Mathematical models of a tick borne disease in a British game bird with potential management strategies

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Abstract

Louping ill virus (LIV) is a tick borne disease that causes mortality in red grouse, an economically important game bird of British uplands. The aim of this thesis is to extend previously published models of LIV , to consider the potential impact of different management strategies. In addition a new route of infection and the seasonal biology of both grouse and ticks will be explored.

Grouse chicks are known to eat ticks as part of their diet in the first three weeks of life which may contribute to virus persistence if chicks consume infected ticks. This novel route of infection is incorporated in to the model which predicts that ingestion increases the range of host densities for which the virus is able to persist. The ingestion of ticks by grouse also reduces the tick population so that for low host densities the ingestion of ticks by grouse reduces the tick population so virus cannot persist.

The model is adapted to take account of the seasonal biology of grouse and ticks. Although the temporal predictions of the seasonal models show some differences the addition of seasonality does not alter the model predictions of when LIV is likely to persist at different grouse and deer densities. Consequently seasonality is felt to be unimportant when considering management strategies.

The treatment of sheep with acaricide in an attempt to reduce the tick population on a grouse moor is currently being trialled in Scotland. We use a model to predict the likely effect of this strategy at different deer densities. The number of ticks found attached to sheep varies so we consider the effect of tick attachment rates as well as acaricide efficacy. Although we predict that acaricide treated sheep can reduce the tick population and therefore LIV in grouse in some circumstances the treatment is less effective in the presence of deer.

Consequently we use a model to make theoretical predictions of the effectiveness of acaricide treated deer as a control strategy for reducing LIV in red grouse. The effect of culling deer on LIV in grouse is also modelled and contrasted with the effect of acaricide use. It is predicted that acaricide treatment of deer could be highly effective, particularly if the deer density is first reduced by culling.

Finally we considered the direct treatment of red grouse with acaricide. Female grouse can be given an acaricidal leg band which protects her directly and indirectly protects her chicks as they acquire some acaricide whilst brooding. Trials have suggested this can reduce tick burdens for individuals. We use the model to determine the potential effect that treating individual broods may have on the whole grouse population. The model predictions suggest that unless acaricide efficacy on chicks is high and long lasting treating individual broods is unlikely to reduce LIV in the whole population but will still provide some benefit for the individuals. The effectiveness of treatment is reduced by higher deer densities.

The success of the management strategies considered in this thesis appear to be restricted by the presence of deer. It may therefore be that a combination of treatments including the treatment of deer may be of the greatest benefit to the grouse population.

Contents

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Chapter 1

Introduction

Mathematical models can be used to help explain empirical data or offer predictions of what might happen under certain conditions. The use of a model can help identify mechanisms within a system and highlight areas where further empirical research is needed. Models do not need to take account of the ethics that may prevent or regulate empirical trials. Whilst mathematical models can provide insights in to biological phenomena they are a simplified view based on assumptions and need to be interpreted with caution.

Ticks are one of the most important vectors of zoonotic diseases throughout the world carrying serious infections that can deplete livestock and wildlife populations as well as infecting humans. Arthropod vectors differ from vectors such as mosquitos that can feed frequently and, being unable to fly, ticks have limited geographical range over which they can spread the infection without the aid of a host. Consequently mathematical models of tick-borne diseases have been developed to consider the particular dynamics of the tick vector, explained in detail below. This chapter presents a summary of the different techniques used to model tick-borne diseases. The work presented in this thesis extends previous work on louping ill virus (LIV), a tick-borne disease that affects red grouse, consequently the papers considering LIV are discussed in greater detail than the literature on other tick-borne diseases.

Tick biology The models presented here are of tick-borne pathogens spread by hard ticks of the Ixodid genus. Ixodid ticks progress through 3 active life stages, larvae, nymph, adult, requiring a blood meal at each stage from a suitable host. Larvae and nymphs will feed on any host but adults generally require larger mammalian hosts to feed successfully. After successfully completing a meal each stage returns to the ground vegetation and moults in to the next stage. For European sheep ticks, *Ixodes ricinus*, depending on timing and climatic conditions this may occur immediately so the tick begins questing again in the same season or the tick may enter diapause and not resume questing until the following summer (Randolph et al., 2002). A diagram of the tick life cycle is shown in Fig. 1.1. Ticks found in temperate zones are less active in colder months and show peak periods of activity in the summer months. The timings of peak questing periods can differ between stages and appear to vary with location and between years (Randolph et al., 2002). Questing ticks have now been recorded in each month of the year

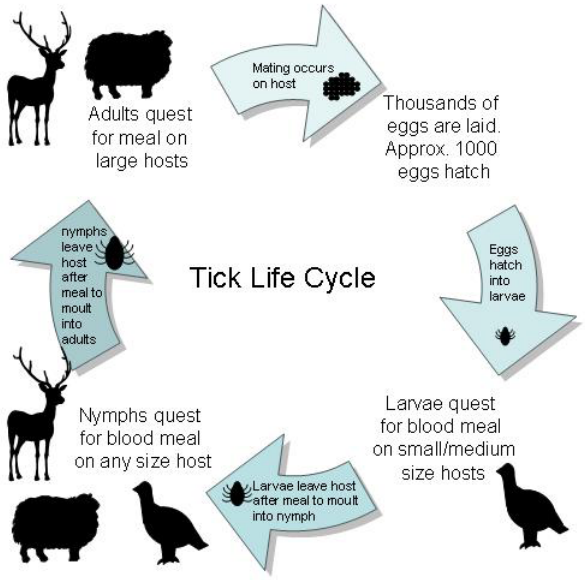


Figure 1.1: The life cycle of the European sheep tick *Ixodes ricinus*

in south west Scotland at temperatures between 3.5 and 29.3°C (Walker, 2001).

Some evidence of transovarial transmission (from adult females to eggs/larvae) has been found for some tick-borne pathogens, e.g., *Anaplasma phagocytophilum*, but not for LIV or *Borrelia burgdoferi*. Systemic/viraemic transmission occurs when ticks feed upon viraemic hosts (hosts experiencing high levels of virus in the blood). A feeding tick receives the pathogen as it takes a blood meal from the infectious host. Similarly an infectious tick can transmit the pathogen to a host as it feeds.

Another route of infection for some tick-borne pathogens is through non-viraemic transmission. This occurs via the skin aided by tick saliva when a susceptible tick feeds close to an infected tick on a host that is not displaying high levels of viraemia in the blood (Labuda et al., 1993).

1.1 Models of tick-borne diseases

Hudson et al. (1995) presented the first published deterministic mathematical model of a tick-borne disease system, namely the LIV system. This and other LIV system models are discussed separately in Sec. 1.2 as they form the basis for this thesis.

A major development in modelling tick-borne disease dynamics came from O'Callaghan et al. (1998) with a fully parameterised SIR type model of Heartwater, a tick-borne rickettsial disease in ruminants of sub Saharan Africa, neighbouring islands and the Caribbean. Heartwater affects cattle, causing major economic losses through mortality and cost for control. The model presented in this paper aimed to describe the conditions affecting the infection dynamics that may help explain the endemic stability noted in some populations that have a high level of infection in all ages of livestock. The cattle population is described by 6 time-delayed, non-linear, ordinary differential equations. The tick vector dynamics are described by coupled, non-linear, delay differential equations.

O'Callaghan et al. (1998) was the first paper to lend theoretical and quantitative support to the concept of endemic stability in the epidemiology of tick-borne diseases in cattle. They found this is

principally due to the protection of calves against disease due to innate and maternal factors. Vertical infection of cattle allows endemic stability to occur at lower levels of tick challenge. However, the incidence of infection is reduced for all levels of tick challenge and makes endemic stability less apparent. The severity of the disease at a population level is related in a non-linear fashion to the level of tick attachment and the rate of infection. This agrees with field data.

O'Callaghan et al. (1999) and others extended this work to develop a model to consider the potential effect of a vaccination for heartwater. Mathematical models provide a more practical alternative to expensive (and sometimes impossible) long term field trials with results obtained in a much shorter time frame. Although models require good estimates of parameters if they are to be used to make predictions about real life scenarios.

By adding vaccination to the previous model O'Callaghan et al. (1999) were able to conclude that the effectiveness of any vaccination protocol will vary according to the timing of the vaccination in an epidemic and the degree of tick challenge. The frequency of revaccination may have a greater effect on population protection than vaccination efficacy. Vaccination changes the rate of infection, usually leading to a slower and smaller epidemic. Vaccination may need to be maintained long term but can be useful if the right circumstances are present in the face of an epidemic.

Similar models have been developed for other tick-borne diseases. Tick borne encephalitis virus (TBEv) is a disease of major importance in parts of Europe. Rosa and colleagues have made a significant contribution to understanding the factors important to the persistence of TBE virus in Europe through the use of mathematical models. Rosa et al. (2003) considered the role of non-viraemic transmission (the ability of a host to transmit the virus without having a viraemic response themselves), feeding times and the aggregation of ticks on hosts. The deterministic SIR type model consists of a set of coupled ordinary differential equations based on that of Gilbert et al. (2001), discussed in Sec. 1.2. The tick population is considered as separate life stages, larvae, nymphs and adults which enables the model to explicitly consider the aggregation of different tick stages on the different hosts, competent hosts which are susceptible to the virus and hosts which are incompetent to the virus but maintain the tick population. The consideration of tick feeding times and aggregation of ticks is a novel aspect of this model.

The reproductive rate of the virus R_0, is classically defined as the number of new infections caused when one infectious individual is added to a susceptible population. If $R_0 > 1$ the virus can persist, if $R_0 < 1$ the virus cannot persist. Rosa et al. (2003) used R_0 derived from the equations to explore thresholds for virus persistence for the viraemic route of infection as well as the non-viraemic routes from nymph to nymph, adult to adult and between stages. The dilution effect occurs when the incompetent host density increases to the point where the virus dies out because infective tick bites are being 'wasted' on hosts that do not transmit virus. When only viraemic transmission is considered the dilution effect of non-viraemic hosts occurs, but the extent of the dilution depends on the parameter choices.

When non-viraemic transmission is also considered then the model predictions show broadly that increasing non-viraemic transmission between stages increases the ability of the virus to persist. If non-viraemic transmission is high enough the dilution effect stops occurring and the virus can persist even

without a viraemic host. Increasing the transmission to larvae has the most dramatic effect as the larvae are able to transmit back to hosts as a nymph and again as an adult.

Aggregation of the ticks on hosts and the effect on the pathogen persistence thresholds is also considered. If nymphs are strongly aggregated on incompetent hosts then for a fixed density of competent hosts R_0 is significantly increased and the density of incompetent hosts for which dilution takes place may double. If there is a strong correlation between adults and larvae this can also cause a large increase in the ability of the virus to persist.

This work is extended in Rosa and Pugliese (2007) by looking in more detail at the questing and feeding phases of the different tick stages, different transmission routes and the density dependence function for ticks. The assumptions made in the formation of the model mainly follow from the previous paper but additional assumptions were necessary as the model was extended. The questing and feeding phases of the tick stages were modelled explicitly which required the assigning of moulting probabilities for each stage after the completion of each meal. It was assumed that no death occurs during feeding but only during questing periods, however the moulting probabilities took in to account the mortality of fed ticks before they emerge as the next life stage. The density dependence of the ticks was no longer assumed to be linear, as this can potentially lead to negative reproduction when tick densities are high. Instead two alternative functions were considered; a negative exponential function depending on tick population and a negative exponential function depending on the average tick load per host.

Similar functions were included for the moulting probabilities of the different tick stages. The model is a series of 15 coupled differential equations for the Larvae, Nymphs and Adult ticks which are modelled explicitly as feeding and questing and then split again according to infection status, either susceptible or infected. The competent host is affected by the virus and was thus modelled as susceptible, infected or immune.

The equations were used to consider the infection dynamics and the threshold host densities that allow pathogen transmission. It was assumed that adult ticks do not feed on competent hosts as is the case for TBEv, rodents transmit virus but do not feed adults and deer are incompetent hosts amplifying the tick population but not virus. Transovarial transmission in ticks and non-viraemic transmission in competent hosts were included independently to consider the impact of tick to tick transmission when co-feeding on the same host. When non-viraemic transmission was included ticks that are exposed but not yet infectious need to be modelled separately so that virus can be acquired in one meal but not transmitted until the next meal after the tick has moulted into the next life stage. Due to the complexities of the model R_0 the reproductive rate of the virus was not calculated. An alternative threshold condition, S_0, calculated as the sum of the different transmission routes within the host and tick populations was used. S_0 can be interpreted in a similar way to R_0 so that if $S_0 > 1$ virus persists and if $S_0 < 1$ it does not.

Rosa and Pugliese (2007) pointed out that when the models are calibrated for TBEv in the Trentino area of Italy the infection cannot persist if only viraemic transmission occurs (i.e. if transovarial and non-viraemic transmission parameters are set to 0). Comparing the transmission routes for reasonable (20-30 per hectare) rodent densities suggests that transovarial transmission would need to be greater than

10%, an order of magnitude higher than empirical estimates. Non viraemic infection would, however, need to be between 30 and 40%, which is comparable with estimates in the literature.

To summarise, Rosa and Pugliese (2007) have improved the realism of previous models by explicitly modelling the different tick stages and phases of feeding and questing. The authors discovered that the effect of the competent host depends on the density dependence of the tick population, with two forms proposed. Empirical evidence suggests that real life may be somewhere between the two. Although the model predictions give some theoretical evidence of competent hosts having a dilution effect on the virus for the model with tick density depending on the total tick population the understanding of tick demography is not sufficiently well understood to verify this. Calibrating the model for TBEv gives theoretical support to the argument that TBE cannot persist without routes of infection other than viraemic host to tick to host transmission, in particular, non-viraemic transmission. Although the authors noted that seasonality may affect the quantitative threshold densities believed the results would be qualitatively similar.

Spatial models Tick-borne diseases are found in many parts of the world although their distribution can be patchy. Ticks are extremely limited in their mobility off a host. However, tick-borne pathogens can be spread over geographical areas by the movement of tick hosts carrying either infected ticks to neighbouring locations or transmitting virus to susceptible ticks in neighbouring locations. Adding space to a mathematical model brings added complexity but also additional insights into observed behaviour.

One way of controlling the spread of tick-borne pathogens could be to prevent the movement of tick hosts by the use of fencing. Various empirical trials of excluding deer from a given area have been conducted but their success at reducing tick densities has been varied. In general it would seem that tick densities are decreased inside large exclosures but increased in small exclosures (Perkins et al., 2006). Pugliese and Rosa (2008) developed a spatial model to consider the effect of excluding deer from different sized areas which helps explain the differences found by the trials. The ticks are modelled as in Rosa and Pugliese (2007) with density dependence on average tick load per host. To allow for the deer exclosure the model is extended over two patches with homogeneous environment so that the density of rodents is equal in both patches. One contains deer and one does not. Rodents can move freely between the two patches, with rate of movement dependent on exclosure size, but to ensure rodent density is constant if one moves out of the exclosure another has to move in. As rodents move they carry with them their tick burden. Virus dynamics are added to the model so that rodents can allow both viraemic and non-viraemic infection.

Assuming parameters are equal inside and outside the exclosure the effect of the exclosure varies for different tick stages and encounter rates as well as for exclosure size. Adding the assumptions that rodent density and the rate at which adult ticks feed on rodents are increased in the absence of deer changes the numerics of the predictions but not the overall conclusion. Increasing densities of rodents above the minimum threshold increases the prevalence of TBEv and also the density of infected nymphs (the stage that present the most risk to humans). Increasing deer densities leads to a rise in prevalence only for low

deer densities, after which the dilution effect begins to occur so that bites are 'wasted' on deer.

If the deer exclosures are large then the density of infected questing nymphs is reduced inside the exclosure (compared to outside). However, as the exclosure size is reduced the density of questing nymphs inside the exclosure rises so that for smaller exclosures there are more infected questing nymphs inside than out. When the exclosure is very small (0.75-1 hectare) the density of infected questing nymphs is twice as high inside than out and could represent a potential TBEv hotspot. The authors do not explain the mechanism for this phenomena but it may be that the deer amplify the tick population but not the virus outside the exclosure. The rodents then carry ticks in to the exclosure where they are able to amplify the virus without the diluting effect from the deer.

The authors acknowledged that a deer exclosure may change the habitat structure but believe this unlikely to vary with exclosure size. Therefore the different results for the different exclosure sizes must be due to another factor, for example the host dynamics. The mathematical models presented in the paper suggest that the effect of exclosure size on the tick density within the exclosure is due to the basic dynamics of the tick-host interactions without assuming habitat heterogenity.

Seasonal models Ticks display peaks of questing activity during the summer months increasing the potential for pathogen transmission between ticks and hosts. The effect of temporally changing tick/host interactions may affect the dynamics of infection, consequently mathematical models have been developed to explore this issue. Ghosh and Pugliese (2004) developed a semi-discrete model of ticks and tick-borne infections, using TBEv as an example. The model of ticks (without virus) assume ticks are active only in the summer and there is no activity over the winter period. Tick stages were considered separately and it was assumed that larvae and nymphs feed one summer but after moulting arrest development so do not quest again until the following summer. Adult females lay eggs after feeding, a proportion of these hatch immediately but the rest do not hatch until the following summer. Any ticks that have not fed die at the end of the summer. The host population was assumed to be constant. Tick activity over the summer was described by three differential equations, whose initial conditions each year were given by an integral based on the variables of the previous year.

Infection was added to the model but without transovarial and non-viraemic transmission. It was assumed that neither the tick nor competent host are affected by the infection. As in previous models the competent host was assumed not to feed adults and the incompetent host fed all stages but did not transmit virus. The analysis of the equations was complicated and the given formula for $R_{0,inf}$ is acknowledged to be difficult to interpret. However, the authors used numerical simulations to explore the model parameters. When parameterised for TBE the model predicted an endemic equilibrium with infection. Increasing the density of each host independently showed that there is an upper and lower limit between which there will be an endemic equilibrium. The dilution effect of the incompetent host was discussed in Rosa et al. (2003) but for much higher host densities. Ghosh and Pugliese (2004) found that the model predicted infection will begin to decrease for densities not much higher than the average found in the wild. There was also a dilution effect seen in the competent hosts, with lower infection

levels occurring at densities approx. double that found in the wild. The authors argued that this could be an important result, i.e. that infection is predicted to die out for competent host densities 4 times that of the average as opposed to 1000 times predicted by Rosa et al. (2003). However, they failed to acknowledge that the infection has no effect on the hosts in their model, but the competent host in Rosa et al. (2003) suffer virus induced mortality. The comparisons drawn are thus not comparing like with like and the interpretations need to be treated with caution.

