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Time-dependent infection probability of classical swine fever via excretions and secretions

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ABSTRACT

Several routes contribute to the spread of classical swine fever (CSF) during outbreaks of this disease. However, for many infected herds in recent epidemics, no route of virus introduction could be indentified. To obtain more insight into the relative importance of secretions and excretions in transmission of CSF virus, a model was developed. This model quantified the daily transmission probabilities from one infectious pig to one susceptible pig, using quantitative data on: (a) virus excretion by infected pigs, (b) survival of virus in the environment and (c) virus dose needed to infect susceptible pigs. Furthermore, the model predicted the relative contribution of secretions and excretions to this daily probability of infection of a susceptible pig. Three virus strains that differed in virulence were evaluated with the model: the highly virulent strain Brescia, the moderately virulent strain Paderborn and the low virulent strain Zoelen. Results suggest that it is highly probable that susceptible pigs in contact with Brescia or Paderborn infected pigs will be infected. For a pig in contact with a Zoelen infected pig, infection is less likely. When contact with blood is excluded, the predicted overall probability of infection was only 0.08 over the entire infectious period. The three strains differed in the relative contribution of secretions and excretions to transmission, although blood had a high probability of causing infection of a susceptible pig when in contact with a pig infected with any strain. This supports the statement that during outbreaks, control measures should ideally be based on the characteristics of the specific virus strain involved, which implies the development of strain-specific measures.

1. Introduction

Classical Swine Fever (CSF) is a highly contagious viral disease that affects both domestic pigs and wild boar. In the recent past, several outbreaks of CSF in Europe that occurred have been difficult to control, especially in areas with a high pig density. During these outbreaks, CSF virus (CSFV) was spread within- and between farms through direct contact between infected and susceptible pigs, and via indirect transmission routes, such as swill feeding (Williams and Matthews, 1988; Farez and Morley, 1997; Fritzemeier et al., 2000; Sharpe et al., 2001), artificial insemination (De Smit et al., 1999; Floegel, 2000; Hennecken et al., 2000), or contaminated mechanical vectors such as clothing and footwear or livestock trucks (Terpstra, 1988; Stegeman et al., 2002; Ribbens et al., 2004, 2007). However, in many cases, the route of virus introduction in farms could not be established (approximately 50% of the cases during the 1997–1998 outbreak in the Netherlands) (Elbers et al., 1999; Fritzemeier et al., 2000; Allepuz et al., 2007). Most of these infected herds were situated close to herds infected earlier in time, and were, therefore, called neighbourhood infections (Elbers et al., 1999, 2001). Subsequent studies on transmission routes

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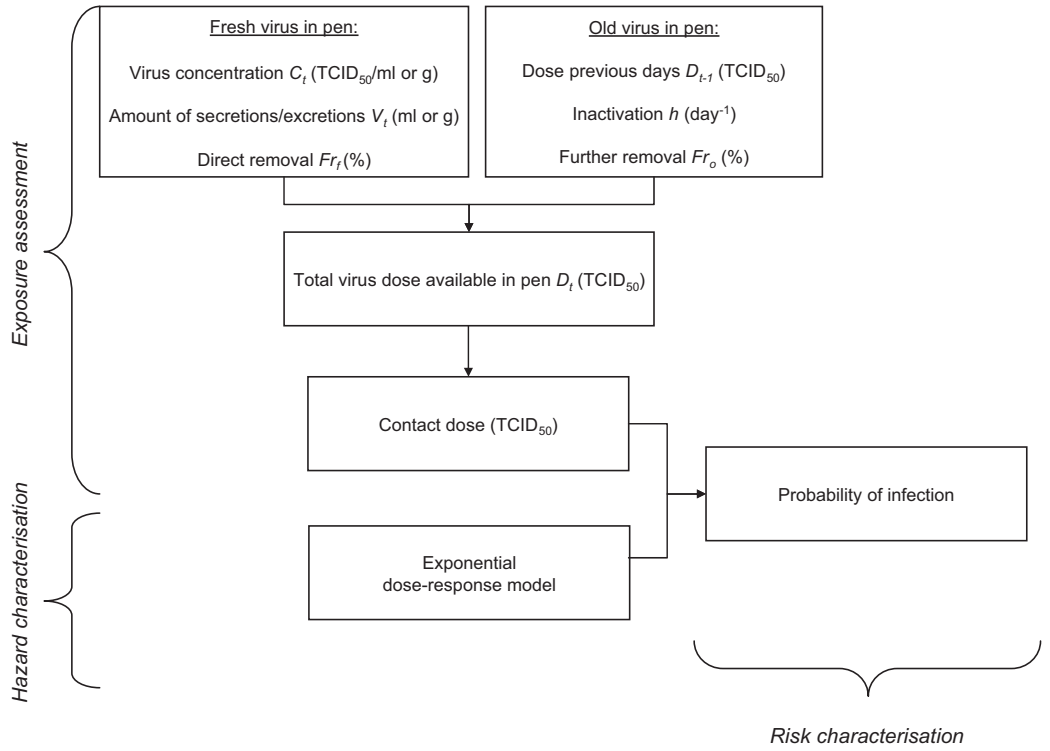

