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Controlling *Salmonella* infections in pig farms: A framework modelling approach

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ABSTRACT

Human salmonellosis is an important food-borne disease and *S.* Typhimurium is the most common serotype attributed to pork products. Under a farm-to-fork strategy, reducing the levels of *Salmonella*-positive pigs entering the slaughterhouse is an important goal. A framework model was developed, where the effect of dynamic (infection characteristics) and non-dynamic (cleanness and disinfection, biosecurity measures, etc.) factors were considered. Four baseline scenarios were created, corresponding to different levels associated with national *Salmonella* monitoring programs, and sensitivity analyses were run for the non-dynamic factors. Moreover, the option of vaccination was incorporated into the model, in order to provide with a tool for the formulation of an optimum vaccination strategy depending on the characteristics of the vaccine.
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1. Introduction

S. Typhimurium is the second most frequent cause of human salmonellosis after *S.* Enteritidis and the most frequently isolated serotype from pork products (EFSA & ECDC, 2009). Despite the fact that efficient control measures are taken and HACCP principles are followed during pork production from slaughter to the consumer, random outbreaks of human salmonellosis are still observed, with serious consequences regarding human illness, occasional fatalities and financial costs (Wong & Hald, 2000). This reinforces the principle that as long as the pathogen is introduced into the slaughter chain through infected pigs, consumers will always be exposed. In this context and under a farm-to-fork strategy, a substantial amount of research has been focused on the herd level of the pork production chain towards the reduction of the prevalence of the pathogen in pigs at time of slaughter.

In previous work (Soumpasis & Butler, 2009), a basic stochastic model for *S.* Typhimurium infection in modern pig farms was developed. That model was targeting only the final stage of the pig production and assumed that the fattening room was a closed system. In this way, the effects of the room size and the starting conditions of infection (population of infectious animals at the first day of the model/fattening period) on the transmission of the pathogen in slaughter pigs were evaluated in isolation of any external factor.

Although this approach was useful for understanding the dynamics of the pathogen, other "non-dynamic" factors should be considered to describe mathematically more realistic farm conditions. Friendship, Mounchili, McEwen and Raji (2009), in their review of on-farm intervention strategies against *Salmonella*, point out several categories of risk factors that could affect the propagation of the pathogen in pig farms. In addition, the import of the pathogen from external sources could alter substantially the dynamics of infection in the farm. Although testing each of these factors stand-alone could yield to some useful results with the assumption that all the others remain unchanged, an integrated approach where all the main risk factors are included and can be studied concurrently is missing. Such an approach would be also very useful, if vaccination was to be tested in order to optimize a vaccination strategy for different levels of infection in the farms.

Therefore, the primary aim of this work was to develop a mathematical framework model for the propagation of *S.* Typhimurium at pig farms taking into account the dynamic and non-dynamic behaviour of the pathogen in the field. Moreover, a second aim was to evaluate the effect of different risk factors, and of the associated intervention strategies, on the propagation of the pathogen in the farm in a more integrated approach. A final aim of this work was to test vaccination by incorporating this event into the model and considering two characteristics; efficacy of the vaccine and level of immunity that it creates. It was here assumed that vaccination can offer the maximum protection, such as the one acquired after natural infection.

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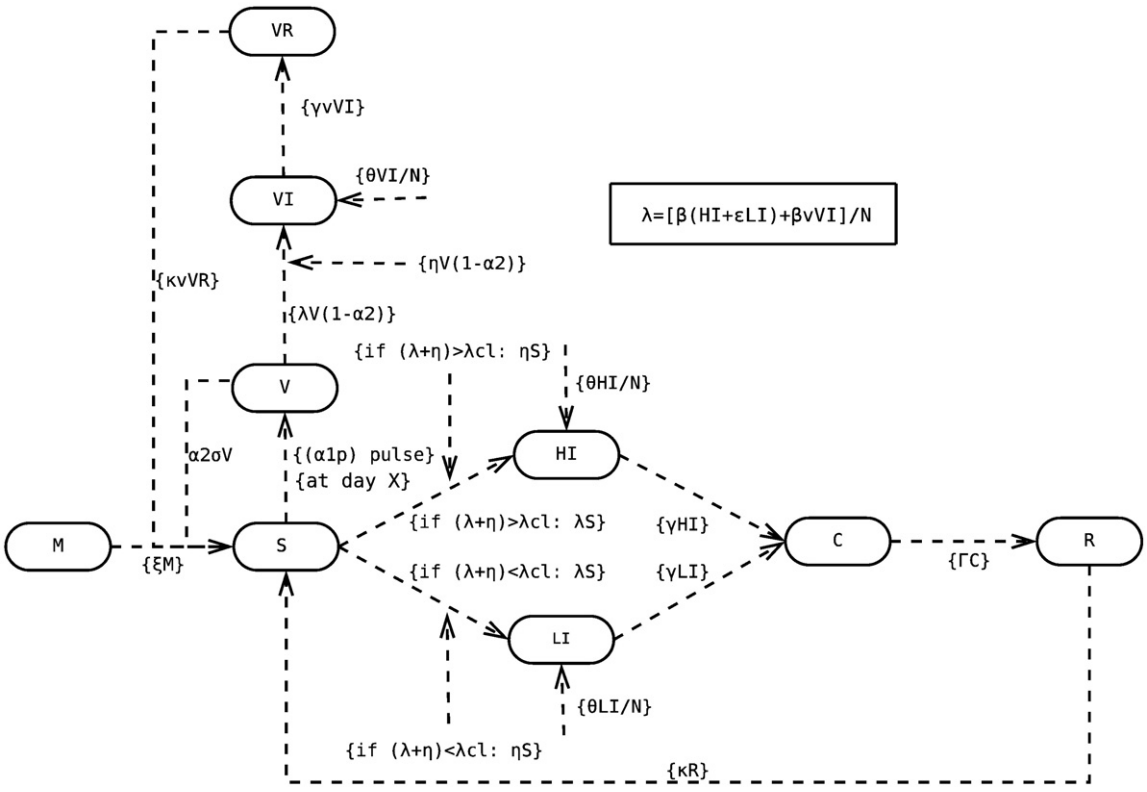

