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Modelling *Salmonella* spread within a farrow-to-finish pig herd

Ferkel

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Abstract – Delivery of infected pigs to the slaughterhouse is a major source of pork meat contamination by bacterial hazards to humans. We propose a model of *Salmonella* spread within a farrow-to-finish pig herd, assuming the prevalence in infected delivered pigs depends on the whole pig life-time and growing process. This stochastic discrete-time model represents both the population dynamics in a farrow-to-finish pig herd using batch management, and *Salmonella* spread. Four mutually exclusive individual health states were considered: *Salmonella*-free, seronegative shedder, seropositive shedder and seropositive not shedding carrier, making the distinction between seropositive animals and shedders. Since indirect transmission is the main route of transmission, the probability of infection depends on the quantity of *Salmonella* in the pigs' environment (Q). A dose effect function is used with two thresholds, assuming saturation in exposure for high Q vs. a minimum exposure for low Q. *Salmonella* is introduced in an initially *Salmonella*-free 150-sow herd. Prevalence of shedders and seroprevalence are calculated over time in batches of sows and pigs, and in groups of delivered pigs, composed of pigs from different batches. The model shows very variable seroprevalence over time within a herd among delivered groups, as well as among replications. The mean seroprevalence and the mean shedding prevalence are 19.3% and 13.8% respectively. A sensitivity analysis shows that the *Salmonella* quantity shed and the maternal protective factor are the most influential parameters on *Salmonella* prevalence in delivered pigs.

swine / *Salmonella* / epidemiological model / population dynamics / environment

1. INTRODUCTION

Human salmonellosis has been a major public health concern for the last two decades in Europe[1]. Pork is regarded as an important source for this food-borne infection, after eggs and poultry meat [9]. According to the European regulation No. EC 2160/2003 on the control of specified zoonotic agents, Member States (i) need to collect data on the prevalence of *Salmonella* serotypes with public health significance in the broiler, chickens and pigs, and (ii) work towards reducing this prevalence. In the pork food chain, several European countries have anticipated this regulation by setting plans which include control measures aiming at preventing the introduction of *Salmonella* in herds and at controlling the within-herd transmission[1]. Indeed, infected pigs do not exhibit clinical signs but do, however, shed *Salmonella* in their environment. They are then responsible for the contamination of susceptible pigs within the herd [7], during transport and lairage,

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[1] EFSA, Opinion of the scientific panel on biological hazards on "Risk assessment and mitigation options of *Salmonella* in pig production", The EFSA Journal (2006) 341:1–131. [on line] http://www.efsa.europa.eu/EFSA/efsa_locale-1178620753812_1178620776028.htm [consulted 03/04/08].

and the contamination of carcasses during the slaughter process [4, 14]. Focussing on reducing the number of *Salmonella* infected pigs at slaughter age can therefore lead to a reduction in pork food chain contamination[2].

As for other pathogens [29], the within-herd *Salmonella* spread is influenced by interactions between animals also called contact structure. The contact structure corresponds to the existence, type (e.g. direct or indirect via the environment), intensity and frequency of contact among animals. In farrow-to-finish pig herds, the reproduction of sows, farrowing and pig growth from birth to slaughter are conducted on a single site. The farrow-to-finish pig herds represent 73% of French pig herds. In these herds, a batch farrowing management system of sows is often implemented, leading to an all-in/all-out housing system for growing pigs [5]. With this management system, theoretically, there is no direct contact between pigs other than sows from different batches. However, in order to deliver finisher pigs with homogeneous weights to the slaughterhouse, producers sometimes need to adapt their management system by mixing pigs from different batches especially during the finishing stage. These adaptations influence the within-herd contact structure and thus can modify *Salmonella* spread. Given the survival of *Salmonella*, the environment is responsible for pig infection both within and between groups due to the contamination of rooms. This contamination level is related to the flows of shedding animals in the rooms.

A better understanding of the key factors that influence within-herd *Salmonella* spread is needed. Indeed, these key factors can be targeted by control measures. Modelling allows the representation of the characteristics and dynamics of both the pig population, including its contact structure and the producer's management system, and *Salmonella* infection [35]. Actually, it is a tool to assess ex-ante the efficiency of control measure effects on key factors [16, 21]. Previously published models on *Salmonella* transmission in pig herds represented only part of the production process, i.e. the growing stages (but not the breeding phase) [13, 15, 31]. Van der Gaag et al. [31] studied the transmission between different groups of pigs on the farm, but did not account for the heterogeneous contact structure resulting from a batch farrowing management system. Models

In this paper, we present a model of *Salmonella* spread within a farrow-to-finish pig herd under a batch management system, while allowing batch mixing in the finishing stage. The aim of this modelling study was to simulate the spread of *Salmonella* to assess the change of its prevalence in sow and pig batches over time.

