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Blue tongue – A modelling examination of fundamentals – Seasonality and chaos

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HIGHLIGHTS

- A deterministic within-farm model for blue tongue disease in cattle is constructed.
- This is important because of the increasing impact of the disease due to continued global warming.
- The model makes use of a recently suggested modification of logistic growth used for the vectors, which can greatly affect early disease dynamics.
- Autonomous oscillations may occur if loss of host resistivity to the virus is included, depending on host–vector coupling coefficients. Adding seasonal vector mortality can then give rise to chaos.
- Placental disease transmission to offspring has little effect on disease progress.

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ABSTRACT

A deterministic mathematical model is developed for the dynamics of bluetongue disease within a single farm. The purpose is to examine widely the possible behaviours which may occur. This is important because of the increasing impact of blue tongue due to global warming. The model incorporates a recently suggested modification of logistic growth for the vectors which can greatly affect early disease dynamics and employs a variable number of up to 10 sequential pools for incubating vectors and for incubating and infectious hosts. Ten sequential pools represent the possible loss of immunity of recovered hosts over a 3-year period.

After formally describing the model, the impact of the two logistic growth scenarios considered is examined in Section 3.1. The scenarios are applied with parameters that give identical long-term consequences but the early dynamics can be greatly affected. In the two scenarios, the effect of varying the assumed constant birth rate (scenario 1) or constant mortality rates (scenario 2) is considered.

If the recovered (and immune) hosts, are assumed to lose their immunity, then, given particular values of the host–vector coupling constants, the system can exhibit autonomous oscillations (Section 3.2).

Seasonality is represented by air temperature, and it is assumed that air temperatures below a threshold can increase vector mortality (Section 3.3). Adding seasonal effects on mortality to the autonomous oscillations resulting from recovered and resistant hosts losing immunity can give rise to chaos (Section 3.4). This could help explain the unusual persistence and re-occurrence of the disease.

Finally (Section 3.5), the roles of host birth and mortality rates in examined, particularly in relation to placental transmission of the virus to offspring. It is concluded that the latter does not make an appreciable contribution to disease dynamics.

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1. Introduction

1.1. Blue tongue disease (BTD) – the essentials

Blue tongue is a non-contagious arboviral disease, where the vector carrying the virus to an uninfected host is an arthropod ("arbo" denotes "arthropod-borne"). BTD can exhibit complex

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dynamics (O'Farrell and Gourley, 2014). Infectious vectors carrying the virus can infect hosts; these are ruminants such as sheep, cattle, deer and goats. Recovery and the loss of resistance of diseased animals is a possibility, so that autonomous oscillations may arise. Uninfected vectors acquire the virus from infectious hosts, which they can incubate and pass on to other hosts when they take a blood meal. The infectious agent of BTD, the bluetongue virus (BTV), is a double-stranded RNA virus, is carried by various species of the female biting midge, *Culicoides* (Charron et al., 2011). Blue tongue (BT) is now a notifiable disease (OIE, 2014).

Similarly, dengue is also a arbovirus-transmitted viral disease, in this case, of humans. Here the virus is carried by *Aedes* mosquitoes (Favier et al. 2005). In both cases, two populations must therefore be considered: hosts, H, and vectors, V, which together determine the overall dynamics of the disease.

1.2. Impact of climate change on BTD – impacts on health, control and economics

Due to climate change, and arguably the effect of higher temperatures on vector survival, some of these "emerging infectious diseases" (EID's) are increasingly spreading into new geographical areas, having economic and animal health consequences which give rise to concern (Purse and Rogers, 2009; Lelli et al., 2011). BTD is being addressed at many different levels: from the molecular level (Maan et al., 2012), to health impact (Sedda et al., 2012; Turner et al., 2012; Elhassan et al., 2014); control (Durel et al., 2015); and to economic impact (Gethmann et al., 2015). Mellor et al. (2009) give an overview of the wider aspects of BTD. Sedda et al. (2012) and Burgin et al. (2013) address the issue of between-farm spread of BTD.

Once infected with BTD, animals can exhibit a range of symptoms: e.g. emaciation, lameness, fever and depression, depending on species, age, breed. In some animals the tongue turns blue, due to lack of oxygen caused by mucosal inflammation.