Gaff and Gross (2007) used a different technique and presented a metapopulation model of tick-borne infection using both space and time to gain better insight in to the spread of tick-borne pathogens. Gaff and Gross (2007) focused on an effort to control an outbreak of human monocytic ehrlichiosis that occurred in Tennesee in the 1990s. Initially a one patch model of the total and infected host and tick densities was described. Deer were assumed to be the only important tick host and pathogen reservoir, remaining infected after receiving the pathogen without any additional mortality or loss to fecundity. There is no vertical transmission in either tick or host.

Unlike other models Gaff and Gross (2007) did not model tick birth separately but incorporate it as part of the overall tick growth rate, which also incorporates the probability of a tick finding a host and survival rate between meals. The tick growth rate was restricted by the maximum number of ticks per deer. The external death rate of the ticks includes desiccation and was manipulated to also include the effect of acaricide. Deer were modelled to have a logistic growth rate restricted by a given carrying capacity as well as suffering external death.

To account for seasonal differences the authors varied the growth and death rates through the year. The authors considered grassy and wooded areas assuming the death rate of ticks to be higher on grassy areas due to the greater impact of weather. A tick control strategy was added to this model to mimic the feeding of deer with acaricide laced corn. The treated corn is given to deer during the months of May -Aug. The acaricide needs time to leave the deer system before the hunting season begins. Although there will actually be a period of waning the model assumed that the acaricide remains fully active for a month after treatment stops. The use of acaricide was modelled as an increase in the external death rate.

The authors first considered the effect of treatment on individual sites (either woody or grassy) and a 2 patch model pairing a grassy area with a wooded area. A 12 patch model was then presented to mimic the study area, which contained 6 sites of tick collection, each with a wooded and grassy area. Total movement was allowed between the grassy and wooded area of each collection site, but movement between sites was related to the distance between them. Treatment sites showed a drop in the tick population in the first year but then the tick population remained the same. The percentage of ticks that are infected did not fall until year 2 but continued to fall in year 3. (Further years were not shown.) Little effect of treatment was shown in the sites without direct treatment suggesting that the effect of treatment does not aid neighbouring sites. The model predictions agreed with the data observed during the study period.

Although the tick dynamics have been captured in a more simple way than other models the model presented by Gaff and Gross (2007) provides useful insight into the likely effects of acaricidal use on

deer in set locations. The authors acknowledged that better research is required to improve estimates of parameter values but believe that the model could be a useful way of predicting the benefits of tick control strategies. By considering space and time explicitly within the model the model could be used to predict the optimum time and place for a treatment to be given.

Next generation matrix models Hartemink et al. (2008) used a completely different technique to develop a next generation matrix model to describe R_0 for tick-borne infections. For simple directly transmitted infections of one host R_0 is often defined as the average number of secondary cases caused by the introduction of an infected individual in to an entirely susceptible population. The size of R_0 can be used to determine whether or not there will be a disease outbreak, and if so gives an indication of the likely size of the epidemic. The interpretation of R_0 is complicated for vector borne diseases of wildlife as often more than one species is involved which can react differently to the infection. The authors argued that previous models of tick-borne infections that have developed an expression for R_0 often give threshold conditions for infection persistence but the quantity is not the traditional R_0 as it cannot be interpreted at an individual level.

Hartemink et al. (2008) categorized individuals by their state, 'type-at-birth', at the time of infection and considered the generations of infected individuals distributed over different states. The growth factor was given by the largest eigenvalue of the matrix and was interpreted as R_0. The 5 types-at-birth (of infection) are 1) tick infected as an egg, 2) tick infected as a larva, 3) tick infected as a nymph, 4) tick infected as an adult, 5) systemically infectious host. The matrix element k_{ij} represents the number of new cases of type-at-birth i caused by one infectious individual of type j during the entire infectious period.

The matrix was parameterised for both TBEv and Lyme disease in Europe. For both pathogens R_0 was given as a function of the proportion of tick blood meals taken from competent hosts, h_c. For both pathogens the size of R_0 increases with h_c but TBEv requires a much greater proportion of meals to be from competent hosts before TBEv can persist. Using sensitivity and elasticity analysis the authors were able to determine which parameters are likely to have the greatest influence on R_0. The survival of ticks to the nymphal stage seemed to be the most influential factor for TBEv. The non-viraemic transmission from larvae to nymphs and viraemic transmission from nymphs to host and hosts to larvae were also identified as important for TBEv. Whilst all the above were also influential on Lyme disease persistence the survival of all tick stages, the number of eggs, the amount of transovarial transmission and the transmission efficiency of larvae also seemed to be important, with non-viraemic transmission less so. The authors noted that there are many parameters which the model is not sensitive to and would not therefore need as accurate an estimate.

The authors also considered the influence of the different types of transmission, concluding that TBEv is highly influenced by non-viraemic transmission, particularly at higher values of h_c (which agrees with Rosa and Pugliese (2007)) but *Borrelia burgdoferi*, the agent of Lyme disease, is mainly sustained through viraemic transmission. It was noted that interchanging the duration of infection in the host for the two

pathogens reverses the results if all other parameters are held constant. This shows the importance of infection duration in transmission of tick-borne pathogens.

The authors claimed that their interpretation of R_0 from the next generation matrix is closer to the classic definition for simple infections as it can be interpreted at the individual level. Whilst the techniques are theoretically applicable to most tick-borne infections the data required to parameterise the model is not available for all infections. However, the sensitivity analysis indicates parameters that are likely to be the most influential which can direct future research to estimate these parameters.

1.2 Models of louping ill virus

The biology of louping ill virus Louping ill virus is the western variant of the TBE complex of viruses. The virus is transmitted by the European sheep tick, *Ixodes ricinus*. This flavivirus affects livestock and wildlife but is rare in humans. (No recent human infection has been recorded. (Health Protection Agency, 2009)) Although LIV can infect horses, pigs and cattle it is mostly found in sheep and red grouse. Disease in sheep manifests itself as tremors, difficulty walking and paralysis and can be fatal, although not all infected sheep develop clinical symptoms. The role of lambs in virus transmission appears to vary as a consequence of maternally acquired immunity which is greater in areas with a high force of infection. Lambs can acquire natural immunity through the colostrum of the ewe which lasts for the first few weeks of life. Lambs that survive infection have life long protection from further infection (Hudson et al., 1995). Although symptoms and fatality vary in sheep it seems the disease has more dramatic effect in naive sheep. Vaccination can be used to protect young or naive sheep, and repeated application should give life long immunity (Hudson et al., 1995). Acaricide dips or pour on can be used to reduce the number of ticks biting.

Experimentally infected red grouse suffered 78% mortality in laboratory experiments (Reid et al., 1978). The virus can cause substantial losses on infected grouse moors, seriously affecting economic revenue from grouse shoots. Adult ticks are rarely found on red grouse and it is important to note that as a result red grouse populations alone cannot sustain the tick population.

No evidence of transovarial transmission has been found for LIV, hence larvae are assumed free of the virus (Gaunt, 1997). Larvae can be infected upon biting an infectious host (unvaccinated sheep or grouse). Infected nymphs can transmit virus to grouse (sheep) upon feeding and susceptible nymphs become infected by feeding from an infectious host. Adult ticks are not important in the transmission cycle for red grouse as they do not generally feed on red grouse but could transmit (or receive) virus when feeding on sheep.

Another route of infection is through non-viraemic transmission which has only been found to occur on mountain hares (Gaunt, 1997). Grouse chicks are known to eat ticks as part of their diet of invertebrates during the first three weeks of life. Gilbert et al. (2004) suggest that the ingestion of infected ticks may be the main route of infection in wild grouse chicks.

Effect of LIV on grouse populations The temporal dynamics of grouse populations appear to differ between sites with and without LIV. Sites with LIV have lower grouse shooting bags, greater breeding losses and a lower density of breeding adult pairs. There appears to be some interaction between the presence of LIV and the parasitic worm *Trichostrongylus tenius*. The worms cause population cycles in red grouse, but these cycles seemed to be dampened on sites with LIV. LIV may also be responsible for population sinks in grouse populations, with LIV infected populations surviving after the immigration of uninfected grouse from neighbouring sites (Hudson et al., 1995).

LIV control Direct treatment of red grouse chicks with pour on acaricide showed some success in reducing tick burden and LIV prevalence (Laurenson et al., 1997) but may not be a practical method for routine management requiring considerable effort to catch and treat the chicks. Leg bands on hen grouse and directly on chicks also showed reduction in tick numbers, with the tags on chicks showing a greater effect. However chick survival was not increased by direct leg bands, possibly to due to adverse effects of the bands themselves (Laurenson et al., 1997). The use of leg bands on hens was further investigated by Mougeot et al. (2008) as a possible strategy that could be applied when female grouse are routinely caught for treatment with an anthelminthic to reduce intestinal worm infections. The chicks of female grouse with treated leg bands were found to have a reduced tick burden up to one month after hatching. Although some success has been noted in these trials treatment of grouse is not yet routinely used. Treatment of wild birds entering the food chain would need to be licensed but could be a feasible part of grouse management.

Grouse alone cannot support the tick population so indirect methods to reduce the tick population may include the removal of alternative tick hosts (e.g. deer, sheep and mountain hares) by culling, fencing or acaricide treatment. There are no published studies on the impact of the removal of deer on LIV incidence but deer are often culled or fenced out of areas due to their importance as tick reproduction hosts (Gray, 1998). The acaricide treatment and vaccination of sheep can reduce the prevalence of LIV on sheep farms Laurenson et al. (2007) but the effect of acaricide treated sheep on LIV prevalence in grouse is not yet fully understood. The preliminary results of the Game and Wildlife Conservation Trust (GWCT) have found that the treatment of sheep with acaricide to kill ticks may decrease tick burdens on grouse in areas with few deer but may be less effective when there are higher deer densities (Smith, c.2006).

Mountain hares host a relatively large tick burden when compared with red grouse and allow non-viraemic transmission to occur. As a result mountain hares have been culled in many areas in an attempt to reduce LIV in grouse. However, this is unlikely to be successful in areas with deer (Gilbert et al., 2001).

Models of LIV The first mathematical models of LIV were proposed by Hudson et al. (1995, 1997). The authors detailed the dynamics of LIV transmission in the grouse - tick - hare system and discussed the factors that may be important to LIV transmission, providing evidence that LIV prevalence may be higher on grouse moors that have mountain hares, whilst acknowledging that the geographical location of the sites may be a confounding factor. The paper brought together the known and unknown factors

(many of which are discussed above) that may contribute to the maintenance of LIV in the field. It was acknowledged that the extent of many of those factors was unknown at that time and that further investigation would need to be undertaken to gain a better understanding of these factors.

The first preliminary models of coupled ordinary differential equations considered the tick stages separately. Using coarse estimates Hudson et al. (1995) were able to demonstrate that hares (or similar mammal host) are required to sustain the tick population but high densities of hares (or similar) may prevent virus persistence as infected tick bites are wasted on these dead end hosts, as they were, at that time, thought to not transmit the virus. (Quantitative predictions were vague with accurate parameter estimates lacking.) It was noted that the parameters that the model was most sensitive to were the tick mortality rate and the probability of a tick biting a host. It was suggested that the tick mortality rate may be increased by the use of acaricide on wildlife, although this had not been tried at the time of publication. Alternatively, ticks require a damp matt layer below the vegetation in which to overwinter. Reducing the availability of overwintering habitat for the tick population may reduce tick numbers (Hudson, 1986b).

Hudson et al. (1997) developed the model presented above by expanding on the details of some of the studies mentioned in Hudson et al. (1995) to explore why LIV persists in areas where sheep are vaccinated and hence no longer contributing to the transmission cycle. Many of these studies were consequently published as individual papers (Gaunt, 1997; Jones et al., 1997; Laurenson et al., 1997, 2000); a summary of the main findings into LIV biology has been given above.

Hosts other than grouse are important for the maintenance of LIV, as grouse themselves cannot support the entire tick population because adult ticks need a larger host. Year to year variations of LIV prevalence are related to the number of nymphs found on chicks. Age prevalence curves show a rise in infection at an early age in grouse which coincides with the seasonal rise of questing ticks. The tick abundance in Northern Scotland is different to that of England suggesting that the tick cycle may be greater than 3 years. LIV prevalence varies spatially; areas with mountain hares show an increase in LIV prevalence compared to sites without hares (Hudson et al., 1997). Detailed studies have found that hares alone account for the difference in prevalence (Hudson et al., 1997). Given that non-viraemic transmission is known to occur in mountain hares and hares host a large number of ticks Hudson et al. (1997) consider hares to be a significant host for ticks and LIV. Alternative hosts can maintain ticks or virus or both, or may waste tick bites if incompetent. Hares have been found to carry 5-15 times as many ticks as sheep and 7-26 times as many larvae and nymphs as red grouse. The calculation of the reproductive rate of the virus for sites with differing levels of host densities supported the view that the virus may not persist in grouse if there are no other hosts. A brief summary of the model in Hudson et al. (1995) was also provided. The models in these papers were very much preliminary with accurate parameter estimates unavailable, however, these early papers provided direction for further studies and were the important first steps in gaining the much improved understanding we now have today.

Norman et al. (1999) provided a more in depth discussion of the mathematical model outlined in the papers by Hudson et al. (1995, 1997). The theoretical exploration of the reproductive rate of ticks and also virus provided potential threshold quantities for viraemic and non-viraemic host densities that allow

virus persistence. Parameter estimates were investigated; for many parameter combinations there is both an upper and lower threshold for non-viraemic hosts above and below which the virus will not persist. The low boundary signifies when there are only just enough non-viraemic hosts for the ticks to persist. The upper boundary occurs when the non-viraemic hosts are diluting the virus, as they are dead end hosts for the virus. Applying the theory to the LIV system Norman et al. (1999) showed that grouse alone cannot sustain the tick population (and hence virus). The simple model provided in this paper gave an insight into host interactions and showed how models may be a useful way of investigating host management strategies that may help control disease or quantify the dilution effect. Although Norman et al. (1999) offered a more detailed analysis of the LIV system than previous papers the model still did not account for non-viraemic transmission in hares or the potential role of lambs.

Laurenson et al. (2000) explored the role of lambs in the maintenance of LIV on sheep farms that have vaccinated ewes for many years using a field study in combination with a mathematical model. They concluded that although theoretical conditions allowed lambs to play a significant role in LIV persistence these conditions were unlikely to be met in most farming situations. Consequently lambs have not formed a part of further models.

Gilbert et al. (2001) considered the importance of red deer in the maintenance of LIV for the first time and explicitly models non-viraemic transmission on mountain hares. Grouse are considered in three disease classes, susceptible G_s, infected G_i and immune G_z. The tick stages are combined and the tick population is divided into susceptible T_s and infected T_i classes. The model of Gilbert et al. (2001) forms the basis of much further work (including this thesis) and therefore I explain it in detail here. The equations are given below.

$$\begin{aligned}
\frac{dG_s}{dt} &= A + (a_g - s_g G)G - b_g G_s - \beta_1 T_i G_s \\
\frac{dG_i}{dt} &= \beta_1 T_i G_s - \Gamma G_i \\
\frac{dG_z}{dt} &= \gamma G_i - b_g G_z \\
\frac{dT_s}{dt} &= (a_t - s_t T)T(\beta_4 H + \beta_5 D) - b_t T_s - \beta_2 T_s G_i - (\beta_4 H + \beta_5 D)T_s - \theta T_s T_i H \\
\frac{dT_i}{dt} &= \theta T_s T_i H + \beta_2 T_s G_i - b_t T_i - (\beta_4 H + \beta_5 D)T_i
\end{aligned}$$

where $\Gamma = \alpha + b_g + \gamma$.

Gilbert et al. (2001) were the first to provide a full explanation of their parameter estimates. The authors use two wild populations with different host community structures to estimate some parameters so that model predictions match the empirical data and two different sites are used to validate model predictions. For reference Table 1.1 outlines the parameters and values. Further explanation is given in the text.

Param -eter	Value (unit)	Explanation
A	1.94 (m^{-1})	Rate of grouse immigration (0 for site C)
a_g	0.167 (m^{-1})	Natural birth rate of grouse (Hudson, 1992)
s_g	$0.000\dot{3}$ $(g^{-1}m^{-1})$	Density dependence constraint on grouse.
b_g	0.087 (m^{-1})	Natural death rate of grouse (Hudson, 1992)
α	5 (m^{-1})	Disease induced death rate of grouse (Reid, 1975)
γ	1.25 (m^{-1})	Recovery rate of infected grouse (Reid, 1975)
a_t	83.33 (m^{-1})	Natural birth rate of ticks
s_t	0.00139 $(t^{-1}m^{-1})$	Density dependence constraint on ticks.
b_t	0.083 (m^{-1})	Natural death rate of ticks
β_1	0.0002 $(g^{-1}m^{-1})$	The rate at which a tick bites and infects a grouse.
β_2	$9.75\beta_1$ $(g^{-1}m^{-1})$	The rate a tick bites a grouse and becomes infected
β_4	$1.84\beta_1$ $(H^{-1}m^{-1})$	The rate an adult female tick bites a mountain hare and reproduces
β_5	$8.82\beta_1$ $(D^{-1}m^{-1})$	The rate an adult female tick bites a deer and reproduces
θ	$3.9\text{x}10^{-7}$ $(H^{-1}m^{-1})$	The rate of non-viraemic transmission between co-feeding ticks on mountain hares

Table 1.1: The model parameters from Gilbert et al. (2001). For the units m denotes month, g grouse, t ticks, H hares and D deer.

The grouse parameters a_g and b_g the natural grouse birth and death rates respectively assume 4 chicks are 'born' per adult pair and that 35% of grouse remain alive at the end of the year (Hudson, 1992). If grouse die at an exponential rate then b_g is the fraction required to die each month. s_g the density dependence of grouse is estimated from the model to gain the required carrying capacity. The disease induced death rate α and recovery rate γ are estimated from experiments conducted in Reid (1975). Grouse die on average 6 days after infection so on average 5 grouse will die per calendar month. Approx. 80% of experimentally infected grouse died so grouse are 4 times more likely to die than recover, hence $\gamma = \alpha/4$. Grouse immigration is known to occur and estimated so that the ratios of susceptible, infected and immune grouse match empirical data. A is the rate at which grouse immigrate and is estimated using the model.

The tick parameters The birth and death rate of ticks a_t and b_t respectively are estimated from limited published data and assume 1000 successful eggs per female each year (83.33 per month) and that ticks will die after 1 year if they have not successfully fed. Although it is acknowledged that density dependence is likely to act on both tick birth and death rates for mathematical simplicity density dependence s_t acts only on the birth and is estimated so that model predictions fit empirical data.