2. MATERIALS AND METHODS

We coupled a mathematical model simulating the population dynamics within a farrow-to-finish herd [19] with an epidemiological model of *Salmonella* transmission. This coupled model is a discrete stochastic model with a time step of one week. This time step is the basic unit for grouping tasks and moving animals between rooms in a batch management system; it is also well suited to the *Salmonella* infection dynamics, since no process occurs under this weekly time-step. This is a compartment model; the same process occurs for all pigs in a batch.

2.1. Population dynamics model

The within-herd population dynamics is based on a farrow-to-finish herd model described in more detail in Lurette et al. [19]. The management system is defined according to the most frequent modalities encountered and described in a survey[3]. It includes both the entire reproduction cycle of sows and the entire growth of pigs from birth to slaughterhouse delivery.

[2] World Health Organization–Guidelines on Prevention and control of *Salmonellosis*, VPH/83.42, Geneva, Switzerland, (1983). [on line] http://whqlibdoc.who.int/hq/pre-wholis/VPH_83.42_(p1-p66).pdf and http://whqlibdoc.who.int/hq/pre-wholis/VPH_83.42_(p67–p128).pdf. [consulted 03/04/08].

[3] Hébert H., Lurette A., Fourichon C., Seegers H., Belloc C., 2007. Batch farrowing implementation in pig herd and influence on contact among animals. Proc. 39th Research Swine Days, Paris, France, 6–8 February 2007, pp. 345–350.

The sow herd is divided into groups of equal size called batches. A batch of pigs corresponds to the litters of a batch of sows. The model calculates the batch size over time.

The reproduction cycle of sows is represented by three successive periods that correspond to the occupation of three rooms: the mating room (M), gestating room (G) and farrowing room (S). Every three weeks, a batch of sows is inseminated. Among the farrowing room occupancy, sows spend part of the time with their piglets. After weaning, sows begin a new reproductive cycle in the mating room. In mating and gestating rooms, several batches of sows are housed together. A replacement occurs in the mating room with gilts from a supplier herd.

Pig growth is represented by three successive stages (X), which correspond to the occupation of three rooms: the farrowing (corresponding to the suckling period $X = S$), the post-weaning ($X = PW$) and the finishing rooms ($X = F$). At each growing stage change, all pigs of a batch leave a room at once and enter another room together after this room has been emptied. The all-in/all-out housing system allows each room to be decontaminated between two batches by a cleaning-disinfecting process followed by a drying period.

Pig growth is variable within a batch and between batches. To deliver groups of pigs with homogeneous weights, producers compose the delivery groups of pigs from several batches. If pigs (below the expected slaughter weight) still remain in a finishing room when it needs to be emptied, producers can mix them with the following batch (three weeks younger).

2.2. *Salmonella* spread and prevalence

Infected animals shed intermittently *Salmonella* in their faeces [17, 22] and, hence, contaminate their room. The model represents the indirect faecal-oral transmission via free-living *Salmonella* in the room (in the faeces, on the room floor, pen separations and pig bodies). We assume that -within a room- all animals are exposed to the same quantity of *Salmonella* infectious units Q. The within-batch transmission process is due to the room contamination. The between-batch transmission occurs (i) via the room due to residual *Salmonella* infectious units in the room between two successive batches after the cleaning-disinfecting process, and/or (ii) via the animals, if infected animals of a batch are mixed with another batch.

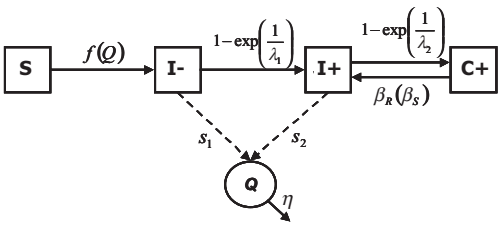

Figure 1. Flow diagram of the *Salmonella* transmission model, representing infection states (F: *Salmonella*-free; Sh: shedder; Shs: seropositive shedder and Cs: seropositve carrier) and transitions between states. The parameters are defined in Table I.

2.2.1. Salmonella *infection states and dynamics*

Salmonella infection does not affect pig demography and growth [4]. Four mutually exclusive health states, measurable with available detection methods (bacteriology and serology [8, 11]), are identified in the literature [22, 33, 34] and retained in this model (Fig. 1): susceptible animals free of *Salmonella* (S), shedding animals (I_-), seropositive shedding animals (I_+), and seropositive carrying animals (C_+). The transitions between infectious states are assumed to be the same for all pigs.

