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Development of simulation technology for modelling Bluetongue disease spread

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Executive Summary

Bluetongue Virus (BTV) is endemic in Northern Australia but southern regions, including sheep-rearing areas that could be impacted by the high mortality of Bluetongue among sheep, are BTV-free. However, the current lack of *Culicoides* midge species that are competent vectors in southern Australia does not mean that the risk of Bluetongue can be ignored. In 2006 and 2008 large and unprecedented Bluetongue outbreaks occurred in northern Europe in areas thought to be safe. The potential future distribution of BTV in Australia is a complex question that may be influenced by changes to weather patterns, the incursion of new competent insect vectors (species of *Culicoides* midge), the incursion of new strains of BTV, or by evolution of the virus itself.

Given that “real world” experimentation in large scale BTV spread is not feasible, a research technique that can address the question challenge is to develop “virtual world” models of BTV which are as realistic as possible, capturing the fundamental features of the underlying physical system.

We have created a computational simulation model of BTV spread in Australia. This model used relevant data sets including livestock locations and weather patterns along with assumptions about unknown factors (such as the characteristics of incursive BTV strains and vectors) to estimate the timing, geographical extent and livestock impact of a hypothetical Bluetongue outbreak.

The benefit of this work to the livestock industry is primarily as a tool for understanding the likelihood and impact of BTV incursions, and for planning possible mitigation measures such as vaccination that could be used in the event of a Bluetongue outbreak.

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1 Background – BTV in Australia

Bluetongue Virus (BTV) is endemic in cattle in Northern Australia but clinical disease in sheep has not been seen, despite the fact that some isolates are known to be pathogenic to Australian sheep. The future distribution of BTV in Australia may be influenced by changes to weather patterns, the incursion of new competent insect vectors (species of *Culicoides* midge), the incursion of new strains of BTV, or by evolution of the virus itself.

The spread of BTV in Northern Europe and its previously unforeseen ability to become endemic in cooler climates has resulted in increased research activities in virus transmission, on detailed reporting of these outbreaks and in the initiation of modelling studies (in the UK, for example). Modelling studies aim to develop techniques to determine the disease spread characteristics (e.g. scale and geographical extent) following an incursion. They would also be used to determine the effectiveness of interventions, such as vaccination, culling and movement bans, in achieving disease free status following an incursion into a previously disease-free area. Appropriate modelling technology is important from a biosecurity perspective, given that “real world” experimentation is not feasible. The research challenge is to develop models which are as realistic as possible, capturing the fundamental features of the underlying physical system.

2 Projective objectives

The overall objectives of the project, as stated in the original research proposal, were as follows:

This project will develop a simulation model for BTV in Australia to predict the future location of the disease following introduction of a novel virus strain or an incursion into a previously disease-free area, and to allow for the determination of optimal control or eradication policies. The simulation technology will be available for use in “real time” to predict disease spread via movement of infected animals and/or midge vectors. This project will apply novel infectious disease modelling methods developed by the Senior Investigator and colleagues at UWA together with techniques which we have developed for capturing the effect of wind on the spread of bushfires. The resulting simulation toolset will be available for use by biosecurity and animal disease managers within state and federal agriculture departments. It will be useful in improving national infrastructure for BTV control and to prepare for BTV incursions occurring in currently disease free areas, as well as being valuable for providing input into planning to achieve disease freedom in areas currently affected (e.g. the Pilbara, WA).

The specific objectives of the project are reviewed in the Discussion section of this report (Section 5).

3 Methodology

3.1 Simulation technology development methodology

This project has applied a novel complex systems modelling methodology developed by the Senior Investigator and colleagues at UWA. This methodology has previously been used in the domain of human and animal infectious disease spread, but also incorporates

techniques developed for simulating wind-driven bushfire spread, which in the context of Bluetongue is relevant to the wind-borne dispersal of midge vectors.

Using this methodology, simulation model development proceeds in a number of phases where individual sub-models are conceptualised, implemented and then applied and validated. This cycle is repeated as each physical phenomenon is added to the model. An overview of the development cycle is given below.

3.1.1 Conceptual model development

First a conceptual “synthetic world” model of the various physical processes that comprise BTV spread over the landscape is developed. This conceptual model is a detailed description of a number of mathematical models for a set of physical processes that, taken together, are sufficient to simulate the spread of BTV from one location (property) to another. The modelled processes cover the full cycle of infection spread through infection of a midge from infectious cattle, development of virus in midge, the possible wind-born transport of the midge, the infection of a susceptible host from the now infectious midge and the development of the virus within the new host. In addition, the movement of cattle and the fluctuation of the midge population is also modelled, as are outbreak intervention measures such as vaccination. The physical process models are based on published scientific literature, where various parameters are inferred from observation or experimentation.

This conceptual model description serves as a blueprint for the development of the software system that simulates BTV spread scenarios, as a vehicle for soliciting feedback from experts, and as a formal presentation of the model in research publications.

3.1.2 Implementation in software

The conceptual BTV spread model component, which is described mathematically, is then implemented in computer software; the resulting suite of software constitutes the simulation model. The software is designed to be readily parameterisable, allowing the core software to be used for a range of host species (including cattle and sheep), vector species that have different habitats and breeding sites, and various patterns host animal movement and spatial heterogeneity.

For this project Java was chosen as the primary implementation language. Extreme portability, a large user base, close to native code level performance, and support of modular object-oriented development make Java an excellent choice for this type of project. In addition to Java, various other standard scripting languages and tools (e.g. Python, R, Excel) were used for data preparation and analysis.

During the project it became apparent that a physically based atmospheric model would be needed to adequately model wind-borne midge dispersal. We chose to use the HYSPLIT model (see Section 3,4,2). We compiled HYSPLIT to run on our simulation server hardware, and invoked HYSPLIT as an extern component from the main Java application – thus combining the modularity of Java and the native-code performance of HYSPLIT (written in C), at the cost of reduced portability, as the system would require HYSPLIT to be re-compiled on any server machine running the simulation software suite.

After all the main simulation software components had been developed and integrated, we developed a graphical user interface (GUI) for the software (see Section3.8).

3.1.3 Application and validation

Using the developed simulator software system, a range of scenarios in Australia where incursion of BTV might occur were simulated. Such scenarios included incursion into otherwise BTV free areas, incursion of novel BTV serotypes or incursion via BTV competence appearing in *Culicoides* species previously thought not to act as vectors. Simulations were then used to determine the dynamics of spread in these scenarios, and the impact of various vaccination intervention strategies, using actual animal demographic data and historical weather data.

In this Methodology section we describe the development of each part of the conceptual model, showing how the whole model was developed in step-by-step fashion. In the following Results section we describe the outcome of the application and validation of each part of the model, and the model as a whole.

3.2 Simulator framework with landscape as discrete cells

The simulated area is divided into a set of approximately similar area spatial *cells*. Each cell has a nominal centroid location (latitude and longitude co-ordinates), and a set of information which captures the state of the landscape represented by the cell. Single cells are the finest level of spatial detail captured by the simulation model: cells are considered to have uniform characteristics throughout their area. The positions and sizes of cells used in a simulation will depend on the landscape and the data sources available.

Each cell has a set of associated data fields that represent the state of the landscape within the cell. These data fields are of several different types.

3.2.1 Geographic data

Cell geographic data includes latitude, longitude, altitude and area. The relative location of cells determines distances and directions between neighbouring cells, which influences the spatial spread of BTV via the dispersal of midges, depending upon prevailing wind conditions. The cell area determines the density of hosts and/or vectors in a cell, which may also influence the transmission of BTV from vector to host and host to vector within a cell.

3.2.2 Weather data

Weather enters into the model as an input data set and is updated for each cell for each (daily) simulation cycle. Temperature influences the model in multiple ways including vector reproduction rate, adult vector survivability, vector biting activity, vector movement activity, and virus development in vector. Wind drives vector dispersal, and other weather variables such as humidity and rainfall may affect vector habitats.

Weather is not actually computed by the model, but is a factor in several of the other processes, and so enters into the model as an input data set. Temperature influences the model in multiple ways: vector population dynamics, vector biting activity, vector movement activity and virus development in vector. Wind drives vector dispersal, and other weather variables such as humidity and rainfall may affect vector habitats.

Initially we obtained, from the Australian Bureau of Meteorology, 3-hourly weather elements (temperature, humidity, rainfall, wind speed and wind direction) for all automatic weather stations (AWS) in NSW for each year in the period 1980-2010. During each simulation cycle, the local weather occurring at each simulation landscape cell was interpolated from the closest AWS. This interpolation was simply the weather of the closest AWS for which valid data exist. As it turned out, this scheme was too approximate, and it was replaced by more sophisticated weather data sources.

A daily historical temperature data set for all of Australia (on a 0.05 degree grid) was obtained from the Bureau of Meteorology and incorporated into the simulation system. This has allowed the generation of *C.brevitarsis* overwintering risk maps and improved validation of the *C.brevitarsis* population dynamics and dispersal model.

Atmospheric data, used by the HYSPLIT component of the model for simulating wind-borne midge dispersal was sourced from a global reanalysis data set freely available from the American NOAA.

3.2.3 Host data

Each cell records the ruminant (cattle and sheep) populations that constitute a potential host population for BTV. In addition to the number and type of host animals in the cell, the current BTV infection state of the host population is recorded. This includes the number of uninfected (susceptible), exposed (infected but not infectious), infectious, and animals that have been infected but have recovered and are currently immune. Vaccinated animals are also placed in the immune state.

ABS Agricultural Census and ABARES Land Use data sets have been used to incorporate spatial heterogeneity of cattle populations into the simulation system. Cattle populations are incorporated both as hosts of BTV and as sources of cattle dung which forms the breeding habit of *C.brevitarsis*.

The ABS data estimates the number of animals present in each ABS SA2-level area. This gives a host density measure; however SA-2 areas are of a variable size and are at too coarse a spatial resolution in areas away from human population. We used high-resolution (approximately 1 km) ABARES land-use raster data to select the subset consisting of the 5 km resolution landscape cells containing hosts (i.e. land use codes for grazing or dairy). All the host-containing cells falling within a SA-2 area then had the cattle and sheep (separately) population of that SA-2 distributed uniformly across them. This ensured that host populations were excluded from the areas within each SA-2 that did not have significant livestock populations such as national parks and built-up areas.

3.3 Temperature-dependent vector population dynamics

One component of the overall BTV spread model is a sub-model that determines the midge density for any area at any time during the simulation; this sub-model is the *vector population model*.

3.3.1 Vector spatial heterogeneity

It is expected that vector density will not be uniform for an entire simulated area. Some spatial variation in vector density will occur due to spatial variation in weather and environment. Two otherwise similar areas may have different vector density due to differences in temperature, rainfall and humidity.

Another source of spatial heterogeneity in vector density is the suitability of a cell as a vector habitat. The rate at which the vector population can grow and the maximum density attainable may depend on factors such as suitability of breeding sites and density of the host population, as at least a minimum number of hosts is required to provide blood meals and (dung) breeding sites. The characteristics that constitute vector habit vary from species to species. For example, in an Australian context the presence of cattle constitutes a required habitat for midge species that require cattle dung for breeding. In other contexts the NDVI greenness index might serve as a proxy for the breeding habitat of other midge species.

Relevant research on spatial and climatic variability of midge habitats includes Animal Health Australia, 2001, Carpenter et al., 2009, Gerry and Mullens, 2000, Purse et al., 2005b, Racloz et al., 2008, Mellor et al., 2009.

3.3.2 Vector temporal variation (population dynamics)

It is assumed that vector populations will change through time, following seasonal patterns such as temperature, rainfall and wind variation. The vector population dynamics model calculates the population density for each cell for each day.

In the case where midge populations are invasive and encroaching into an area they did not previously inhabit, or where wind dispersal significantly alters population density patterns, a dynamic vector population model is needed. A dynamic model consists of birth and death rates (or birth rates and life spans) as functions of current population, temperature and local habitat. A conventional population dynamics model (Wilson and Bossert, 1971) can be fitted to data for the midge species and habitats of interest (as for example was done by the research team in Karl et al. 2014).

In order to correctly model the virus prevalence, the population model needs to determine not only the population change from day to day, but also the effective birth and death rates (or alternatively birth rate and life span). This is necessary because in the absence of new vector infections, the virus prevalence in the population will decay as old vectors die and are replaced new uninfected vectors. The rate at which this occurs is dependent on the turnover (or lifespan) of the vector population. It is assumed that there is no vertical or vector-to-vector virus transmission.

Relevant research on temporal variability of midge vector populations includes Murray, 1995, Gerry and Mullens, 2000, Racloz et al., 2008, Klingseisen et al., 2011, Bishop et al., 2000, Bishop et al., 2004, Mullens et al., 2004a

Culicoides population density is critical in modelling the spread of BTV, as transmission is only possible where hosts and competent *Culicoides* vector species are co-located. *Culicoides* density affects BTV transmission in at least two ways: higher *Culicoides* density means more biting and greater rate of BTV transmission between hosts in the same area,

and higher *Culicoides* density means more midges being dispersed to neighbouring areas, possibly carrying BTV with them. The size of the vector population thus directly impacts the spatial spread and rate of transmission.

The density of *Culicoides* populations is climate dependent, and in some areas is highly seasonal. Thus in order to simulate the spread of BTV throughout the year in different climatic zones, a dynamic model of *Culicoides* population that takes into account seasonality is required. In areas where the climate is favourable for *Culicoides* vector species, *Culicoides* may be present and active all year round. In other areas, the *Culicoides* population and/or activity may become very low during winter, possibly curtailing BTV spread. In still other areas, the climate may support incursions of *Culicoides* populations (and BTV spread) during summer, but extended cold winters may render *Culicoides* locally extinct.

Note that it is possible to model BTV spread without explicitly representing vector populations by using, for example, a simplified approach where BTV transmission is approximated as a host-to-host or farm-to-farm transmission process (de Koeijer et al., 2011). However this does not allow us to explicitly model spatial spread of BTV via midge dispersal, nor BTV spread in areas where the presence of a *Culicoides* population may vary seasonally or from year to year. We have not adopted this highly approximate approach.

The vector population dynamics model described below has been implemented in the BTV spread simulator software application. When supplied with initial vector populations and daily temperature time series, the simulator generates adult and immature *Culicoides* population time series for each landscape cell. Figs. 10-16 appearing in the Results section were generated from simulator output.

3.3.3 Landscape cell vector population state

At the most abstract level, each landscape cell contains data representing the state of the *Culicoides* population in that cell. The landscape cell may be in one of three *vector population states*, namely *active*, *inactive* or *extinct*.

1. **Active**, indicating that *Culicoides* are present in the cell, that the *Culicoides* breeding cycle is on going, and *Culicoides* are active and capable of transmitting BTV. In cold conditions, breeding and development rates may slow to the point where they are exceeded by the adult death rate, in which case the population will fall, and may become inactive.
2. **Inactive**, indicating that adult population numbers are very low, and BTV transmission may be reduced or absent. If temperatures subsequently rise, a cell in the inactive state may become active as immature *Culicoides* emerge. Alternatively, sufficiently cold and sustained conditions may kill or render unviable all adult and immature *Culicoides*, making the vector population extinct in the cell.
3. **Extinct**, indicating that no viable *Culicoides* are present in any life stage (adult, larva, or pupa). As a result changes in weather or habitat conditions will not cause any change in the cell population state. If new midges are transported to the cell and the conditions are favourable, the cell state may then transition into an active state.

These states and the possible transitions between them are illustrated in Fig. 1.

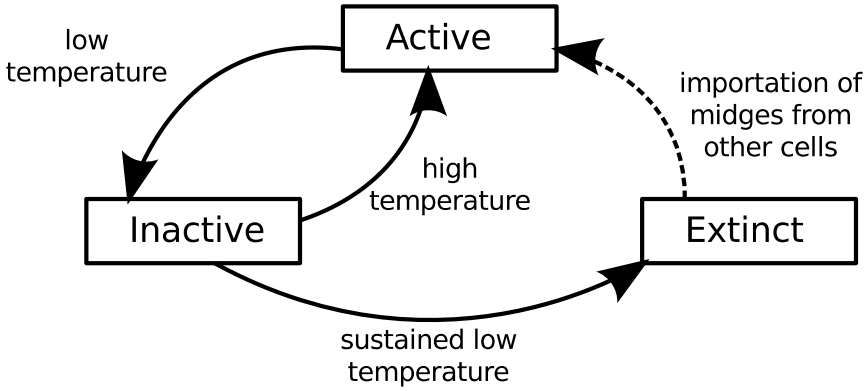


Fig 1 : Landscape cell vector population state

Cells in the active and inactive states have two additional numeric attributes representing the population density of the adult and immature *Culicoides* (taken to include all pre-adult stages) in the landscape cell.

3.3.4 Landscape cell vector population dynamics

It is assumed that population density evolves according to a logistic population model (Wilson and Bossert, 1971). This is a standard model in population biology where populations grow exponentially when unconstrained by resources, but where population growth rate decreases with increasing population density, representing competition for some finite resource required for population growth. In our model there are 3 on-going processes that modify the density of the adult and immature populations, and we assume that the rate at which these process occur is temperature dependant. Temperature is a critical factor in the *Culicoides* population dynamics model, and the model's temperature dependencies are described below in section 2.2 .

1. **Oviposition**. Adult female *Culicoides* perform a cycle of blood feeding followed by oviposition (egg-laying). The rate at which adult *Culicoides* lay eggs (increasing the immature *Culicoides* population in the model) depends both on temperature and on the current immature *Culicoides* population density. It is assumed that there is a population limiting factor for immature *Culicoides* (i.e. cow dung for *Culicoides brevitarsis*), and that the oviposition rate decreases linearly from a maximum level to zero as the immature population increases from zero to the limiting density for the immature population. Density limited *Culicoides* larval development at survival is reported in (Akey et al., 1978).
2. **Maturation**. *Culicoides* larva hatch from eggs and develop into pupae, from which they emerge as adults. The rate at which immature *Culicoides* mature into adults (increasing the adult population in the model) is assumed to be a function of temperature.
3. **Death**. Adult and immature *Culicoides* are assumed to die at a particular rate, which is temperature dependant for adults, with mortality becoming higher at higher temperatures. Note that the immature "lifespan" is the mean time in which an immature *Culicoides* dies, *given* that it has not matured into an adult.

The relationship between these processes is illustrated in Fig 2. In addition to these processes, vector dispersal will also alter cell vector population states, with adults being moved out of some cells and into others.

