens cannot contribute to

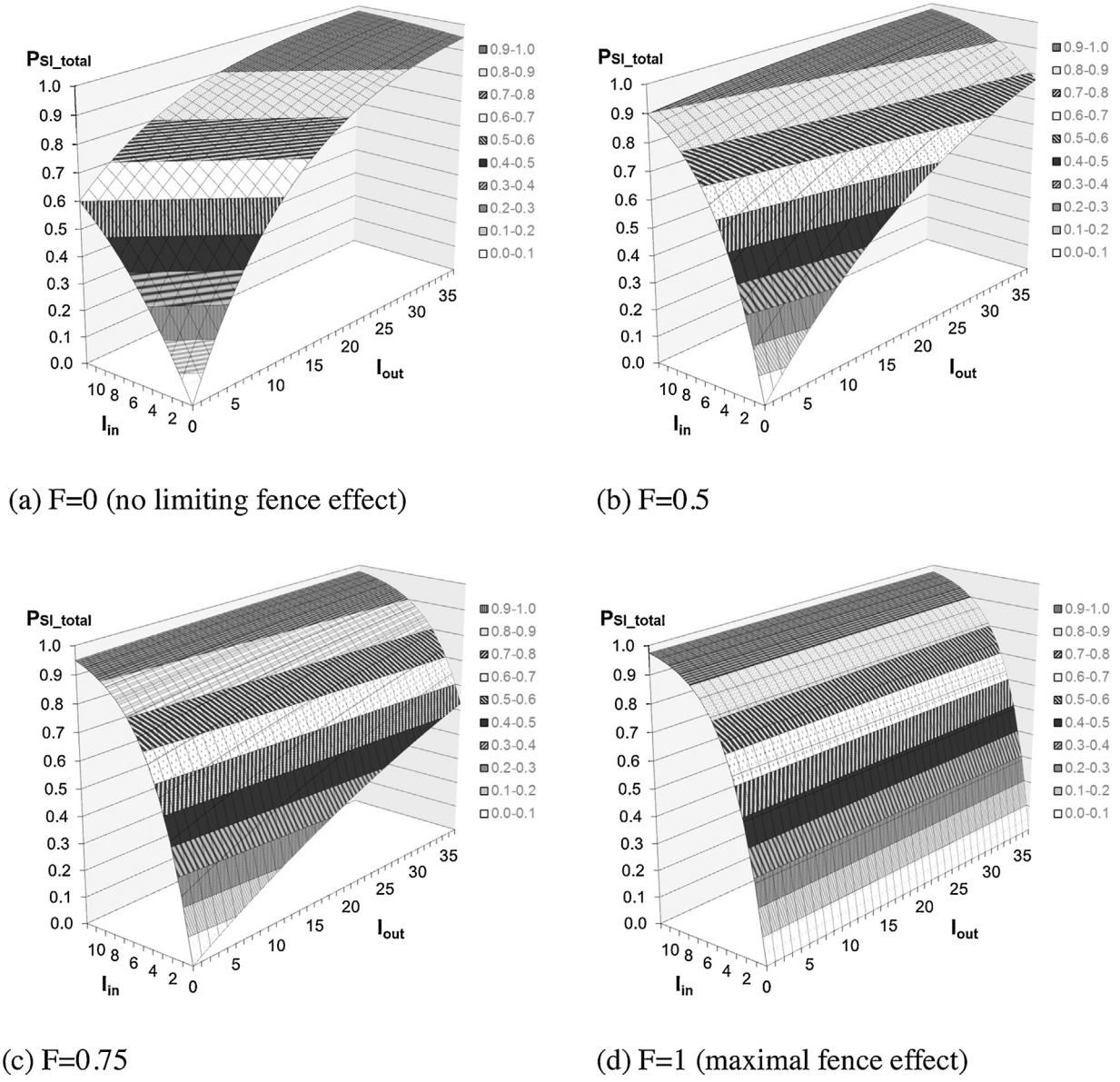

Fig. 3. Visualisation of the consequence of subdivision of the compartment on the per-head probability of susceptible pigs to get infected (β = 4). Depending on the fence effect the number of infectious pigs from the same (I_{in}) and adjacent pens (I_{out}) contribute differently to the per head infection probability (P_{SI_total}) for a susceptible pig.

the infection probability P_{SI_total} for the susceptible pig. Increasing the effect of fences (Fig. 3a. . .d) enlarges the contribution of the infectious pen mates to P_{SI_total}, whereas the impact of the infectious pigs from the adjacent pens decreases (and vice versa).