The latent period between *Salmonella* ingestion and bacteria shedding in the faeces lasts less than 24 h[4]. We neglect this state, assuming it has no effect on *Salmonella* infection dynamics over the model time step. We assume that the seroconversion delay is shorter than the duration of the shedding period. The literature data are not in favour of a recovery of pigs, so we do not consider any state in which pigs recover from infection. We do not model a come back to a seronegative carrier state. Indeed, this come-back, if it exists, would require more time than available for pigs to reach their slaughter weight (on average 178.5 days in our model). Moreover, no data showing a return to the seronegative status for pigs [4] or sows are available.

[4] Blaha, T., Solano-Aguilar, G., Pijoan, C., The early colonization pattern of *Salmonella* Typhimurium in pigs after oral intake, Proc. 2nd International Symposium on Epidemiology and Control of *Salmonella* in Pork, Copenhagen, 1997, pp. 71–73.

2.2.2. *Coupling the herd and the epidemiological models*

In the coupled model, the demographic processes are applied before the epidemiological processes. The number of pigs in batch b at time t in growing stage X in each health state is denoted by: $S^X(t,b)$, $I_-^X(t,b)$, $I_+^X(t,b)$, $C_+^X(t,b)$. At each time step t, pigs of batch b are distributed in the four health states, the total of which being $P^X(t,b)$.

2.2.3. *Transition from susceptible to shedding state*

The probability of infection depends on the quantity of *Salmonella* infectious units in room r at time t, $Q(t,r)$, and on the number of pigs in batch b located in room r (i.e. in growing stage X) at time t, $P^X(t,b)$. Assuming homogeneous mixing of pigs within a batch, each pig of batch b located in room r is exposed at time t to $\left(\frac{Q(t,r)}{P^X(t,b)}\right)$ denoting the quantity of *Salmonella* infectious units per pig in room r at time t. To represent a dose effect relation, the probability of infection $f\left(\frac{Q(t,r)}{P^X(t,b)}\right)$ is assumed to be an increasing function of the logarithm of the number of infectious units Q with two plateaus (Fig. 2):

$$\begin{cases} Log\left(1+\dfrac{Q(t,r)}{P^X(t,b)}\right)=0 \Rightarrow f\left(\dfrac{Q(t,r)}{P^X(t,b)}\right)=0 \\ 0 < Log\left(1+\dfrac{Q(t,r)}{P^X(t,b)}\right) \le q_1 \\ \quad \Rightarrow f\left(\dfrac{Q(t,r)}{P^X(t,b)}\right)=a_1 \\ q_1 < Log\left(1+\dfrac{Q(t,r)}{P^X(t,b)}\right) \le q_2 \\ \quad \Rightarrow f\left(\dfrac{Q(t,r)}{P^X(t,b)}\right) \\ =a_1+\dfrac{a_2-a_1}{q_2-q_1}\left(Log\left(1+\dfrac{Q(t,r)}{P^X(t,b)}\right)-q_2\right) \\ Log\left(1+\frac{Q(t,r)}{P^X(t,b)}\right) > q_2 \\ \quad \Rightarrow f\left(\dfrac{Q(t,r)}{P^X(t,b)}\right)=a_2 \end{cases}$$

with q_1 the inferior threshold of the logarithm of the *Salmonella* infectious units per pig below which the probability of infection is low and equal to a_1, and q_2 the saturation threshold above which the probability reaches its maximum a_2. The infection probability is nil for no *Salmonella* in room r ($Q(t,r) = 0$).

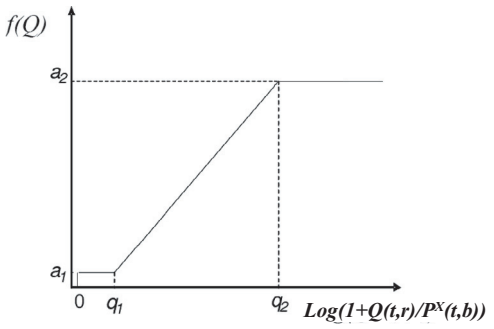

Figure 2. Probability of infection as a function of the number of *Salmonella* infectious units Q per animal in the room. $P(t,b)$ is the number of animals in batch b at time t located in the room. (see Tab. I for parameter description). The corresponding equation is given in the text.