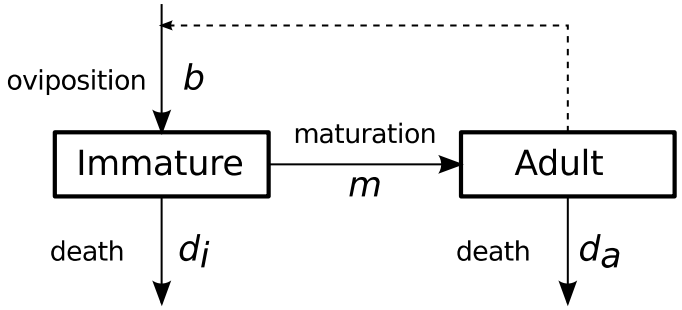


Fig. 2 : Vector population dynamics model

The model vector population dynamics *within* a landscape cell can be described as an ordinary differential equation (ODE) system as follows.

$$\frac{dp_i}{dt} = b\left(1 - \frac{p_i}{p_{imax}}\right)p_m - d_i p_i - m\,p_i$$

$$\frac{dp_m}{dt} = m\,p_i - d_m p_m$$

The symbols appearing in the equations are described in Table A1 in the Appendix.

The implemented model differs from this ODE description in a number of ways.

1. The model implements a discrete-time difference equation with one-day time steps; evolution is deterministic for large vector populations, and stochastic for small populations (where the distinction between “large” and “small” is parameterisable). Thus cell populations can become extinct when the population is small and net growth rate is negative. This is important as it correctly models the situation where vectors completely die out in a cell, so that the population will not recover without external seeding even if conditions later become favourable for population growth.
2. The immature *Culicoides* population density limit factor is not (1 - p_i / p_{imax}) but max(0,1 – p_i / p_{imax}), i.e. when $p_i > p_{imax}$, the oviposition rate is zero and does not become negative.

The nature of the ODE based population model means that while the average lifespans of adult and immature *Culicoides* matches observed lifespans, the age distribution of populations may not be correct (being skewed to more younger individuals). If it is the case that there are important population dynamic or BTV infection processes that depend upon

Culicoides age distribution, additional life stages could be introduced to give more realistic lifespan distributions.

3.3.5 Temperature dependent model parameterisation

Three critical temperature dependencies of *Culicoides* population biology are captured in the model; these are the *activity* of adults, the *maturation rate* of immature Culicoides, and the *lifespan* of adults.

1. **Activity**. The flying, biting and oviposition activity of *Culicoides* midges is temperature dependent (Mellor et al., 2000). The period between ovipositions ranges from over 10 days at 13° C down to 2 days at 35° C (Mullens et al., 2004a) (note, data for *C. sonorensis*). This is consistent with other estimates of gonotrophic period of 3.5 – 4.5 days in summer (Birley and Boorman, 1982) (data for *C. obletus* group).
2. **Maturation Rate***.* Experimental data for *C.brevitarsis* indicates that maturation time ranges from more than 35 days at 17 ° C down from 10 days at 36° C (Bishop et al., 1996). This is consistent with other estimates, e.g. 11-24 days for the egg to adult period (Campbell and Kettle, 1976).
3. **Lifespan***.* Adult *Culicoides* survive for periods from 20 days at temperatures below 10° C down to 5 days at temperatures above 20° C (Mullens et al., 2004a). Immature *Culicoides* survive for approximately 28 days (Bishop et al., 1996).

As a result of these temperature dependencies, the behaviour of the population model has two important temperature regimes.

At high temperatures, adult activity is high, giving a high oviposition rate. Although adult mortality increases with temperature, the oviposition rate also increases, meaning that fecundity does not decrease with increasing temperature. In addition, the immature maturation period is short and most immature *Culicoides* emerge and do not die in the immature state. In this temperature regime, the population grows until the oviposition rate is limited by the immature population density.

At low temperatures, adult activity is low, giving a low oviposition rate. What is more, the immature maturation period is long, becoming comparable to the immature lifespan, and immature mortality becomes significant. With fewer immature *Culicoides* emerging and the slow rate of oviposition, the immature and adult populations decline and eventually becomes extinct. Note that due to the relatively long immature lifespan, the immature population may take several months to become extinct after the initial crash of the adult population.

3.4 Vector dispersal

In order for BTV to spread from one location to another, either infected host animals or infected vectors must move. Two types of vector movement are considered.

3.4.1 Short-range diffusion

None of the midge vector species of interest exhibit self-propelled long range movements. In the absence of wind, they can be assumed to move by a slow random diffusion process (Rudd and Gandour, 1985, Kareiva, 1983). This process moves a small proportion of vectors to nearby cells, with the numbers falling off rapidly with distance. Research relevant to the

dispersal of midges and spatial spread of bluetongue (without the explicit consideration of wind-blown midges) includes de Koeijer et al., 2011, Ducheyne et al., 2011, Pioz et al., 2011.

In the absence of wind or other directional stimuli, insect dispersal can be modelled as a diffusion process (Rudd and Gandour, 1985, Kareiva, 1983). We assume that adult midges move at random, and the number of midges crossing into a neighbouring cell is proportional to the density of midges in the cell, as shown in Fig. 3.

Three studies report diffusion coefficients for *Culicoides* species: 60.11 m^2/s for *C. impunctatus* (Rudd and Gandour, 1985, Kettle, 1951) and 12.9 m^2/s for *C. variipennis* (Lillie et al., 1981). We have failed to find such quantified dispersal data for *C. brevitarsis*: we use the greater *C. impunctatus* value unless otherwise stated, since for the purposes of this project over-estimating midge dispersal is preferable to under-estimation.

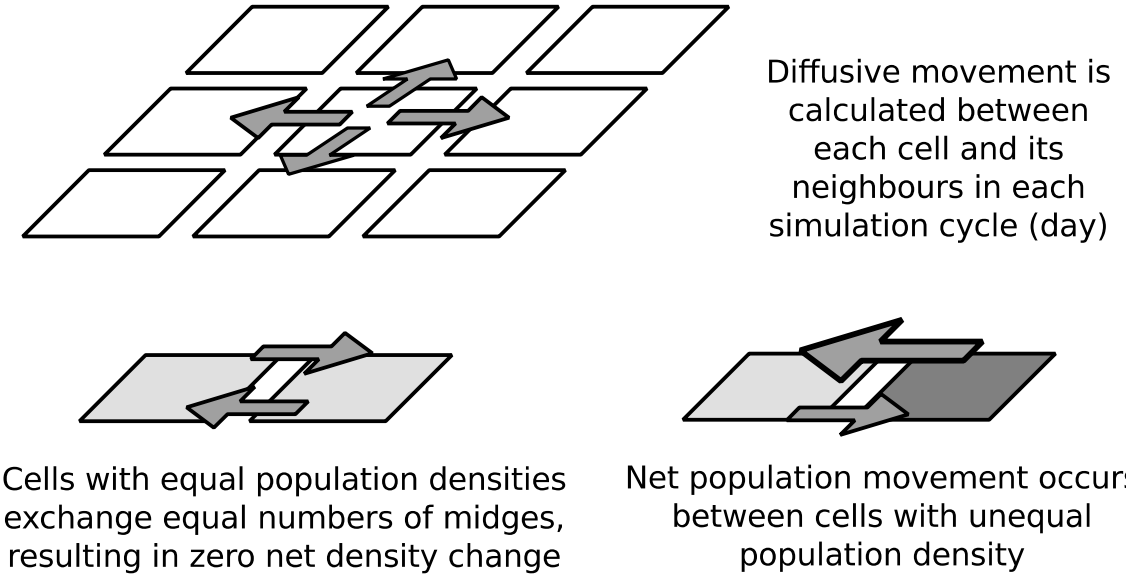


Fig. 3

When applied to a rectangular array of cells the diffusive transport process described above implements the forward Euler numerical solution to the diffusion equation (Atkinson, 1978).

3.4.2 Wind-borne dispersal

Culidoides midges are dispersed by the wind (Murray, 1991, Bishop et al., 1995b). Our model of midge dispersal is based on field studies of *C. brevitarsis* flying behaviour (Murray, 1987) and long-distance dispersal (Bishop et al., 1995b, Bishop et al., 2000), however the model is parameterised so that other midge species can also be represented.

For the purposes of wind dispersal, each 1-day simulation cycle is broken in to 3-hour wind sub-cycles. It is assumed that adult (female) midges take to the air from dusk when the temperature is 18 degrees C or greater and the wind speed is 8 km/h or less (Murray, 1987).

We assume that during each wind sub-cycle for which these conditions hold, a fixed proportion of the midges in a landscape cell take flight and are transported downwind, as illustrated in Fig. 4. It is assumed that this proportion of midges are removed from the source cell, and distributed over the cells within a dispersal footprint.

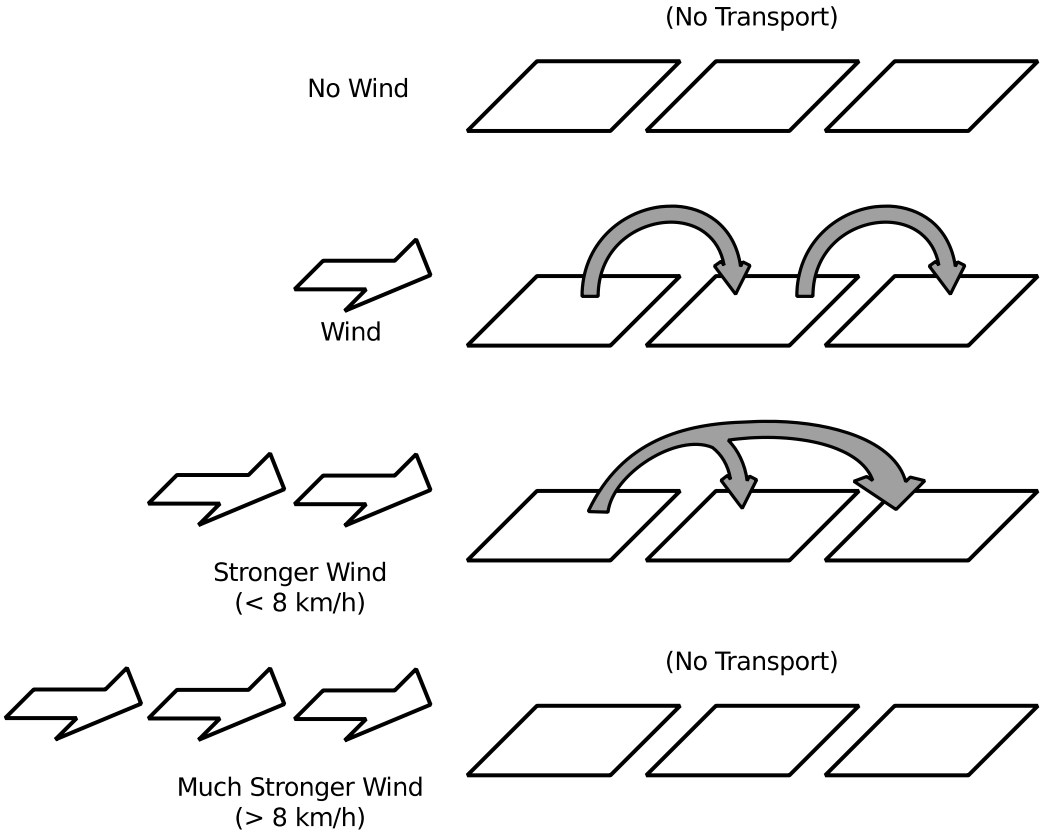


Fig. 4

The initial model used 3-hourly wind direction with fan-shaped footprint, based on surface winds from BOM automatic weather stations (AWS). While this did provide an approximate model for wind-borne midge spread, an improved sub-model based on the HYSPLIT atmospheric dispersal model was incorporated.

Previously, the simulator used surface winds recorded at the nearest AWS to determine wind-borne transport of midges from each simulation cell. In our previously published research outcome of this project we found that simulated *C.brevitarsis* spread in NSW best matched observed spread if wind-borne transport occurs at speeds up to 4 times the recorded surface (i.e. 10m) wind speed, indicating that midge wind transport may occur at higher altitudes where winds are stronger (Kelso and Milne, 2014).

Research in Australia and Europe has found that upper atmosphere (at altitudes up to 3,000m) winds are capable of transporting *Culicoides* midges and BTV quickly over long

distances (Purse et al., 2005a, Ducheyne et al., 2007, Hendrickx et al., 2008, Eagles et al., 2014). This research used atmospheric transport algorithms that model the three-dimensional movement of air. We investigated the Numerical Atmospheric Modelling Environment (NAME), developed by the UK Met Office (Jones et al., 2007) and Hybrid Single Particle Lagrangian Integrated Trajectory model (HYSPLIT) , developed by the US NOAA Atmospheric Resources Laboratory and the Australian Bureau of Meteorology (National Oceanic and Atmospheric Administration, 2015). HYSPLIT has been successfully used for modelling of wind-borne transport of *Culicoides* midges to Australia from Indonesia (Eagles et al., 2012, Eagles et al., 2014, Eagles et al., 2013).

We obtained the HYSPLIT software from NOAA / ARL, and have incorporated it into our *Culicoides* / BTV spread simulator as an alternative model of wind-borne midge transport. The HYSPLIT dispersal model (specifically, the deposition process that distributes midges along the calculated trajectory track) is considerably more computationally intenstive than the previous AWS-based dispersal mechanism. However, if repeated simulations are being made of the same area for the same time period (which is the case if, for instance multiple BTV introduction or intervention scenarios are being simulated), the results of the trajectory and deposition calculations are cached and re-used. For a six month simulation for a typical 12,000 cell landscape, this reduces computation time from 11 hours to less than 20 minutes.

We conducted tests to benchmark HYSPLIT-based midge dispersal against the previous surface wind AWS-based dispersal. The tests showed that, as intended, the HYSPLIT dispersal model predicts wind transport and down-wind deposition of midges with higher spatial and temporal resolution compared to the AWS-based dispersal model (see Fig. 5 below for an illustrative example). The HYSPLIT-based dispersal model provides higher temporal and spatial resolution due to the fact that (a) HYSPLIT uses gridded atmospheric wind data and interpolates between grid points, whereas the AWS model uses data from the closest AWS (of which there are 138 in the simulation area), and (b) HYSPLIT follows trajectories at 1 hour time resolution whereas the AWS model uses 3-hourly time steps (the resolution of the available AWS data). Furthermore, since the HYSPLIT model takes into account upper-atmospheric winds it provides a more accurate prediction of wind-borne midge dispsersal compared to the AWS-based model which is based on surface winds only.

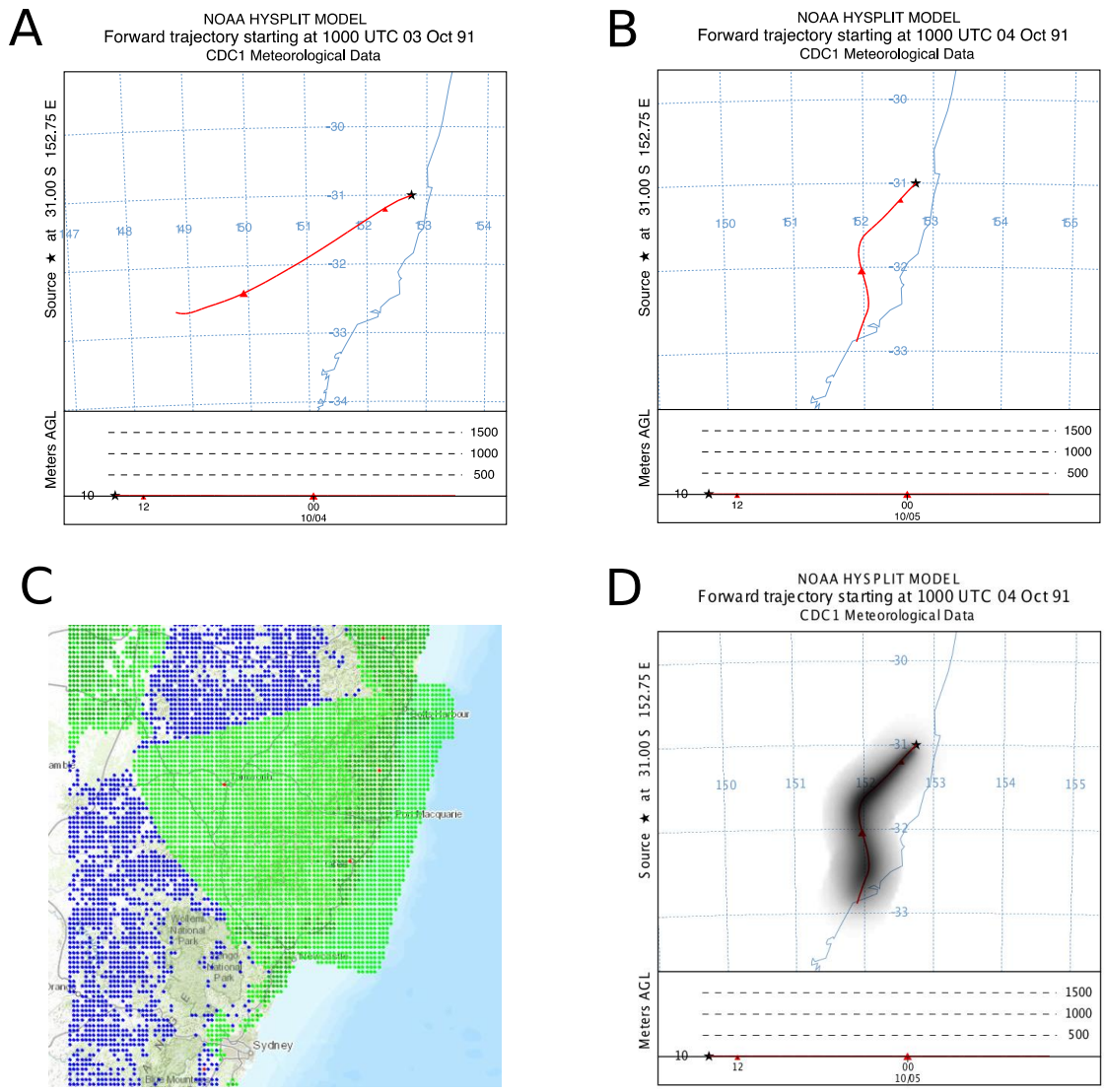


Fig. 5

This Fig. 5 is an example comparison of HYSPLIT-based versus AWS-based wind-borne midge dispersal. The top row (A and B) show HYSPLIT trajectories from a sample point on the 3rd and 4th of October 1991. The bottom row left (C) shows AWS-based midge dispersal for the same dates: a wind transport event is evident as a large green wedge shape. Bottom row right (D) shows a single 24 hour HYSPLIT based midge dispersal from one point on the 4th of October.