2.5. Parameterization

For an assumed value of R_0 and after evaluating (2), the transmission rate β was set constant in (5) for all scenarios. The considered R_0-values (i.e. 0.3–4.8) cover the 95%-confidence interval for the R_0-point estimate from earlier experiments (Maurice et al., 2002). Additionally, the fence effect (F) was increased in steps of 0.10 from 0 to 0.9. For perfect fences (i.e. F = 1, no contact between pigs in adjacent pens) (5) is collapsed into (1) with $N = 12$, which was used to validate the model against the contact experiments (Maurice et al., 2002).

2.6. Simulation routine

The combinations of R_0 and F values determine 160 simulation scenarios (i.e. 16×10). For each scenario the simulation run was repeated 1.000 times to cover stochastic variability of the outcome measures. Each run was initialised by infecting a random pig in a random pen. The simulation outcome was described on the individual pig and on the pen level. Firstly, the mean total number of pigs ever infected during an outbreak (NIPG) was recorded to compare scenarios to those of the simulation of a non-compartmentalised pig population. Additionally, the crowding of infections in single pens was measured by the average number of infected pigs per infected pen. Secondly, also to visualise spatial spread, the number of infected pens (NIPN) was averaged over 1.000 repetitions where a pen was considered infected when at least one pig in the pen became infected. An outbreak at compartment level with NIPN ≥ 12 (i.e. 50%) was considered major, while outbreaks with NIPN ≤ 4 (i.e. at most one sixth) were considered of minor size. As the infection process has an expected bimodal outcome especially for larger R_0

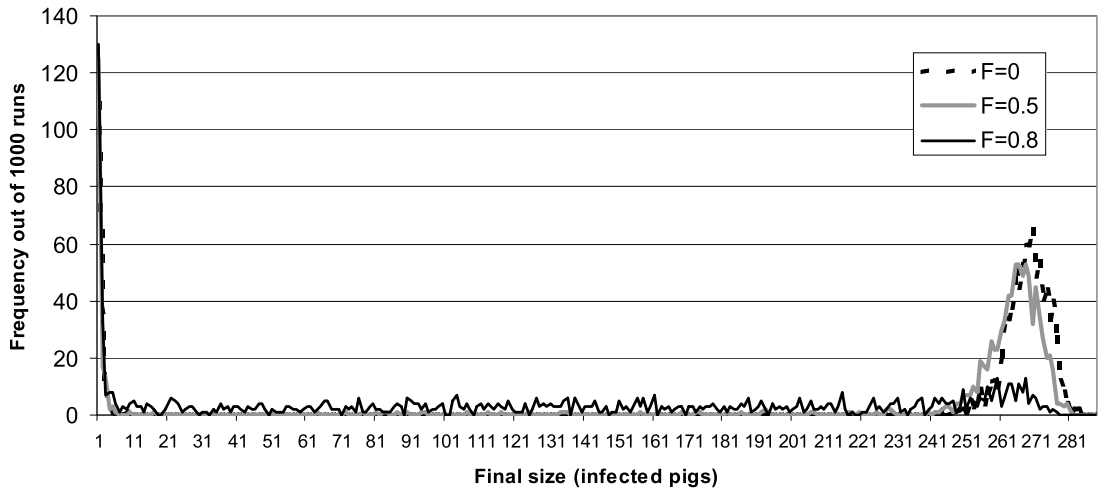

Fig. 4. The frequency distribution of possible final size values found out of 1000 simulated EMCV-outbreaks for $R_0 = 3$ in a group of 288 pigs in a compartmentalised housing. Different graphs reflect different fence effects, resp. F = 0.0, 0.5 and 0.8.

values (see Fig. 4), results additionally were described using the probability to observe outbreaks with at least a specified threshold of infected pens (50% and 80%; Fig. 5).