A fixed degradation rate η is applied on Q during each time step. Q is upgraded by the number of infectious units shed by pigs in each room. The shedding of *Salmonella* depends (i) on the growing stage because finishing pigs and sows produce more faeces than piglets with a similar mean number of CFU/gr and, therefore are assumed to shed a larger quantity of *Salmonella* than piglets; and (ii) on the serological status. The change in Q in room r is represented by:

$$Q(t,r(t,b)) = (1-\eta)\,Q(t-1,r(t,b)) + s_X^1 I_-^X(t,b) + s_X^2 I_+^X(t,b),$$

where $s_X^1 = \pi_X s$ and $s_X^2 = \pi_+ \pi_X s$, with s, the quantity of *Salmonella* shed by a seronegative finishing pig, π_+, the relative shedding of a seropositive compared to a seronegative finishing pig, and π_X, the relative shedding of a pig in growing stage X compared to a seronegative finishing pig. In the farrowing room, where sows and piglets are housed together, the change in Q includes both shedding piglets and shedding sows.

At each cleaning-disinfecting process, when a batch of pigs leaves a growing room, the *Salmonella* quantity in room r is updated:

$Q(t,r) = (1-\nu^r)\,Q(t-1,r)$, with ν^r the proportion of *Salmonella* infectious units eliminated by the cleaning-disinfecting process in room r ($\nu^r < 1$, under field conditions, this elimination process is never complete).

In batch b, the number of newly infected pigs in growing stage X at time t is drawn by a binomial law: $Inf^X(t,b) = Bin\left(S^X(t,b), \varepsilon f\left(\frac{Q(t,r)}{P^X(t,b)}\right)\right)$, with ε, a protective factor. Actually, the presence of passive immunity has been demonstrated in piglets at birth [3]. This passive immunity reduces the susceptibility of piglets to *Salmonella* infection [23]. We then assume that the susceptibility of piglets is reduced during the suckling period compared to other growing stages. In our model, this immunity is considered both variable among litters and partial. Indeed, it has been shown that piglets from seropositive sows can be infected at weaning [23]. Hence, the maternal protection is represented by a lower probability of infection.

2.2.4. Transition from seronegative to seropositive shedding state

The seroconversion probability p_S at each time step is given by: $p_S = 1 - \exp\left(\frac{-1}{\lambda_1}\right)$, with λ_1 the average seroconversion delay. The seroconversion delay has been shown to range from one to two weeks [3, 20]. The number of animals in a batch that become seropositive at time t is drawn by a binomial law:

$$Sero^X(t,b) = Bin\left(I_-^X(t,b), p_S\right).$$

2.2.5. Transition from seropositive shedding to seropositive carrier state

The probability to stop shedding *Salmonella* $p_E(t)$ depends on the shedding period duration and is given by: $p_E(t) = 1 - \exp\left(\frac{-1}{\lambda_2(t)}\right)$. The duration $\lambda_2(t)$ is recalculated at each time t to represent a variation between batches due to environmental factors such as temperature or biological factors such as stress and health status [4]. We assume that it follows a lognormal distribution with parameters ($\alpha_{\lambda 2}$, $\sigma_{\lambda 2}$). The number of pigs which become seropositive carriers (C_+) at time t is drawn by a binomial law:

$$Stop^X(t,b) = Bin\left(I_+^X(t,b), p_E(t)\right).$$

2.2.6. Transition from seropositive carrier to seropositive shedding state

The back and forth transition between the seropositive carrier (C_+) and shedding (I_+) state represents the intermittence of shedding and can occur several times during an animal's lifetime.

The probability β_R of shedding reactivation for carrier pigs is fixed. The number of pigs in batch b affected by this transition at time t is: $\mathrm{Re}A^X(t,b) = Bin\left(C_+^X(t,b), \beta_R\right)$. Stressful conditions such as weaning can increase the reactivation of *Salmonella* shedding [4]. So, a different probability β_S (with $\beta_S > \beta_R$) is applied at the weaning of piglets and: $\mathrm{Re}A^{PW}(\tau,b) = Bin\left(C_+^{PW}(\tau,b), \beta_S\right)$, with τ denoting for the date of weaning of batch b.