Tick biting and transmission rates The transmission rate of LIV from ticks to grouse β_1 is taken as the biting rate of infected nymphs on grouse since nymphs are the only tick stage that bite grouse that can be infected. The biting rates of different tick stages on different host animals had been counted on different sites and were used to relate all biting rates to the number of nymphs biting grouse. The rate at which the virus is transmitted non-viraemically on the mountain hare θ is unknown; although it is known to occur there is no available data that quantifies non-viraemic transmission. However, it is estimated using the model so that model predictions of grouse densities match empirical data.

This model is quite different from the previously published models; ticks are no longer considered at different life stages and the non-viraemic transmission of the mountain hare is included. The reproduction of the tick population depends on the availability of mammalian hosts. As explained previously the different tick stages take part in the transmission cycle in different ways. This is now incorporated in

to the model indirectly through the calculation of the attachment rates of susceptible and infected ticks on the different hosts depending on which tick stages are involved. The contributions of the three tick hosts differ; grouse amplify virus but not ticks, deer amplify ticks but not virus and hares amplify both. By changing host densities the authors were able to explore the conditions under which the virus might persist and possible parasite mediated apparent competition.

The thresholds for ticks and grouse were calculated in much the same way as in previous work, but here $R_{0,virus}$ also depends on non-viraemic transmission, and the carrying capacity includes immigration of grouse into sink populations.

The results confirmed that grouse alone cannot sustain virus, nor can deer alone, but together usually would (except for some low host densities). This is because grouse transmit the virus whilst deer sustain the tick population. Apparent competition is occurring as the grouse population is reduced by the virus; this is unusual as deer are not amplifying the virus only the vector. High deer densities cause a dilution effect because infectious tick bites on deer are wasted as deer do not transmit virus.

The virus usually persists in the presence of both hares and grouse with both hosts amplifying the virus and hares also amplifying the vector. Apparent competition also occurs between grouse and hares. Due to the non-viraemic transmission by mountain hares the virus can persist with hares alone. If all three hosts are concurrent then the likelihood of virus persistence is increased. Adding hares to an environment with deer and grouse reduces the chance of dilution as hares amplify the virus. Adding deer to a hare and grouse environment increases the amplification of the tick population so that virus almost always persists.

Gilbert et al. (2001) used the model to make predictions of the host densities for which LIV is likely to persist. In contrast, Laurenson et al. (2003) used the mathematical model to help identify the mechanisms behind the results of a large scale field experiment in which the density of hares is reduced to 0 over a period of 8 years in an attempt to reduce tick abundance and LIV prevalence in grouse. To incorporate the reduction in hare density the model was run for one year then stopped with initial grouse densities given by the densities at the study site before the treatment began and with the hare density as year 1 of the study. The model was run for the next year beginning with predicted densities of grouse and ticks but the hare density reduced to that of the treatment site. The following years were dealt with in the same way. In order to determine the likely factors mountain hares may contribute to the LIV system the model was run under 3 scenarios; i) hares can transmit virus and are hosts of the tick; ii) hares cannot transmit virus but are tick hosts; iii) hares can transmit virus but are not tick hosts.

The experimental reduction of mountain hares seemed to reduce the tick population to less than 1% of the original level. The prevalence of LIV was significantly reduced and breeding success in red grouse increased, relative to the control. However, there was no significant change to the relative grouse density.

The model simulations for the 3 scenarios all showed a decline in seroprevalence year on year as the hare density was reduced. The model showed the best fit to the data when non-viraemic transmission was included. The authors noted that the relative importance of hares as tick hosts and virus transmitters was changed when the order of magnitude of the tick population was altered by an order of magnitude,

suggesting the role of hares may change according to specific situations. The qualitative predictions seemed to be robust to parameter changes.

Norman et al. (2004) took the model of Gilbert et al. (2001) and Laurenson et al. (2003) and performed a more in depth theoretical exploration of the model and the effects of different parameters. The effect of mountain hares on the tick population and non-viraemic transmission being the main focus of this paper. The model no longer included immigration and the calculation for the death rate from disease used the maximum number of days observed between infection and death, instead of the mean number of days used in previous papers. Consequently the recovery rate was also reduced (since recovery is calculated to be 1/4 of the death rate). The death rate of ticks is assumed to be approx 1/3 of that given in previous papers. The relative biting rates of ticks on grouse and hares also differed from previous models. Although many of the parameters were estimated using field/experimental data there are some parameters for which there is little information and the model was used to make the estimates. These parameters are the transmission parameter β (the probability of an infected tick biting a susceptible grouse per unit time), the non-viraemic transmission parameter θ (the rate at which non-viraemic transmission occurs between two ticks feeding on a mountain hare) and the density dependence of ticks s_t. The effect of changing these parameters on the position of the $R_0 = 1$ curve in the hare - grouse plane was explored in detail. As non-viraemic transmission was the focus of this paper, red deer were not considered.

The authors analysed the equations to find threshold conditions for the density of hares required for ticks to survive. Noting that for this threshold to be low the natural death rate of ticks needs to be low, but the birth rate and biting rate of female adult ticks on hares need to be high.

The effect of hares on virus dynamics was also explored. To begin with non-viraemic transmission was omitted so that the effect on the $R_0 = 1$ curve of changing β and s_t could be seen. For large β the $R_0 = 1$ curve bent back on itself so that for some grouse carrying capacities there was an upper and lower threshold for hares above and below which the virus will not persist, as there are too few ticks or the virus is diluted as hares are assumed not to transmit virus. When s_t was varied the grouse carrying capacity required for virus persistence increased by a factor of 10 as s_t increased by a factor of 10.

s_t and β were set at intermediate values and non-viraemic transmission was included at different rates. The addition of non-viraemic transmission made the virus more likely to persist, especially at lower grouse carrying capacities. There was no longer an upper boundary of hares for virus persistence. Indeed, for high enough levels of non-viraemic transmission the virus could persist at realistic hare densities even without grouse, the viraemic host. The same qualitative results were found for two values of β although the quantitative predictions differed and the higher value of β saw the dilution effect still occurring at low non-viraemic transmission levels. The authors used their model to conclude that non-viraemic transmission could be important in the dynamics of LIV making disease in grouse more likely. However, as red deer are not included these results may not be applicable to estates that have deer.

Spatial effects This model was not extended again until Watts et al. (2009) who tested the model presented in Gilbert et al. (2001) with new field data before extending it to consider the spatial effects

of roaming deer. Initially the model of Gilbert et al. (2001) was used to predict the prevalence of LIV in ticks and grouse and the density of red grouse at new sites for which Watts et al. (2009) had data on host densities. The model underestimated the extent of LIV predicting persistence at 3 of the 6 sites, although LIV was detected in ticks collected at all sites. Watts et al. (2009) argued that the model appeared to overestimate LIV prevalence in grouse, however they only provided the LIV prevalence for grouse at two sites. At site 2 the authors found no LIV in red grouse around the area from where the ticks were tested but the ticks themselves had the highest prevalence in ticks on any site found in that year (2004). The authors have not tried to explain this discrepancy between high virus prevalence in ticks but not in grouse from around the same area. However, careful reading shows that the model was run with host densities from 2005, not 2004 when the grouse were tested for LIV. Had testing occurred in 2005 grouse from this site may have shown LIV antibodies if they had been bitten in the previous year by one of the many infected ticks. Watts et al. (2009) accepted in the discussion that the available data has limitations which may influence predictions.

The model performed better at predicting the relative number of ticks per site but seemed to underestimate relative tick density at site 2. The model predictions of LIV prevalence in ticks was lower than empirically found on some sites but higher on others. Watts et al. (2009) accepted that it is difficult to compare model predictions with empirical data as the LIV prevalence in ticks varied substantially between years on some sites. The predicted prevalence in ticks was sensitive to grouse population variables. The authors recognised that using the summer grouse density as a proxy for carrying capacity is not ideal when LIV is present as the grouse population will be reduced by disease. However, Watts et al. (2009) found that using a higher carrying capacity (the highest recorded by Gilbert et al. (2001)) was not sufficient to raise the predicted equilibrium grouse density to empirical levels. This was achievable but only by incorporating unrealistically high immigration levels.

Watts et al. (2009) went on to develop a spatial model consisting of two patches between which red deer were allowed to move. Three scenarios were considered; i) symmetrical deer movement between a site without hares and a site with hares, ii) asymmetrical deer movement between two identical sites, iii) varying deer movement between neighbouring sites of the study.

Scenario 1: The grouse density on the site with no hares is unaffected by deer movement if the neighbouring site has no hares. However, as the hare density on the neighbouring site increases the effect of increasing deer movement is enhanced so that at hare densities above approx 5 per km^2 a small increase in deer movement dramatically reduces grouse density. The seroprevalence in grouse shows a corresponding effect; grouse densities are low when seroprevalence is high.

Scenario 2: The asymmetrical movement of deer between 2 otherwise identical sites saw the highest LIV prevalence in grouse on the site where deer immigration was greater than emigration. The grouse population was correspondingly smaller.

Scenario 3: The authors assumed that deer could move between study sites 3 and 4 with host densities ($H < 1, D = 10, K_g = 16$) and ($H = 3, D = 5, K_g = 54$) respectively. Tick numbers are highest on either site when immigration of deer is high and emigration is low. There are more ticks at site 3 so emigration

from 3 to 4 had most effect, even when deer movement was equal between sites. LIV prevalence was higher at site 4 than site 3, even though site 3 had more ticks. This can be explained by the higher hare density at site 4. Hares have more effect on prevalence than deer due to their ability to transmit virus. The authors state that 'although LIV can be introduced to a site by ticks roaming on red deer it cannot then reach high levels unless other factors are also acting.'

Watts et al. (2009) concluded that the movement of deer between sites is important when considering the persistence of LIV. Even a small move from a LIV endemic area to an area without LIV can allow LIV persistence. The culling of hares may not work if deer are able to roam through from neighbouring sites. The authors argued that ordinary differential equation SIR type models may be more suitable for determining general patterns rather than modelling specific scenarios. They suggested that the incorporation of grouse density dependence in the birth rate may not reflect the pattern of density dependence that appears to occur during autumn recruitment after grouse chicks suffer disease induced mortality. The authors discussed the limitations of the model given the lack of detailed information on the spatio-temporal interactions of the hosts and ticks and suggest areas for future study.

The idea of spatial movement of LIV through deer movement is considered further by Jones et al. (2010). Jones et al. (2010) developed a simple 1 dimensional reaction diffusion model of deer movement between different habitat types. The model is an extension of that given in Gilbert et al. (2001), with the total grouse density made up of the susceptible, infected and recovered grouse but now the model has to specifically determine which ticks are on a host and which are off a host. Off the host the ticks can be susceptible or infected. If a susceptible grouse is bitten by an infected tick it is assumed to become infected immediately and any susceptible ticks that were attached or become attached also become infected, so that an infected grouse cannot carry susceptible ticks. (The accuracy of this assumption has little effect on the model predictions.) A recovered grouse can however carry both susceptible and infected ticks without any transmission occurring. Ticks are also modelled attaching to deer without reference to infection status as this is not affected by the deer. The model considers the average tick per host at a particular point in space and not the total number of ticks. The system of partial differential equations approximates the one dimensional movement of deer between forest and moorland habitats. This requires further assumptions to those of Gilbert et al. (2001); in particular, as both infected and susceptible ticks are modelled attaching to grouse in all classes a new attachment rate was calculated and ticks remain attached for a mean period. The grouse can move within the moorland but cannot pass into the forest. Deer however can roam freely between the two. No hosts can leave the closed area. The attachment rate of ticks on deer depends on the number of ticks already attached and saturates to a maximum. After detaching from deer the ticks reproduce at a given rate and then die without taking another meal. The forest and moorland differ in their effect on tick mortality with ticks suffering greater mortality in the moorland patches, with an immediate effect occurring at the boundary.

Jones et al. (2010) considered the effect of deer movement on the tick distribution and found that increasing the rate (distance) of deer movement into the moorland increased the density of ticks found in the moor. With this increase in ticks came a reduction in grouse survival. The effect was greatest

closest to the forest edge, with some levels of deer movement showing grouse survival to have doubled 100m from the boundary.

The model predicted that habitat size and structure plays an important part in viral persistence. If the proportion of forest to moorland is increased with the overall area remaining fixed then the proportion of grouse surviving falls. One moor with forest either side, with equal areas of total forest and moorland, was predicted to have a greater proportion of infected grouse than one moor and one neighbouring forest. If the relative size of the forest to a constant size moorland is increased then the number of grouse surviving is decreased. Similarly increasing the patchiness, i.e. more strips of alternating forest and moorland but with the same relative area, reduced the grouse survival. This is because the highest density of ticks is found close to the forest edge and if there are a greater number of forest edges there will be a greater area with a high tick density and thus low grouse density. The movement of grouse within the moorland had an homogenising effect on the reproductive rate of the virus so that it is decreased closer to the forest and increased further away. However patchiness reached a saturation point when the effect of the forest edges begins to overlap on the moorland strips.

The model may not provide numerically accurate simulations but the predictions are qualitatively useful and suggest that habitat management may be important. Restricting deer access to woods or preventing them from entering moorlands from forested habitat may reduce LIV prevalence. Similarly reducing the number of woods neighbouring moorland may be beneficial. Jones et al. (2010) did however present caveats, acknowledging that high tick densities do not necessarily mean there will be high LIV prevalence (indeed there was no LIV in the forests despite high tick densities). They also recognised that ticks are not distributed evenly at any spatial scale, but aggregated in patches. This small scale heterogeneity could potentially be modelled stochastically. More detailed information on questing tick densities and attachment rates/periods is needed to make quantitative predictions. There is the potential to model 2-D movement but the added complexity may not actually reveal anything new.

The work in this thesis takes the same modelling approach as Norman, Gilbert and colleagues, with some developments, to test the role of different management strategies in controlling LIV prevalence and tick numbers.

1.3 Aims, Approach and Thesis Layout

The aim of this thesis is to build upon previous published models of the LIV-grouse-tick-deer disease system to predict the effectiveness of various potential tick/virus management strategies. The importance of alternative routes of transmission and the seasonal biology of the system are also considered.

The models in this thesis extend from the non-linear coupled differential equations published previously, described in detail in Gilbert et al. (2001). The models are run in Mathematica, version 7, to give simulations and predictions for short and long term dynamics of the grouse and tick populations in different disease classes. The predicted equilibrium density of grouse and tick populations at varying host densities are also compared to assess when the virus may persist.

The equilibrium density predictions of grouse and ticks are used to assess the efficacy of management strategies at different host densities. If the grouse are predicted to survive at a higher economically viable density or for a greater range of host densities with the management strategy the strategy is deemed successful.

For each model presented sensitivity analysis is performed to determine the relative influence of different parameters and also first order interactions of potentially linked parameters. Where new parameter estimates are made the model predictions are explored within the plausible range to ascertain the effect of possible errors in the estimation. For non seasonal models algebraic analysis is also performed to ascertain whether the equilibrium is stable for given parameter sets. The details of these analyses are given in Appendix B

Chapter 1 provides background information on models of tick-borne diseases, the biology of the louping ill system and the rationale for this thesis.

Chapter 2 considers the inclusion of young grouse ingesting ticks as an additional route of virus transmission and assesses the impact of this on model predictions of grouse density and virus prevalence.

Chapter 3 explores the management strategy of using acaricide treated sheep as a form of tick control. The model predictions are used to assess how effective they might be at reducing virus prevalence and increasing grouse densities and the conditions under which they may or may not be expected to work. The chapter is presented as a published paper with additional material.

Chapter 4 discusses the incorporation of the seasonal biology of grouse in to the model, presented here in two different ways. The within year dynamics, long term behaviour and equilibrium densities of grouse and ticks at different deer densities are presented.

Chapter 5 considers the seasonal questing behaviour of ticks, treating them as either active in the summer or inactive in the winter. The within year dynamics, long term behaviour and equilibrium densities of grouse and ticks at different deer densities are presented

Chapter 6 explores the management strategy of treating wild deer with acaracide as a form of tick control. The model predictions are used to assess how effective they might be at reducing virus prevalence and increasing grouse densities and the conditions under which they may or may not be expected to work.

Chapter 7 looks at the management strategy of acaricidal leg bands attached directly to grouse hens. The model predictions are used to assess the potential effect leg bands may have on virus prevalence and grouse densities under different conditions.

Chapter 8 brings together a discussion of the models presented detailing their merits and limitations. Firstly the models that explore the effects of adding biological realism/complexity to the model are discussed. Secondly, the predictions of the models that consider potential management strategies are evaluated and possible improvements noted. A brief outline of future work that could be developed from that presented here is also given.

Appendix A provides a brief outline of the fieldwork undertaken to aid parameter estimation.

Appendix B contains all the models presented in this thesis.

Appendix C contains the analysis of the individual models.

Chapter 2

Modelling the role of grouse chicks ingesting ticks as a route of infection

2.1 Ingestion as a route of infection

It was originally assumed that louping ill virus (LIV) transmission was principally via bites from ticks that had become infected after feeding from an infected host or through non-viraemic transmission (see Sec. 1.2 for further details). Gilbert et al. (2004) discovered that a key route of infection for LIV in red grouse could be through the ingestion of infected ticks. It is known that the chicks of red grouse eat invertebrates during their first three weeks of life (Hudson, 1986a; Park et al., 2001). This can include ticks on the heather as well as those removed during preening, of themselves and siblings. The importance of this route of infection for red grouse was examined in Gilbert et al. (2004). The methods are explained briefly below and form the basis from which the incorporation of ingestion was developed in the model.

The feeding experiment of Gilbert et al. (2004) fed 8 red grouse chicks LIV infected suckling mouse brain, 8 chicks LIV infected ticks and 4 chicks uninfected ticks as a control. From this experiment it was estimated that the probability of a grouse becoming infected after consuming an infected tick is 0.109. To ascertain that LIV could be transmitted back to ticks after infection from ingesting infected material 4 of the 8 chicks that were fed infected ticks were infested with uninfected ticks, but 3 chicks had not become infected. The grouse that was successfully infected by consuming LIV infected ticks and infested with uninfected ticks transmitted the virus back to ticks. For simplicity and because we have no significant evidence to suggest otherwise we assume in the model that grouse infected by ingesting infected ticks behave in the same way as grouse infected by a tick bite; grouse to tick transmission is assumed to occur each time a susceptible tick bites an infected grouse.

Twenty-two wild red grouse chicks were captured and the number of ticks found per dropping was counted and compared to the number of ticks biting the grouse at the same time. To assess how many droppings were made per day chicks were kept in an enclosure for 24 hours and the number of droppings was counted. From the experiments of Gilbert et al. (2004) we are able to conclude that chicks consumed

an average of 5.4 ticks per day. (Unfortunately there is insufficient information to calculate the standard error.) The conclusion of this study was that up to 73% of LIV infections in red grouse in the first season could be due to ingestion.