3.5 Bluetongue Virus transmission

In order to model the transmission of BTV it is necessary to represent the current *infection state* of both the host (ruminant) and vector (*Culicoides* midge) populations in each landscape cell, and the *transmission dynamics* that change the infection state over time due to infection and progression of the disease. An overview of the transmission model is shown in Fig. 6.

In this stage the 4 processes required to model the full BTV transmission cycle are added:

1. vector-to-host transmission
2. virus development in host (including BTV-caused host mortality)
3. host-to-vector transmission
4. virus development in vector

The competence of a midge species to transmit a particular virus strain can be modelled by the β_{vector} transmission parameter, which determines the probability of a midge becoming infected from a bite on an infectious host. β_{host} might also vary with midge and virus species, although evidence suggest that this value is very high (a single bite from an infected midge being highly likely to infect a susceptible host).

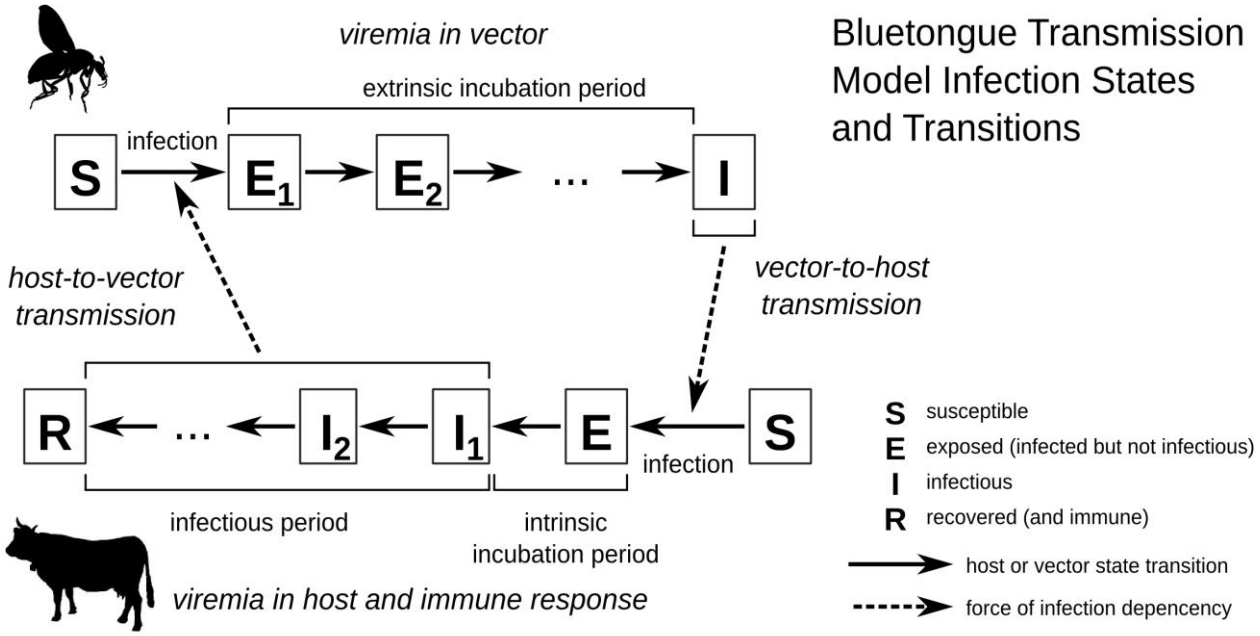


Fig. 6

3.5.1 Host infection states

The host population of a landscape cell is represented by an integer number of host animals (e.g. cattle or sheep) The infection state of the host population is represented by partitioning the host population into a number of sub-populations. These are the susceptible (S), exposed (E – infected, but not yet contagious), infectious (I_n – N sequential infectious states), and recovered (R – hosts that have been infected, have recovered, and are now immune to the disease). Note that N different infectious stages are included so that the distribution of infectious periods can be realistically modelled (with a single stage only an exponentially decreasing distribution of infectious duration can be modelled). The host infection state is represented independently for each host species present; for example landscape cells containing cattle only, sheep only, or both cattle and sheep are possible. The vector population in a cell is common to all host species present, so infection present in one species can spill over to another via infected vectors.

3.5.2 Vector infection states

The vector population of a landscape cell is represented by a vector *population density* which is also partitioned into subpopulations: susceptible (S), exposed (E_n – N sequential states for midges infected but not yet contagious), and infectious (I). As above, N different exposed stages are included so that the distribution of incubation periods can be realistically modelled. This N is not necessarily the same as the N for host I stages. Note that these states apply to the adult female midge population: the immature *Culicoides* life stages (eggs, larvae, and pupae) do not participate in BTV transmission.

3.5.3 Transmission dynamics

The simulation of BTV transmission proceeds in discrete time steps representing one day. In each time step, the infection states of each landscape cell are updated based on the current infection state of the cell and the weather in the cell on that day. The spread of BTV involves four processes that change the host and vector infection states with time; Bluetongue can also change (reduce) the host population state due to deaths caused by infection. The computation of each of these processes proceeds in the same way, calculating the subset of the host or vector population that transitions from a *source* subpopulation state to a *target* subpopulation state (e.g. from source S to target E). In the case of infection-caused mortality, the source subpopulation is reduced without increasing any other subpopulation. The computation proceeds as follows.

- A *transition rate* is calculated i.e. the inverse of the average period of time before the state transition will happen to the host or vector. The transition rate for a process can depend on the current host population infection state, vector population infection state, and weather in the landscape cell.
- A *transition probability* is calculated; that is, the probability that the state transition will occur in the simulation cycle. If the transition rate calculated above is λ and the simulation cycle duration is *dt*, the transition probability is:

$$P_{trans} = 1 - e^{-\lambda\, dt}$$

- A *subpopulation change* is stochastically chosen based on the transition probability and the size of the source subpopulation (*n*) of the process. Since the transition probability applies to each member of the source subpopulation, the number of subpopulation members that undergo the transition in one simulation cycle (*k*) is drawn from the distribution:

$$k = \text{Binomial}(n, P_{trans})$$

- In the case of the midge population (or for large host populations), suitable approximations of the binomial distribution are used. The population states are updated by subtracting *k* from the source subpopulation state and adding it to the target subpopulation state.

The four Bluetongue transmission processes are as follows. The numerical values of the parameters named below are given in Table A2 in the Appendix.

1. **Vector-to-host transmission**. As infected midges bite susceptible hosts, hosts become infected. The rate for the host S $\rightarrow$ E transition (i.e. the force of infection on each host) is:

   $\lambda_{host} = \beta_{host} * A(T) * (N_{vector} / N_{host}) * (I_{vector} / N_{vector})$

   where

β_{host}	is the basic probability of host infection from a bite from an infectious vector.
A(T)	is a temperature dependant activity factor, which represents the increased rate of vector flight and biting at increased temperatures.
N_{vector}, N_{host}, I_{vector}	are the total vector, total host, and infected vector populations respectively. The ratio (N_{vector} / N_{host}) is the per-host vector density in the landscape cell, and the ratio (I_{vector} / N_{vector}) is the proportion of infected vectors.

2. **Viraemia in host**. Host infection is assumed to follow a SEIR (Susceptible-Exposed-Infectious-Recovered) pattern. The host population in a cell is partitioned into S, E, I, and R stages (with multiple E and I stages as described below).

   The intrinsic incubation period of the virus is assumed to be a fixed number of days, constant for a host-virus pair. The number of animals in each stage (day) of incubation is tracked, with newly infected animals added to stage 1, animals in the final incubation stage becoming infectious, and animals in intermediate stages moving forward one stage.

   The host infectious period is handled similarly to the intrinsic incubation period (although possibly with weekly rather than daily stages), with a host and virus specific infectious period.

   Animals exiting the infectious stages enter a recovered stage, and are assumed to be neither infectious or susceptible to infection for the remainder of the simulation.

   Mortality from Bluetongue is modelled by a (host and virus species-specific) constant daily mortality rate for animals in the infectious stages, with numbers of dying animals generated stochastically. This is only of significance in areas where sheep are present

   The rates for host transitions from E $\rightarrow$ I, and the rates for the $I_n \rightarrow I_{n+1}$ and I $\rightarrow$ R are assumed to be constant and are governed by the intrinsic incubation period and infectious period, respectively.

3. **Host-to-vector transmission**. The rate of the vector S $\rightarrow$ E_1 transition (i.e. the force of infection on each vector) is:

   $\lambda_{vector} = \beta_{vector} * A(T) * (I_{host} / N_{host})$

where

β_{vector} is the basic probability of vector infection from a bite from on an infectious host.

A(T) is a temperature dependant activity factor, which represents the increased rate of vector flight and biting at increased temperatures.

I_{host} is the total number of infected hosts (i.e. the sum of all of the In host subpopulations).

N_{host} is the total host population. The ratio (I_{host} / N_{host}) is thus the proportion of infectious hosts.

4. **Viraemia in vector**. Vector infection is assumed to follow the SEI (Susceptible-Exposed-Infectious) pattern. The total vector density is partitioned in to S, E and I stages (with multiple E stages).

   The extrinsic incubation period of the virus is assumed to last for a number of days which is a function of the temperature, with the quantity of newly infected vectors transitioning from the S stage to the first E stage, and the quantity in each E stage proceeding to the next until the I stage is reached. Note that because the length on the incubation period can change with temperature, a shortening incubation period will move several of the final E stages to the first I stage.

   The infectious stage is assumed to last until the end of the vector's life. Vectors are assumed to die (and be removed from S, E and I stages) at a rate determined by the current temperature dependent mean lifespan. Lifespans are thus assumed to be exponentially distributed; if this is too unrealistic then additional life stages will be introduced. Research relevant to the dynamics of virus development in midge vectors includes (Gerry et al., 2001, Mullens et al., 2004a, Mellor et al., 2009, Carpenter et al., 2006).

   The rate of the $E_n \rightarrow E_{n+1}$ and $E \rightarrow I$ transitions is governed by the extrinsic incubation period, which is a temperature dependent parameter.

When multiple host species (cattle and sheep, for example) are simulated, the activity factor *a* in the λ_{host} calculation for each host species is partitioned amongst host species taking into account relative host species numbers and possible host preference amongst vectors. The force of infection on vectors λ_{vector} is summed over host species, also taking into account host preference in *a*.

Vectors in a cell form a single pool that can bite any host species present although possibly prefering biting some species over others.

3.6 Vaccination Interventions

Vaccination is an effective measure for preventing BTV transmission, provided that a vaccine is available that is effective against the particular virus serotype causing the outbreak. For example, BTV-8 was unexpectedly introduced into northern Europe and began spreading in 2006. No vaccine was initially available and large numbers of infections

occurred. A vaccine effective against BTV-8 was developed and deployed in varying degrees in Germany, France and the UK in 2008. This played a role in ending the BTV-8 incursion, and may have slowed the spread of BTV-1 that was subsequently introduced in 2008 (the vaccine was also effective against BTV-1) (Gubbins et al., 2010, Pioz, 2014, Baetza, 2014). BTV-1 is active in Australia; as are other serotypes that are not covered by any current vaccine, for example BTV-21.

We have implemented a flexible reactive vaccination model, in which detected host infections trigger a vaccination response, during which host animals within the landscape cell and all cells within a certain distance are vaccinated.

In order to support the vaccination sub-model, three new data fields are recorded for each cell: the date on which vaccination was triggered in the cell (which is initialised to “never”), the number of susceptible vaccinated hosts, and the number or hosts who have been made immune through vaccination.

The vaccination sub-model process occurs in two phases.

3.6.1 Infection detection and breakthrough infections

Each time a host animal becomes infected (which occurs in the transmission process), there is an independent fixed probability that the infection will be detected. The first time an infection is successfully detected in a cell, that cell, and all unvaccinated cells within the vaccination radius, have their vaccination date changed from “never” to the current cycle plus a delay. While in the simulation the detection event is computed at infection time, in fact the actual detection would happen later, after an incubation period, and vaccination response would occur after this due to necessary logistics. The delay parameter accounts for these factors. All further detection events occurring in the cell are ignored – it is assumed that vaccination will only be triggered once for a cell.

If an infection occurs in a cell that has been vaccinated, possible “breakthrough” infections are accounted for. This means that if say N out of S susceptible hosts become infected, then the number of susceptible-but-vaccinated hosts (which is separately recorded for each cell as part of the cell’s vaccination state information) is reduced stochastically by binomial (N, N/S).

3.6.2 Vaccinated cell process

Once a cell is marked with a vaccination date, it progresses through a sequence of events

Delay. As noted above, there is a delay period after an infection has been detected but before the marked vaccination date.

Vaccination. On the marked vaccination day, a certain percentage (the vaccination coverage parameter) of host animals in the cell are vaccinated. If there are S susceptible out of a total of T hosts, then the number of susceptible-but-vaccinated hosts is stochastically set to binomial (S, coverage).

Immunity development. Vaccines do not instantly provide immunity: there is an immunity development time which we model as a stochastic process with rate set by the mean-time-to-immunity (TTI) parameter. Susceptible-but-vaccinated hosts that become immunte

through vaccination are deducted from the susceptible-but-immune count, have their immunity status set to R (i.e. can no longer become infected), and are added to the immune-through-vaccination count.

Note that all vaccination model parameters can be made host species and BTV serotype specific. For simplicity, detection of infection in any host species or with any serotype triggers a generic vaccination response in which all host species are vaccinated (with a vaccine appropriate to that serotype). Each parameter of the vaccination model is listed in Table A5 in the Appendix with baseline values and supporting references.

3.7 Simulation Algorithm

In order to simulate BTV spread across the landscape each of the previously described process sub-models is executed for each cell. This updates the simulation state from one day to the next, and this process is repeated for the duration of the simulation. The simulation algorithm proceeds as follows.

Initialisation

Simulations are initialised by setting all the data fields (described in section 2) for each cell, according to the intended spread scenario. This will consist primarily of populating cells with host and vector populations, which will be susceptible for incursion scenarios and a mix of susceptible, exposed, infectious and recovered states for endemic infection scenarios.

Weather series and a set of animal movement events should be prepared or generated for the simulation.

Simulation Cycle

The following algorithm is used to simulate the spread of BTV across the landscape.

1. The geographic area of interest is selected, and the landscape within that area is partitioned into discrete landscape cells, and the geographic data for the cell recorded.
2. A weather series is loaded for a set of weather stations in the simulated area. From these the weather at any cell for each day of the simulation can be interpolated.
3. Vector and host population data are initialised. Host populations are initialised from records of cattle and sheep holdings, while vector populations are initialised based on *Culicoides* habitat data and season. The BTV infection states of host and vector populations are initialised according to the simulation scenario. For incursion scenarios all populations are initially susceptible; while for scenarios including BTV-endemic areas the infection states are a mix of susceptible, exposed, infectious and recovered states.

Note that the initialisation performed in steps 1-3 can be saved and re-used for multiple simulations.

4. Simulations proceed in a series of daily cycles, each of which updates the simulation state data for every cell from one day to the next. This is performed as follows:

   for each cell C

update weather from weather series
update vector density using vector population model
    remove vectors according to vector death
    add new susceptible vectors according to oviposition and maturation
advance vector infection state (E to I)
for each host species
    advance host infection state (E to I, I to R)
for each host species
    calculate new host infections and update host state
for each host species
    calculate force of infection on vectors
calculate new vector infections and update vector state
for each cell within vector diffusion range from C
    disperse midges by diffusion
for each cell within wind dispersal footprint from C
    disperse midges by wind transport
for each cell in which a new host infection occurred
    calculate the detection phase of the vaccination process
for each cell in which vaccination is active
    calculate the number of vaccinated hosts that acquire vaccine immunity and update host state
for each cell
    write out a snapshot of all requested cell statistic for later analysis (typically the host and vector population and infection states)

5. The outcome of a simulation run is a complete trace of all animal infection events occurring in the simulation on a cell-by-cell basis. All aspects of infection spread can then be examined, including the rate at which new animals and farms become infected, the geographical pattern of spread, the relationship between vector population dynamics and infection spread, and the expected final attack rate. In addition, the daily *Culicoides* population and its infection state are also recorded.

The computer memory required to run a simulation depends on the size of the area simulated, and time required depends on the area and the number of days simulated. To give indicative values, our simulations of north-eastern NSW used 12,220 landscape cells and covered an area of approximately 720 km (north-south) by 440 km (east-west). On a circa 2014 desktop computer, a six-month simulation takes approximately 11 hours, or 20 minutes if the HYSPLIT wind transport component has been pre-computed.

3.8 Graphical User Interface

We developed a graphical user interface (GUI) application program for the simulator system. The simulator GUI allows the user to manage one or more simulation projects, each of which allows the user to specify and then modify all the spatial datasets, scenario starting conditions and model parameters needed to run a simulation. Upon running a simulation, the simulation setup is recorded for future reference, the simulation is run, and then the simulation result is presented as a series of map snapshots that shows the state of the midge and vector population in each simulation landscape cell as time progresses.

We developed an initial prototype of the user interface in Java, using the ESRI ArcGIS Java Runtime framework to provide the map display component. However, for ease of access and to avoid propriatry software licensing costs which would impede deployment, we recreated and continued development using a web browser interface. This interface employs Google Maps, Javascript and HTML on the client side to display simulation outputs; while on the back (server) end it employs a web server (Apache) interfaced to the the Java BTV simulator application via the Common Gateway Interface (CGI). A set of simulation output process scripts takes the raw simulator time-series outputs and generates the necessary data files that are requested by the client browser pages. Using a web-based user interface has the advantage of requiring no client-side deployment to users (other than access to the internet and a web browser). The BTV spread simulator could thus be offered as a service, with the server computing infrastructure scaled to demand as needed. Screen shots of the simulator interface are shown in Figs. 7 and 8.