3. Simulation results

The subdivision of the compartment into 24 pens by introducing fences prevented random mixing of all pigs in the compartment. Thus results are found by comparing simulations, either in terms of the expected number of infected pigs or pens, with different fence effect relative to the control simulation of 288 randomly mixing pigs.

3.1. Number of infected pigs

At F = 0 (the fence is only considered a "physical" barrier separating the pigs into various pens, precluding contacts between pigs in non-adjacent pens) outbreaks resulting from either small or large values of R_0 on average were comparable to those for a randomly mixing population (i.e. R_0 either 0.6 or 3.0 in Table 2). For R_0-values fairly above 1 the average size of the outbreaks among the 288 pigs in a compartmentalised housing was considerably smaller compared to outbreaks in a randomly mixing group of 288 pigs (i.e. R_0 either 1.2 or 1.8 in Table 2). For increasing values of F, the expected average number of infected pigs was further reduced over the whole range of R_0-values.

To illustrate the impact of the fence on the probability to observe "major" outbreaks, Fig. 4 gives the frequency distribution of the final size values (number of infected pigs per model run; cf. Fig. 2 without fence) for 1000 simulated outbreaks with $R_0 = 3$ and an increasing fence effect (resp. F = 0.0, 0.5 and 0.8). The probability to observe outbreaks with 270 infected pigs (see Section 2.3) is reduced from 0.40 at F = 0 (compared to 0.86 at random mixing) to approximately 0.03 at F = 0.8.

3.2. Number of infected pens

Fig. 5a and b shows the probability to observe major outbreaks affecting at least 50% (NIPN ≥ 12) and 80% (NIPN ≥ 20) of the pens, assuming different values of R_0. For F = 0 (background line in Fig. 5a) and R_0 values below 1.2 the probability that resulting outbreaks affect more than 12 pens was only 10%; whereas R_0 values above 1.8 produced such outbreaks with more than 50% chance. However, increasing the fence effect up to F = 0.8 (i.e. 80%) reduced the probability of major outbreaks below 50% for values even up to $R_0 = 2.7$ (Fig. 5a). Only for R_0 values beyond 4.0 outbreaks of major size are observed with more than 50% chance even at maximum fence effect (F = 0.9; Fig. 5a). Furthermore, in order to observe outbreaks affecting at least 80% of the pens (Fig. 5b) and assuming, e.g. an 80% fence effect (F = 0.8), already R_0-values beyond 3.3 were required.

4. Discussion

Although estimated close to one, experimental R_0-estimates on EMCV transmission suggested potential for major outbreaks among randomly mixing pigs (Maurice et al., 2002). However, finishing pigs usually are kept in compartmentalised housings. We used a simulation model to evaluate direct pig-to-pig EMC virus spread. Starting from one group of 288 randomly mixing pigs, virtual fences were introduced to simulate the course of EMCV outbreaks in a realistic compartment set up.

4.1. Initial model input

Starting point in the simulations were experimental data (Maurice et al., 2002), indicating a MLE of $R_0 = 1.24$ with a CI of 0.39–4.35. For all these R_0-values in the CI the corresponding final sizes (total number of infected animals) potentially could be expected, albeit with a reduced probability compared to the MLE. Therefore all these R_0-values were included in the simulations. The stochastic modelling did not produce one single outcome but covered a broader distribution. Simulation results were compared on their probability of occurrence of major outbreaks, as the expected bimodal outcomes prohibited comparison of means. The definition of major outbreaks in terms of the number of infected pens was arbitrarily set to 50% (12 pens). This might have led to an underestimation of the probability to observe "major outbreaks" for values below the specific (unknown) R_0, for which less than 50% of pens infected could be considered as major outbreak already.