A second approach in the same paper compared the proportion of infected grouse expected if the only route of infection was from tick bites with the actual proportion found to be infected from blood samples collected during the grouse shooting period. This comparison concluded that up to 98% of LIV infections in the first season of a red grouse could be due to ingestion.

These studies suggest that ingestion may be an important route of LIV infection for red grouse. Previous models (Gilbert et al. (2001); Laurenson et al. (2003); Norman et al. (2004)) have not included ingestion as a route of infection, assuming that all LIV infection in red grouse is due to tick bites. The addition of ingestion as an extra route of infection may be expected to increase the ability of LIV to persist in the red grouse and tick population at lower host and grouse densities. This chapter tests this hypothesis by modifying the model presented in Gilbert et al. (2001) and using the same parameter values to compare the model predictions for virus persistence at different grouse and tick host densities with and without ingestion added. The model is run over the scenarios presented in Gilbert et al. (2001); that is with deer, with mountain hares and with both deer and mountain hares. The deer and hare densities are chosen to reflect those found at the sites given in Gilbert et al. (2001); site C, with deer only and no mountain hares (Fig. 2.1); site M2, with hares only and no deer (Fig. 2.2); site P, with one deer per $km^2$and hares (Fig. 2.3a). The grouse and deer/hare densities at these sites are marked on the corresponding plots.

The grouse and tick densities predicted by this model are lower than those found on many grouse moors (Gilbert, unpublished data). The model is therefore re-parameterised to predict more realistic estimates of current grouse and tick densities. The effect of varying the new parameter value estimates is tested to ensure consistent model predictions. The most realistic model is then put forward.

2.2 The Ingestion Model

From the results and methods described in Gilbert et al. (2004) it was apparent that whilst all the ticks that are consumed by the grouse will die not all of them will infect the grouse, even if they themselves are infected. This meant that the rate at which the ticks infected the grouse via ingestion was not equal to the rate at which the grouse ate the ticks. This had to be incorporated into the model, given below. The new parameters are β_3, the rate at which grouse ingest ticks and P, the rate at which infected ticks infect grouse through ingestion. The new terms are boxed in the equation below.

$$
\begin{aligned}
\frac{dG_s}{dt} &= A + (a_g - s_g G)G - b_g G_s - \beta_1 T_i G_s \boxed{-P\beta_3 T_i G_s} \\
\frac{dG_i}{dt} &= \boxed{P\beta_3 T_i G_s} + \beta_1 T_i G_s - \Gamma G_i \\
\frac{dG_z}{dt} &= \gamma G_i - b_g G_z \\
\frac{dT_s}{dt} &= (a_t - s_t T)T(\beta_4 H + \beta_5 D) - b_t T_s - \beta_2 T_s G_i \boxed{-\beta_3 T_s G} \\
&\quad -(\beta_4 H + \beta_5 D)T_s - \theta T_s T_i H \\
\frac{dT_i}{dt} &= \theta T_s T_i H + \beta_2 T_s G_i \boxed{-\beta_3 T_i G} - b_t T_i - (\beta_4 H + \beta_5 D)T_i
\end{aligned}
$$

where $\Gamma = \alpha + b_g + \gamma$.

Many of the parameters values have been estimated from biological evidence. This was explained in detail in Gilbert et al. (2001) and summarised in Section 1.2 of this thesis. For ease of reference Table 2.1 gives the parameter definitions, values used and citations.

Param -eter	Value (unit)	Reasoning
A	1.94 (m^{-1})	Rate of grouse immigration (0 for site C)
a_g	0.167 (m^{-1})	Natural birth rate of grouse (Hudson, 1992)
s_g	$0.000\dot{3}$ $(g^{-1}m^{-1})$	Density dependence constraint on grouse. Section 2.3.1
b_g	0.087 (m^{-1})	Natural death rate of grouse (Hudson, 1992)
α	5 (m^{-1})	Disease induced death rate of grouse (Reid, 1975)
γ	1.25 (m^{-1})	Recovery rate of infected grouse (Reid, 1975)
a_t	83.33 (m^{-1})	Natural birth rate of ticks (Gilbert et al., 2001)
s_t	varied $(t^{-1}m^{-1})$	Density dependence constraint on ticks. Section 2.3.1
b_t	0.083 (m^{-1})	Natural death rate of ticks (Gilbert et al., 2001)
P	0.109	The proportion of infected ticks that infect a grouse when ingested (Gilbert et al., 2004)
β_1	varied $(g^{-1}m^{-1})$	The rate at which a tick bites and infects a grouse. Section 2.3.1
β_2	$9.75\beta_1 (g^{-1}m^{-1})$	The rate a tick bites a grouse and becomes infected (Gilbert et al., 2001)
β_3	$12\beta_1$ $(g^{-1}m^{-1})$	The rate ticks are ingested by grouse. Section 2.3.1
β_4	$1.84\beta_1$ $(H^{-1}m^{-1})$	The rate an adult female tick bites a mountain hare and reproduces (Gilbert et al., 2001)
β_5	$8.82\beta_1$ $(D^{-1}m^{-1})$	The rate an adult female tick bites a deer and reproduces (Gilbert et al., 2001)
θ	$3.9\text{x}10^{-7}$ $(H^{-1}m^{-1})$	The rate of non-viraemic transmission between co-feeding ticks on mountain hares

Table 2.1: The ingestion model parameters. For the units m denotes month, g grouse, t ticks, H hares and D deer.

2.2.1 The Reproductive Rate of the Virus, R_0

In order to find the threshold densities of grouse, deer and mountain hare above which the virus persists we consider the reproductive rate of the virus, R_0. If the value of R_0 is above 1 then the virus will persist, below 1 the virus will die out.

The R_0 equation for the virus can be obtained by considering the situation without virus where grouse and ticks are both at their carrying capacities, K_g and K_t, respectively, with the density of deer, D and hares, H. If we were to introduce one infected tick we would expect this tick to produce R_0 infections in ticks.

The equation for R_0 can be derived by considering the model equations. The infected tick is expected to live for $(b_t + \beta_3 K_g + \beta_4 H + \beta_5 D)^{-1}$ units of time and infect $(\beta_1 + P\beta_3)K_g(b_t + \beta_3 K_g + \beta_4 H + \beta_5 D)^{-1}$

grouse through tick bites and ingestion of ticks. In turn each of these grouse live for Γ^{-1} units of time and will infect $(\beta_2 K_t)\Gamma^{-1}$ ticks. The tick would also infect $(\theta K_t H)(b_t + \beta_3 K_g + \beta_4 H + \beta_5 D)^{-1}$ other ticks through non-viraemic transmission. Combining these together gives

$$R_0 = \frac{\beta_2(\beta_1 + P\beta_3)K_g K_t + \Gamma\theta K_t H}{\Gamma(b_t + \beta_3 K_g + \beta_4 H + \beta_5 D)}$$

with

$$K_t = \frac{(a_t - 1)(\beta_4 H + \beta_5 D) - b_t - \beta_3 K_g}{s_t(\beta_4 H + \beta_5 D)}$$

and

$$K_g = \frac{a_g - b_g}{s_g}.$$

R_0 can also be derived from the stability conditions required for the system to attain equilibrium. This is explained in detail in Appendix C.

Drawing the curve described by $R_0 = 1$ allows us to see the combinations of host densities that are expected to allow virus persistence and those that are not. The $R_0 = 1$ curve describes the expected cut off point for virus persistence; above the curve virus persists, below it does not. A comparison of the curve for the model simulations run with and without ingestion will show any differences in the threshold densities of grouse and deer (or hares) caused by the addition of ingestion. (Eg. Fig. 2.1)

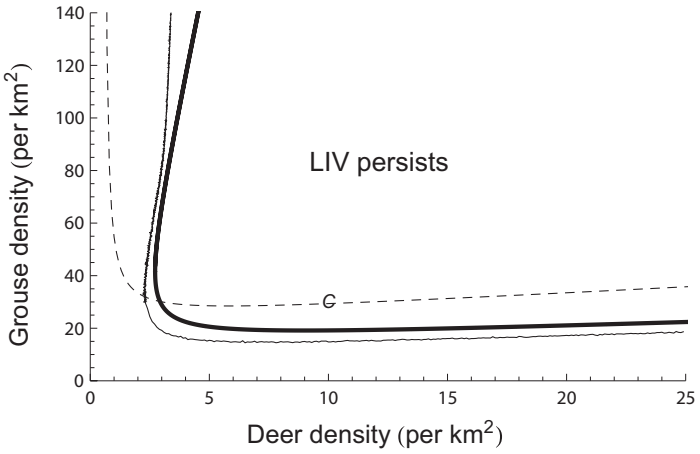


Figure 2.1: The effect of ingestion on the predicted area of pathogen persistence with grouse and deer only. The thick solid line represents $R_0 = 1$ with ingestion. The thin solid line represents the pathogen persistence threshold with ingestion. The dashed line represents $R_0 = 1$ without ingestion.

Interestingly, the addition of ingestion to the model has lead to a positive feedback mechanism within the virus dynamics which the traditional form of R_0 is unable to capture. When ingestion of ticks by red grouse is included in the model as a route of infection this has the dual effect of i) decreasing the susceptible population of red grouse due to increasing infection transmission and ii) increasing the tick population by reducing the number of grouse that can consume them. The increase in tick numbers increases the chances of the remaining grouse becoming infected which in turn decreases the grouse population further and enables the tick population to increase further. This positive feedback mechanism is not captured by the existing formula for R_0. The disease free equilibrium densities for grouse and ticks, K_g and K_t respectively, are used in the calculation of R_0 which do not take into account the extra

dynamics caused by ingestion. However, if the model predictions for the grouse and tick densities at disease induced equilibrium (for different deer densities) are used in the equation for $R_0 = 1$ instead of K_g and K_t then a second curve is obtained (Thick solid line, Fig. 2.1). This curve matches the model predictions for when virus does and does not persist. This is determined by assessing whether or not the model predictions for the grouse population at equilibrium has reached carrying capacity; the virus does not persist if the grouse reach carrying capacity and does persist if the grouse do not reach carrying capacity. The curve joining the points at which the grouse are first predicted to no longer reach each carrying capacity will be referred to as the pathogen persistence threshold (PPT) and is distinct from the conventional $R_0 = 1$ curve. (It should be noted that the $R_0 = 1$ and PPT curves are identical when ingestion is not included in the model as the feedback mechanism is not present.)

2.3 Model predictions

The effect of adding ingestion to the model was first considered by adding ingestion to the model presented in Gilbert et al. (2001). A discussion of how this parameter was chosen is given in Sec. 2.3.1. All other parameters values were kept the same. The following plots show the $R_0 = 1$ curve with and without ingestion, as well as the PPT, denoted by a thick solid line, dashed line and thin solid line respectively.

For each scenario the $R_0 = 1$ curve is compared for the model run with and without ingestion. The PPT curve is also compared to the $R_0 = 1$ curve to determine the level of infection that occurs in model predictions but that is not captured by R_0.

Disease persistence with deer and grouse only Fig. 2.1 shows the effect of ingestion, comparing the $R_0 = 1$ curve without ingestion (dashed) to the PPT curve (thin solid), when the only tick hosts in addition to grouse are deer. At low deer densities (< 2 per km^2) the addition of ingestion shows the virus is predicted not to persist. In contrast, LIV is predicted to persist without ingestion. This may seem counterintuitive as ingestion is another route of infection but the ingestion of ticks is also a mechanism for reducing the tick population. When the deer densities are low the tick population is of a sufficiently small size for the grouse to consume a high enough proportion of the tick population to reduce the tick density below that necessary for virus persistence.

At higher deer densities (> 2 per km^2) the model predicts that virus persists for lower grouse carrying capacities when ingestion is included compared to when ingestion is not included. At deer densities above 2 per km^2 the associated tick density is too high for the consumption of ticks by grouse to have a significant impact on the tick population size. Instead the ingestion of ticks becomes an important additional route of infection causing virus to be predicted to persist when it is predicted not to without ingestion.

Gilbert et al. (2001) discusses the apparent 'dilution effect' occurring for high deer densities. The model presented in Laurenson et al. (2003) predicts that as well as a lower limit of deer per km^2 below which the virus does not persist there is also an upper limit of deer per km^2 above which the virus does not persist. This is due to the number of 'wasted' tick bites on deer: deer are incompetent hosts for virus transmission so bites from infected ticks on deer are wasted because the virus is lost from the system.

If the number of tick bites on deer that are loosing virus from the system is too high the virus cannot persist as there is no longer a large enough reservoir of infected ticks to transmit the virus to the grouse. Consequently, although the deer enhance the tick population high numbers of deer actually make it more difficult to maintain the virus in the system. The model with ingestion still displays the dilution effect at very high deer densities although this is less apparent in Fig. 2.1, restricted to common deer densities.

Fig. 2.1 shows however that the ingestion model has an upper as well as lower threshold for virus persistence for the grouse carrying capacity. At low grouse carrying capacities (< 18 per km^2) the virus cannot persist as there are too few grouse to maintain the virus. For certain deer densities, the virus is also predicted not to persist at higher grouse densities. For example, considering the PPT curve, if there are 3 deer per km^2 then the virus is predicted not to persist for grouse carrying capacities below approx. 18 per km^2. The virus is also predicted not to persist for grouse carrying capacities above approx. 60 per km^2. This is not only dilution but the effect of the grouse consuming ticks and reducing the tick population below that necessary for virus persistence at high grouse densities.

The PPT closely follows the $R_0 = 1$ curve with ingestion for most grouse densities. However, for high grouse carrying capacities the virus is predicted to persist above approx. 3.5 deer per km^2 regardless of grouse density although $R_0 < 1$. This could reflect the contribution to the rate of infection made by the ingestion of ticks which is not taken account of by the $R_0 = 1$ curve.

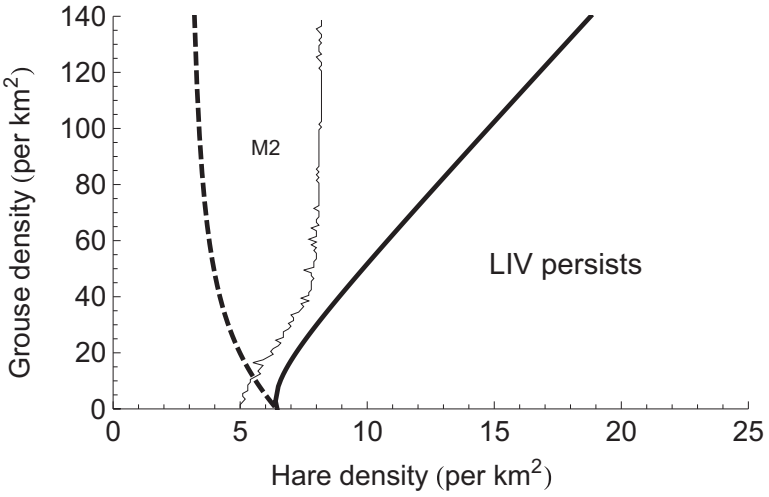
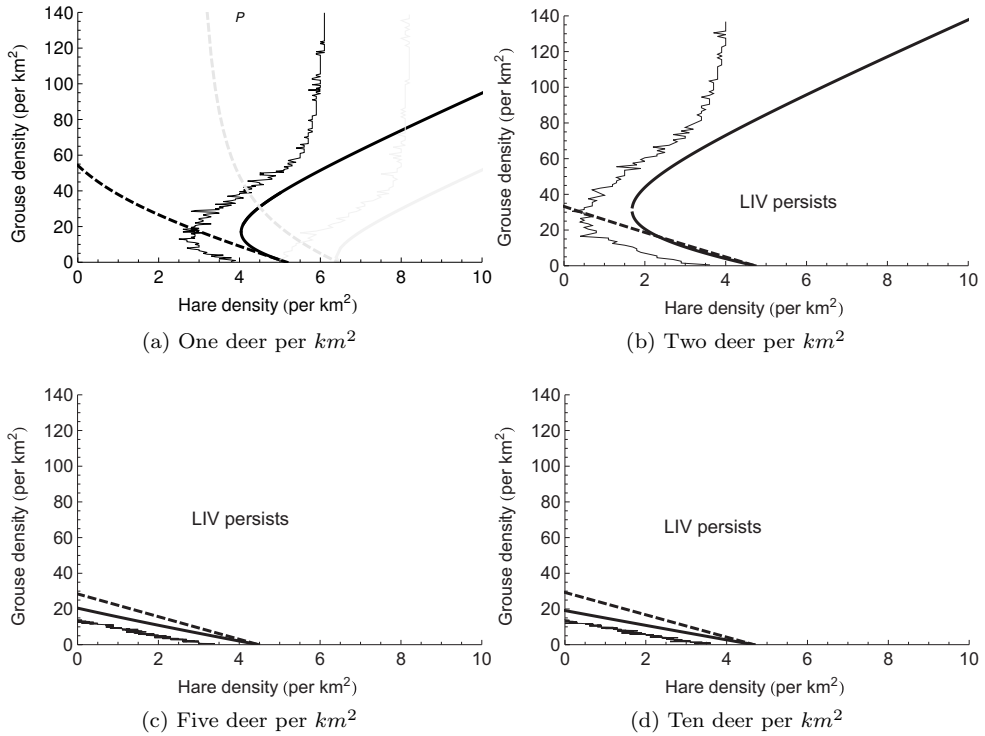


Figure 2.2: The effect of ingestion on the predicted pathogen persistence with grouse and mountain hares only. The thick solid line represents $R_0 = 1$ with ingestion. The thin solid line represents the pathogen persistence threshold with ingestion. The dashed line represents $R_0 = 1$ without ingestion.

Disease persistence with mountain hares and grouse only Fig. 2.2 represents a situation with no deer, but mountain hares and consequently non-viraemic transmission. With these parameter values ingestion has a dramatic effect on the position of the $R_0 = 1$ curve: there is a large area of parameter space in which the virus will persist when ingestion is not included (dashed line) in the model but will not persist when ingestion is included in the model (thick solid line). i.e. For all non zero grouse densities a larger hare density is required for LIV persistence when ingestion is included in the model. This could be explained by the fact that, with ingestion, grouse eat a significant proportion of the tick population such that there are insufficient ticks to sustain the virus. More hares are therefore required as additional

competent tick hosts. The PPT curve shows more parameter space where the virus persists than the $R_0 = 1$ curve suggests for the ingestion model. The PPT curve indicates there may be a threshold for mountain hares (approx. 7.5 per km^2) above which the virus persists irrespective of grouse carrying capacity. Ingestion is a significant route of infection but loss of ticks through ingestion has limited effect on the tick population. For very low grouse carrying capacity (< 10 grouse per km^2) the PPT curve shows the virus persists with ingestion included in the model but not without. At these low grouse densities the grouse are not at sufficiently high densities to impact on the tick population and ingestion is a significant route of infection. Although such low grouse densities are not viable on a working grouse moor it is interesting to note this behaviour from a theoretical point of view.