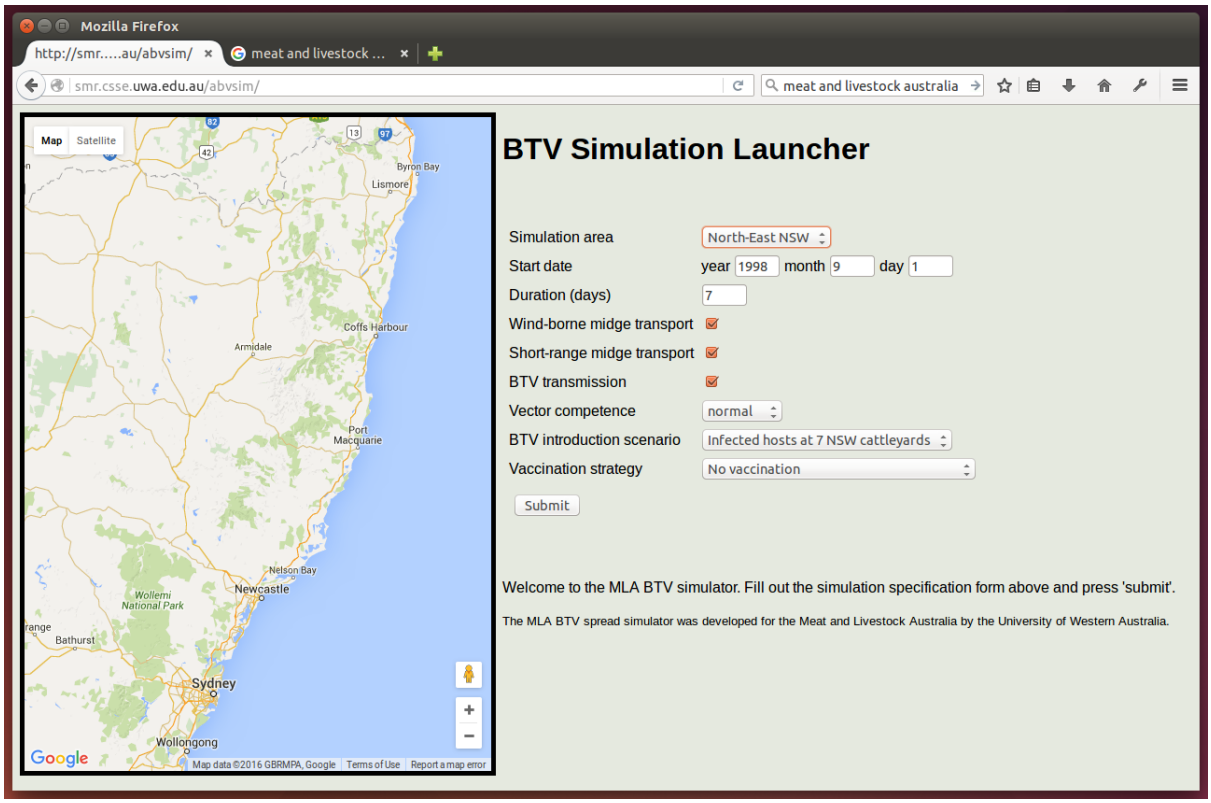
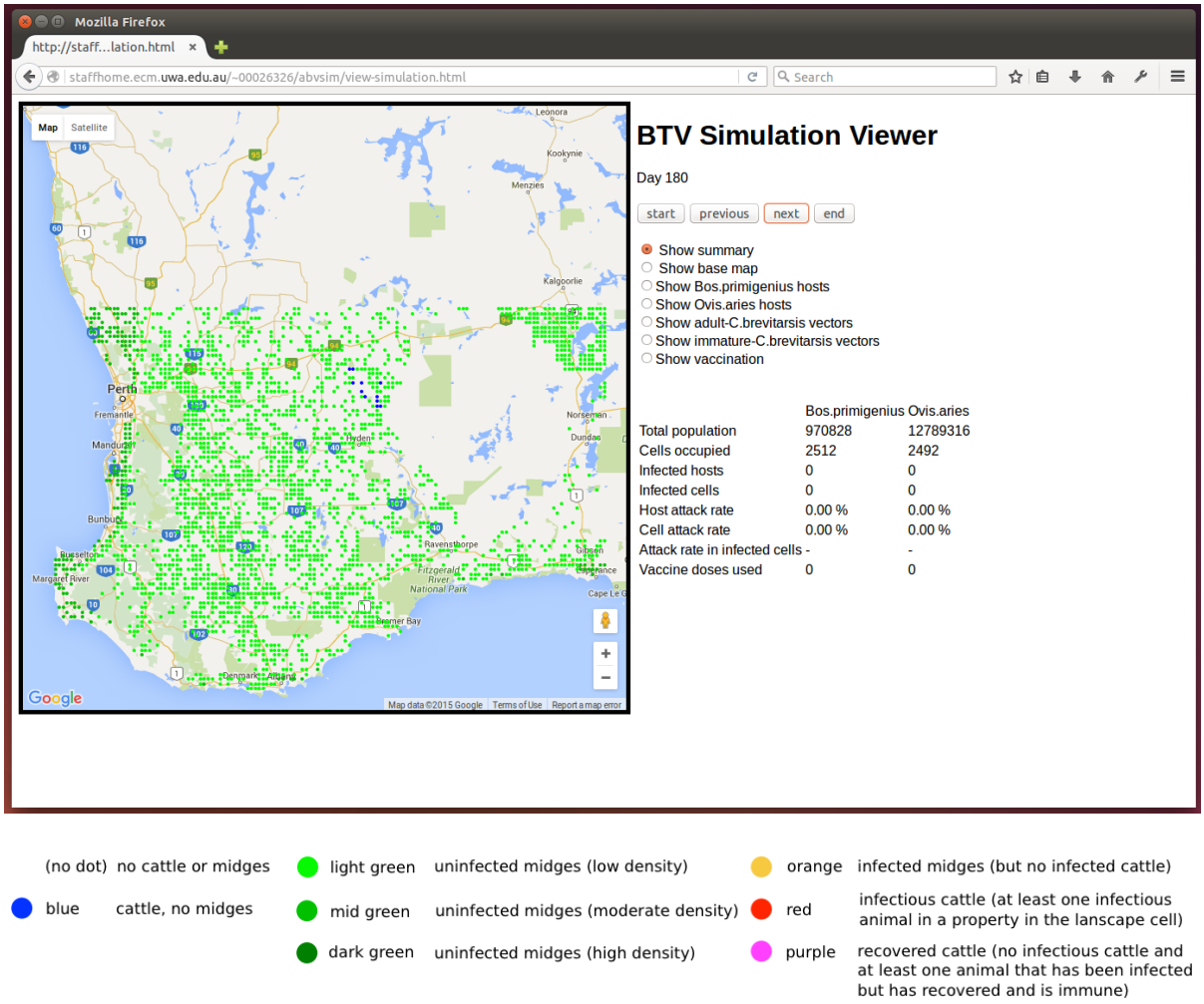


Fig. 7

Fig. 7 is a screenshot of the BTV spread simulation launcher web page. Shown in the right part of the figure is the simulation setup control panel, which allows the simulation scenario to be edited. On the left is the simulation area selected.



(no dot) no cattle or midges
blue cattle, no midges
light green uninfected midges (low density)
mid green uninfected midges (moderate density)
dark green uninfected midges (high density)
orange infected midges (but no infected cattle)
red infectious cattle (at least one infectious animal in a property in the lanscape cell)
purple recovered cattle (no infectious cattle and at least one animal that has been infected but has recovered and is immune)

Fig. 8

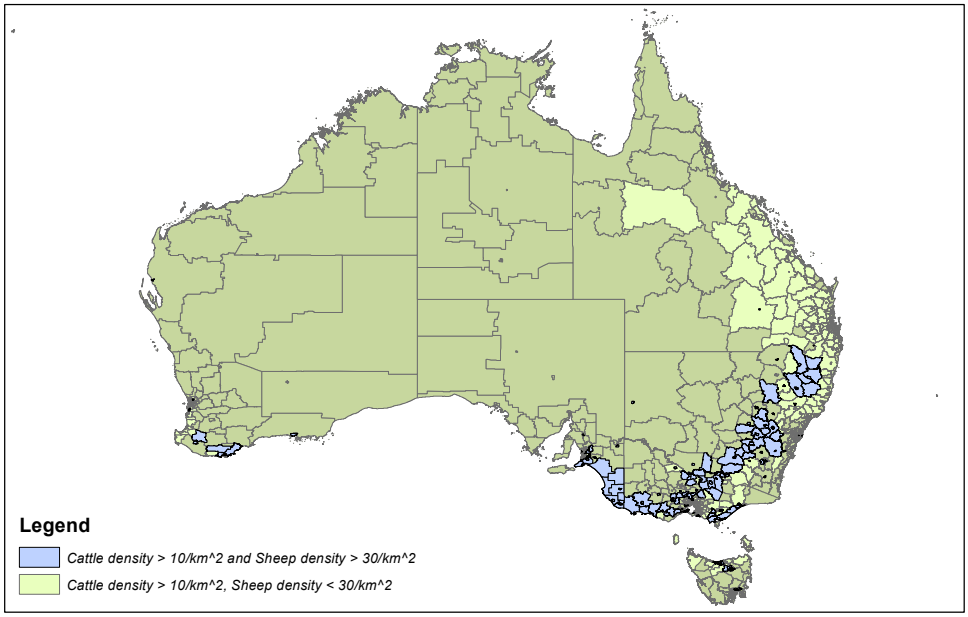
Fig. 8 shows the simulation results viewing interface of the simulator. On the left panel a map of the simulation area is displayed. Each coloured dot overlayed on the base map display represents the state of one simulation landscape cell. In this simulation each cell is approximately 5km by 5 km. The right panel shows information about the simulation, including statistics on the final infection outcome; the right panel also contains controls for stepping through the simulation snapshot sequence and changing the view colouring mode.

4 Results

4.1 Landscape cells and host density mapping

Fig. 9 shows an Australia-wide map of cattle and sheep density derived from ABS agricultural census data. This map highlights areas in which cattle and sheep co-occur. These areas are of special importance for potential BTV spread via *C.brevitarsis*, since brevitarsis requires cattle dung as breeding habitat, and Bluetongue has a high mortality rate in sheep.

Cattle and Sheep Co-Location - Australia



Source: ABS Agricultural Commodities data set (7121.0) 2010-2011

Fig. 9

We constructed landscape cell data sets for three specific areas: North-Eastern NSW, Eastern Victoria, and South-West WA. Cattle and sheep density maps for these areas are shown in Figs. 10, 11 and 12.

In these maps, density of livestock is shown in three levels of blue shading, with lightest to darkest ranging from 1-100 animals, 100-1000 animals, and over 1000 animals respectively. Cattle are shown in the left panels and sheep are shown on the right

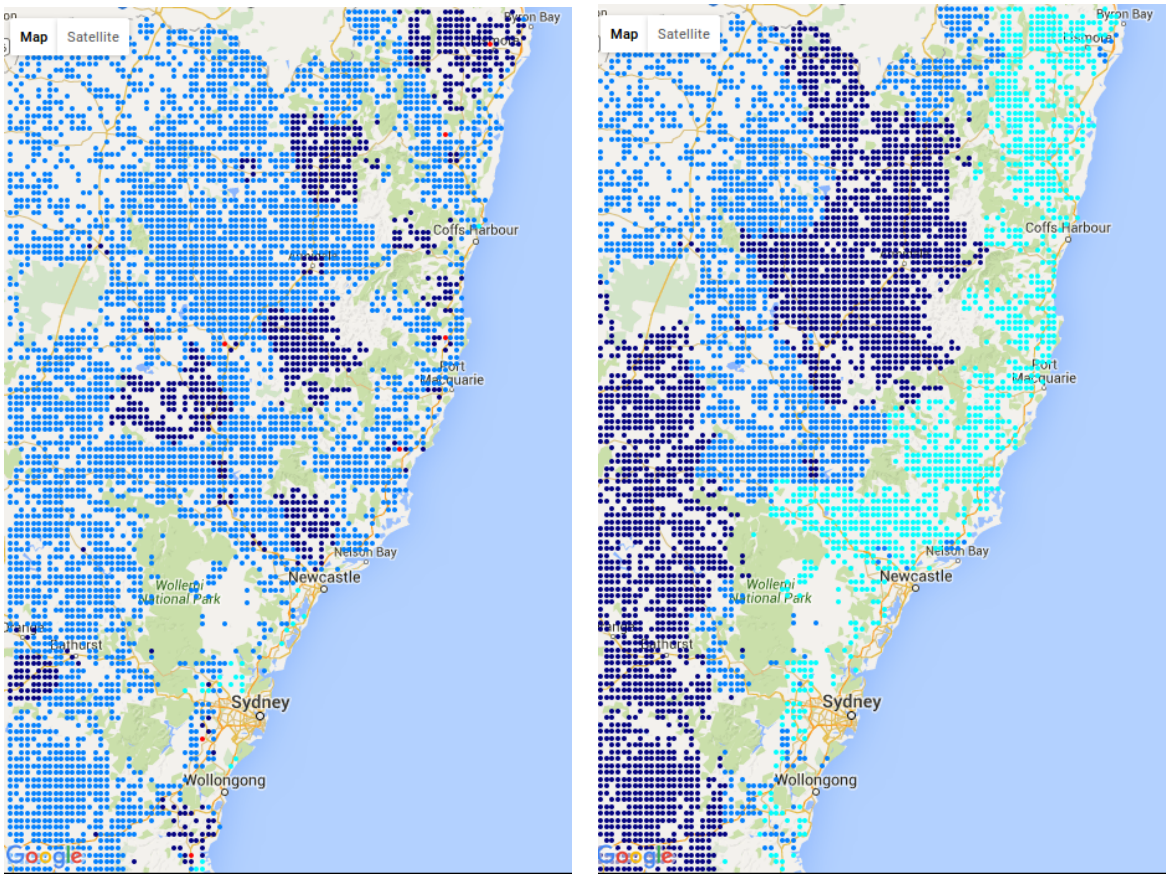


Fig. 10 Livestock density in New South Wales

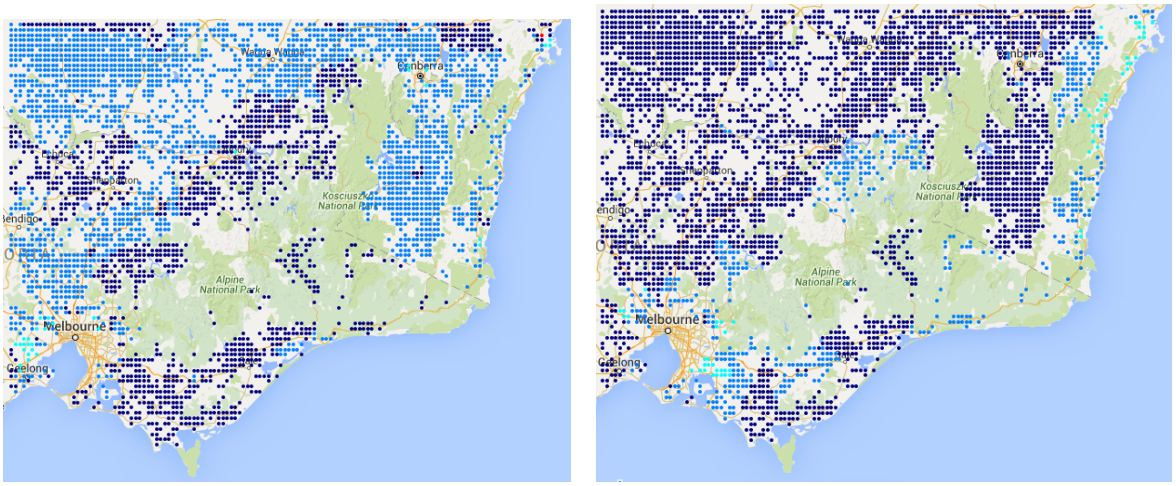


Fig. 11 Livestock density in Victoria

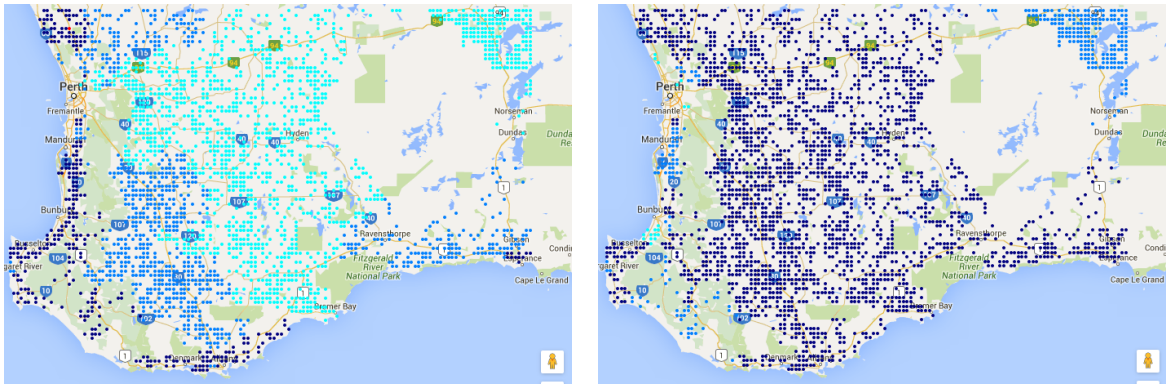


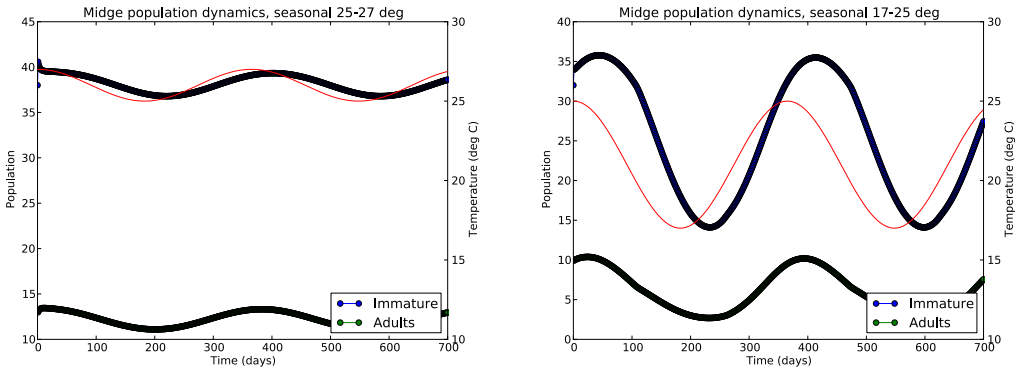
Fig. 12 Livestock density in South-West WA

4.2 Climate dependant *Culicoides brevitarsis* population dynamics

The vector population model described above is capable of representing *Culicoides* population dynamics of at least three distinct climatically driven patterns, each of which has different consequences for BTV incursion and transmission. Simulations of these climatic scenarios are presented below. Synthetic annual temperature series, based on the highest and lowest mean monthly temperatures at various locations on Australian's eastern coast, were used to demonstrate that the population dynamics model was capable of exhibiting different observed *C.brevitarsis* population regimes.