4.2. Evaluation of the fence effect

The introduction of fences as "physical barrier" between pens (F = 0, the fence is only a physical barrier separating the pigs into various pens, precluding contacts between pigs in non-adjacent pens) in itself already reduced the final size of EMCV outbreaks. For $R_0 < 1.24$ (Maurice et al., 2002) the probability to observe outbreaks with e.g. more than 100 infected pigs was reduced from about 17%

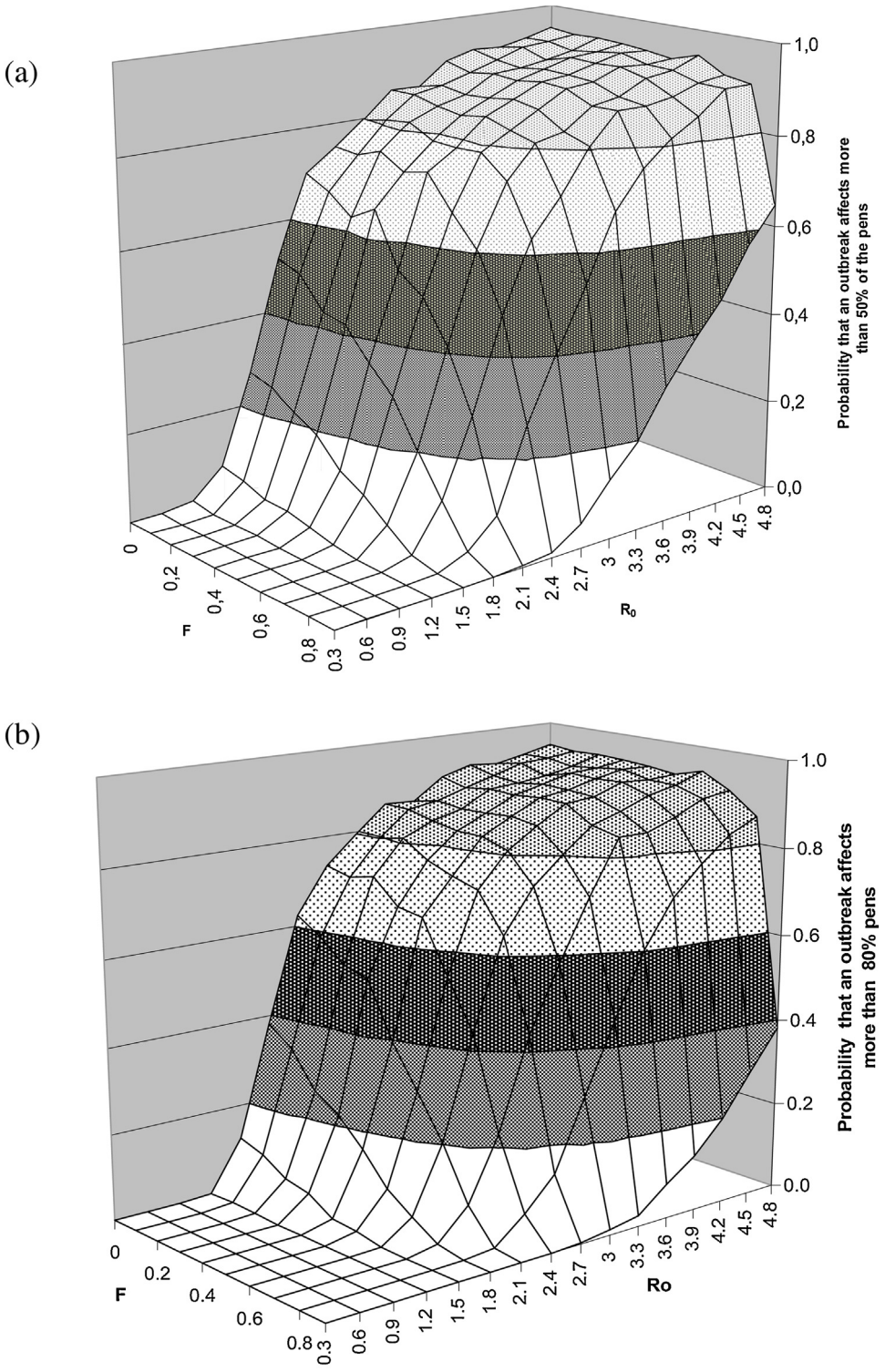

Fig. 5. Impact of the R_0-value and the fence effect (F) on the probability to observe simulated EMCV outbreaks that affect more than (a) 50% and (b) 80% of the pens.