The model prediction of fewer, rather than more, host combinations with virus persisting when ingestion is included in the model is surprising. The hypothesis presented in Sec. 2.1 proposed the opposite, that ingestion would increase the area of virus persistence. It is interesting that Fig. 2.2 suggests for these model parameters the grouse are able to consume such a proportion of the tick population as to be able to reduce it below the density required for virus persistence. This may be unrealistic and further suggests the need to re-estimate some of the parameters. If the parameter values are adjusted to allow a larger (and perhaps more realistic) tick density the proportion of the tick population consumed by the grouse should have less of an effect on the tick population dynamics.



(a) One deer per km^2 (b) Two deer per km^2

(c) Five deer per km^2 (d) Ten deer per km^2

Figure 2.3: The effect of ingestion on the predicted pathogen persistence at different deer densities with mountain hares and grouse. The thick solid line represents $R_0 = 1$ with ingestion. The thin solid line represents the pathogen persistence threshold with ingestion. The dashed line represents $R_0 = 1$ without ingestion. (Note that for comparison Fig. 2.2 has been added as faint curves to Fig. 2.3a)

Disease persistence with mountain hares, deer and grouse. Fig. 2.3 shows the effect of including different deer densities in the model with grouse and mountain hares. Comparing Fig. 2.2 with Fig. 2.3a

shows the inclusion of one deer per km^2 (as in site P of Gilbert et al. (2001)) has the effect of reducing the predicted number of hares needed to allow the virus to persist (Fig. 2.3a). This is because the deer now hosts a proportion of the tick population so fewer hares are needed to sustain them. The $R_0 = 1$ (thick solid) and PPT (thin solid) curves with ingestion included have now also changed shape compared to Fig. 2.2 at low grouse densities so that they bend back on themselves. This gives for particular mountain hare densities, an upper and lower limit of grouse densities between which the virus can persist. For example, Fig. 2.3a shows (for the PPT curve) when there are 3.5 hares per km^2 the virus is predicted to persist for grouse numbers between approx. 5 and 30 per km^2. Below 5 grouse per km^2 there are too few grouse for LIV persistence. Above 30 grouse per km^2 the number of ticks being eaten is large enough to reduce the tick population to the point where the virus cannot persist. However, between 5 and 30 grouse per km^2 the density of grouse is such that the number of ticks eaten is enough to cause the virus to persist in the grouse population but not sufficient to reduce the tick population to the point where it cannot sustain the virus. (This was apparent to a lesser extent in Fig. 2.1 for some deer densities.)

Fig. 2.2 showed a small area where the virus was predicted to persist with ingestion but not without ingestion. Fig. 2.3a shows a similar small area for grouse densities below approximately 20 per $km^2$and hare densities between approximately 3 and 5 per km^2 where the virus is predicted to persist when ingestion is included but not without ingestion (comparing PPT with $R_0 = 1$ for no ingestion). A similar pattern of behaviour occurs for deer densities of 2 per km^2(shown in Fig. 2.3b) and 3 per km^2(not shown).

With higher deer densities the combination of host densities for which virus is predicted to persist increases dramatically. The model predicts the same qualitative behaviour for all reasonable deer densities above 4 per km^2 (not shown). Figs. 2.3c, 2.3d show the area of virus persistence for 5 and 10 deer per km^2 respectively. The $R_0 = 1$ curves with ingestion are slightly lower than those without, suggesting that ingestion is primarily acting as an extra route of infection and causing the virus to persist when it would not without ingestion. The PPT line is lower still indicating that the formula for R_0 is consistently underestimating the area of virus persistence. However, for all reasonable deer densities the R_0 and PPT curves show the same pattern of behaviour and differences between numerical predictions are small. For deer densities greater than 4 per km^2 the virus is predicted to persist for almost all grouse and hare densities and the effect of ingestion is negligible in comparison to the effect of the increased deer density. The virus is predicted not to persist only for very low densities of grouse and mountain hares and will persist otherwise. Deer carry large tick burdens and are important reproduction hosts (Gray, 1998). The presence of deer allows the ticks to reproduce more readily and a larger tick population will increase the opportunity for virus transmission. At these deer and tick densities the ingestion of ticks by grouse has very little effect on the tick population and is insignificant compared to the impact of increasing the deer density.

The effect of ingestion

The hypothesis made in Sec. 2.1 was that including ingestion as a route of infection in the model of Gilbert et al. (2001) would increase the ability of the virus to persist so that there would be a larger area of parameter space in which the virus is predicted to persist. Adding ingestion has not had the simple amplifying effect that was expected; the impact of ingestion on the tick population had not been considered. Although for some deer and hare densities the inclusion of ingestion in the model does predict that the virus persists when it had not without ingestion there are many deer and hare densities for which the inclusion of ingestion actually predicts the virus does not persist when it does without ingestion. The ingestion of ticks by grouse is not purely an extra method of virus transmission but it also reduces the tick population. The magnitude of the effect that the ingestion of ticks has on the tick population may be unrealistically large. It seems unlikely that grouse could decimate the tick population to the extent predicted by this model, suggesting that the model needs to be re-parameterised. The fieldwork outlined in App. A suggests that the tick population on a typical grouse moor is much larger than predicted here. The model could therefore be improved by increasing the predicted tick population.

2.3.1 Re-parameterising the ingestion model

The parameters used in Sec. 2.3 are from Gilbert et al. (2001) and predict low densities of ticks (in the tens of thousands per km^2) but the number of ticks on a grouse moor may be considerably higher (in the millions per km^2, Appendix A). If the model is re-parameterised to predict a higher tick density the effect of ingestion on the tick population should not be as dramatic, as a lower proportion of the whole tick population will be eaten by the grouse. The prediction of a larger tick population should allow the grouse to become infected by this route without unrealistically reducing the tick population. This requires s_t, the density dependence of ticks and β_1, the rate at which nymphs bite grouse to be re-estimated.

A, the rate of immigration will also be removed from the equations. The purpose of the ingestion model is to gain a better understanding of the impact that ingestion of ticks by grouse may have on the virus dynamics. It is recognised by Gilbert et al. (2001) that immigration does not occur for all sites, although it may be likely for areas with LIV. The value for A given in Gilbert et al. (2001) is calculated using model estimations and assumed the same for those sites where immigration is thought to occur. Immigration does not always occur and there is no biological data on which to base an estimate. To avoid error from another unknown parameter we have chosen to remove immigration from the model.

The density dependence of ticks, s_t The density dependence of ticks s_t is incorporated into the reproductive function for the tick population, although in actual systems it may be that the density dependence occurs in the development of one life stage to another. It is not possible to incorporate density dependence in this way as the tick stages are not modelled separately. However, we believe that having the density dependence of ticks as part of the reproductive function is sufficient to capture the dynamics of the tick population. However there is no empirical evidence for the value of this parameter, consequently the model is used to assess which value of s_t gives the most biologically plausible estimates

for the grouse and tick densities. (Realistic predictions reflecting known densities of breeding grouse on estates with LIV would be in the region of 20-25 (Gilbert et al., 2001) grouse and millions of ticks per km^2 (App. A)). The model predictions for different values of s_t will be compared to assess the robustness of the model to different choices to ensure that the pattern of behaviour predicted is not a quirk of a particular set of parameter values. We can then be confident that the overall pattern of prediction is likely to be correct even if the particular quantitative predictions are not accurate.

The transmission parameter, β_1 The rate at which a grouse is bitten and infected by a tick β_1 can only involve the nymphal stage as adults do not generally bite grouse and the larvae are not infected. The rate at which nymphs bite grouse was estimated in Gilbert et al. (2001) from tick counts of nymphs on deer and grouse as well as from model predictions. However, these counts were performed in 1998 on one site (C). Although it has been assumed in all published models that the ratio of tick bites on grouse to tick bites on deer will remain constant from site to site there is no evidence that this is the case. On the contrary tick counts on different mammals at different sites given in Laurenson et al. (2003) show the ratios to be different. Kirby et al. (2004) argue that the tick burden on red grouse chicks is rising based on counts from 13 estates in Scotland. It may be that the estimate of β_1 is now out of date. As we do not have recent data on the number of tick bites on red grouse and deer the model will be used to estimate β_1 and the robustness of the model to the value of β_1 will be assessed in the same way as for s_t outlined above.

Including mountain hares in the model also requires the inclusion of non viraemic transmission (NVT). Although it is known that non-viraemic transmission occurs (Jones et al., 1997) there is insufficient data to form a biologically based estimate of the rate at which it occurs. Consequently the value of the parameter θ that reflects the rate of NVT in the model was estimated in Gilbert et al. (2001) by using the model to ensure biologically realistic predictions. However, as stated above the predicted tick density using the parameter values of Gilbert et al. (2001) does not reflect a typical grouse moor and so the value for θ would need to be recalculated. To avoid the complications of having a model with several unknown variables mountain hares and NVT have been removed.

The Ingestion Parameter

The studies of Gilbert et al. (2004) indicate that a high proportion of LIV infections in grouse during their first season may stem from the grouse ingesting the ticks. The first method of the study suggested that on average grouse chicks eat approximately 5.4 ticks per day, but are only bitten by 0.051 ticks per day. So on average a chick is 107 times more likely to eat a tick than be bitten by one. It is assumed that the ratio of tick bites to ticks ingested is constant for all grouse and tick densities. However, this only occurs for the first three weeks of life, so taking this into account the rate at which grouse ingest ticks given by β_3 is $6\beta_1$ ($3/52 \times 107 \approx 6$). The model assumes that all grouse, rather than just chicks, are eating ticks for three weeks each year. This is not accurate, as some grouse are adults, but calculating the proportion of the grouse population that are chicks is non trivial. This issue is investigated further

in Chapter 4.

Using an alternative method Gilbert et al. (2004) conclude that up to 98% of infections in the first season could be from ingestion. If this is the case then throughout the first season a grouse is 49 times more likely to get infected through ingestion than by being bitten. However, the first season (June-Aug) lasts for only $\frac{90}{365}$ of the year and the proportion of grouse infected having consumed an infected tick is 0.109. From this the upper limit for β_3 is $111\beta_1$ ($49 \times \frac{90}{365} \div 0.109 \approx 111$).

This estimate seems unfeasibly large and it is noted in Gilbert et al. (2004) that their estimate may be conservatively high due to their tick counts being performed in June when the tick count is probably highest. Looking at how the model runs with various estimates shows that for any value of β_3 above $65\beta_1$ the grouse reach their carrying capacity of 80 per km^2 and the tick population is unrealistically reduced (see Table 2.2).

Both methods give a percentage of infections thought to be due to ingestion, with the lower one being 73%. If we use 84% the midpoint of this and the upper limit of 98%, then on average a grouse is 5.25 times more likely to get infected through ingestion than by being bitten. Using this and arguing as above (i.e. $5.25 \times \frac{90}{365} \div 0.109$), we get $\beta_3 = 12\beta_1$. This does not have a much more dramatic effect than the lower estimate of $6\beta_1$. (See Table 2.2).

The effect of ingestion in the model can only be established when the rest of the model parameters are constant. To determine the impact of ingestion and compare different choices for the ingestion parameter particular values for s_t and β_1 need to be chosen. Keeping all other parameters the same and running numerical simulations on Mathematica suggest that $\beta_1 = 0.00002$ and $\beta_1 = 0.00003$ give reasonable predictions of tick densities 17 million and 25 million respectively ($s_t = 0.000002$). The density dependence parameters were chosen to give a reasonable carrying capacity for ticks in the absence of grouse and disease. For ticks to give a predicted carrying capacity in the millions per km^2 $s_t = 0.000002$. The actual predicted tick population depends on host density. Consequently $s_t = 0.00002$ and $\beta_1 = 0.00003$ have been used in the model to compare the effect of different ingestion parameters within this section. However, further investigation into the effect of s_t and β_1 on the area of virus persistence is described in Sec. 2.3.1.

β_3	R_0	G^*	T^*
0	1.19	28.83	2.55 million
$5\beta_1$	1.23	20.40	2.49 million
$6\beta_1$	1.23	19.28	2.48 million
$7\beta_1$	1.23	18.29	2.48 million
$8\beta_1$	1.24	17.39	2.47 million
$9\beta_1$	1.24	16.58	2.46 million
$10\beta_1$	1.25	15.84	2.46 million
$11\beta_1$	1.25	15.16	2.45 million
$12\beta_1$	1.25	14.54	2.45 million
$13\beta_1$	1.26	13.97	2.44 million
$14\beta_1$	1.26	13.45	2.44 million
$15\beta_1$	1.26	12.96	2.44 million

β_3	R_0	G^*	T^*
$20\beta_1$	1.27	10.97	2.42 million
$30\beta_1$	1.28	8.41	2.41 million
$40\beta_1$	1.29	6.82	2.39 million
$50\beta_1$	1.30	5.73	2.39 million
$60\beta_1$	1.30	4.95	2.38 million
$65\beta_1$	0.00006	80	16.31
$70\beta_1$	1.6×10^{-7}	80	0.04

Table 2.2: The effect of changing β_3 on model predictions with 10 deer per km^2 ($\beta_1 = 0.00003$, $s_t = 0.00002$)

The possibility of using an estimate for β_3 based on the proportion of ticks eaten each day was considered. The term, $\beta_3 T_i G_s$ in the equations would become $NT_i G_s$, where N is the proportion of the tick population consumed by the grouse. However, although it is known approximately how many ticks grouse chicks ate during the experiment in Gilbert et al. (2004) the number of ticks that were available to them remains unknown. Let n be the total tick population available during the experiment. Approximately 5 ticks are eaten per day, so $N = \frac{5}{n} \times \frac{21}{365}$. (As mentioned previously the chicks only eat ticks 3 weeks, or 21 days, each year.) The results of varying N by powers of ten to account for the possible variation in the tick population size are shown in Table 2.3. Varying N, the proportion of ticks consumed, by powers of ten does not have a great effect on the model predictions. Although it may seem reasonable to assume that grouse consume a proportion of the tick population there is no evidence to direct a choice for this proportion and varying the proportion has little impact on model predictions. Consequently I have chosen to reject this as a possible form for the ingestion parameter and instead used $\beta_3 = 12\beta_1$ as my estimate because this is based on the empirical evidence of Gilbert et al. (2004)

N	R_0	K_g	K_t
$3. \times 10^{-7}$	1.19	28.81	2.55 million
$3. \times 10^{-6}$	1.12	28.59	2.55 million
0.00003	1.20	26.61	2.53 million
0.0003	1.25	15.84	2.461 million
0.003	0	80	0

Table 2.3: Changing the proportion of ticks ingested by grouse, N, (with 10 deer per km^2)

Varying the ingestion parameter, β_3 The effect of varying β_3, the number of ticks consumed by grouse, on LIV persistence is tested by considering the area in parameter space of pathogen persistence. With the new parameter values for s_t and β_1 we expect that the area of LIV persistence will increase as β_3 increases because ingestion is an extra route of infection but now the effect on the tick population will be small.

The effect of changing β_3 on the area of pathogen persistence is shown in Figure 2.4. (For ease of reading I have only included the $R_0 = 1$ curves in this plot. As will be shown in Sec. 2.3.1 the PPT curves follow the same pattern as the $R_0 = 1$ curves, but give a greater area of pathogen persistence.)As predicted increasing the rate of ingestion increases the area of parameter space in which LIV persists. However, whilst the virus is predicted to persist more readily when ingestion is included for low grouse densities (< 50 per km^2) the opposite is true for higher grouse densities (> 50 per km^2). At high grouse and low deer densities the tick population is low enough that the grouse ingesting ticks can have a detrimental effect on the size of the tick population, leaving too few ticks for virus persistence. At these high grouse densities as the rate of ingestion increases more deer are required for the tick population to increase enough for virus persistence.

The rate of ingestion is assumed (in the model) to be related to the rate of virus transmission, i.e. the rate at which nymphs bite grouse. The effect of ingestion will therefore alter as β_1, the transmission parameter, alters. One would expect that decreasing β_1 would decrease the area of virus persistence as a

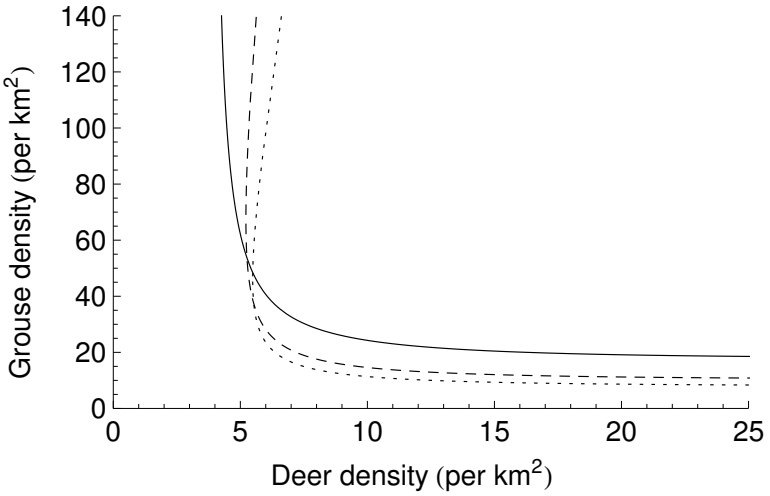
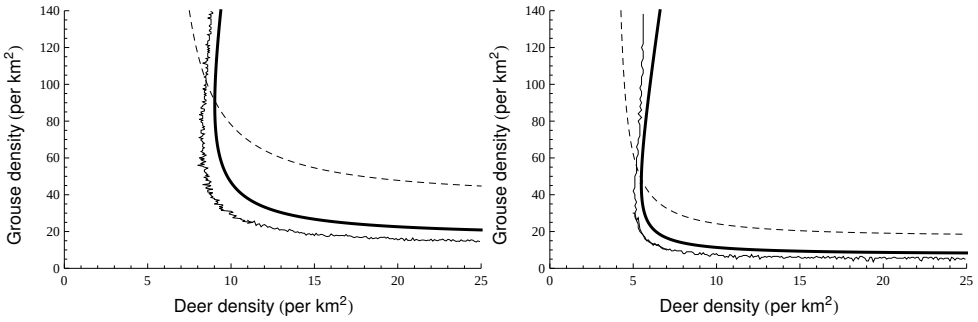


Figure 2.4: The effect of changing the β_3 the rate of ingestion ($\beta_1 = 0.00003$) on the $R_0 = 1$ curve, without ingestion(solid line), with ingestion at rate $\beta_3 = 7\beta_1$ (dashed line) and $\beta_3 = 12\beta_1$ (dotted line).

smaller rate of transmission makes it harder for the virus to persist, however the general trend of model behaviour for different grouse and deer densities should remain. To ensure that the general pattern of predictions remains consistent I have plotted the areas of virus persistence with and without ingestion for the two 'reasonable' values of β_1 (Sec. 2.3.1).