4.2.1 *Brevitarsis* endemic areas

In these areas a *Culicoides* population density sufficient for BTV transmission is continually maintained. The population remains in the high-temperature regime, although the population may seasonally fluctuate as the activity and breeding rate varies with mean daily temperature. (Klingseisen et al., 2011, Ward et al., 1995). This population dynamics scenario is illustrated in Figs. 13 and 14, which show simulation output of population time series for areas where the mean temperature varies seasonally from 25-27° C and 17-25° C respectively.



Figs. 13 (left) and 14 (right)

4.2.2 Areas in which the *Brevitarsis* population undergoes large fluctuations but does not become extinct.

In these areas the population is in the high-temperature regime in spring, summer and autumn but falls into the low-temperature regime for a period during winter. There may be times of the year in which the adult *Culicoides* population becomes very low (and may incapable of transmitting BTV, although this is uncertain), but the *Culicoides* population recovers each year without external introduction when the temperature rises and surving immature *Culicoides* emerge and re-start the breeding cycle (Bishop et al., 1996). This scenario is illustrated in Fig. 15, which shows simulation output for an area where the mean temperature seasonally varies from 13-21° C.

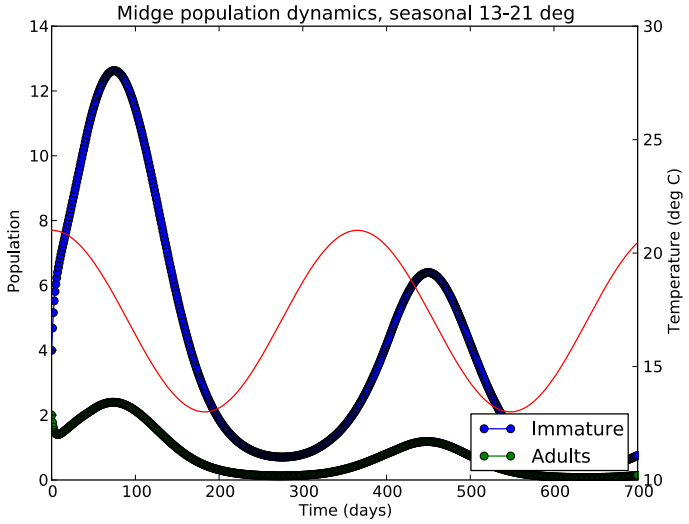


Fig. 15

4.2.3 Areas in which *Brevitarsis* can only survive seasonally.

In these areas incursions may result in a population becoming established with a high-temperature regime in summer, but in winter the population reverts to the low-temperature regime for such a period that both the adult and immature populations become extinct (Bishop et al., 1996). This is illustrated in Fig. 16, which has conditions one degree cooler than Fig. 15.

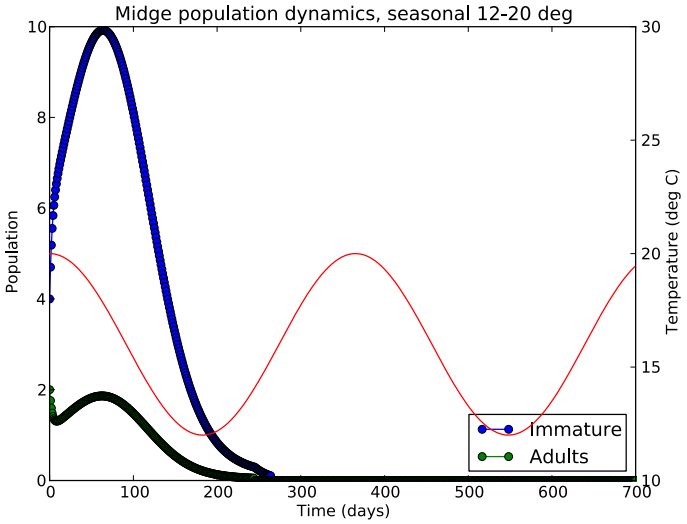


Fig. 16

To validate the population dynmics model, we examined three locations representative of these regions and ran the population dynamics model with actual annual temperature series (rather than the synthetic sinusoidal series shown above).

Endemic populations where adults are present year-round can occur near the northern NSW state border, for example Byron Bay (latitude 28.64° south). Fig. 17 shows the daily mean temperature and simulated population curves over one year (2003).

South of these areas are other areas in which the adult population disappears (i.e. falls below levels where it is detectable by a trapping program) during winter but where larvae survive and quickly re-establish adult populations once the temperature increases. Fig. 18 shows temperature and population curves for Taree (latitude 31.8896° south) which is approximately 384 km south of Byron Bay.

Further south still are areas in which imported populations can survive during summer and autumn but where longer winter cold periods render both the adult and immature population extinct. Fig. 19 shows temperature and population curves at Nowra (latitude 32.03° south) which is approximately 390 km south of Taree. In this simulation a population is assumed to be present at the beginning of the year, due to movement from further north. The population grows in summer, declines in autumn, and the adult population becomes zero during winter. Somewhat later, the immature population (not shown) also becomes zero.

High altitude (Great Dividing Range) and far southern regions are such that any introduced midge population fails to establish a breeding population due to low temperatures.

By using actual daily temperature data, we verified that the population dynamics model is consistent with the *C. brevitarsis* population regimes in the expected locations (Bishop et al., 1995a, Bishop et al., 1995b, Bishop and McKenzie, 1994, Murray, 1991, Murray and Nix, 1987).

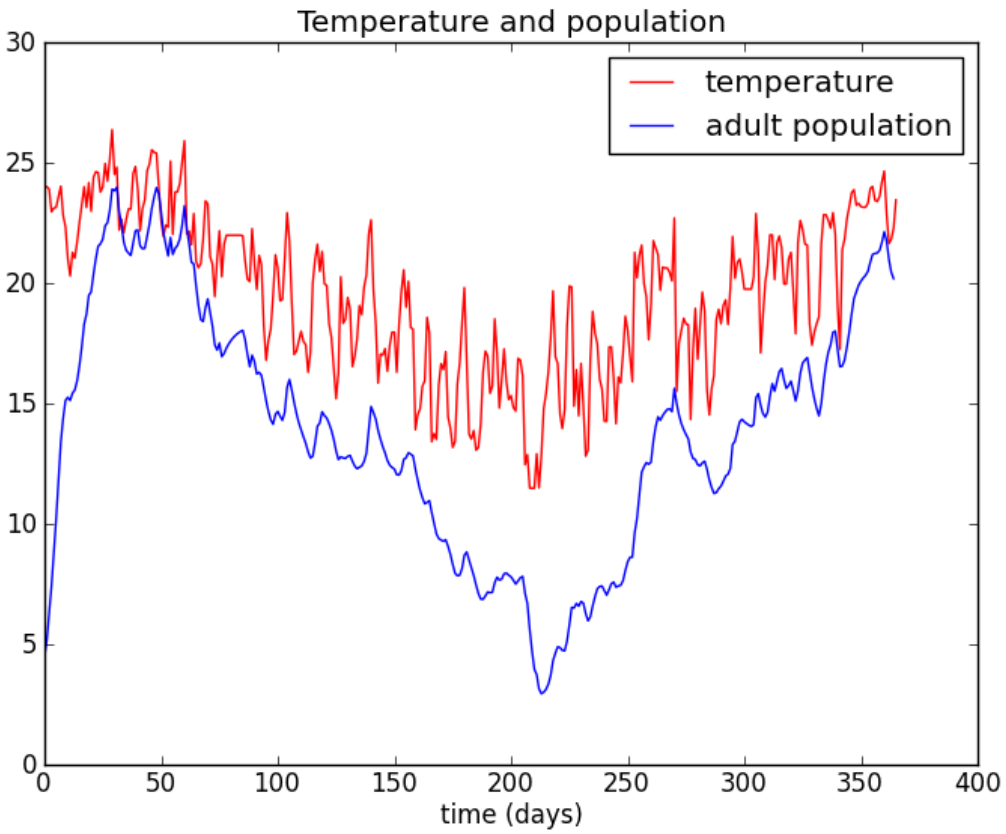


Fig. 17 Temperature and simulated midge population at Byron Bay

This simulation shows that temperature and breeding activity are at a maximum in January and February, explicitly reflecting known temperature dependant population dynamics. The midge population grows during this period and peaks at the end of February. The population then slowly declines with declining temperatures, as the rate of newly emerging midges does not keep pace with midge death. Temperature and breeding activity reaches a minimum in July and begins to increase at the end of July. By the end of August the population begins to increase as the larvae from the increased breeding activity begin to emerge.

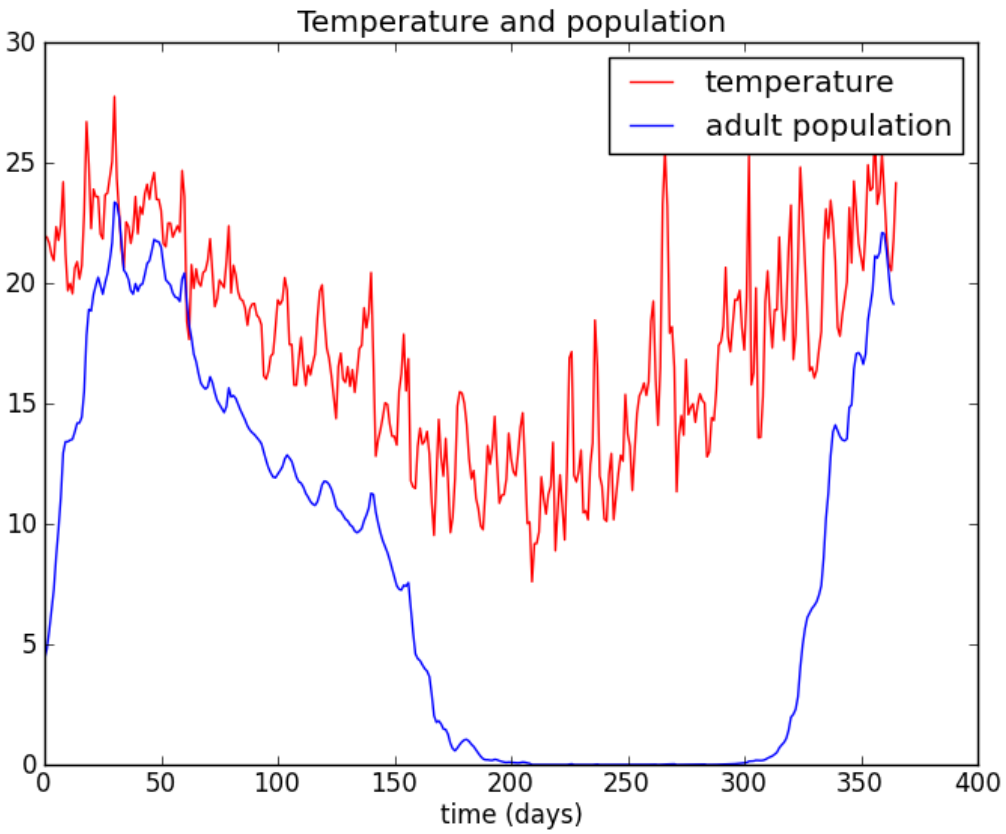


Fig. 18 Temperature and simulated midge population at Taree

The population curve here during summer and autumn is similar to that described in Fig. 17 above, except that the population fluctuation is larger due to the greater seasonal temperature variation. At the coldest part of winter the adult population falls to zero: all the adults die due to low temperatures, and the low temperature (below 13° C) also mean that no larvae emerge. Once the temperature increases however, surviving larvae emerge and establish a breeding cycle once more.

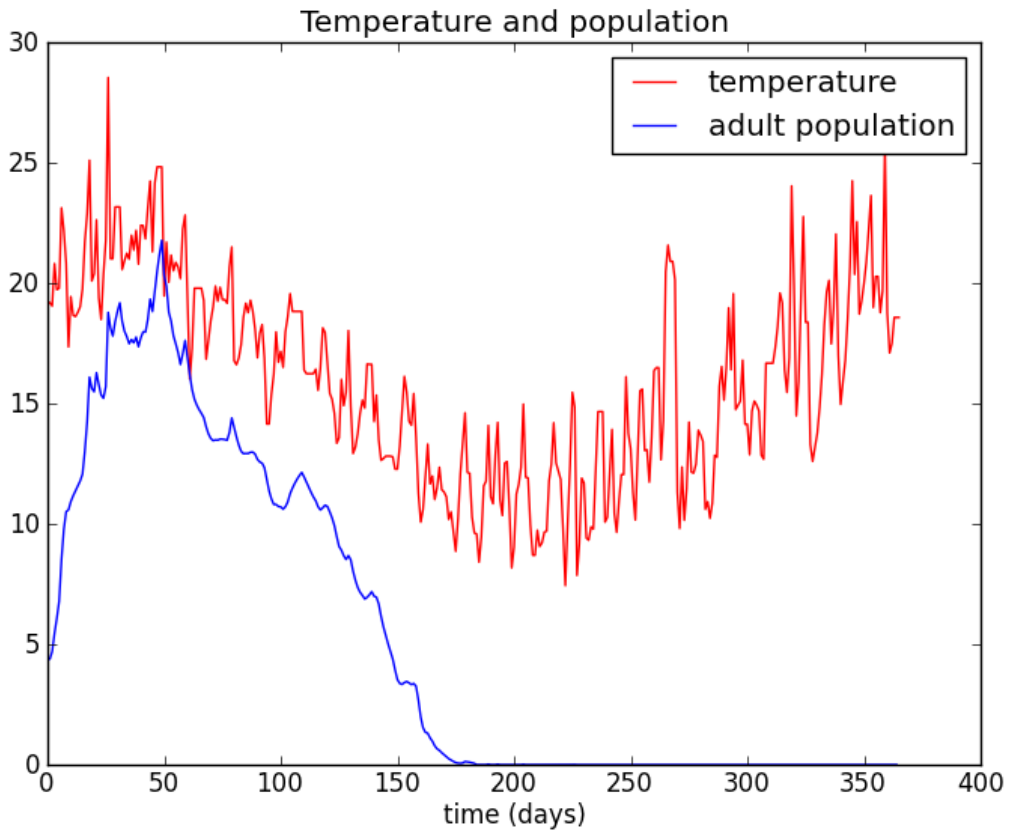


Fig. 19 Temperature and simulated midge population at Nowra

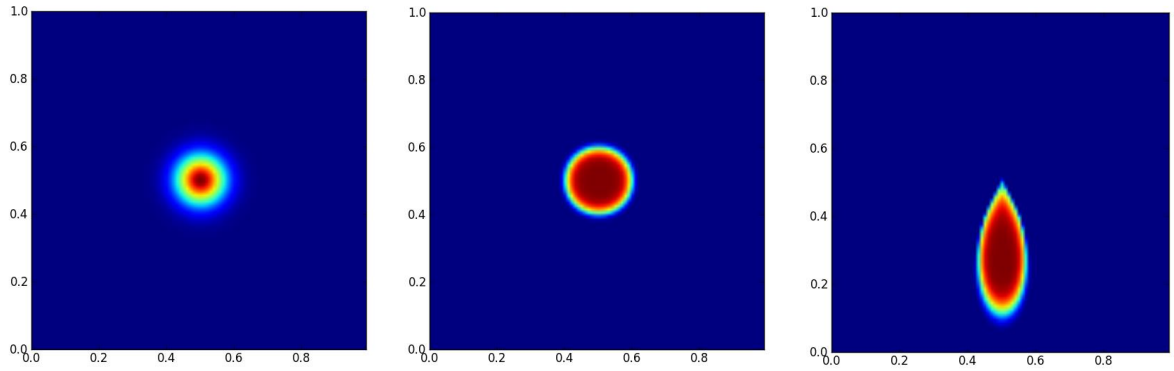
The population curve for Nowra is similar to that in Fig. 18 above, however the longer cold winter period below 13 degrees results in the immature population becoming extinct over winter. Without further importation of midges, the population therefore does not recover in spring.

We note that midge populations reported in the literature from trapping programs (Murray, 1991, Murray, 1987) are considerably more “noisy” than the population curves appearing above, possibly due to the fine-grained effect of rainfall and humidity variability on *Culicoides* survival and reproduction. This could be added to the model if necessary.

4.3 Combined *Culicoides* population dynamics and dispersal

Before using the combined *Culicoides* population dynamcis and dispersal models on real-world geographic and climate data, we first ran the combined model in several idealised test settings.

Fig. 20 below further illustrates the combined effect of the population dynamics model and the dispersal models. In Fig. 20, a grid of 100x100 cells is used with an initial population occupying a single cell in the centre.



On the left an initial point population diffuses over time, but without any population growth dynamics (the population is constant and just spreads over a greater area). In the centre, the same diffusion process operates, but population dynamics are also in effect. Notice that the central population is maintained at a high level, and that the population has diffused further, since the higher central population maintains a high the rate of diffusion. On the right, the same point population undergoes population dynamics and wind-driven dispersal by a constant northerly wind. The result is similar to combined diffusion and population dynamics, but the directional nature of wind dispersal has created a teardrop-shape oriented downwind.

Fig. 20 Combined population dynamics and dispersal.

4.3.1 NSW seasonal incursion simulations

Midge trapping research programs have documented the spread of *C. brevitarsis* in the Hunter Valley area e.g. (Bishop et al., 1995b). Typically, midge populations are detected in the Manning Valley area in November, having spread from the endemic areas to the north. In the following months, midges are successively detected at increasing distances from the Manning Valley.