Table 2
Modelling results of the mean number of infected pigs (NIPG) out of 288 pigs shown for representative combinations of R_0 and F. The number of infected pigs per infected pen (I/pen) measures crowding of infections per pen of 12 pigs. Values are shown after averaging all 1000 repetitions and rounding to the nearest natural.

R_0	No fence	F = 0	F = 0.5	F = 0.9	I/pen
0.6	*3*	2	2	2	1.4 – 1.9
1.2	*29*	12	9	5	3.1 – 3.4
1.8	*136*	95	53	12	7.6 – 5.9
3.0	*232*	230	218	55	11.1 – 9.7

in the scenario without the pen structure to about 1% in the "fence as a physical barrier only" scenario (F = 0), due to the limited mixing of pigs. The probability to observe outbreaks affecting more than 50% of the pens remained below 10%. The impact of the fences on the final size of simulated outbreaks further increased as soon as they were also assumed to reduce the direct contacts between pigs in adjacent pens (F > 0; see i.e. Fig. 4 for $R_0 = 3$).

For fences that, e.g. because of their height, require increased efforts from pigs in adjacent pens to get in contact (from reaching out with the head until standing up against the wall), a reduction in contacts up to 80% was considered realistic. Model outcomes indicated that for F = 0.8 the probability for an EMCV outbreak to affect at least 50% of all pens (major outbreak) was below 0.5 for R < 3. Calculating the one sided 95% CI for the experimental data (Maurice et al., 2002) narrowed down the upper limit for R_0 to a value of 3.64. Although a maximal fence effect (F = 0.9) still could reduce the probability of major outbreaks below 0.5, especially for the R_0-values between 3 and 3.6 of the tail of the CI, major outbreaks could not be ruled out beforehand.

4.3. Comparison to field data

From a recent field data set a MLE of $R_0 = 1.36$ (CI: 0.93–2.23) was obtained for EMCV, based on 6 fully sampled pens (with 15 pigs each) (Kluivers et al., 2006). In this field study pens were treated as independent groups and a single introduction per pen was assumed. When multiple introductions per pen were assumed, both the MLE and the corresponding CI for R_0 were even further reduced. Incomplete sampling in the remaining pens resulted in R_0-estimates between 0.8 and 1.7 (Kluivers et al., 2006). The available point estimates clustered around the threshold value of $R_0 = 1$, and the consideration of the CI from Kluivers et al. (2006), tend to further narrow down the range of plausible R_0-values to below $R_0 = 3$. Moreover, in the field study at least 80% of the pens were found infected (Kluivers et al., 2006). Model calculations indicated that for F = 0, R_0-values beyond 2.0 would be required in order to assure reproduction of this field observation with a probability of at least 50% (Fig. 5b). When a fence effect of 60% is considered reasonable (F = 0.6), an $R_0 > 2.5$ is needed (i.e. above the upper limit of the CI = 2.23; Kluivers et al., 2006) to have a minimum parsimonious agreement with the observed 84% infected pens, while for F = 0.8 only R_0-values >3.3 would be sufficient.

Both from the transmission experiments (pairwise and group experiments) as from the field study (6 fully sampled pens) the point estimates for R_0 varied between 0.7 and 1.7, while the combined R_0-estimates were 1.24 and 1.36 respectively. Model calculations indicated that for none of these point estimates the field observation (80% infected pens) was reproduced at least with 50% chance, regardless of the fence effect (F). Hence, either all studies had jointly bad luck and underestimated the R_0 for EMCV, or single introductions are unlikely to cause the observed virus spread patterns as in the field (Kluivers et al., 2006).