2.2.7. Complete epidemiological model

The resulting equations used to update the number of pigs or sows in batch b in growing stage X are:

$$\begin{cases} S^X(t,b) = S^X(t-1,b) - Inf^X(t,b) \\ I_-^X(t,b) = I_-^X(t-1,b) + Inf^X(t,b) \\ \qquad\qquad - Sero^X(t,b) \\ I_+^X(t,b) = I_+^X(t-1,b) + Sero^X(t,b) \\ \qquad\qquad - Stop^X(t,b) + ReA^X(t,b) \\ C_-^X(t,b) = C_-^X(t-1,b) + Stop^X(t,b) \\ \qquad\qquad - ReA^X(t,b) \\ Q(t,r) = (1-\eta)\,Q(t-1, r(t,b)) \\ \qquad\qquad + s_1^X I_-^X(t,b) + s_2^X I_+^X(t,b) \end{cases}$$

2.3. Simulation

2.3.1. Parameters used in the model

The herd model is calibrated by integrating knowledge from various sources, from published data to experts' knowledge, so as to obtain a realistic representation of such a pig herd. The values chosen for the parameters used in the epidemiological model are shown in Tab. I. The infection function derives from unpublished data[5]. The proportion of eliminated bacteria during

[5] Fravalo P., Cariolet R., Proux K., Salvat G., The asymptomatic carrying of *Salmonella enterica* by pigs : results obtained from a model of experimental infection, Proc. 35th Research Swine Days, Paris, 4–6 February 2003, pp. 393–400.

Table I. Definition and values of the parameters used in the *Salmonella* infection dynamics model within a farrow-to-finish pig herd*.

Notation	Description	Value	Source
q_1	Inferior threshold of infection below which the probability of infection is the lowest	$\text{Log}(10^4)$	[a]
q_2	Saturation threshold above which the probability reaches a maximum value	$\text{Log}(10^6)$	[a]
a_1	Minimum infection probability	10^{-6}	[a]
a_2	Maximum infection probability	0.08	[a]
λ_1	Seroconversion delay	2 weeks	[3][b]
λ_2	Shedding period duration	Lognormal distribution: Mean: $\alpha_{\lambda 2} = 4$ weeks s.d.: $\sigma_{\lambda 2} = 1.8$ weeks	[6, 17, 22]
β_R	Weekly probability of shedding reactivation	0.2	[a]
β_S	Weekly probability of shedding reactivation due to stress	0.4	[a]
ε	Protective factor – passive immunity	0.75	[a]
η	Weekly survival probability of *Salmonella*	0.4	[25]
ν	Proportion of *Salmonella* infectious unit (S.i.u.) eliminated by the cleaning-disinfecting process:		[27, 28]
	• in mating and gestating rooms:	0.80 S.i.u^{-1}	
	• in farrowing, post-weaning and finishing rooms:	0.999 S.i.u^{-1}	
s	*Salmonella* infectious units (S.i.u.) shedded by a seronegative shedding finishing pig or sow	Normal distribution Mean $= 5.10^4$ S.i.u s.d. $= 10^2$ S.i.u	[a]
$\pi_{S,}\ \pi_{PS}$	Ratio of shedding for piglets and post-weaner compared with a shedder finisher pig	1/10 (piglets/finisher), 1/2 (post-weaner/finisher)	[a]
π_+	Ratio of shedding for a seropositive animal compared with a seronegative shedder pig	0.8 (seropositive/seronegative)	[a]

* The values chosen for parameters used in the epidemiological model are estimated based on experimental data reported in the literature.

[a] Parameters' values are assumed.

[b] From Fravalo P., Cariolet R., Proux K., Salvat G., The asymptomatic carrying of *Salmonella enterica* by pigs: results obtained from a model of experimental infection, Proc. 35th Research Swine Days, Paris, 4–6 February 2003, pp. 393–400.

the cleaning-disinfecting process used in this model is estimated from data of the cleaning-disinfecting process obtained in poultry [27] and pig trailers [28]. The cleaning-disinfecting process in the mating and the gestating rooms is considered worse than in other rooms since several batches of sows are housed together in these facilities and these rooms are never emptied.

2.3.2. Initialisation

The model is initialised by assigning a number of sows and pigs to each batch. The total number of sows is 150, distributed into seven batches. In a batch, 18 sows are inseminated on average. The number of piglets at farrowing is 180 on average. Four gilts are recruited at each reproduction cycle. Based on data from French supplier herds, the gilts are distributed in the four health states and the seroprevalence is exponentially distributed with a mean probability of 0.05[6].

2.3.3. Simulation outputs

The model was implemented with Scilab 4.0[7]. Results were obtained from 150 replications for a given parameter set (Tabs. I and II) over 400 weeks to assess the possible long-term variations in *Salmonella* prevalence. The number of replications allows us to obtain a stable distribution for the simulated results.

The first output was the average prevalence of shedding (including pigs in states I_- and I_+) and of seropositive animals (including pigs in states I_+ and C_+) in a batch of pigs from birth until slaughterhouse delivery, and in a batch of sows at the end of the service, the gestation and the suckling periods. The results were obtained over all batches and all replications, i.e. 37 000 batches of pigs (125 batches per replication over 400 weeks) and seven batches of sows during each reproduction cycle.