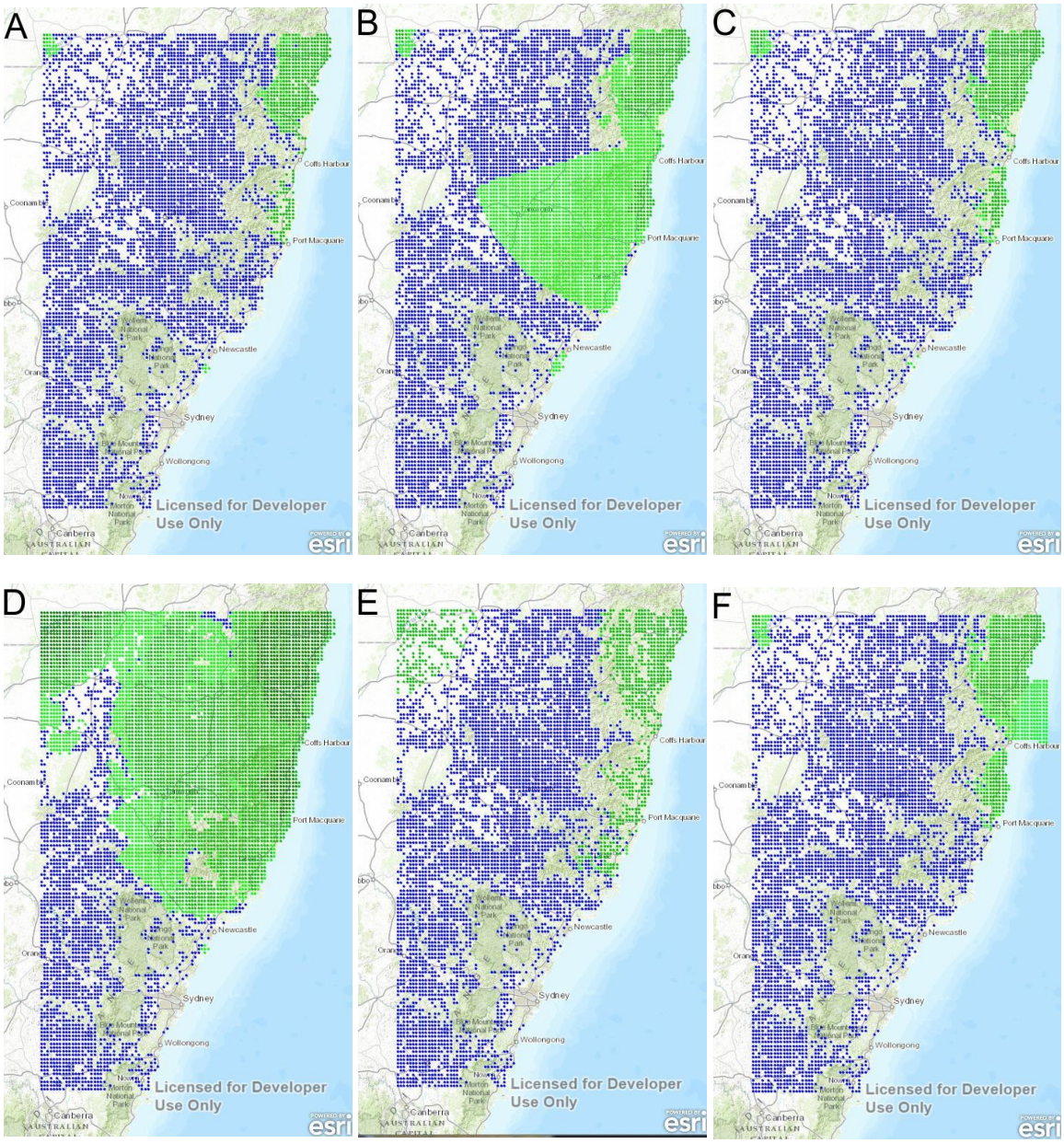


Fig. 21

Fig. 21 shows a simulation of annual *C.brevitarsis* population dynamics and wind-borne dispersal, NSW, starting October 1 1991. Blue indicates the presence of cattle (and thus habitat for *C.brevitarsis*) and shades of green indicate adult midge population density from lowest (light green) to highest (dark green). The top left frame (A) shows the midge population at the start of the simulation on the 1st of October. This time after winter represents the most restricted extent of *brevitarsis* in NSW. Midges are present on the north-east coast, and in an area north of the Hastings valley. The top middle frame (B) is one week later: a wind transport event (visible as the large green wedge shape) has spread a low density of midges as far south as the Manning valley and as far west as Tamworth. However, one week later in the top right frame (C) it can be seen that all these areas are now midge-free. This is for two reasons. Firstly, some of these areas (including national parks) do not have a cattle population, and thus *C.brevitarsis*, which requires cattle dung as

breeding habitat, cannot colonise these areas. In the simulation, midges can enter these areas and survive for a time (possibly to be blown to other locations), but will eventually die without establishing a breeding cycle. Secondly, even in locations with a cattle population, at this time in mid-October temperatures are too low for midge populations to survive the low inland and highland minimum temperatures.

The bottom left frame (D) is 6 months after the start of the simulation, when the midge population is near its greatest extent. Midge populations have established as far south as the Hunter valley, and in some inland areas. These populations have become established following wind transport events in the preceeding months (not shown) when midges from the endmic areas have been blown southwards and westwards. Unlike the wind transport event shown in frame (C), these events occurred in the summer months when the temperature upon arrival was suitable for breeding, allowing dense populations to become established. The bottom middle frame (E) is 9 months after the start of the simulation, in winter 1992. Midge populations have retreated from inland areas that are now too cold to support the *brevitarsis* breeding cycle. The bottom right frame (F) is one year after the simulation start, and the midge population has retreated and is now very similar to that at the simulation start.

The pattern of spread varies from year to year; Fig. 22 (reproduced from (Bishop et al., 1995b)) shows this spread during 1992/1993. Using this data set of estimated midge arrival times, we ran an extensive series of simulation experiments to validate the combined midge population dynamics and dispersal model.

This validation process is documented in a publication in PLoS ONE (Kelso and Milne, 2014). We briefly summarise the outcome of the validation process here. Fig. 23 shows simulated arrival time contours (in a similar form as the observed data in Fig. 22); Table 1 and Fig. 24 present monthly simulated arrival times together with the corresponding averaged observed arrival times from the years 1990-1993, thus comparing simulated and actual arrival times for all locations where arrival of midges was recorded. Arrival times are denoted in months, with November = 1, December = 2, etc.

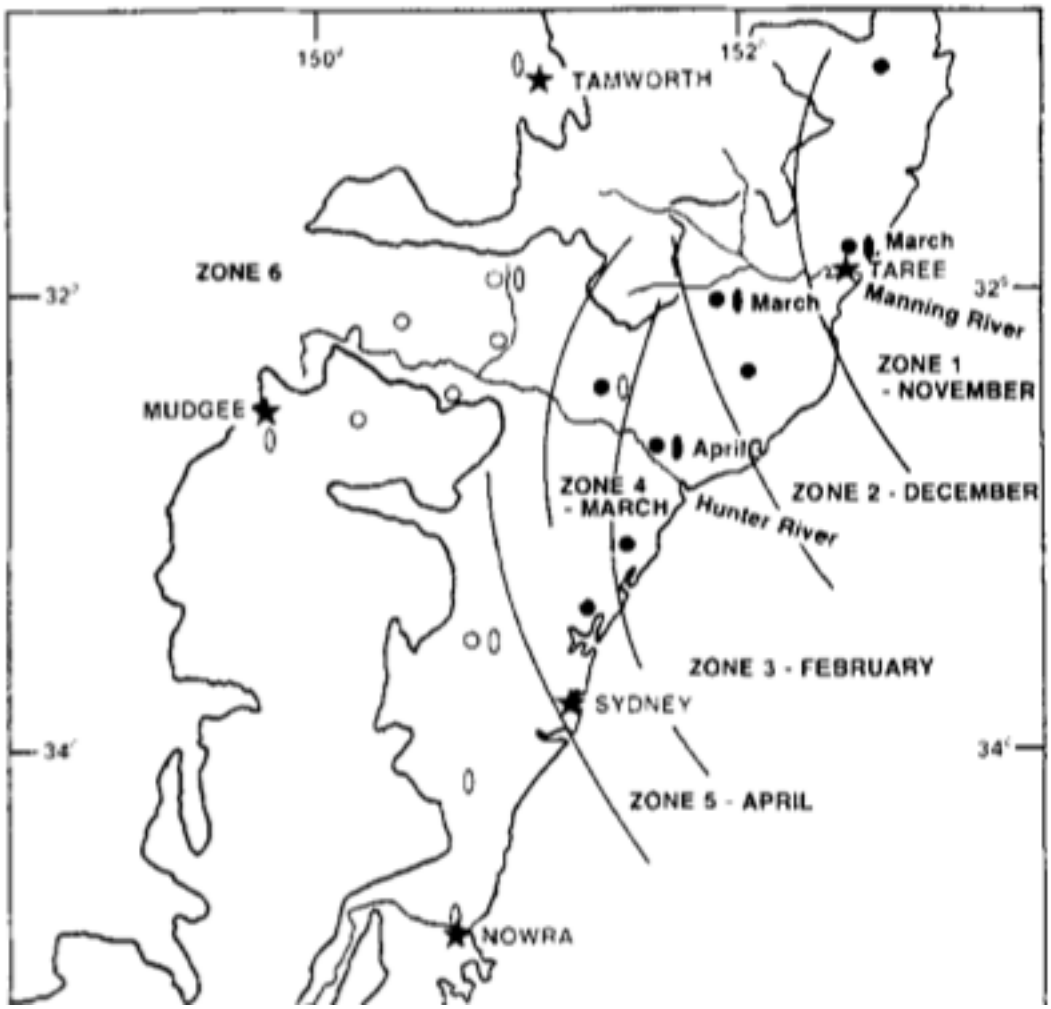

150°
152°
TAMWORTH
March
TAREE
32°
March
Manning River
ZONE 6
ZONE 1
- NOVEMBER
MUDGEE
April
ZONE 4
- MARCH
ZONE 2 - DECEMBER
Hunter River
ZONE 3 - FEBRUARY
SYDNEY
34°
ZONE 5 - APRIL
NOWRA

Fig. 22

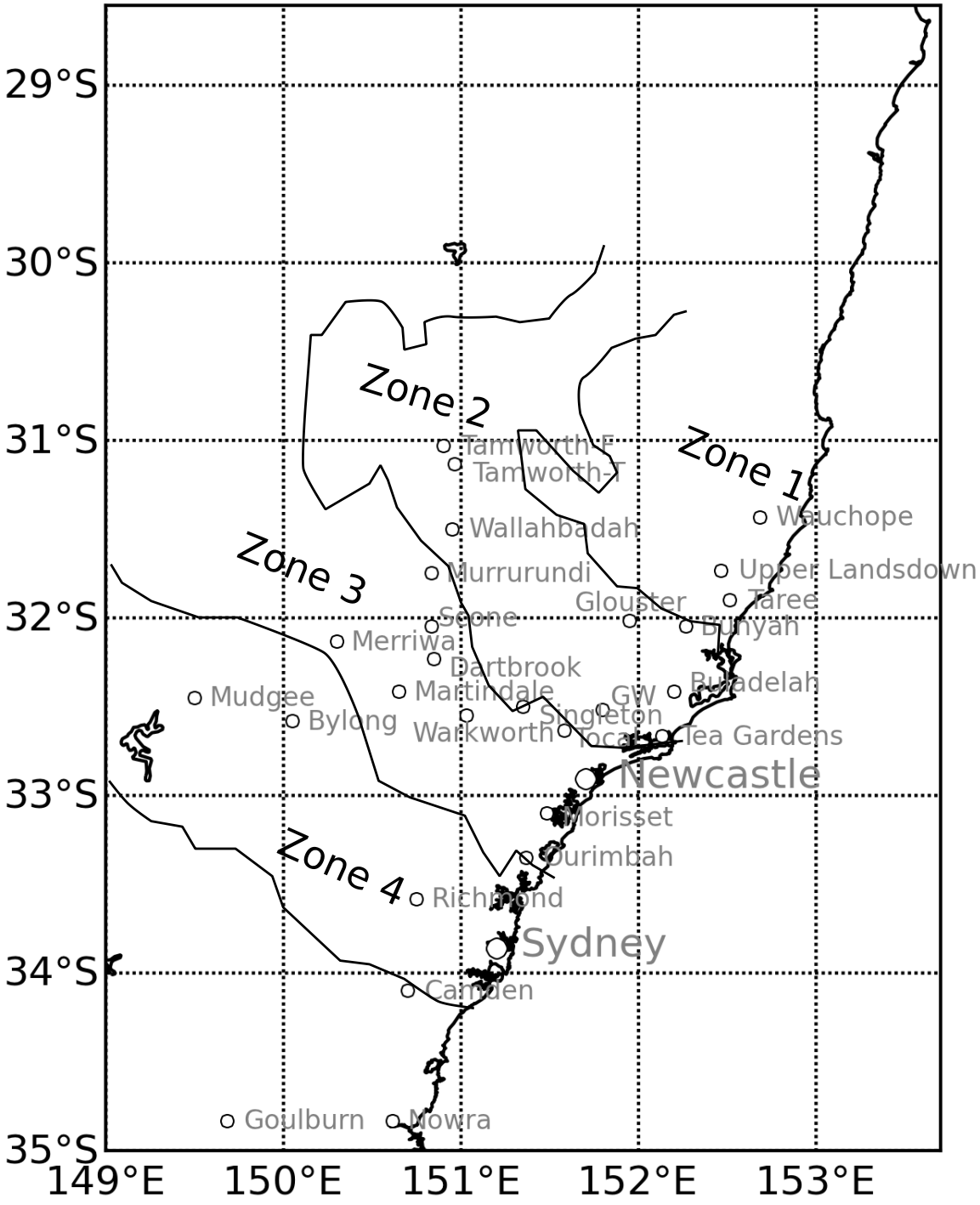
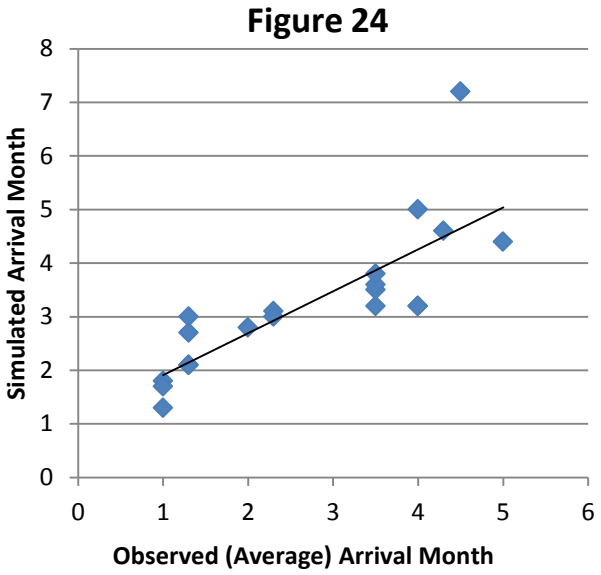


Fig. 23 Simulated first months of midge arrival with Zone 1 in November, Zone 2 in December etc.

Table 1 Simulated versus observed midge arrival times

Site	Arrival Month	
	Observed (Average)	Simulated
Buladelah	1.3	2.7
Bunyah	1.3	2.1
Bylong	5	4.4
Camden	4	5.0
Dartbrook	4	3.2
Glenwilliam	2	2.8
Glouster	1.3	2.1
Martindale	3.5	3.5
Morisset	3.5	3.6
Nowra	4.5	7.2
Ourimbah	3.5	3.8
Richmond	4.3	4.6
Scone	4	3.2
Singleton	2.3	3.1
Taree	1	1.8
Tea Gardens	1.3	3.0
Tocal	2.3	3.0
Upper Landsdown	1	1.7
Warkworth	3.5	3.2
Wauchope	1	1.3
Correlation coefficient:	**0.81**	

Figure 24



At stations for which arrival year could not be unambiguously determined (namely Ourimbah in 1993) the average of the candidate arrival months was used. Only stations that recorded both simulated and at least two years of observed arrival were included (this excluded Goulburn, Merriwa, Mudgee, Murrurundi, Tamworth and Wallahbadah).

4.4 BTV Transmission

4.4.1 Transmission model qualitative validation (single location)

The behaviour of the combined midge population dynamics and BTV transmission models was first qualitatively validated with a series of simulation experiments representing the transmission of BTV in a single herd (i.e. in a single spatial location). Except where stated, the experiments used the scenario settings shown in Table A3 in the Appendix.

BTV spread in an established midge population. In the first experiments, the midge population (adult and immature) was initialised to equilibrium values for the host population size for the particular experiment temperature. BTV was then introduced via either a single infected host or a single infected midge, for temperatures 28^{o}C and 20 ^{o}C. Unless stated

otherwise, parameters for these experiments are as given in the Appendix in Table A5. Results are shown in Table 2.

It was found that at the higher temperature, a Bluetongue outbreak instigated by an infected midge was more likely. This is because at higher temperature the midge activity (biting rate) was higher and the chance that the infected midge would successfully infect a cow before dying was higher. In the case where BTV was introduced by an infected cow, an outbreak occurred in 10 out of 10 simulations. Although the chance of midge infection per bite was very low (1%), the 20-day host infectious period and the large number of biting midges inevitably resulted in multiple infected midges and an outbreak.

Table 2 Transmission model experiment results

Scenario	Probability of outbreak (n=10) Introduction by infected midge / cow	Day of full host infection	Day of full host recovery
		Mean (s.d.) where outbreak occurred	
Established midge population			
28°C	80% / 100%	22.5 (5.3)	72.1 (5.1)
20°C	60% / 100%	46.1 (1.9)	96.3 (7.4)
No initial midge population (incursive infective midge)			
28°C	50%	35.4 (0.5)	84.8 (4.6)
20°C	0%*	-	-

* in 2/10 simulations, a single cow was infected

Higher temperatures also resulted in a more rapid outbreak, due again to greater adult midge activity, but also due to shorter EIP (extrinsic incubation period) and faster immature midge maturation. Once started, outbreaks at a particular temperature were similar whether they originated from an infected vector or an infected host. Figs. 25 and 26 show the infection state dynamics of typical 28°C and 20 °C outbreaks, respectively. Note that although the infected midge population peaks at over 8000 (800 per cow), this is a small fraction of the total adult midge population.