4.4. Role of pigs as reservoir host

The results, combined with the short vireamic period and the often observed early death of infected pigs (Maurice et al., 2002; Acland, 1989), indicate that pigs should not be considered as reservoir host for EMCV in compartmentalised pig farms under usual rearing conditions. Therefore other pen-to-pen infection mechanisms of EMCV by e.g. manure and farmer, or multiple introductions by e.g. rodents (Spyrou et al., 2004; Maurice et al., 2002; Joo, 1999; Seaman et al., 1986) might need to be considered to understand reported outbreaks affecting the majority of pens in commercial pig houses (Kluivers et al., 2006; Koenen et al., 1999).

We have recognised the possible re-activation of EMCV in a pig previously recovered from the disease by Dexamethasone treatment (Billinis et al., 1999; compare also Brewer et al., 2001). How dexamethasone treatment relates to stress levels deemed reasonable in a usual pig fattening compartment is not known. However, such experimentally induced immunosuppression was found previously not to impact the insights from transmission modelling in Aujeszky's Disease virus (Stegeman et al., 1997; Van Nes et al., 1998), a virus that can also be reactivated by Dexamethasone treatment.

It cannot be excluded that the observational data applied in our quantitative comparison are consequent to reactivation of the infection in the majority of the recovered pigs (making that incompatible with the model output). However, the limited involvement of pigs in fully diagnosed pens (between 10% and 60% in field data, i.e. Kluivers et al., 2006) does not point to frequent reactivation occurring prior to data collection. This is reasonable because stress levels comparable to Dexamethasone treatment would not be expected in conventional pig fattening. Therefore, the issue of EMCV reactivation was not further considered in the modelling study.

4.5. Future research/model implications

Consequently the model results combined with the findings by Kluivers et al. (2006) also question the need for additional experiments to narrow down the CI around the point estimate of $R_0 = 1.24$ (Maurice et al., 2002) below a value of 2.2 for pig-to-pig EMCV spread in compartmentalised housings. Not only because such experiments are considered both unethical and expensive, but also since the model calculations indicate that for all values up to $R_0 = 2.4$ (Kluivers et al., 2006) the probability to observe major outbreaks already could be kept below 0.50 for $F \geq 0.7$. In addition earlier studies reported difficulties or even failures to prove contact transmission among pigs in experiments (Foni et al., 1993; Christianson et al., 1990; Horner and Hunter, 1979; Littlejohns and Acland, 1975). During an Aujeszky's disease control programme a reproduction number greater one was found between vaccinated fatteners but no large outbreak could be observed in the population, most likely also because of the compartmentalization of the farms (Van Nes et al., 1996). For randomly mixing pigs in e.g. group housing systems, it may still remain important to ultimately assess whether R_0 for EMCV in pigs is above or below one. At the herd level this could imply whether these farms can suffer major EMCV outbreaks while more compartmentalised farms would not.

Basic and vital assumption in the modelling approach was that EMCV transmission only resulted from direct pig-to-pig contact (no airborne spread), which allowed for the use of the same β for both within- and between pen spread. Although multiple transmission routes might be involved for other diseases, the applied basic principle to mechanistically evaluate the within versus the between pen spread by direct contact between animals in itself remains valid and informative.

In the SIR approach the transmission rate beta (β) is constructed from "a number of contacts per time unit" times "the probability of successful transmission of infection during such a contact between a susceptible and an infectious animal", of which only the first one actively can be manipulated by zootechnical measures. In the current modelling approach the effect of the fence implicitly reduced the number of contacts per time unit between pigs in adjacent pens. Effective application of fences to prevent virus spread requires further analysis of the actual contact patterns among pigs in adjacent pens.

5. Conclusion

In conclusion the model outcomes indicated that, combining the current estimates and information on direct pig-to-pig transmission, a single introduction of EMCV in compartmentalised pig housings is unlikely to cause major EMCV outbreaks by direct pig-to-pig transmission alone.

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