Secondly, at each slaughterhouse delivery and over the whole simulation duration, the prevalence of shedding pigs and the seroprevalence were calculated. Given the total number of replications, 30 000 groups were delivered, the distribution of which was then calculated for the two prevalences.

2.4. Sensitivity Analysis

To assess the influence of parameter variation on the results, a sensitivity analysis was performed on all epidemiological parameters of the model. A one-at-a-time analysis [30] was used on these parameters, which were increased and decreased separately by 25% from their initial values (Tab. I). The sensitivity analysis was performed over 150 replications across the whole simulation period. This analysis was conducted on the mean prevalence of shedding and seropositive pigs in groups of delivered pigs.

3. RESULTS

3.1. Prevalence of *Salmonella* infection in batches of growing pigs and sows

The mean prevalence of shedding and seropositive pigs increased over time until reaching 18% and 22% at the slaughterhouse delivery respectively (Fig. 3). Seroprevalence was higher than the shedding prevalence during the finishing period, and then at slaughter age as shown in the distributions (Fig. 4). The seroprevalence became non-null at two weeks of age. A break down of the curves occurred at each room change but it was more marked for the prevalence of shedding. Individual trajectories of *Salmonella* prevalence in a batch of pigs either stood nil over the growing period or increased until the pig delivery. The mean prevalence of shedding sows did not vary over the three reproduction stages, but the prevalence was highly variable between batches (Tab. II).

3.2. Prevalence of *Salmonella* infection in groups of delivered pigs

There was a high variability in the prevalence of shedding and in the seroprevalence in groups of delivered pigs (Fig. 4) using the set of parameters given in Table I. Variations occurred between and within replications, even for two successive delivered groups. The seroprevalence and the prevalence

[6] Lejolivet A., Prévalence des salmonelles dans le schéma de sélection d'un groupement de producteurs de porcs, pratiques à risques et intervention sur l'excrétion, Veterinary thesis report, Nantes Veterinary School, France, 2004.

[7] Available on line : http://www.scilab.org [consulted 16/01/08].

Table II. Prevalence of shedding sows and of seropositive sows according to the reproductive period

Reproductive period	Prevalence of shedding sows			Prevalence of seropositive sows		
	Mating	Gestating	Lactating	Mating	Gestating	Lactating
Median	21.6	21.4	22.3	41.3	42.1	42.1
5th Percentile	5.1	4.8	5.5	14.1	14.1	14.3
95th Percentile	41.2	39.8	41.1	60.4	60.4	60.5