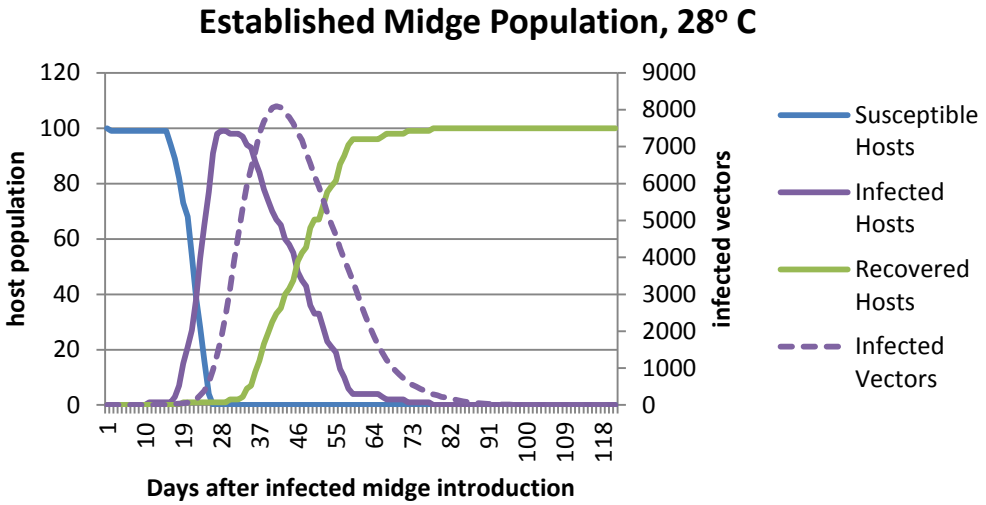


Fig. 25

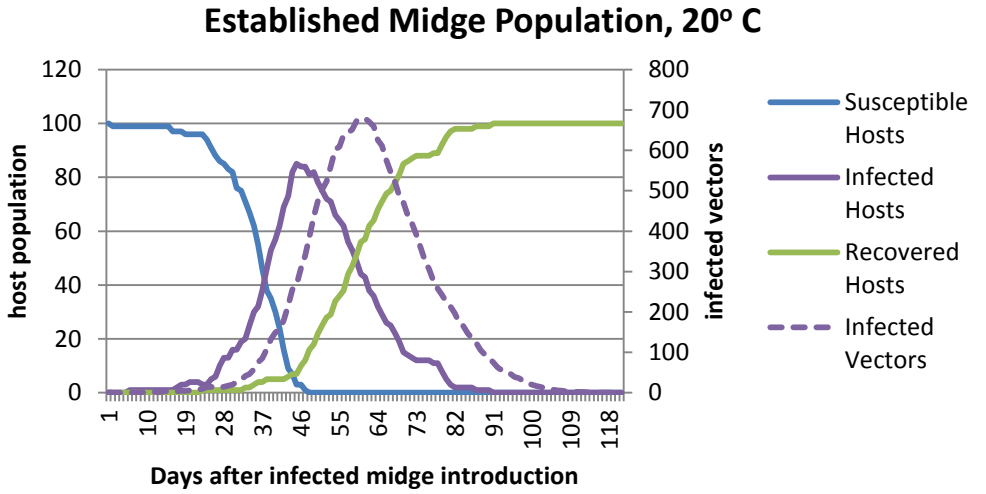


Fig. 26

BTV spread from an incursive infected midge. In the second experiments, the midge population (adult and immature) was initialised to zero, and a single infected midge was introduced, for temperatures 28°C and 20°C. Results are shown in the lower part of Table 2.

The most notable feature of the incursive infected midge scenario is that outbreaks are less likely: at 28°C a single infected midge caused an outbreak 50% of the time (compared to 80% for the established midge population), and at 20°C no outbreaks occurred, although in 2 out of 10 simulations a single cow was infected. The infection state dynamics of a typical 28°C incursive infected midge outbreak is show in Fig. 27. These outbreaks are similar to the established population outbreaks, except they are effectively delayed, as very rapid epidemic growth does not occur until a sufficiently large midge population becomes established.

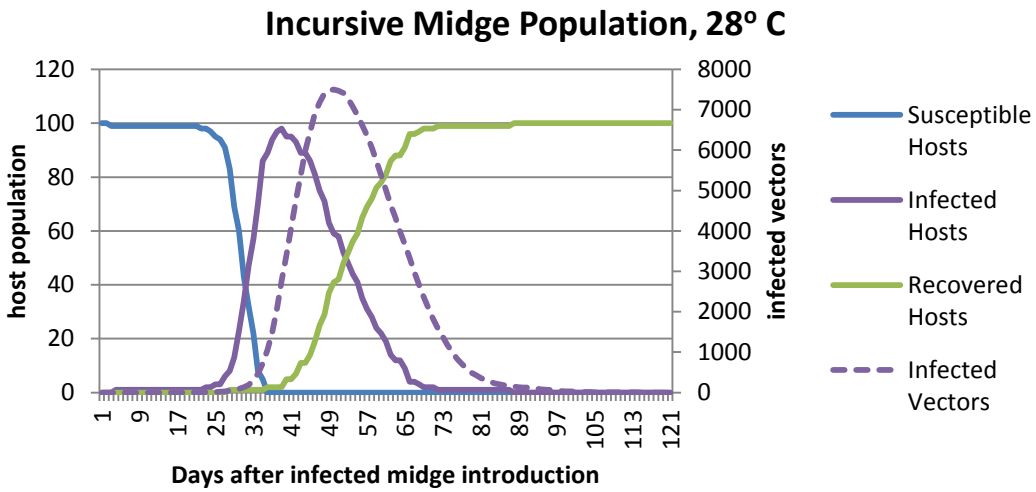


Fig. 27

Although additional quantitative validation will be conducted, the results of this initial experiment series are *prima facie* plausible. In particular, the ability of a single introduced midge to infect a cow without causing a Bluetongue outbreak may model the observations of Bluetongue seroconversions in sentinel cattle herds in NSW without any corresponding Bluetongue outbreak (reported for example in (Bishop, 1996)).

4.4.2 BTV transmission, vector population dynamics and vector dispersal (full model)

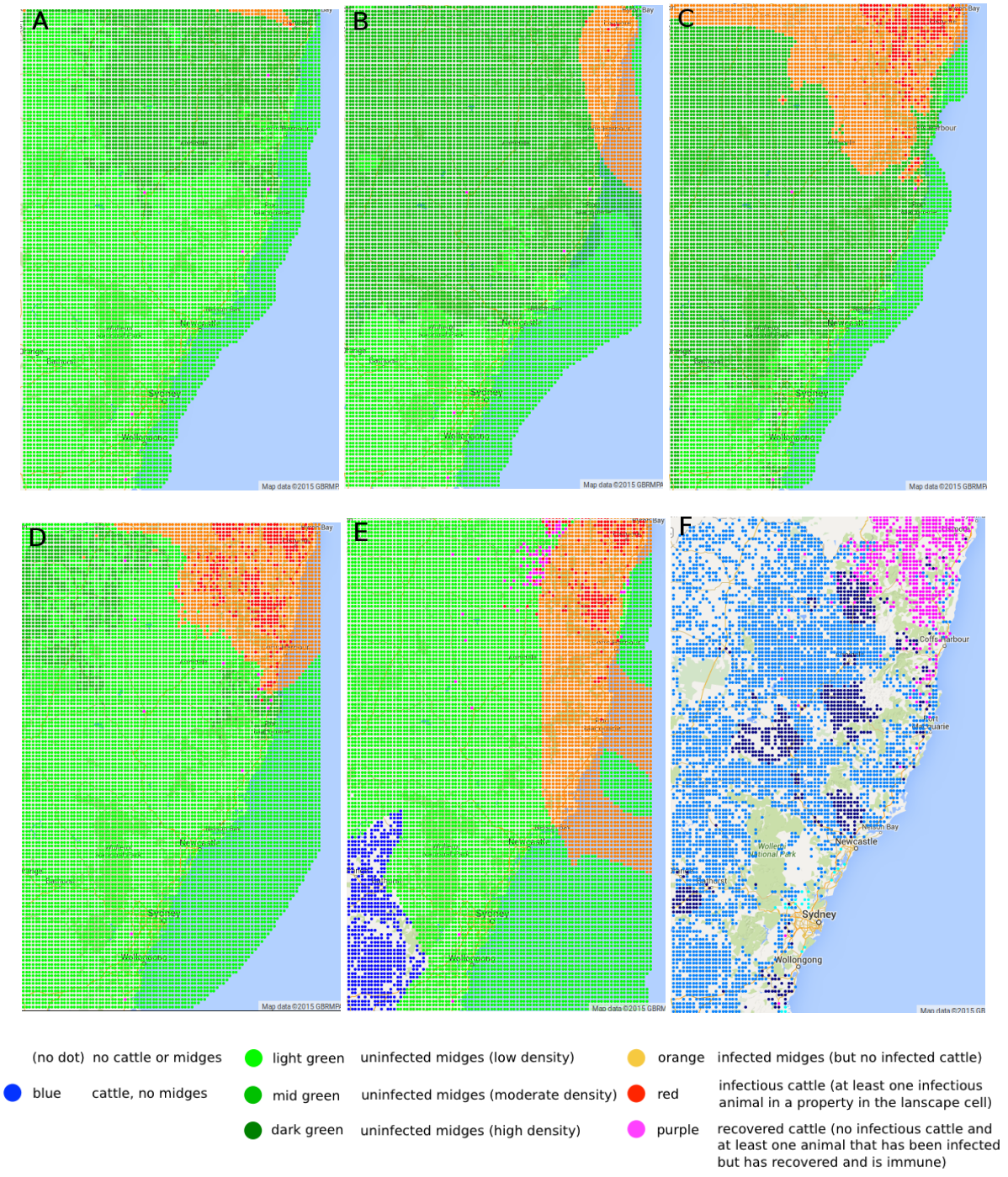


Fig. 28

Fig. 28 shows an example of a simulation where all components of the simulation model work together to generate a hypothetical Bluetongue outbreak in Northern NSW. In this scenario, one infected animal was introduced in each of 7 locations (Lismore, Grafton, Kempsey, Taree, Tamworth, Camden and Nowra – all locations having livestock saleyards). This simulation begins on the 1st of January in 1999. Experiments showed that infected cattle introduced earlier were much less likely to trigger Bluetongue outbreaks; and that

introductions through to March were even more likely to produce outbreaks due to higher temperatures and midge densities. Crucially, in this scenario, the *C.brevitarsis* vector competence parameter has been increased so that midges are 10% likely to become infected from a bite on an infected host. This value is much higher than any estimates for *C.brevitarsis*, which is one reason why Bluetongue outbreaks of the size simulated here do not occur in NSW.

The top left frame (A) is 30 days after simulation start. Infected midge populations have been generated and have are being blown to the north-west from Lismore (seen as the yellow trail of cells, indicating the presence of infected midges). No additional infections in cattle or sheep in other cells have been generated at this point.

The top middle frame (B), which shows the outbreak 60 days after the start of the simulation shows that midges have carried BTV to other cells around Lismore (shown as red dots indicating the presence of infected cattle) . Note that these cells are not adjacent to any of the introduction cells, indicating that BTV is being spread by wind-borne midges, rather than short range movement, which would leave a sequence of adjacent infected cells.

After 90 days (top right frame C) wind-borne and local movement of infected midges has created a large outbreak in the area near Lismore and Grafton. The large yellow area indicates that infected midges are now being generated by multiple infected source cells – several isolated red cells can be seen surrounded by a halo of yellow infected midges.

After 120 days (bottom left frame D) the outbreak has expanded further, although by this time (the beginning of May) temperatures and midge populations have begun to decline, which can be seen by the contraction of the darker green areas indicating high midge density..

After 6 months (bottom centre frame E) the outbreak has stop spreading. There are still cells with active host infections (show in red) but the purple cells indicate that there are cells in which transmission has ceased, leaving a record of infected (but no longer infectious) cattle. A clear wind transport event can be seen carrying infected midges as far as Forster; however with low temperatures and lack of midges these midges do not create any new centres of BTV transmission. It can be seen that there are several cells inland in the northern tablelands area, but that these did not lead to any onward transmission. The scenario of isolated cattle infections that do not lead to any further local spread is consistent with data recorded by the National Arbovirus Monitoring Program (NAMP), which occasionally finds that single animals in sentinel herds in various locations in NSW have been exposed to BTV (i.e. have seroconverted for BTV). Our simulation results suggest that this exposure may occur by wind-borne midges from distant locations, and that outbreaks do not occur because local *brevitarsis* populations are not sufficiently dense and/or active enough to support transmission. A feature of note (also seen in frame E) is a wind transport event which has blown a cloud of infected midges out to sea (where they will perish and play no further part in the simulation).

The bottom right frame F shows the state of the cattle population after the outbreak has ceased in September. The spatial extent of the outbreak is shown by the presence of purple cells.

We would like to reiterate that this outbreak scenario was only achieved by increasing the vector competence of *C.brevitarsis* beyond the level supported by any estimate appearing in the literature.

5 Discussion

Our findings support the current understanding that the BTV free areas in Australia, both cattle, and mixed cattle and sheep areas, are protected by the lack of competent *Culicoides* species capable of permanently inhabiting temperate regions. This is due to (a) the low vector competence of *C.brevitarsis* to all BTV serotypes currently active in Australia, and (b) seasonal distribution of *brevitarsis* in temperate regions.

We have qualitatively reproduced BTV and *Culicoides* activity in southern Australia. The most important unanswered question following from this study is: can these results be made quantitative, given the available data ? The potential exists to use the simulation model to run large numbers of outbreak scenarios with different values for uncertain parameters, such as the timing and location of the introduction of BTV, the vector competence levels and absolute midge populations. These simulations could be used to develop maps of areas at risk of Bluetongue, as well as the potential effectiveness of interventions. Although several rich data sources on Australian *Culicoides* and BTV activity exist, including the NAMP/Animal Health Australia programme, it is not known whether these could provide the necessary constraints on unknown parameter values.

The plan of staged development of a complex, interacting simulation system worked very well. One difficultly identified during the project was that the initial temperature data set and wind dispersal model were limited; this required further refinement, using BOM gridded spatio-temporal temperature data and the HYSPLIT dispersal model. The initial plan did not require use of a highly accurate atmospheric dispersal model for wind-borne transport; however once this was incorporated into the system (in the form of HYSPLIT) the value of this became apparent in the improved quality of the wind-transport patterns. While this functionality turned out to be superior to what was envisioned, the effort required did come at the cost of reduced time for model validation activity.

To enable future access to the UWA Bluetongue simulator environment, the software will be placed in the UWA repository along with an instruction manual. This will permit use of the system by BTV researchers and make it available to use during future BTV outbreaks.

5.1 Specific project objectives

Phase 1

1) Development of “synthetic world” model of BTV spread over the landscape, involving four component sub-models incorporating explicit animal (transportation) and insect vector (wind) movement.

2) Feedback on this initial model obtained from midge and virus reference group following presentation by UWA researchers.

3) Possible redesign of model components following feedback.

4) Validation of synthetic model by reference group complete.

2), 3) and 4) Feedback from the reference group emphasised the importance of being able to represent multiple host species, multiple vector species, and multiple BTV serotypes (although, not all at the same time). Based on this we redesigned the underlying data model to make it highly modular to ensure that particular initial assumptions (such as vector *C.brevitarsis* requiring cattle dung as breeding habitat) were not hard-coded into the structure.

Phase 2

1) Development of "Belgian" or other model completed.

2) Validation of this model using outbreak data from Belgian partners (or other alternative data if available) and reference group.

3) Jounal paper reporting modelling results submitted.

1), 2) and 3) On further investigation, the use of European BTV spread data sets that was initially intended would have been of limited use, given that the European Bluetongue outbreaks were qualitatively different from potential Australian outbreaks due to the existence of dense livestock and competent midge populations over extended areas. This contrasted sharply with southern Australian conditions where *C.brevitarsis* is the most prevalent, competent vector, and is only seasonally present. Understanding and correctly modelling *C.brevitarsis* population dynamics, movement and seasonal occurrence was thus of primary importance. We devoted considerable effort to this *Culicoides* sub-model and published a research paper specifically on the subject before moving on to the addition of BTV transmission to the model.

Phase 3

1) Development of Australian Region Scenarios completed.

2) Simulation of unmitigated outbreaks in these regional scenarios complete.

1) and 2) Once Australia-wide or global data sets had been sourced (from the BOM, ABS, ABARES, Animal Health Australia and NAMP, NOAA), we targetted the initial NSW simulation model to further areas of interest in WA and Victoria.

3) Analysis of these simulations by reference group completed.

Feedback received from Peter Walker and colleagues at CSIRO AAHL.

4) Experimentation with control interventions conducted for all regional scenarios.

See note on 1) and 2) above.

5) Completion of policy paper reporting on simulation results and impact of interventions, in collaboration with reference group.

6) Journal paper report on results completed.

7) Results disseminated to State and Commonwealth agriculture depertments and to producer groups as required. To include demonstration of the simulation technology to disease and biosecurity managers.

5), 6), 7) While we believe our simulation model qualitatively reproduces *C.brevitarsis* and BTV transmission in Australia, we have not analysed our simulation results in sufficient depth to draw well-supported quantitative conclusions. This work is ongoing and will culminate in the generation of at least one more research publication.

6 Conclusions/recommendations

There is no specific reason to suspect that the conditions limiting Bluetongue in Australia will not persist; however further reseach is warranted to assess the ongoing risk of (a) the incursion of a BTV serotype for which a southern *Culicoides* species is a competent vector; and (b) changes in distribution of *Culicoides* species, perhaps due to climate change.

Should further research indicate a substantial risk of BTV incursion, the simulation software tools developed in this project will enable planners to assess the likely area affected, magnitude and duration of a Bluetongue outbreak beginning in different locations, at different times of the year, and in different annual weather conditions. Furthermore, prospective vaccination intervention strategies can be assessed for these different possible outbreaks ahead of time to gauge their ability to mitigate the outbreak.

The need for ongoing collection of data on both midge vectors and circulating BTV strains is highly important and necessary to underpin detailed disease spread simulation models such as that developed at the University of Western Australia.

7 Key messages

This project raises the general awareness of producers to the risk of Bluetongue outbreaks: what areas might be affected, how fast a Bluetongue outbreak might unfold, and what opportunities for mitigation they may have should an outbreak occur (vaccination, animal movement bans). A version of the simulation software would serve as an awareness-raising or educational tool if further developed. The benefit would be a more informed industry-base that could participate in informed decision-making on mitigation planning, and better informed producers in the face of an actual outbreak (in terms of vaccine uptake and compliance with movement bans).

8 Bibliography

AKEY, D. H., POTTER, H. W., et al. 1978. Effects of rearing temperature and larval density on longevity, size, and fecundity in the biting gnat Culicoides variipennis. *Annals of the Entomological Society of America,* 71**,** 411-418.

ANIMAL HEALTH AUSTRALIA 2001. The history of bluetongue, Akabane and ephemeral fever viruses and their vectors in Australia 1975-1999. Animal Health Australia.

ATKINSON, K. E. 1978. *An introduction to numerical analysis,* New York, Wiley.

BAETZA, H.-J. 2014. Eradication of bluetongue disease in Germany by vaccination. *Veterinary immunology and immunopathology,* 158**,** 116-119.

BIRLEY, M. & BOORMAN, J. 1982. Estimating the survival and biting rates of haematophagous insects, with particular reference to the Culicoides obsoletus group (Diptera, Ceratopogonidae) in southern England. *The Journal of Animal Ecology,* 51**,** 135-148.

BISHOP, A., BARCHIA, I., et al. 1995a. Last occurrence and survival during winter of the arbovirus vector Culicoides brevitarsis at the southern limits of its distribution. *Australian Veterinary Journal,* 72**,** 53-55.

BISHOP, A., KIRKLAND, P., et al. 1995b. Distribution and seasonal movements of Culicoides brevitarsis Kieffer (Diptera: Ceratopogonidae) at the southern limits of its distribution in New South Wales and their correlation with arboviruses affecting livestock. *Australian Journal of Entomology,* 34**,** 289-298.