(a) $\beta_1 = 0.00002$ (b) $\beta_1 = 0.00003$

Figure 2.5: The effect of ingestion on LIV persistence, with different transmission rates. The thick solid line represents $R_0 = 1$ with ingestion. The thin solid line represents the PPT with ingestion. The dashed line represents $R_0 = 1$ without ingestion. ($s_t = 0.000002$, $\beta_3 = 12\beta_1$)

Comparing Fig. 2.5a and 2.5b shows that both values of β_1 display the same general qualitative behaviour as the model using the parameter estimates of Gilbert et al. (2001) but the numerical estimates for the densities of grouse and deer are different. In all cases the virus persists over a greater range of grouse densities when ingestion is included. This is because it is now easier for the grouse to become infected with the inclusion of an extra route of infection via the oral route. However, in each case there is also a point where the model predicts more deer are needed to sustain the tick population when ingestion is introduced because ingestion by grouse reduces the tick population. The point at which this occurs varies depending on the choice of β_1. (The predicted PPT curves suggest this is approx. 9 deer and 105 grouse when $\beta_1 = 0.00002$ and approx. 5.5 deer and 55 grouse per km^2 when $\beta_1 = 0.00003$.)

The dilution effect no longer occurs for reasonable deer densities. Although Fig. 2.5b indicates there is a very small range of deer densities (between approx. 5 and 6 deer per km^2 in this case) where there

exists an upper and lower limit for the grouse carrying capacity. As discussed in Sec. 2.3 this could be explained by the fact that below the lower limit there are too few grouse for virus persistence and above the higher limit the grouse density is sufficient to reduce the tick population so there are not enough ticks for virus persistence.

As predicted the smaller value of β_1 (0.00002) decreases the area above the curve for which the virus persists and the larger value (0.00003) increases it. A lower value of β_1 reduces the rate at which infected nymphs bite grouse so it is harder for the virus to persist.

The density dependence of grouse The carrying capacity of grouse has been set at 80 grouse per km^2 following Laurenson et al. (2003). It became apparent that in general this carrying capacity for grouse was too low. The carrying capacity of grouse is given by

$$K_g = \frac{a_g - b_g}{s_g}.$$

A grouse carrying capacity of 80 grouse per km^2 gives an estimate of 0.001 for s_g. Although empirical evidence suggests that maximum grouse counts vary from site to site a carrying capacity of 240 grouse per km^2 is not unreasonable for summer densities (Laurenson et al. (2003), Gilbert, unpublished data). This gives the estimate $s_g = 0.000\dot{3}$ which will be used from now on.

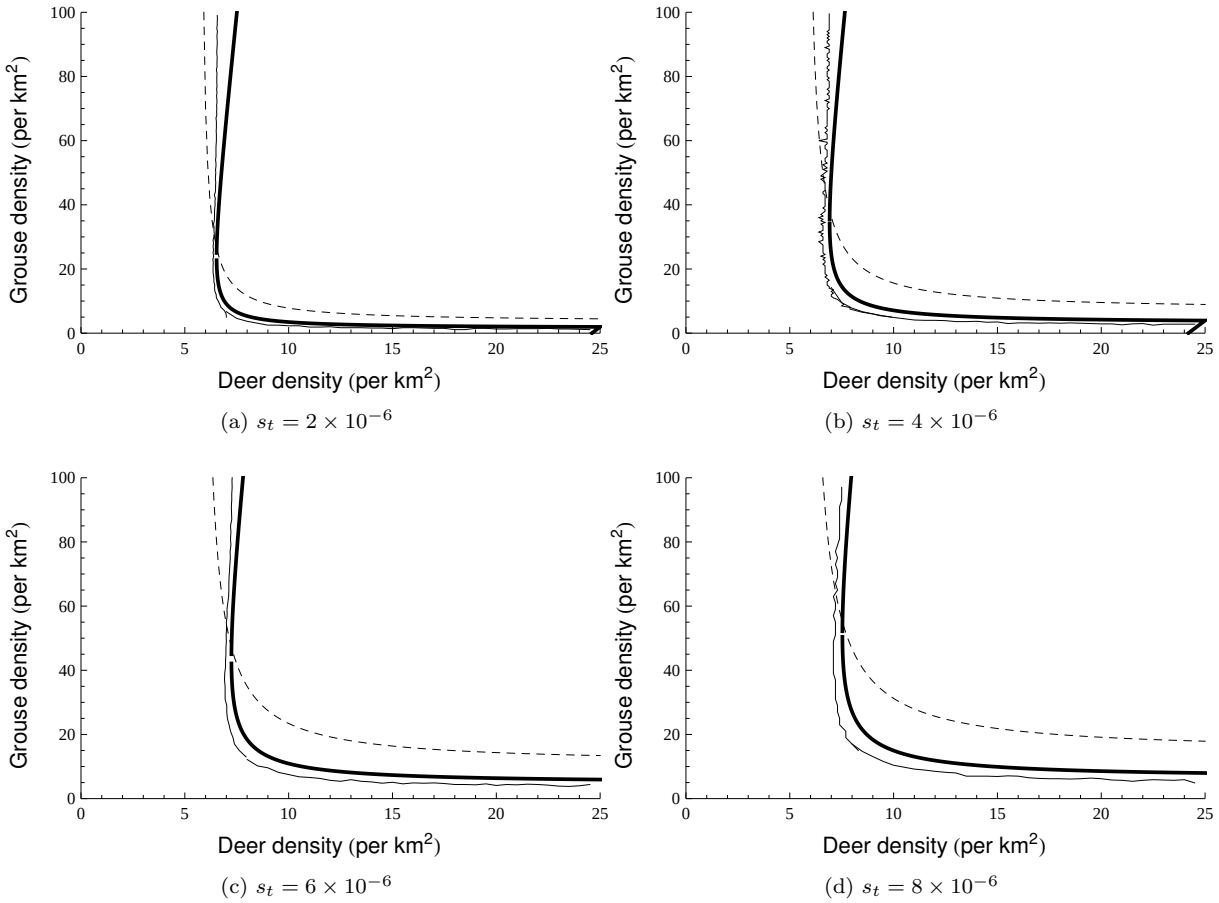
Varying the Density Dependence of Ticks, s_t

The model was used to estimate s_t the density dependence of ticks by systematically varying the density dependence and using the value that predicts the most realistic grouse and deer densities. As there is no empirical evidence for s_t it is important to establish that the model predictions are not overly sensitive to variation in s_t. If varying s_t were to have a disproportionate effect on the predictions and qualitative predictions were a quirk of those parameter choices it would be difficult to have confidence in the model predictions.

Changing the density dependence of ticks is not a simple matter as it depends on the tick reproduction host densities (as well as grouse densities when ingestion is included in the model). There are no definitive estimates of the actual densities of ticks found in the field. I therefore undertook the fieldwork outlined in App. A to gain a better understanding of the number of ticks that may be found on a grouse moor. The experiment suggests that although the tick densities on grouse moors vary hugely they could regularly be expected to be in the tens of millions per km^2.

Varying s_t in the model has a dramatic effect on the predicted grouse densities as well as the tick densities. To keep the grouse densities with infection (and ingestion) reasonable (20-25 per km^2) I also varied β_1. By altering both β_1 and s_t there are a few options which give reasonable numbers of both grouse and ticks.

Fig. 2.6 shows the effect of increasing the density dependence of ticks on predicted pathogen persistence. As can be seen increasing s_t decreases the area of virus persistence by increasing the grouse carrying capacity and deer density needed for LIV persistence. Increasing the density dependence of the



(a) $s_t = 2 \times 10^{-6}$ (b) $s_t = 4 \times 10^{-6}$

(c) $s_t = 6 \times 10^{-6}$ (d) $s_t = 8 \times 10^{-6}$

Figure 2.6: The effect of changing s_t when $\beta_1 = 0.00002$. The thick solid line represents $R_0 = 1$ with ingestion. The thin solid line represents the pathogen persistence threshold with ingestion. The dashed line represents $R_0 = 1$ without ingestion.

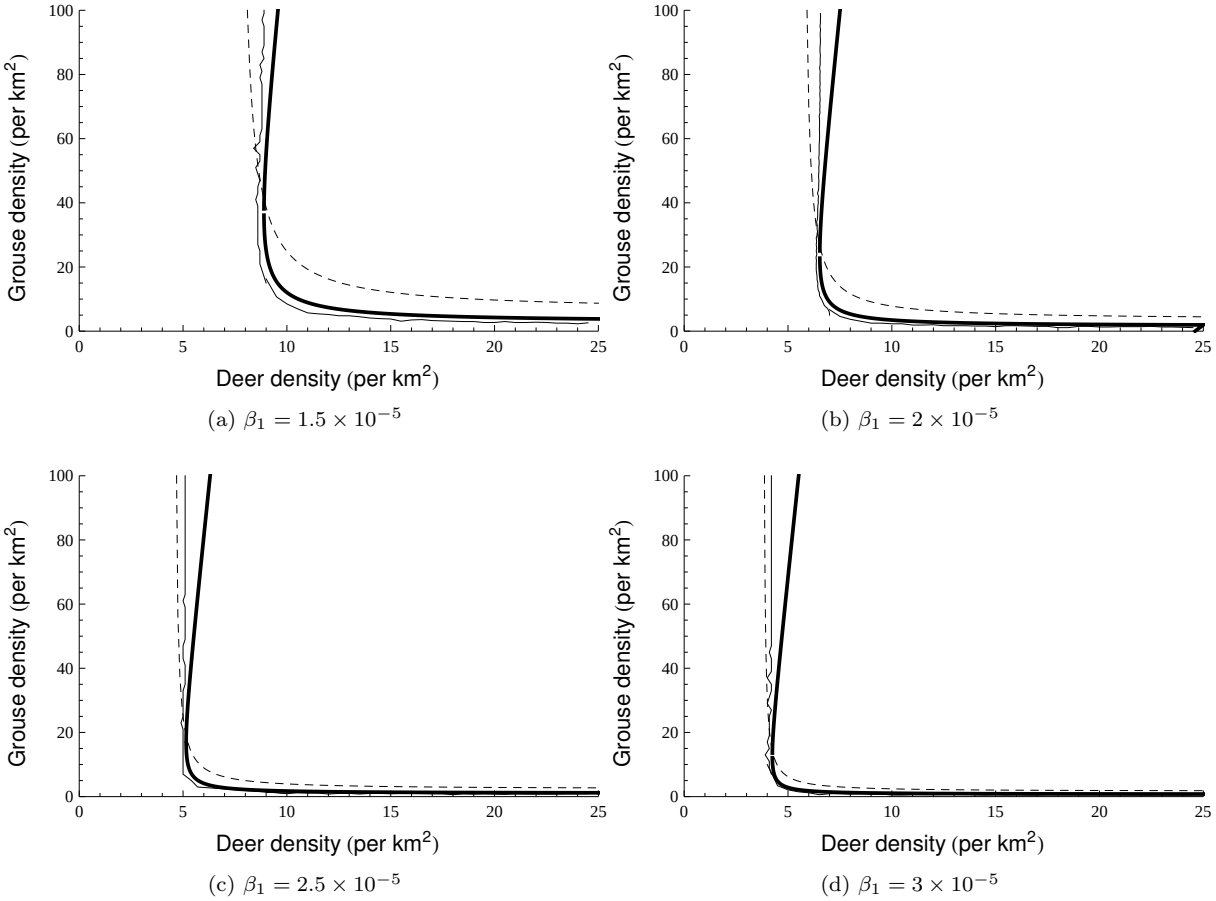
ticks decreases the size of the predicted tick population. With a smaller tick population the chance of a tick being able to find and infect a grouse is reduced and the virus will not be able to spread so readily. Consequently larger densities of grouse and deer are required to provide the opportunity for enough virus transmission to allow the virus to persist.

Increasing s_t also increases the impact of including ingestion in the model. With a greater degree of density dependence the predicted tick population will not be as large so the impact of the grouse ingesting them will be greater. However, the overall pattern of behaviour is the same for all values of s_t. Including ingestion in the model increases the area of pathogen persistence. For high deer densities ingestion increases the ability of LIV to persist but for certain lower deer densities including ingestion allows the grouse to consume enough ticks to reduce the tick population sufficiently to prevent virus persistence. The predicted grouse and deer densities where this change occurs varies depending on s_t.

Varying the Transmission Parameter, β_1

The rate at which nymphs bite grouse and infect them was also chosen using model predictions so the effect of varying β_1 also needs to be considered.

Fig. 2.7 considers the effect on pathogen persistence of changing β_1 whilst keeping s_t constant ($s_t = 2 \times 10^{-6}$). The effect of increasing β_1 is to increase the area in parameter space where virus is predicted



(a) $\beta_1 = 1.5 \times 10^{-5}$ (b) $\beta_1 = 2 \times 10^{-5}$

(c) $\beta_1 = 2.5 \times 10^{-5}$ (d) $\beta_1 = 3 \times 10^{-5}$

Figure 2.7: The effect of changing β_1 when $s_t = 2 \times 10^{-6}$. The thick solid line represents $R_0 = 1$ with ingestion. The thin solid line represents the pathogen persistence threshold with ingestion. The dashed line represents $R_0 = 1$ without ingestion.

to persist by reducing the grouse carrying capacity and deer density necessary for LIV persistence. The effect is greatest for the deer density so that with a greater transmission rate the virus will persist at much lower deer densities. When the chance of a tick finding and infecting a grouse is high fewer ticks are needed to transmit the virus, and consequently fewer deer are needed to sustain the tick population.

There is also an effect on the grouse carrying capacity suggesting the virus will persist with fewer grouse when the transmission rate is higher because a greater number will become infected.

Increasing β_1 also seems to have an effect on the pattern of LIV persistence. For larger values of β_1 (Fig. 2.7d) the curve displays a sharp transition between deer and grouse densities which do or do not allow virus persistence rather than a gradual change. For low enough deer densities the virus cannot persist, regardless of grouse density. Similarly, for low enough grouse densities the virus will not persist regardless of deer density. Whilst this is true for all values of β_1, here there is not a gentle transition from one case to the other.

The impact of including ingestion in the model decreases as β_1 increases, even though β_3 will increase. At high rates of virus transmission the predicted grouse population is significantly reduced by the virus and so there are fewer grouse ingesting the ticks. Consequently the effect of ingestion on the tick population is slight and few grouse are infected in this way.

The Tick Population Size

As both the tick density dependence and transmission parameter have an impact on the size of the predicted tick population they will both effect predictions for which grouse and deer densities will allow LIV to persist.

Tick Density Dependence, s_t Increasing s_t means that there is a greater density dependence factor acting on the number of ticks that are recruited in to the population, and so increasing s_t results in a lower reproduction rate and a reduced tick population. Conversely, decreasing s_t lowers the effect of density dependence so more ticks reproduce and there is an increase in the tick population.

Transmission Parameter, β_1 Changing the transmission parameter β_1 also has an effect on the size of the tick population, with an increase in β_1 resulting in an increased tick population. All the β_i are linked together, with each β_i, $i > 1$ being a multiple of β_1. β_5 describes the rate at which ticks bite deer and reproduce. If we increase β_1 then we also increase $\beta_5 = 8.82\beta_1$, consequently this leads to a predicted increase in the number of female ticks feeding on deer and reproducing, and hence a larger tick population.

An increased tick density increases the area in which the virus is predicted to persist because there are ample vectors to find a host and transmit the virus. Similarly a large tick population is more able to reproduce and continue the population. However, if there are fewer ticks then the virus will not persist as readily because it will be more difficult for those ticks to find a host and virus transmission to occur. Consequently fewer ticks need more available hosts to improve their chances of locating a meal. Both grouse and deer are important for virus persistence: grouse so ticks can transmit the infection, deer to enable the ticks to reproduce and maintain the tick population.

The virus cannot persist if the tick density is too low. It is therefore interesting to consider for which grouse and deer densities the tick population is predicted to persist and to compare this to the PPT. One would expect that the grouse and deer densities for which the tick population can survive are slightly lower than the densities required for virus persistence. The reproductive rate of the tick population, r_t, can be calculated from the equations, giving

$$r_t = \frac{a_t(\beta_4 H + \beta_5 D)}{b_t + \beta_3 K_g + \beta_4 H + \beta_5 D}.$$

This can be explained in the same way as R_0. If we add one tick to an area with H hares, D deer and grouse at their carrying capacity, K_g, then that tick would be expected to live for $(b_t + \beta_3 K_g + \beta_4 H + \beta_5 D)^{-1}$ units of time and produce $a_t(\beta_4 H + \beta_5 D)$ offspring per unit time. During the course of its life that tick would produce r_t young. (This can also be calculated from model analysis. See Appendix C, Sec. C.1.1 for details.)

However, the formula for r_t underestimates the reproduction of the tick population in the same way as R_0 underestimates virus persistence. It does not take in to account the reduced effect on the tick population that a reduced (due to virus) grouse population has. Consequently the model simulations

have been used to estimate both the PPT and tick survival threshold (TST).

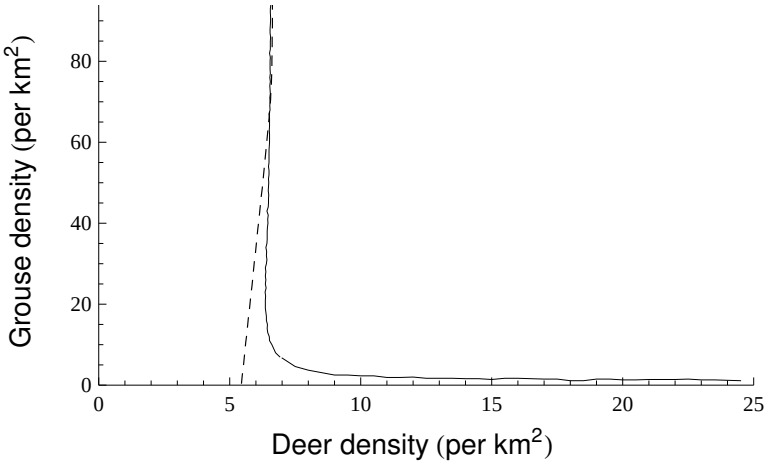


Figure 2.8: Comparing the tick survival threshold (dashed line) with the pathogen persistence threshold (solid line). ($\beta_1 = 0.00002$, $s_t = 0.000002$)

Figure 2.8 shows that the PPT curve asymptotes to the TST curve. The area between the PPT curve and the TST line shows the deer and grouse densities for which the tick population is predicted to be surviving but not at a high enough level to allow the virus to persist. (At high grouse densities it appears as though the virus is persisting when the tick population is not surviving. This is obviously not biologically possible but is a consequence of the step size used for varying host densities. Using a smaller step size renders too large a file.) This suggests that the grouse ingesting ticks may not necessarily reduce the tick population to zero but enough to prevent the virus persisting. (When ingestion is not included the R_0 curve asymptotes to the threshold number of deer required for tick survival, 3.81. Not shown.)