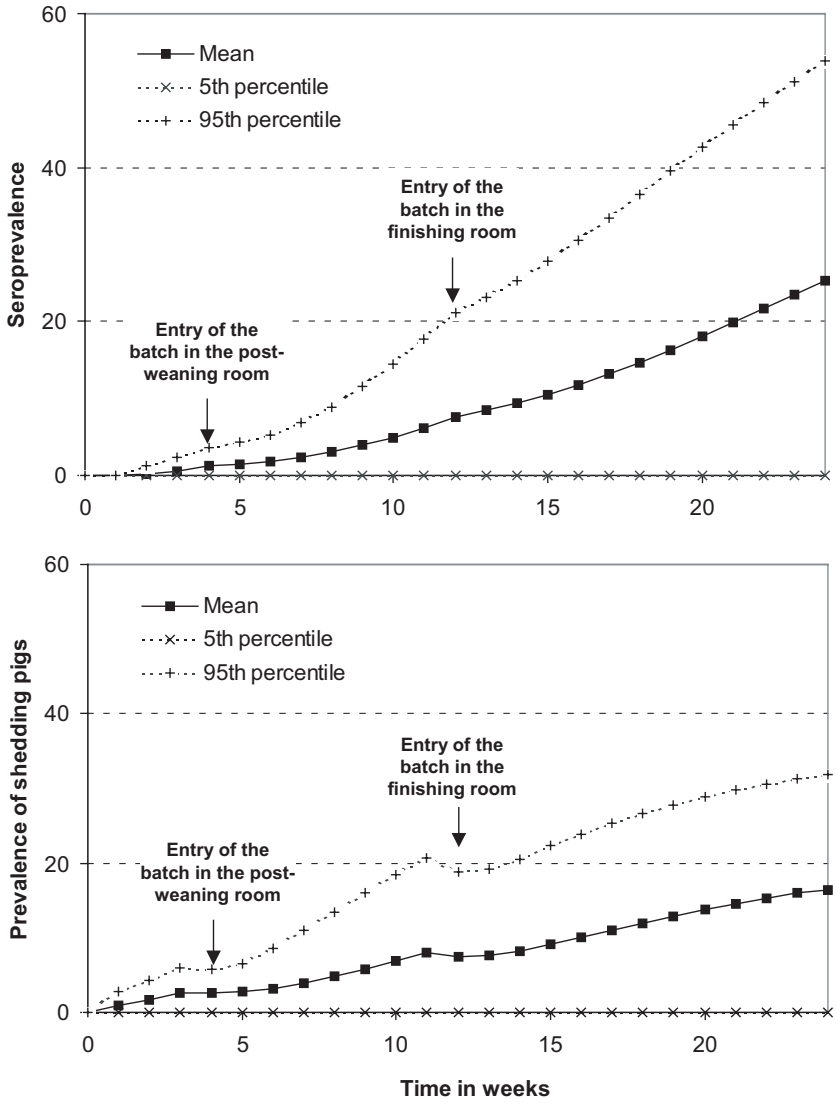

Figure 3. Distribution of the seroprevalence and the prevalence of shedding pigs in groups of pigs delivered to the slaughterhouse. All the groups encountered in 300 replications over a 400-week simulation period are considered, corresponding to 30 000 groups of delivered pigs.

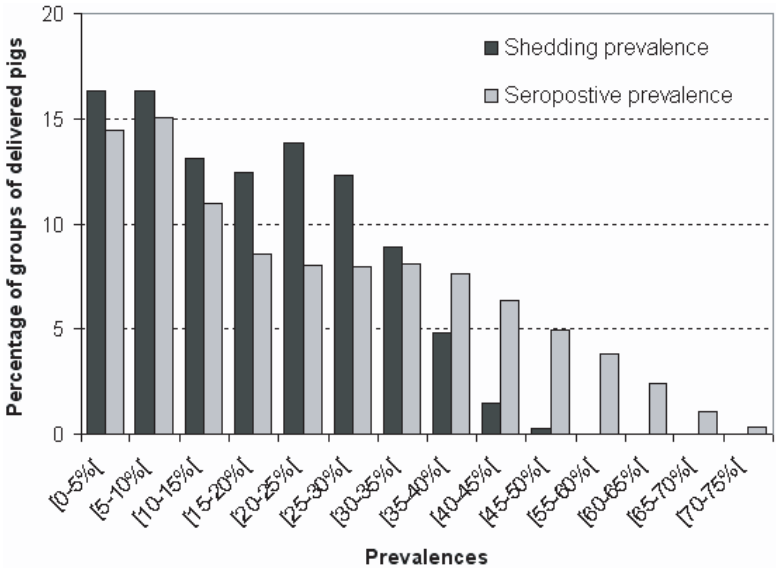

Figure 4. Mean seroprevalence and shedding prevalence in a batch of pigs from birth until slaughterhouse delivery (average over all batches encountered in 150 replications on a 400-week simulation period corresponding to 18.750 batches of pigs).

of shedding in groups of delivered pigs were not identically distributed. The seroprevalence ranged from 0% to more than 75% (mean of 22.6%) whereas the shedding prevalence ranged from 0% to 50% (mean of 16.4%). The prevalence of shedding and the seroprevalence for a given group of delivered pigs were, however, highly correlated (Pearson coefficient, $r = 0.96$).

3.3. Sensitivity analysis

The range of variation of the results was the same for the prevalence of shedding and seropositive pigs. Only six parameters contributed for more than 5% to the output variation in the range of variation tested: s, ε, a_1, a_2, and ν. The variation in the prevalence of shedding in groups of delivered pigs differed according to the parameter tested (Fig. 5) and was symmetric around the default value for s, ε, a_1, and a_2. In the range of variation tested, the increase in the quantity of *Salmonella* shed (s) and the decrease in the protective factor (ε) increased the prevalence by more than 40%. A decrease in ν led to an increase of the mean seroprevalence whereas an increase of this parameter value did not modify this output.

A variation in a_1 and/or a_2 induced a variation between –13% and +10% from the results obtained with the default values of these parameters. A higher effect of a_1 was observed.

4. DISCUSSION

This epidemiological model represents both the population dynamics, including animal flows between the farm rooms, and *Salmonella* spread within a farrow-to-finish pig herd under a batch management system.