In Northern Europe outbreaks of BTD are seasonal, due to the seasonal activity of the midges and their temperature-dependent life-cycle (Wilson and Mellor, 2009). They can therefore occur on a time scale which is not commensurate with any autonomous oscillations arising from loss of host resistivity (Section 3.2). Thus, our system of ODE's fulfils some of the requirements for possible chaos (Acheson, 1997, pp. 156–157). Control measures may include good hygiene (not using a single needle to administer medication to a herd or a flock); use of insecticides to kill midges; housing the animals indoors during times of midge biting activity; quarantining or slaughtering infected animals. Vaccines have been developed against some serotypes of BTV, of which there are many.

The economic impact of BTD is sufficient to cause genuine apprehension in the agricultural community, and, as mentioned above, has led to BT becoming a notifiable disease (OIE, 2014). Due to the lability of prices, it is difficult to model economic consequences in a meaningful way other than retrospectively.

1.3. Simplicity versus complexity in disease modelling

The present model (Fig. 1) is regarded by the authors as being "simple" – it comprises a number of ordinary differential equations many of which are the same – there are good biological reasons why it is realistic to include sequential compartments (E.g. see Appendix A of Thornley and France, 2012). Even in a simple model, many parameters remain uncertain, and

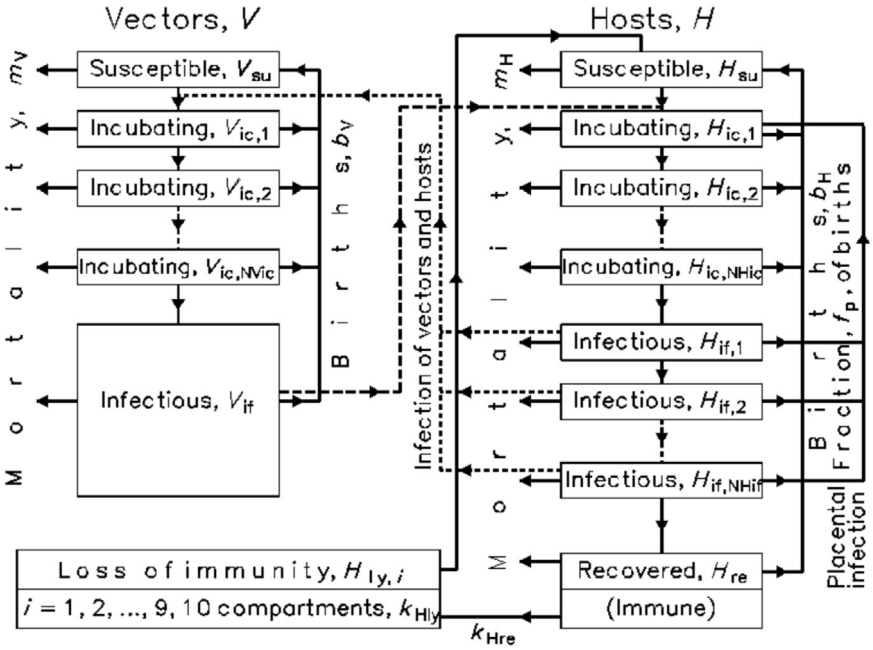

Fig. 1. Vector–host scheme for blue tongue disease. State variables are shown in the boxes. Vectors, V, can be susceptible (su), incubating (ic) with up to N_{Vic} stages, and infectious (if) with a single stage (this assumes that all infectious vectors have the same infectivity, and that there is no "recovered" vector condition). Hosts, H, can be susceptible (su), incubating (ic) with up to N_{Hic} stages, infectious (if) with up to N_{Hif} stages, or recovered (re). Recovered (immune) hosts can be moved, with rate constant, k_{Hre}, into a 10-compartment sequence, $H_{ly,i}$, $i = 1,\ldots, 10$, where there is gradual loss of immunity, with rate constant k_{Hly} between the 10 compartments. Rate constants (d^{-1}) for birth, b_V, and mortality, m_V, of vectors and of hosts, b_H and m_H, are given next to the arrows which denote these processes. Infection of susceptible hosts by infectious vectors and susceptible vectors by infectious hosts are indicated by the medium and short dashed lines and arrows. Placental infection of calves is represented by fraction, f_p, of births from infectious hosts. See Eqs. (11), (16) and (22) for the state-variable differential equations and Table 1 for definitions of symbols, parameter values and units.

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