The re-parameterised model

After considering the model predictions it would seem that using $\beta_1 = 0.00002$ and $s_t = 0.000002$ give the most biologically plausible predictions. The values chosen have an element of uncertainty but knowing that the pattern of predictions is robust to changes in β_1 and s_t allows us to be confident that although the numerical predictions may not be accurate the general pattern of behaviour will be as predicted.

2.3.2 The Overall Effect of Ingestion

The hypothesis presented in Sec. 2.1 proposed that the inclusion of ingestion in the model would increase the ability of the virus to persist. In general the addition of ingestion to the re-parameterised model increases the area of virus persistence, so that the virus persists at lower host densities. However, there are some points, which vary with parameter choice, at which the inclusion of ingestion causes the virus to be predicted not to persist when it otherwise would without ingestion. This is especially apparent in the model using the parameter values of Gilbert et al. (2001). For some low deer densities there are grouse densities above which the grouse population is sufficiently large that the ingestion of ticks reduces the predicted tick density below the threshold required for the virus to persist. Consequently to allow virus persistence at these higher grouse densities the predicted number of deer must increase. This seems to

be reasonable because on average each grouse chick ingests a total of 113.4 ticks (5.4 ticks a day for 21 days a year) (Gilbert et al., 2004). Deer can carry 2-300 per week (Gilbert, unpublished data) so high numbers of grouse could potentially eat the equivalent number of ticks that a deer might carry.

The process of extending a previously published model to include another transmission mechanism has also led to the re-parameterisation of the model presented in Gilbert et al. (2001). This is the result of new data collected and current understanding of grouse, deer and tick densities on grouse moors. The estimation of parameters is always difficult when presented with few data or data that are conflicting. However, by varying in turn the parameters β_1 and s_t which are both influential but chosen by comparing model output to biologically realistic densities it has been possible to show that the overall qualitative behaviour of the model remains consistent. The model cannot be used to provide precise densities of grouse and deer that can be expected to allow (or not) virus persistence, but rather to suggest how varying densities affects the likelihood of virus persistence.

With the new parameter values the dilution effect from 'wasted bites' on deer, seen in the model of Gilbert et al. (2001), is no longer occurring at biologically sensible host densities. This is probably due to the estimated high numbers in the tick population. There are now so many ticks that huge numbers of deer would be needed for enough bites to be 'wasted' on the incompetent hosts and the virus to die out.

2.4 Summary

Gilbert et al. (2004) suggested that ingestion of infected ticks by red grouse chicks could be an important route of LIV infection, with up to 98% of LIV infections being attributed to ingestion during their first season. The addition of ingestion to a previously published mathematical model adds some support to this but has also highlighted the impact that grouse ingesting ticks may have on the tick population. The model predicts that for high deer densities the inclusion of ingestion increases the ability of LIV to persist, but for some low deer densities the grouse are able to consume enough of the tick population to reduce it below the threshold necessary for virus persistence.

Although simplifying assumptions (e.g. averaging the ingestion of ticks by grouse chicks over the year) needed to be made in order to incorporate ingestion in to the model the model predictions reflect the belief that ingestion is an important route of infection. The inclusion of ingestion within the model with new parameter values suggests that ingestion does increase the range of host densities that allows virus persistence. (Although for low deer and high grouse densities ingestion may reduce the ability of the virus to persist.) Gilbert et al. (2004) found a higher proportion of grouse chicks were infected with LIV than they would expect if the only route of infection was through tick bites. This is consistent with the model predictions for higher deer densities, where there is a greater likelihood of virus persistence with ingestion compared to without. There is no mention of the deer density on the study site in Gilbert et al. (2004) but as many grouse estates have deer it would not be unreasonable to assume that there were deer present.

The model adapted from Gilbert et al. (2001) has been re-parameterised to reflect current estimates of grouse and tick densities on a grouse moor. This was done by using the model itself as there is little empirical evidence on which to base estimates of the tick density dependence and transmission parameter. Although using the model may not give an accurate estimate of these parameters their effect on the model predictions was assessed and although quantitatively different the pattern of model predictions remains consistent for different parameter estimates.

The introduction of grouse ingesting ticks has lead to an interesting and, as far as we are aware, novel feedback mechanism that means the traditional form of R_0 is no longer an appropriate measure of virus persistence. By ingesting ticks grouse are increasing their chance of becoming infected and consequently the grouse population is reduced. As a result fewer ticks are consumed, the tick population is increased and the chances of a grouse becoming infected is increased further. This cycle then continues until equilibrium is reached. The traditional formula for R_0 is unable to account for the feedback mechanism and consequently underestimates the ability of the virus to persist.

The model to be used in further chapters will be the ingestion model (Sec. 2.2) using the parameter values presented in Gilbert et al. (2001) (Table 2.1) with the following exceptions, $\beta_1 = 0.00002$, $s_t = 0.000002$ and $\beta_3 = 12\beta_1$.

Chapter 3

The potential role of acaricide treated sheep as a management strategy for ticks and LIV

This chapter is presented as an edited version of a paper published in *Theoretical Ecology*, available online from 20 May 2010 (DOI: 10.1007/s12080-010-0080-2). The final publication is available at www.springerlink.com. http://www.springerlink.com/content/7487387811473886/

An additional discussion of analytical analysis is provided in App. C.

Controlling tick borne diseases through domestic animal management: a theoretical approach

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Abstract

Vector-borne diseases are of global importance to human and animal health. Empirical trials of effective methods to control vectors and their pathogens can be difficult for practical, financial and ethical reasons. Here, therefore, we use a mathematical model to predict the effectiveness of a vector-borne disease control method. As a case study we use the tick-louping ill virus system, where sheep are treated with acaricide in an attempt to control ticks and disease in red grouse, an economically important game bird. We ran the model under different scenarios of sheep flock sizes, alternative host (deer) densities, acaricide efficacies and tick burdens. The model predicted that, with very low deer densities, using sheep as tick mops can reduce the tick population and virus prevalence. However, treatment is ineffective above a certain threshold deer density, dependent on the comparative tick burden on sheep and deer. The model also predicted that high efficacy levels of acaricide must be maintained for effective tick control. This study suggests that benignly managing one host species to protect another host species from a vector and pathogen can be effective under certain conditions. It also highlights the importance of understanding the ecological complexity of a system, in order to target control methods only under certain circumstances for maximum effectiveness.

3.1 Introduction

Vector-borne diseases are of global importance to human health, animal welfare, economics and biodiversity. In Europe ticks are the most important vector of zoonotic pathogens, which include *Borrelia burgdorferi* the agent of Lyme borreliosis and the tick borne encephalitis (TBE) complex of viruses. The abundance and distribution of *Ixodes ricinus* ticks in the British Isles are increasing (Scharlemann et al., 2008; Pietzsch et al., 2005; Kirby et al., 2004). Theoretically, reducing vector populations will mitigate disease incidence. Here we use mathematical models to explore the effectiveness of tick control strategies in reducing ticks and disease prevalence and increasing the population of susceptible species. We use the louping-ill virus (LIV) system as a particularly interesting case study because land managers are currently attempting to kill ticks on one species in the hope of increasing the population of another species. However, the theory could be applied to any vector-borne pathogen system.

LIV causes a tick borne disease of great importance to livestock farmers and game keepers as it causes symptomatic infection in both sheep (*Ovis aries*) and red grouse (*Lagopus lagopus scoticus*). LIV infection can lead to severe illness and death in both animals, with up to 80% of experimentally infected red grouse dying from the disease (Reid, 1975). Between 1985 and 2003 a rise in the tick burdens was found on red grouse chicks on 13 sites in Scotland (Kirby et al., 2004). This suggests that red grouse chicks may be at an increasing risk of contracting LIV.

The Biology of Louping Ill Virus LIV is transmitted by the three stage sheep tick which feeds on a variety of hosts. Each stage (larva, nymph, adult) requires a blood meal from a vertebrate host before moulting into the next life stage. Following reproduction the adults die. It is important to note that adult *I. ricinus* ticks rarely feed on red grouse so grouse alone cannot sustain the tick population.

Ticks acquire the virus after feeding from an infected host. There is no transovarial transmission so newly hatched larvae do not carry virus (Gaunt, 1997). Once a tick is infected it can pass on the infection to a host when it takes the next feed during the next life stage. In addition red grouse chicks feed on various invertebrates during the first three weeks after hatching and can acquire the infection after ingesting an infected tick (Gilbert et al., 2004).

Control Strategies of Louping Ill Virus Sheep can be vaccinated against the virus and treated with acaricide to kill ticks which try to attach. This, when conducted properly, can reduce LIV prevalence in sheep farms (Laurenson et al., 2007). Red grouse however cannot easily be treated in a cost effective manner, although tick burdens have been successfully reduced experimentally on small numbers of grouse using acaricidal wing tags (Laurenson et al., 1997) and treating hens with permethrin coated leg bands (Mougeot et al., 2008). This is unlikely to be practicable on a commercial basis.

Mountain hares (*Lepus timidus*) have been shown experimentally to allow LIV transmission non-viraemically between co-feeding ticks (Jones et al., 1997). As a result some grouse managers are conducting extensive culls of mountain hares in an effort to reduce LIV prevalence in red grouse. However, models predict that culling mountain hares can reduce LIV in red grouse only in the absence of other

tick hosts, such as red deer (Gilbert et al., 2001). Therefore, red deer (*Cervus elaphus*) are also culled in some areas due to their importance as tick reproduction hosts (Gray, 1998), even though they do not transmit LIV.

A more benign method of controlling LIV in red grouse could be using sheep as 'tick mops'. In sheep tick mop experiments sheep are actively being used to try and 'mop up' the tick population by killing those ticks that try to attach. The sheep are treated every six weeks with acaricide and put out on the moor in the hope that they will reduce the tick population and thus reduce LIV in the grouse population. The sheep are also vaccinated against LIV. Variable success in reducing LIV prevalence in sheep has been recorded in Northern England (Laurenson et al., 2007). However it is not known how effective sheep mops are at reducing LIV in red grouse, or in areas with alternative tick hosts, e.g. mountain hares and red deer. The Game and Wildlife Conservation Trust (GWCT) is currently conducting trials to test the effects of sheep tick mops on the tick burden of red grouse chicks in the presence of alternative tick hosts in Scotland.

A theoretical approach It is important to understand the factors which affect tick population dynamics to understand how ticks and tick borne diseases might be controlled. This is especially true when empirical trials of tick control methods are made difficult through practical and ethical constraints. Mathematics has a well established history of use in describing the dynamics of tick borne diseases (Cooksey et al. (1990), O'Callaghan et al. (1998), Rosa and Pugliese (2007), Hartemink et al. (2008)). Our aim is to investigate theoretically the effectiveness of controlling a vector-borne disease in one species by reducing the vector population through the management of a second species. The management of one species to control disease in another species is an interesting but not a novel concept. Other applications of this theory include culling badgers (*Meles meles*) to control bovine tuberculosis in cattle (Donnelly et al. (2006), Woodroffe et al. (2006)) and culling bison (*Bison bison*) to control brucellosis in cattle (USDA-APHIS, 2009).

This study involves a more benign treatment strategy, acaricide use as opposed to culling, and is also unusual in that livestock are being managed to control a wildlife disease. Our case study of the LIV system aims to test the effectiveness of sheep tick mops at (i) reducing *I. ricinus* tick populations, (ii) reducing LIV prevalence in red grouse and (iii) increasing the red grouse population. The LIV system is particularly interesting because a large number of hosts interact; grouse, sheep, deer and mountain hares all contribute to the persistence of the pathogen.

An SIR type mathematical model of coupled differential equations for grouse and ticks is used to answer the following questions; 1) How does the addition of a treated sheep flock affect ticks, LIV and grouse compared to grouse moors with no sheep at all? 2) How do alternative hosts, such as deer, impact on the effectiveness of treated sheep? 3) What is the impact of different flock sizes on the effectiveness of treated sheep? 4) How does the efficacy of the acaricide impact on the effectiveness of treated sheep? Our ultimate goal is to provide a critical flock size and acaricide efficacy level and describe how this is affected by the presence of other host species.

Table 3.1: The model parameters. For the units m denotes month, g grouse, t ticks, S sheep and D deer.

Param -eter	Value (unit)	Explanation and Justification
a_g	$0.167(m^{-1})$	Natural birth rate of grouse. Grouse have four chicks per pair on average Reid (1975)
s_g	$0.000\dot{3}$ $(g^{-1}m^{-1})$	Density dependence constraint on grouse. Estimated from model
b_g	0.087 (m^{-1})	Natural death rate of grouse. (Reid, 1975)
α	5 (m^{-1})	Disease induced death rate of grouse, approx. 6 days after infection (Reid, 1975)
γ	$1.25(m^{-1})$	Recovery rate of infected grouse. Calc. from α as 80% infected grouse die (Reid, 1975)
a_t	83.33 (m^{-1})	Natural birth rate of ticks. Assumed adult females hatch 1000 eggs a year (Gilbert et al., 2001)
s_t	0.000002 $(t^{-1}m^{-1})$	Density dependence constraint on ticks. Estimated from model
b_t	$0.083(m^{-1})$	Natural death rate of ticks. Ticks estimated to live for 3 years (Gilbert et al., 2001)
P	0.109	Proportion of infected ticks that infect a grouse when ingested. (Gilbert et al., 2004)
β_1	$0.00002(g^{-1}m^{-1})$	Rate at which a tick bites and infects a grouse. Estimated from model
β_2	$9.75\beta_1$ $(g^{-1}m^{-1})$	Rate at which a tick bites an infected grouse and becomes infected. (Gilbert et al., 2001)
β_3	$7\beta_1(g^{-1}m^{-1})$	Rate at which ticks are ingested by a grouse. See §3.2.3
β_5	$8.82\beta_1(D^{-1}m^{-1})$	Rate at which an adult female tick bites a deer and reproduces. (Gilbert et al., 2001)
β_6	see §3.2.3 $(S^{-1}m^{-1})$	Rate at which an adult female tick bites a sheep and reproduces. See §3.2.3.
β_7	see §3.2.3 $(S^{-1}m^{-1})$	Rate at which a larvae or nymph bites a sheep. See §3.2.3.
d	varied $(S^{-1}m^{-1})$	Efficacy level of the acaricide. Varied for comparisons.

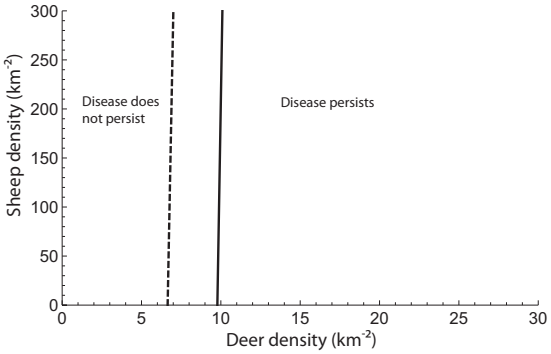
3.2 Methods

3.2.1 The sheep model

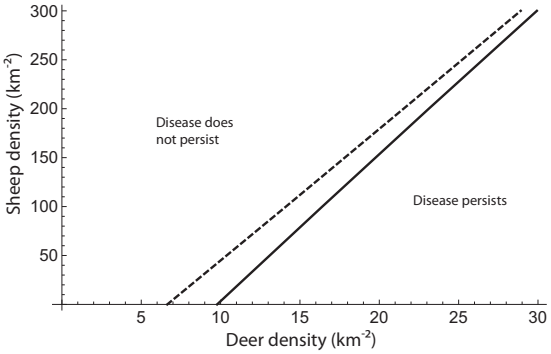
The model is an extension of that developed in Gilbert et al. (2001). The grouse population, G, is split in to three classes: susceptible, G_s, infected, G_i and immune, G_z. The tick population, T, is split in to two classes: susceptible, T_s and infected, T_i. (NB. The different tick stages are combined here and any differences are incorporated into the parameter values.) Deer, D, are included as tick reproduction hosts.

As a result of recent work (Porter et al, unpublished) the model has been extended to include ingestion of ticks as an additional route of infection in red grouse. Young grouse eat invertebrates, including ticks, for the first three weeks after hatching. In Gilbert et al. (2004) it was highlighted that a high proportion of chicks may be infected with LIV as a result of ingesting infected ticks. The model has been adapted to take this in to account. In the model below the terms that are underlined describe these additional ingestion terms.

Sheep can be vaccinated against LIV and treated with acaricide. Consequently sheep are no longer considered important tick hosts and have previously been ignored in models concerning the dynamics of LIV (Gilbert et al., 2001; Laurenson et al., 2003). However, in the case of sheep being used as tick mops they should be included because they play an active role in tick removal. Sheep feed all 3 stages of the tick: larvae, nymphs and adults. Only the adult ticks can reproduce to continue the population cycle. In the model we assume all stages of tick attaching to the sheep may be killed by the acaricide. The effect of killing an adult female tick is greater than that of killing a larva or nymph because it will prevent her from potentially laying 1000 eggs. The two terms in the model that relate to the tick biting rate on sheep are β_6 for adult females and β_7 for larvae and nymphs. These reflect the proportion of the different tick life stages that make up the total tick burden on sheep. The efficacy of the acaricide was also investigated



(a) low sheep tick burden



(b) high sheep tick burden

Figure 3.1: The areas of disease persistence for different sheep and deer densities, with sheep having a) a low tick burden or b) a high tick burden. See The Sheep Parameters section below for an explanation of low and high tick burdens. The solid line represents the line given by solving $R_0 = 1$. The dashed line represents the disease persistence threshold line from model simulations. The area in between the lines denotes where the model predicts the disease persists but $R_0 < 1$. Sheep are treated with acaricide of 100% efficacy.

and is given by d, $0 \leq d \leq 1$, where d represents the proportion of ticks attempting to attach to sheep killed by the acaricide. These terms describing the role of acaricide treated sheep are highlighted in the boxes in the model below.