In previously published models of *Salmonella* spread within a pig herd [13, 15, 31], the batch management system was not explicitly considered. In our model, the batch management system is modelled in which pigs of different ages are housed separately. This sub-group division can induce, in our model, an heterogeneity in the infection dynamics due to a heterogeneity of the contact structure and, then, a variability in the prevalence of groups of delivered

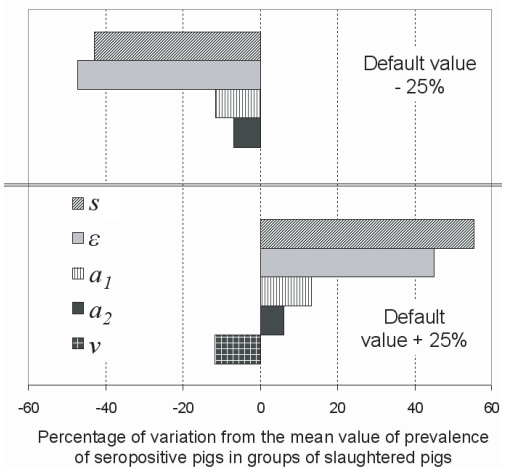

Figure 5. Sensitivity of the prevalence of carrying pigs in groups of delivered pigs to variations in the *Salmonella* survival (ν), *Salmonella* shedding quantity by a finisher pig (s), the maximum (a_1) and the minimum (a_2) infection probabilities, and the maternal protective factor (ε).

pigs, as is observed in field conditions [6]. This influence of the sub-group division has already been shown for dairy herds [35].

Our results suggest that the representation of the breeding period allows to take into account the early infection of piglets that seems to influence *Salmonella* infection dynamics. Indeed, the simulated prevalence of shedding and of seropositive sows in a batch is variable over reproductive life. The resultant variability in both the farrowing room contamination and the maternal antibody transmission can then induce a variation in piglet infection. Moreover, given the major effect of the maternal protection shown by the sensitivity analysis, additional data concerning both the infection of piglets during the suckling period and the protective effect of maternal antibodies or post-infection antibodies are needed because this topic is poorly documented in the literature.

In this model, several modelling assumptions have been made concerning the representation of the room contamination and the infection probability.

Where our model differs from the previously published models studying *Salmonella* prevalence in pigs at slaughter age, is that the latter used a transmission based on the number of shedding pigs in the group considered [15] and in other groups of the herd [13, 31], whereas we considered the room contamination. The residual contamination of a room influences the prevalence of shedding pigs, as shown by the break in the curve pattern at each change of room. This result, associated with the influence of the survival of bacteria shown with the sensitivity analysis, highlights the importance of taking into account both the animal flows in the rearing rooms and the change in *Salmonella* quantity in each room over time, especially in order to test control measures at the herd level.

We chose to use an infection function with two plateaus, which seems to be more adapted than a linear function to the results of experimental data[5]. Linear functions are generally used, however, non-linear function have been shown to suit well realistic conditions [1]. Whereas a non-linear transmission is becoming increasingly well recognised for describing insect-parasitoid interactions [12, 20], it is rarely used for viral or bacterial transmission (e.g. feline retrovirus in cats [10]; bovine tuberculosis [1]). Moreover, as shown in the sensitivity analysis, the two probability thresholds influence the prevalence of *Salmonella* infection. Supplementary

experimental data would help for a better estimation of these infection parameters. We did not consider airborne transmission, which has been characterised as a negligible transmission route for *Salmonella* [26].

Model validation using field data needs to be performed with slaughter prevalence data obtained from a single herd with constant characteristics (batch management, hygiene practices, herd size, etc) which are not available. Given that distributions reported in the literature are built on seroprevalence obtained from several herds[8] [32], only a qualitative validation can be done. However, consistently with observed data in field conditions [6, 18], our model described both the high variations in prevalence between consecutive groups and the existence of seronegative groups of delivered pigs in infected herds.

A high correlation between the seroprevalence and the prevalence of shedding in delivered groups of pigs was simulated. The difference with observational studies [8, 24], which showed discrepancies between seropositive and shedding prevalences, could be reduced by taking into account the sensitivity of the detection tests.

An increasing prevalence over time in a batch of pigs was simulated in this study. This increase is related to the health states and transitions modelled, and has been shown in the literature [3, 17]. Our model differs from other models, which, probably because they considered a recovery state [13], simulate a seroprevalence fade-out at the end of the finishing period.

As shown with the sensitivity analysis, the model suggests that, to reduce *Salmonella* infection at slaughter age, efforts should be made: (i) to reduce the quantity of *Salmonella* shed by infected animals (or fastening the removal of contaminated faeces), or (ii) to decrease the susceptibility of pigs to infection. These reductions could be obtained in the field with acid feed, vaccination, or genetically improved resistance as already shown in chickens [2]. Thanks to its structure, our model can be further used to assess these approaches and other batch management systems and several levels of hygiene.

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