Fig. 1. Schematic outline of the risk model for estimation of the time-dependent infection probability of classical swine fever via excretions and secretions (calculations per d.p.i.).

responsible for these neighbourhood infections, using questionnaires or by analysing outbreak data (Elbers et al., 2001; Crauwels et al., 2003; Mintiens et al., 2003), were only partly successful in elucidating specific routes and their relative importance. As a consequence, the effectiveness of measures other than ring-based control measures cannot be quantified and evaluated.

Although routes of virus introduction in neighbourhood farms remain largely unknown, several studies have indicated that distance is an important variable in neighbourhood spread. The probability of a neighbourhood infection decreases with increasing distance to the primary infected herd (Koenen et al., 1996; Boender et al., 2008). Measures to control CSF outbreaks are to a large extent based on this relationship, for instance, preventive depopulation of herds or emergency vaccination in a certain radius around infected farms (Nielen et al., 1999; Mangen et al., 2001; Backer et al., 2009). The application of these control measures has far-reaching consequences, both ethically because of mass destruction of uninfected pigs, and economically because of prolonged trade restrictions resulting from vaccination (Meuwissen et al., 1999; Boklund et al., 2008). Application of such measures should therefore ideally be avoided.

Studies on transmission routes during outbreaks often apply top-down approaches, using available outbreak data to define important contacts between herds (Elbers et al., 2001; Stegeman et al., 2002). However, also bottom-up approaches to study transmission routes between herds can be used (Ribbens, 2009). Such an approach would use quantitative data on underlying mechanisms of transmission, building a model that starts at the level of transmission from one infectious pig to one susceptible pig, and ultimately reaching the level of between herd transmission.

In the present study, a bottom-up approach was used to study transmission from one infectious pig to one susceptible pig, using quantitative data on (a) virus excretion by infected pigs, (b) survival of virus in the environment and (c) virus dose needed to infect susceptible pigs. A model was constructed to evaluate the relative contribution of blood, saliva, conjunctival fluid, nasal fluid, faeces, urine and the air to spread of CSFV, and the time-dependent probability of infection of a susceptible animal via these secretions and excretions. Three virus strains that differed in virulence were evaluated with the model.

2. Model description

A risk model was constructed in Microsoft Excel 2003 (Microsoft Corporation, Redmond, WA, USA) and @Risk 4.5.3 (Palisade, 2004) to predict the daily probability that a susceptible pig is infected with CSFV via virus excreted in different excretions and secretions of one infected pig. The basic steps of microbial risk assessment (MRA) were applied, i.e., exposure assessment, hazard characterisation, and risk characterisation (Anonymous, 1999; Haas et al., 1999). An outline of the model is presented in Fig. 1, and an overview of the abbreviations used in model equations is presented in Table 1. Experimental data (Bouma