BISHOP, A. & MCKENZIE, H. 1994. Overwintering of Culicoides spp.(Diptera: Ceratopogonidae) in the Hunter Valley, New South Wales. *Australian Journal of Entomology,* 33**,** 159-163.

BISHOP, A., MCKENZIE, H., et al. 1996. Effect of temperature regimes on the development, survival and emergence of Culicoides brevitarsis Kieffer (Diptera: Ceratopogonidae) in bovine dung. *Australian Journal of Entomology,* 35**,** 361-368.

BISHOP, A. L. 1996. The dispersal of Culicoides brevitarsis in eastern New South Wales and associations with the occurrences of arbovirus infections in cattle. *Australian Veterinary Journal,* 73**,** 174-178.

BISHOP, A. L., BARCHIA, I. M., et al. 2000. Models for the dispersal in Australia of the arbovirus vector, Culicoides brevitarsis Kieffer (Diptera: Ceratopogonidae). *Preventive Veterinary Medicine,* 47**,** 243-254.

BISHOP, A. L., SPOHR, L. J., et al. 2004. Effects of altitude, distance and waves of movement on the dispersal in Australia of the arbovirus vector, Culicoides brevitarsis Kieffer (Diptera: Ceratopogonidae). *Preventive Veterinary Medicine,* 65**,** 135-145.

CAMPBELL, M. & KETTLE, D. 1976. Number of Adult Culicoides Brevitarsis Kieffer (Diptera: Ceratopogonidae) Emerging From Bovine Dung Exposed Under Different Conditions in the Field. *Australian Journal of Zoology,* 24**,** 75-85.

CARPENTER, S. 2006. Oral Susceptibility to Bluetongue virus of Culicoides (Diptera: Ceratopogonidae) from the United Kingdom. *Entomological Society of America,* 43**,** 73-78.

CARPENTER, S., LUNT, H. L., et al. 2006. Oral susceptibility to bluetongue viurs of Culicoides (Diptera: Ceratopogonidae) from the United Kingdom. *Journal of Medical Entomology,* 41**,** 73-78.

CARPENTER, S., WILSON, A., et al. 2009. Culicoides and the emergence of bluetongue virus in northern Europe. *Trends in Microbiology,* 17**,** 172-178.

DE KOEIJER, A. A., BOENDER, G. J., et al. 2011. Quantitative analysis of transmission parameters for bluetongue virus serotype 8 in Western Europe in 2006. *Veterinary Research,* 42**,** 53.

DUCHEYNE, E., DE DEKEN, R., et al. 2007. Quantifying the wind dispersal of Culicoides species in Greece and Bulgaria. *Geospatial Health* 1**,** 177-189.

DUCHEYNE, E., LANGE, M., et al. 2011. A stochastic predictive model for the natural spread of bluetongue. *Preventive Veterinary Medicine,* 99**,** 48-59.

EAGLES, D., DEVESON, T., et al. 2012. Evaluation of long-distance dispersal of Culicoides midges into northern Australia using a migration model. *Medical and Veterinary Entomology,* 26**,** 334-40.

EAGLES, D., MELVILLE, L., et al. 2014. Long-distance aerial dispersal modelling of Culicoides biting midges: case studies of incursions into Australia. *BMC Vet Res,* 10**,** 135.

EAGLES, D., WALKER, P. J., et al. 2013. Modelling spatio-temporal patterns of long-distance Culicoides dispersal into northern Australia. *Preventive Venerinary Medicine,* 110**,** 312-22.

GERRY, A. C. & MULLENS, B. A. 2000. Seasonal Abundance and Survivorship of Culicoides sonorensis (Diptera: Ceratopogonidae) at a Southern California Dairy, with Reference to Potential Bluetongue Virus Transmission and Persistence. *Journal of Medical Entomology,* 37**,** 675 - 688.

GERRY, A. C., MULLENS, B. A., et al. 2001. Seasonal transmission of bluetongue virus by Culicoides sonorensis (Diptera: Ceratopogonidae) at a Southern California dairy and evaluation of vectorial capacity as a predictor of bluetongue virus transmission. *Journal of Medical Entomology,* 28**,** 197-209.

GUBBINS, S., SZMARAGD, C., et al. 2010. Assessing the consequences of an incursion of a vector-borne disease I. Identifying feasible incursion scenarios for bluetongue in Scotland. *Epidemics,* 2**,** 148-54.

HENDRICKX, G., GILBERT, M., et al. 2008. A wind density model to quantify the airborne spread of Culicoides species during north-western Europe bluetongue epidemic, 2006. *Prev Vet Med,* 87**,** 162-81.

JONES, A., THOMSON, D., et al. 2007. The UK Met Office's next-generation atmospheric dispersion model, NAME III. *In:* BORREGO, C. & NORMAN, A. L. (eds.) *Air Pollution Modeling and its Application XVII.* Springer.

KAREIVA, P. 1983. Local movement in herbivorous insects: applying a passive diffusion model to mark-recapture field experiments. *Oecologia,* 57**,** 322-327.

KARL, S., HALDER, N., et al. 2014. A spatial simulation model for dengue virus infection in urban areas. *BMC Infect Dis,* 14**,** 447.

KELSO, J. K. & MILNE, G. J. 2014. A spatial simulation model for the dispersal of the bluetongue vector Culicoides brevitarsis in Australia. *PLoS One,* 9**,** e104646.

KETTLE, D. 1951. The spatial distribution of Culicoides impunctatus Goet. under woodland and moorland conditions and its flight range through woodland. *Bulletin of Entomological Research,* 42**,** 239-291.

KLINGSEISEN, B., CORNER, R. J., et al. 2011. Developing spatio-temporal prediction models for arbovirus activity in Norhtern Australia based on remotely sensed bioclimatic variables. *Surveying and spatial sciences biennial conference 2011.* Wellington, New Zealand.

LILLIE, T. H., MARQUARDT, W. C., et al. 1981. The flight range of Culicoides variipennis (Diptera: Ceratopogonidae). *The Canadian Entomologist,* 113**,** 419-426.

MELLOR, P., BAYLIS, M., et al. (eds.) 2009. *Bluetongue*: Academic Press.

MELLOR, P., BOORMAN, J., et al. 2000. Culicoides biting midges: their role as arbovirus vectors. *Annual Review of Entomology,* 45**,** 307-340.

MELVILLE, L., WEIR, R., et al. Characteristics of naturally-occurring bluetongue viral infections of cattle. ACIAR PROCEEDINGS, 1996. 245-250.

MULLENS, B., GERRY, A., et al. 2004a. Environmental effects on vector competence and virogenesis of bluetongue virus in Culicoides: interpreting laboratory data in a field context. *Veterinaria Italiana,* 40**,** 160-166.

MULLENS, B. A., GERRY, A. C., et al. 2004b. Environmental effects on vector competence and virogenesis of bluetongue virus in Culicoides, interpreting laboratory data in a field context. *Veterinaria Italiana,* 40**,** 160-166.

MURRAY, M. 1987. Local Dispersal of the Biting-Midge Culicoides-Brevitarsis Kieffer (Diptera, Ceratopogonidae) in Southeastern Australia. *Australian journal of zoology,* 35**,** 559-573.

MURRAY, M. 1991. The Seasonal Abundance of Female Biting-Midges, Culicoides-Brevitarsis Kieffer (Diptera, Ceratopogonidae), in Coastal South-Eastern Australia. *Australian journal of zoology,* 39**,** 333-342.

MURRAY, M. & NIX, H. 1987. Southern Limits of Distribution and Abundance of the Biting-Midge Culicoides-Brevitarsis Kieffer (Diptera, Ceratopogonidae) in Southeastern Australia-an Application of the Growest Model. *Australian journal of zoology,* 35**,** 575-585.

MURRAY, M. D. 1995. Influences of vector biology on transmission of arboviruses and outbreaks of disease: the Culicoides brevitarsis model. *Veterinary Microbiology,* 46**,** 91-99.

NATIONAL OCEANIC AND ATMOSPHERIC ADMINISTRATION. 2015. *Air Resources Laboratory - HYSPLIT - Hybrid Single-Particle Lagrangian Integrated Trajectory model* [Online]. Available: http://www.arl.noaa.gov/HYSPLIT_info.php [Accessed 29th June 2015].

O'CONNELL, L. 1994. *Entomological aspects of the transmission of arboviral diseases by Culicoides biting midges*, University of Bristol.

PIOZ, M. 2014. Did Vaccination Slow the spread of Bluetongue in France. *PLoS ONE,* 9.

PIOZ, M., GUIS, H., et al. 2011. Estimating front-wave velocity of infectious diseases: a simple, efficient method applied to bluetongue. *Veterinary Research,* 42**,** 60.

PURSE, B. V., MELLOR, P., et al. 2005a. Climate change and the recent emergence of bluetongue in Europe. *Nature Reviews Microbiology,* 3**,** 171-181.

PURSE, B. V., MELLOR, P. S., et al. 2005b. Climate change and the recent emergence of bluetongue in Europe. *Nature Reviews Microbiology,* 3**,** 171-181.

RACLOZ, V., VENTER, G., et al. 2008. Estimating the temporal and spatial risk of bluetongue related to the incursion of infected vectors into Switzerland. *BMC Veterinary Research,* 4**,** 42.

RUDD, W. & GANDOUR, R. W. 1985. Diffusion model for insect dispersal. *Journal of economic entomology,* 78**,** 295-301.

WARD, M., FLANAGAN, M., et al. 1995. Infection of cattle with bluetongue viruses in Queensland, Australia: results of a sentinel herd study, 1990–1992. *Veterinary Microbiology,* 45**,** 35-44.

WILSON, E. O. & BOSSERT, W. H. 1971. *A primer of population biology,* Sunderland, Massachusetts, Sinauer Associates, Inc. Publishers.

9 Appendix

9.1 Appendix A : Additional model details

Table A1

symbol	*description*
	variables
p_i , p_m	population, immature and mature
t	time
	parameters
b	birth (oviposition) rate, function of temperature
d_i , d_m	death rate, function of temperature
m	maturation (emergence) rate, function of temperature
p_{imax}	immature population density at which oviposition becomes zero
	boundary conditions
$p0_i$, $p0_m$	population at time zero
$T(t)$	temperature at time t

The rate parameters b, d and e are assumed to be functions of temperature. We use simple linear ramp functions as described in Table A2.

Table A2

parameter	*maximum value*	*minimum value*	*source*
b	0.4 (mean oviposition of 1 viable egg every 2.5 days) above 35° C	0 below 10° C	(Mullens et al., 2004a)
d_i	0.035 (mean 28 day lifespan)	same as maximum	(Bishop et al., 1996)
d_m	0.2 (mean 5 day lifespan) above 20° C	0.05 (mean 20 day lifespan) below 10° C	(Mullens et al., 2004a)
m	0.1 (mean 10 days from oviposition to emergence) above 35° C	0 below 10° C	(Bishop et al., 1996)

Note that the particular model parameter values (e.g. reproduction rates, life spans, temperature dependencies) given above represent an initial model based on the Culicoides literature. It is expected that parameter values will be refined though interaction with experts.

Table A3 Transmission model parameter values

parameter	meaning	value	source
β_{host}	Probability of infection of host resulting from a bite form an infectious vector.	0.9	(O'Connell, 1994)
A(T)	Temperature-dependent vector activity factor: average number of vector bites per day. See referenced source for equation.	0.360 per day @ 28°C 0.205 per day @ 20 °C	(Mullens et al., 2004b)
β_{vector}	Probability of infection of vector resulting from a bite on an infected host.	0.01	(Carpenter, 2006)
EIP(T)	Temperature dependent extrinsic incubation period: mean duration from vector infection to infectiousness.	6.8 days @ 28°C 17.4 days @ 20 °C	(Mullens et al., 2004b)
EIP stages	Number of vector E stages	2	(Mullens et al., 2004b)
HIP	Host infectious period: mean duration from host infectiousness to cessation of viral shedding.	20.6 days	(Melville et al., 1996)
HIP stages	Number of host I stages	5	(Mullens et al., 2004b)

The parameters reported here correspond to values suitable for *C. brevitarsis* as vector and cattle as host. Note that implementation allows for multiple host and vector species with different parameters for each. The model also includes Bluetongue-caused mortality in hosts (an additional rate for a death transition is calculated in state 2.3.2 above), which is necessary for modelling sheep populations.

Table A4 Vaccination parameters

parameter	typical values	references
VE, cattle	75%	[Baetza 2014], [Gubbins 2012]
VE, sheep	95%	[Baetza 2014], [Gubbins 2012]
host to vector VE	95%, 0%	[Gubbins 2012]
viremea duration reduction	50%	[Gubbins 2012]
coverage	70%, 95%	[Baetza 2014], [Szmaragd 2010 UK], [Pioz 2014]
reactive vaccination radius	2km, 20km, 50km, 100km	[Szmaragd 2010 UK], [Sumner 2013]
time-to-protection, cattle	14 days	[Gubbins 2014]
time-to-protection, sheep	60 days	[Gubbins 2014]
immunity development	linear over time-to-protection	[Szmaragd 2010 UK]
sheep vs cattle vector preference*	0.205	[Ayllón see 31 in Bessell 2014 Schmallenberg paper]
delay to vaccination activation after detection	2 days, 21 days	[Szmaragd 2010 UK]
vaccination rate	(not modelling vaccination logistics resources)	
per-case detection probability, cattle	50%	
per-case detection probability, sheep	95%	

Table A5 Transmission model experiment settings

Setting	Value
Cattle population	100 (fully susceptible)
Temperature	28^{o}C / 20 ^{o}C
Initial midge population (adult)	210,000 / 130,000
Initial midge population (immature)	500,000 / 500,000
Bluetongue transmission parameters	(see Table A3)
Limiting immature midge density	5000 per cow
Oviposition rate	20 per day (for 20^{o}C and above)
Mean immature period	13.8 days at 28^{o}C; 14.2 days at 20 ^{o}C
Mean adult lifespan	5.0 days

9.2 Appendix B : Glossary

Abbreviations

ABARES	Australian Bureau of Agricultural and Resource Economics and Sciences
ABS	Australian Bureau of Statistics
AHA	Animal Health Australia
ArcGIS	ESRI's primary Geographic Information Systems software product
ARL	Atmospheric Research Laboratory (of the United States NOAA)
AWS	Automatic Weather Station
BOM	Bureau of Meterology (Australia)
BTV	Bluetongue Virus (BTV-1, BTV-2 etc denote different BTV serotypes)
CGI	Common Gateway Interface (an internet protocol for constructing web-based interfaces)
GIS	Geographical Information Systems
GUI	Graphical User Interface
ESRI	A commercial software provider of GIS products
HTML	Hypertext Markup Language
HYSPLIT	Hybrid Single Particle Lagrangian Integrated Trajectory Model (an atmospheric dispersion modelling software suite developed by the US NOAA ARL and the Australian BOM)
Met Office	Meterology Office (UK)
MLA	Meat and Livestock Australia
NAME	Numerical Atmospheric-dispersion Modelling Environment (an atmospheric dispersion modelling software suite developed by UK Met Office)
NAMP	National Arbovirus Monitoring Program
NOAA	National Oceanic and Atmospheric Administration (United States)
NSW	New South Wales
SA-2	Statistical Area Level 2 (the second finest geographical region sizes used by the ABS, containing a population from 3,000 to 25,000 people)
TTI	Time To Immunity (the mean time from an animal being vaccinated until full immunity to infection)
UK	United Kingdom
UWA	The University of Western Australia
VIC	Victoria

Symbols

Mathematical symbols (variable or parameter names) used in the text are described below. Full details of all symbols appearing in equations and formula can be found in the text accompanying the formula.

infection dynamics model

For infection dynamics symbols, subscripts (e.g. N_{host}) denote the population to which the symbol applies (e.g. host or vector).

S E I R — These refer to the number or proporation of a vector or host population that is in a particular infection state.

S — Susceptible

E — Exposed (infected, but not yet infectious)

I — Infectious (infected and infectious)

R — Recovered or Removed (post-infection i.e. previously infected but now no longer symptomatic or infectious)

N — N denotes the total size of the population in all infection states

β — The per-bite probability of infection. A subscript denotes the species potentially being infected (vector or host).

λ — The rate of transition of a host or vector from susceptible to exposed (or infected) state, also known as the force of infection. A subscript denotes the species potentially being infected.

population dynamics model

p

p_i — A variable denoting the size of the immature vector population including eggs, larvae and pupae.

p_m — A variable denoting the size of the adult vector population.

p_{imax} — A model parameter; the landscape cell immature population at which competition for resources amongst immature vectors reduces the maturation rate to zero.

9.3 Appendix C : Reference Group Feedback

Professor Peter Walker, Chief Research Scientist at the CSIRO Australian Animal Health Laboratory, provided feedback from himself and his colleagues JB Duchemin, Debbie Eagles and Peter Durr

Overall, we feel that the report is very good, particularly with respect to careful consideration of biotic and abiotic factors that would influence transmission and spread of BTV infection. These, of course, are limited by data availability and the need in some cases to translate data obtained on exotic midge species. The importance of obtaining good, basic, biological data to underpin this and other models is an issue that should be addressed.

Nevertheless, the model appears to reasonably approximate the seasonal population dynamics and dispersal dynamics for *C. brevitarsis*, and to provide a useful basis for predicting BTV transmission dynamics in NSW. It would be very informative to see how well the model deals with other scenarios, particularly with respect to other potential vector species that do not breed in cattle dung. Also, although vaccination interventions have been built into the model, these appear to remain untested. We would also like to see sensitivity analyses applied to the key assumptions of the model to identify points of potential weakness. However, these comments reflect unexplored opportunities rather than any implied criticism of the model itself.

The primary issue of concern is the long-term utility of the modelling effort. The report states that "the simulation software tools developed in this project will enable planners to assess the likely area affected, magnitude and duration of a bluetongue outbreak beginning in different locations, at different times of the year, and in different annual weather conditions" (p.49). However, it is not clear how that will be facilitated. The history of many projects such as this is that when the modelling is needed (say 10 years' time), the creators will have moved on and the code may have been lost (e.g., due to a server upgrade). To overcome this, current best practice is to deposit the code into an open source repository such as Github. Alternatively, the report should state that the primary code will be deposited into a secure repository (UWA?) and provide exact details for future access.

Response from Milne and Kelso:

To enable future access to the UWA Bluetongue simulator environment, the software will be placed in the UWA repository along with an instruction manual. This will permit use of the system by BTV researchers and make it available to use during future BTV outbreaks.