Fig. 1. Flow diagram of model describing spreading of *Salmonella* within pigs with different risk-mitigating options including vaccination. The model has the following classes *M*: Maternally Immune, *S*: Susceptible, *HI*: High Infectious, *LI*: Low Infectious, *R*: Recovered, *V*: Vaccinated, *VI*: Infectious (from vaccinated) and *VR*: Recovered (from vaccinated).

2. Material and methods

2.1. Development of a baseline model

A mathematical model was developed to better understand the dynamics of *Salmonella* in pig farms. This model was based on previous work (Soumpasis & Butler, 2009), where a stochastic model had been developed for the fattening rooms of an All-In-All-Out (AIAO) pig farm. That model had been parametrised using data from clinical trials and by simulating a field experiment with natural infection within a typical intensive farm (Beloeil, Chauvin, Proux, Rose, Queguiner, Eveno et al., 2003) and had been validated against observed surveillance data. It consisted of five classes, Susceptible (*S*), High Infectious (*HI*), Low Infectious (*LI*), Carriers (*C*), and Recovered (*R*), and it was targeting the last part of pig production, the fattening. In this way, pigs that were introduced into the model (either bought from another farm or moved from another house to the fattening room) were already categorised in one of the above classes, forming the starting conditions of infection. In that model, the effects of the different starting conditions of infection and the size of compartment on different infection metrics (probability of disease extinction, mean age of extinction and prevalence of different classes at slaughter age) were evaluated.

The new model is also stochastic using Gillespie's "τ-leap" efficient algorithm (Gillespie, 2001) and incorporates two types of imports (Keeling & Rohani, 2007), external and internal, as well as vaccination. Moreover, it looks at dynamic and non-dynamic aspects of infection and covers the three of the four stages of fattening pig production: weaning, growing and fattening. According to this methodology, all the possible events of an infection are defined in the first place, including the cases of external and internal imports (Table A.3 in Appendix A). For small time steps, the number of events that will happen per time step can be approximated by a Poisson distribution, which has as a mean the rate of each event. This rate can be found from the differential equations forming the deterministic model (Appendix B). In each time step, these Poisson distributions for each event are sampled and the resulting number of individuals is transferred from the population of one class to the population of another (Table A.3 in Appendix A) (Gillespie, 2001; Keeling & Rohani, 2007).

The model developed consists now of nine classes: Maternally Immune (*M*), Susceptible (*S*), High Infectious (*HI*), Low Infectious (*LI*), Carriers (*C*), Recovered (*R*), Vaccinated (*V*), Vaccinated Infectious (*VI*) and Vaccinated Recovered (*VR*) (Fig. 1). The last three classes cover the case of vaccination, allowing also for partial immunity of vaccination. In the latter situation, a proportion of the vaccinated pigs can still become infected, an event that will trigger a stronger immuno-response with higher antibody titres. The model starts during the weaning phase where piglets lose their maternal immunity. If the piglets are bought at growing age, the weaning part of the model can be omitted and the pigs can be introduced at approximately the 80th day, distributed among the different classes of the model (either randomly or fixed).

Two types of infectious classes (*HI* and *LI*) with different effect on the transmission of the pathogen were used in the model because it explains the observed experimental and surveillance data better than if only one class of infectious pigs was used (Soumpasis & Butler, 2009). In short, *S.* Typhimurium is usually observed in pigs as a subclinical infection that produces no overt symptoms. However, when pigs are challenged with large doses of the pathogen they may develop an acute/high propagation syndrome with clinical symptoms (Boyen, Haesebrouck, Maes, Immerseel, Ducatelle and Pasmans, 2008; Boyen, Pasmans, Immerseel, Donn, Morgan, Ducatelle et al., 2009; Fravalo, Cariolet, Proux and Salvat, 2003; Loynachan & Harris, 2005; Griffith et al., 2006). Thus, the model includes two syndromes: a high propagation and a low propagation syndrome. The two syndromes

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