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Inferences about the transmission of Schmallenberg virus within and between farms

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ABSTRACT

In the summer of 2011 Schmallenberg virus (SBV), a *Culicoides*-borne orthobunyavirus, emerged in Germany and The Netherlands and subsequently spread across much of Europe. To draw inferences about the transmission of SBV we have developed two models to describe its spread within and between farms. The within-farm model was fitted to seroprevalence data for cattle and sheep farms in Belgium and The Netherlands, with parameters estimated using approximate Bayesian computation. Despite the short duration of viraemia in cattle and sheep (mean of 3–4 days) the within-farm seroprevalence can reach high levels (mean within-herd seroprevalence >80%), largely because the probability of transmission from host to vector is high (14%) and SBV is able to replicate quickly (0.03 per day-degree) and at relatively low temperatures (threshold for replication: 12.3 °C). Parameter estimates from the within-farm model were then used in a separate between-farm model to simulate the regional spread of SBV. This showed that the rapid spread of SBV at a regional level is primarily a consequence of the high probability of transmission from host to vector and the temperature requirements for virus replication. Our results, obtained for a region of the UK in a typical year with regard to animal movements, indicate that there is no need to invoke additional transmission mechanisms to explain the observed patterns of rapid spread of SBV in Europe. Moreover, the imposition of movement restrictions, even a total movement ban, has little effect on the spread of SBV at this scale.

1. Introduction

During the summer of 2011 dairy cattle in Germany and The Netherlands were reported to be affected by an unknown disease causing a short period of clinical signs including fever, diarrhoea and reduced milk production (Hoffmann et al., 2012; Muskens et al., 2012). Subsequent metagenomic analysis identified the causative agent to be a novel orthobunyavirus (Hoffmann et al., 2012), which has since become known as Schmallenberg virus (SBV). From November 2011 onwards malformations in newborn lambs and calves associated with SBV were reported

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in Germany, The Netherlands, Belgium, France, Luxembourg, Great Britain, Italy and Spain (European Food Safety Authority, 2012a). The detection of SBV RNA in *Culicoides* biting midges (De Regge et al., 2012; Elbers et al., 2013) suggested that, in common with many other bunyaviruses, SBV is a vector-borne disease.

When a new infectious disease emerges there is little or no information available on its epidemiology or transmission dynamics. In this situation it is possible to use other diseases (ideally ones with some relationship to the novel disease) to provide a framework in which to investigate the potential impact of the emerging disease. In the case of SBV several early studies used models parameterised using data on Akabane virus (a related *Culicoides*-borne virus) and bluetongue virus (BTV) (an unrelated, but well-studied *Culicoides*-borne virus) when exploring scenarios for the spread of SBV (European Food Safety Authority, 2012a,b; Bessell et al., 2013). However, suitable data, notably from seroprevalence surveys (Elbers et al., 2012; Gache et al., 2013; Méroc et al., 2013a,b; Veldhuis et al., 2013), are now becoming available and allow inferences about the transmission of SBV to be drawn directly.

In this study we used a stochastic compartmental model, whose structure is similar to one previously developed for BTV (Gubbins et al., 2008; Szmaragd et al., 2009), and fit this to data on the seroprevalence of SBV in cattle and sheep farms in Belgium (Méroc et al., 2013a,b) and The Netherlands (Veldhuis et al., 2013). Parameters in the model were estimated using approximate Bayesian computation (Marjoram et al., 2003; Toni et al., 2009; Sunnaker et al., 2013). This allows us to avoid calculating a computationally unfeasible likelihood function for the model and instead generates distributions of parameters which are consistent with the within-farm seroprevalence data according to a set of predefined goodness-of-fit metrics.

Once the within-farm parameters had been estimated, their consequences for the spread of SBV at a regional level were explored by incorporating them into a separate, between-farm model adapted from one previously used to describe the transmission of BTV (Turner et al., 2012). Sensitivity analyses were then carried out to explore whether parameter estimates for SBV can account for the different rate of regional spread compared to BTV.

2. Materials and methods

2.1. Transmission of SBV within a farm

2.1.1. Data

To infer epidemiological parameters for SBV we used data on the within-farm seroprevalence for cattle and sheep farms from Belgium (Méroc et al., 2013a,b) and The Netherlands (Veldhuis et al., 2013). In total, 422 cattle and 82 sheep farms from Belgium and 543 cattle and 342 sheep farms from The Netherlands were included in the analysis. From each data-set we extracted the number of animals (i.e. cattle or sheep) on the farm, the number of animals sampled, the number of positive samples and the NUTS (Nomenclature of Units for Territorial Statistics) level 2 (NUTS2) region for each farm (European Union, 2011). For The Netherlands, the date of sampling was also extracted.

Temperature data were obtained from the European Commission Joint Research Centre MARS Meteorological Database, which provides daily meteorological data spatially interpolated on a 50 km by 50 km grid cell. Specifically, we extracted the daily minimum and daily maximum temperatures for 2011 and computed the midpoint of these for the pixel closest to the centroid of each NUTS2 region for Belgium and The Netherlands to use in the simulations.

2.1.2. Modelling approach

The within-farm dynamics of SBV were described by a stochastic compartmental model (Fig. 1; Table 1), which was adapted from an earlier model for BTV (Gubbins et al., 2008; Szmaragd et al., 2009). The model includes a single host species (cattle (C) or sheep (S)), with the total host population (H_i) divided into the number of susceptible ($X^{(i)}$), infected (and infectious) ($Y^{(i)}$) and recovered ($Z^{(i)}$) animals (where i indicates the species). The duration of viraemia was assumed to follow a gamma distribution with mean $1/r_i$ and variance $1/n_i r_i^2$. To incorporate this in the model the infected class ($Y^{(i)}$) is sub-divided into n_i stages each of mean duration $1/n_i r_i$ (Lloyd, 2001). The vector population (N) is divided into the number of susceptible (S), latent (i.e. infected, but not yet infectious) (L) and infectious (I) individuals. To allow for a more realistic gamma distribution for the extrinsic incubation (i.e. latent) period (EIP) (Carpenter et al., 2011), the latent class (L) is subdivided into k stages each of mean duration $1/k\nu$ (so the mean duration of the EIP is $1/\nu$). Once infectious, midges remain so for life. Vector mortality occurs at a constant rate μ in all classes and is balanced by the recruitment of susceptible vectors, so that the total vector population remains constant.

The force of infection for host species i, λ_i, is given by

$$\lambda_i(t) = bam_i\theta(t)\frac{I(t)}{N}, \tag{1}$$

where b is the probability of transmission from an infected vector to a host, a is the reciprocal of the time interval between blood meals for the vector (related to the biting rate), $m_i(=N/H_i)$ is the vector-to-host ratio,

$$\theta(t) \propto \exp\left(b_0 + \sum_{j=1}^{2} b_{1j}\ \sin\left(\frac{2j\pi t}{365}\right) + b_{2j}\ \cos\left(\frac{2j\pi t}{365}\right)\right), \tag{2}$$

is the seasonal vector activity on day t (Sanders et al., 2011), normalised so the maximum value is one, and I/N is the proportion of bites which are from infectious vectors. The force of infection for vectors, λ_V, is

$$\lambda_V(t) = \beta a \frac{1}{H_i}\sum_{j=1}^{n_i} Y_j^{(i)}(t), \tag{3}$$

where β is the probability of transmission from an infected host to a midge.