In this model mountain hares and the role they play in non-viraemic transmission (NVT) are not being considered. Many places where there are concerns about ticks and LIV that are using sheep as 'tick mops' have few hares as a result of culling in an attempt to control ticks. The inclusion of NVT also brings an added complexity to the model. Consequently the terms for hares (compared to Gilbert et al. (2001)) have been dropped and the findings of this study will apply only to hare free environments. (NVT has been discussed in detail in Norman et al. (2004)).

$$
\begin{aligned}
\frac{dG_s}{dt} &= (a_g - s_g G)G - b_g G_s - \beta_1 T_i G_s - \underline{P\beta_3 T_i G_s} \\
\frac{dG_i}{dt} &= \underline{P\beta_3 T_i G_s} + \beta_1 T_i G_s - \Gamma G_i \\
\frac{dG_z}{dt} &= \gamma G_i - b_g G_z \\
\frac{dT_s}{dt} &= (a_t - s_t T)T(\beta_5 D + \boxed{\beta_6(1-d)S}) - b_t T_s - \beta_2 T_s G_i \\
&\quad -\underline{\beta_3 T_s G} - (\beta_5 D + \boxed{\beta_6 S + d\beta_7 S})T_s \\
\frac{dT_i}{dt} &= \beta_2 T_s G_i - \underline{\beta_3 T_i G} - b_t T_i - (\beta_5 D + \boxed{\beta_6 S + d\beta_7 S})T_i
\end{aligned}
$$

where $\Gamma = \alpha + b_g + \gamma$.

3.2.2 The Reproductive Value, R_0

The reproductive value of a virus is a useful aid in determining the factors that will allow the virus to persist or cause it to die out. R_0 is defined as the number of new disease cases caused by adding one infected individual to a totally susceptible population. If the value of R_0 is less than one the disease will

not persist, for R_0 greater than one the disease will persist. R_0 can be found by analysing the equations using the methods of Norman et al. (2004). (These are explained in App. **??** of this thesis.) For the sheep model R_0 is given by:

$$R_0 = \frac{\beta_2(\beta_1 + P\beta_3)K_gK_t}{\Gamma(\beta_5 D + \beta_6 S + d\beta_7 S + \beta_3 K_g + b_t)}$$

where K_g and K_t denote the carrying capacity of grouse and ticks respectively and are given by

$$K_g = \frac{a_g - b_g}{s_g}$$

and

$$K_t = \frac{(a_t - 1)(\beta_5 D + \beta_6(1-d)S) - b_t - \beta_3 K_g - d(\beta_6 + \beta_7)S}{s_t(\beta_5 D + \beta_6(1-d)S)}.$$

However, due to the interesting dynamics that the ingestion of ticks by grouse chicks adds to this system using R_0 in this form underestimates the potential for disease spread. The ingestion of ticks included as a route of infection and a mechanism for tick removal causes a feedback loop in the system once sheep and/or deer densities are sufficient to allow LIV transmission. When the virus is able to establish it reduces the grouse population, this then allows the tick population to increase (as there are fewer ticks being ingested). The increased tick population increases the potential for disease transmission which further reduces the grouse population and so on.

Fig. 3.1 shows the line $R_0 = 1$, calculated from the equations given above and also the pathogen persistence threshold (PPT) line, which is the estimated threshold for disease persistence using model simulations to detect when the virus does and does not persist. It can be seen in Fig. 3.1 that the $R_0 = 1$ line is to the right hand side of the PPT line, giving an area between the two lines where the disease is persisting even though $R_0 < 1$. This is because the estimate of R_0 from the equation is unable to account for the feedback within the system. In our discussion we refer to the PPT line rather than $R_0 = 1$, as this is the threshold of disease persistence given by the model simulations.

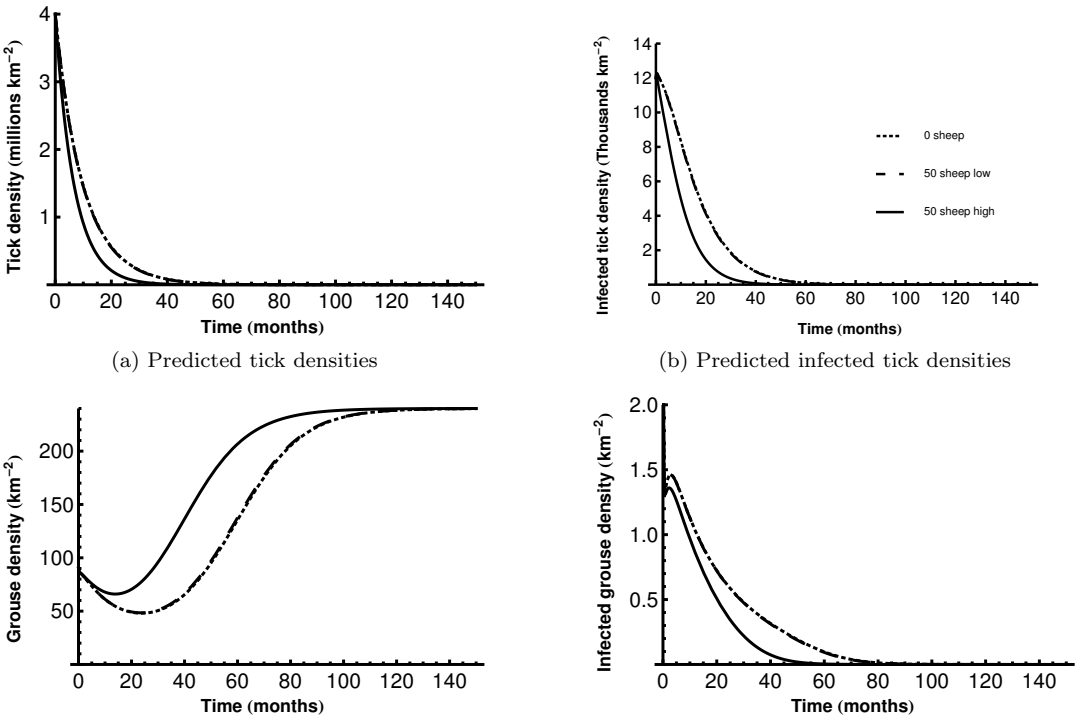
3.2.3 Parameter Estimation

Many parameters values have been published previously and their estimation is more fully explained in Gilbert et al. (2001). Parameter values we use are summarised in Table 3.1. Justifications for estimates made in this paper are explained in the text.

The density dependence parameters, s_g, s_t

The density dependence parameter for grouse, s_g, is estimated from the model to ensure that when there is no disease grouse reach a carrying capacity of 240 per km^2. (Grouse counts of this magnitude have been recorded by Gilbert, unpublished data; Laurenson et al. (2007); GWCT red grouse counts, Game and Trust (c.2009))

We estimated the density of ticks on heather moorland by combining information on the number of



(a) Predicted tick densities

(b) Predicted infected tick densities

(c) Predicted grouse densities

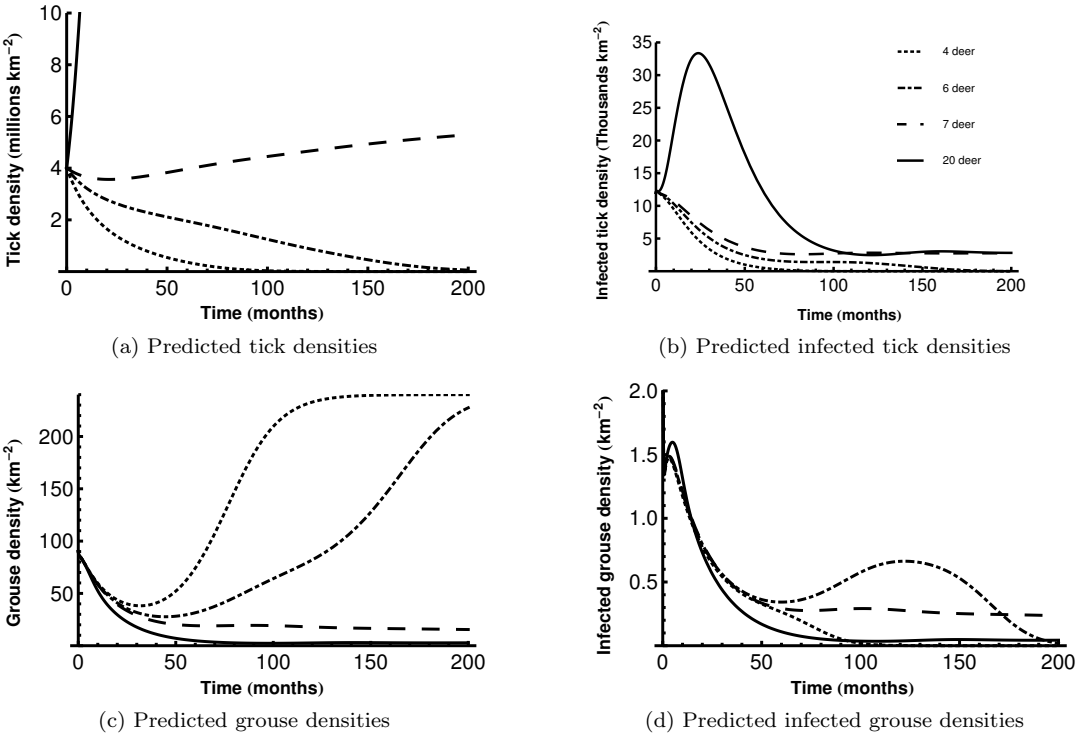
(d) Predicted infected grouse densities

Figure 3.2: The predicted effect of adding sheep treated with acaricide of 100% efficacy ($d = 1$) on a)tick, b)infected tick, c)grouse and d)infected grouse densities over time. No other hosts were present. The dotted line (mostly hidden by the dashed line) represents no sheep. The dashed line 50 sheep per km^2 with low tick burden. The solid line represents 50 sheep per km^2 with high tick burden.

nymphs counted during field surveys with the efficiency of the survey method, then extrapolating up to a km sq. The survey method used 10m long blanket drags (Gray and Lohan, 1982). We found 1.26 ± 0.20 (mean $\pm$ s.e.) nymphs per blanket drag, with a maximum of 50, over 9 areas representative of a typical grouse moor. We then tested blanket drag efficiency by adding a known number of nymphs to four 1x1m patches of heather moorland known to not contain ticks previously, and subsequently repeatedly dragging and counting the nymphs collected. The proportion of ticks collected was approximately $1.3\% \pm 0.3$ (mean $\pm$ s.e.). From this we can estimate a very approximate 9.7 ± 1.1 million (mean $\pm$ s.e.) nymphs per km sq, with maximum 385 million. Therefore the tick density dependence, s_t, is estimated to ensure that ticks are able to reach a carrying capacity in the tens of millions in the absence of sheep tick mops. Actual tick density predictions from the model vary with host availability.

The Ingestion Parameter, β_3

Gilbert et al. (2004) suggested that 73-98% of infections of grouse in their first season may stem from the grouse ingesting the ticks. If we take the midpoint, 84%, then a grouse is 5.25 times more likely to get infected through ingestion than by being bitten during its first season. The first season is from early June when chicks hatch, to August/September when they are shot and questing nymphs begin to decline and is taken as 90 days. The first season lasts for only $\frac{90}{365}$ of the year, and the chance of infection from ingesting an infected tick is 0.109 (Gilbert et al., 2004). Therefore, our estimate for the ingestion parameter, the rate at which ticks are ingested by grouse, now becomes $\beta_3 = 12\beta_1$ (i.e. $5.25 \times \frac{90}{365} \div 0.109$), where β_1 is the rate at which a tick bites and infects a grouse.



(a) Predicted tick densities

(b) Predicted infected tick densities

(c) Predicted grouse densities

(d) Predicted infected grouse densities

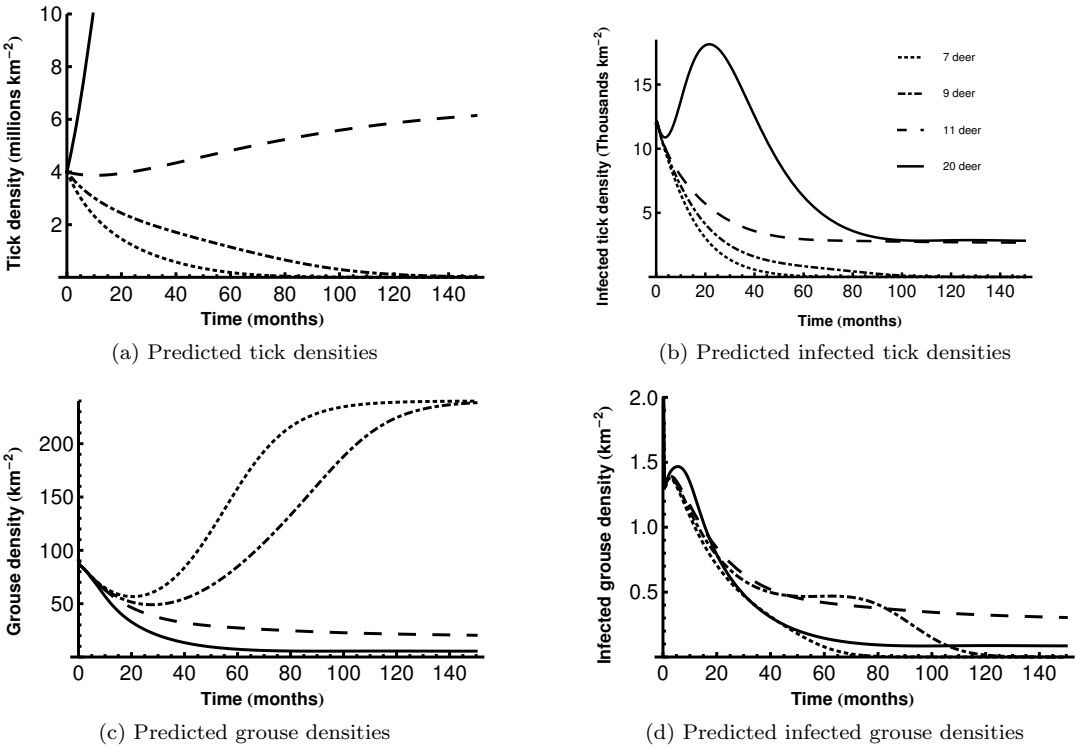
Figure 3.3: The effect of different deer densities on the effectiveness of sheep tick mops on a)tick, b)infected tick, c)grouse and d)infected grouse densities using the low sheep tick burden model. There are 50 sheep per km^2 treated with acaricide of 100% efficacy. The dotted line represents 4 deer per km^2. The dot-dashed line represents 6 deer per km^2. The dashed line represents 7 deer per km^2. The solid line represents 20 deer per km^2.

The Sheep Parameters, β_6, β_7

As described in Gilbert et al. (2001) the transmission parameters (β_i) are calculated based on the ratio of ticks on grouse and the relevant mammalian host on the same estate. We do not have our own recent data of tick burdens on untreated sheep and grouse at the same site but as the two sites in Gilbert et al. (2001) found 9 and 9.3 nymphs per grouse we are relating our estimates of ticks on sheep to ticks on grouse assuming 9 nymphs per grouse to form a crude yet biologically realistic estimate.

We found that the number of ticks attached to untreated sheep varies considerably. Our sample, collected from from 11 untreated sheep on 1 farm on a grouse moor in Scotland, ranged from 0 to 11, with a mean of 4.27 ± 1.25 (mean $\pm$ s.e.). In addition the box plots of tick counts on the head and ears of sheep in Ogden et al. (2002) show great variability and in Laurenson et al. (2000) the number of adult female ticks found on lambs varies hugely, from a mean of 0.04 ± 0.04 (mean $\pm$ s.e.) on one farm compared to a mean of 24 ± 1.6 (mean $\pm$ s.e.) on another. This makes estimating the tick biting rate on sheep difficult.

We ran the model with different values of β_6 and β_7 to assess the effect this has on tick and grouse densities. We found that varying the tick biting rate on sheep within the range we found empirically has very little effect on model output. Consequently we chose to work with $\beta_6 + \beta_7 = 0.75\beta_1$. That is assuming the total tick burden on sheep is 75% of the grouse nymph burden. The tick burden on sheep covers both adult and juvenile ticks. Our data showed that approximately 80% of ticks on sheep are adults so that $\beta_6 = 0.8 \times 0.75\beta_1 = 0.6\beta_1$ and $\beta_7 = 0.2 \times 0.75\beta_1 = 0.15\beta_1$.



(a) Predicted tick densities

(b) Predicted infected tick densities

(c) Predicted grouse densities

(d) Predicted infected grouse densities

Figure 3.4: The effect of different deer densities on the effectiveness of sheep tick mops on a)tick, b)infected tick, c)grouse and d)infected grouse densities using the high sheep tick burden model. There are 50 sheep per km^2 treated with acaricide of 100% efficacy. The dotted line represents 7 deer per km^2. The dot-dashed line represents 9 deer per km^2. The dashed line represents 11 deer per km^2. The solid line represents 20 deer per km^2.

Laurenson et al. (2003) gives an estimate of the tick burden on sheep and grouse at the same site. This would give estimates of $3.43\beta_1$ and $43.48\beta_1$ for β_6 and β_7 respectively. However, the paper explains that only the adult ticks were counted on sheep and the immature tick burdens were estimated using the ratio 1:5:8 for adults:nymphs:larvae derived from Ogden et al. (1998). Our own data finds a very different ratio of adults to nymphs and larvae on sheep. Consequently we feel it is more thorough to consider the results of the model using both empirical data sets, i.e those estimated in Laurenson et al. (2003) giving a high relative sheep tick burden (approx. 47 times the grouse nymph burden) and our own sheep tick counts giving a low relative sheep tick burden (approx. 0.75 times the grouse nymph burden). The two parameter sets will be referred to as high sheep tick burden and low sheep tick burden respectively.

Please see Appendix C, Sec. C.2.2 for a sensitivity analysis of the parameter estimates.

3.3 Results

The model was simulated over the following scenarios to predict the effect treated sheep would have on grouse and tick densities and LIV prevalence in grouse. In all cases the model was run both with a high sheep tick burden and a low sheep tick burden. 1) 50 treated sheep were added to a grouse moor with no alternative hosts, compared to no sheep. 2) 50 treated sheep were added to grouse moors with varying deer densities. 3) The treated sheep flock size was varied for a given deer density. 4) The acaricide level was varied for a given sheep flock size and deer density.

3.3.1 How does the addition of a treated sheep flock affect ticks, LIV and grouse compared to grouse moors with no sheep at all?

If we consider a scenario of grouse and ticks only then the tick population will die out through lack of hosts for reproduction, since grouse feed only immature ticks. Although a grouse and tick only environment is not biologically realistic it is interesting to consider mathematically the effect of adding treated sheep. If we add to the model a flock of 50 treated sheep (as in GWCT experiments) treated with acaricide of 100% efficacy per km^2 we would expect the decline of the tick population to speed up. Fig. 3.2a shows that the addition of treated sheep with a low tick burden (dashed line) has virtually no effect on the speed at which the tick population declines or grouse reach carrying capacity (Fig. 3.2c) when compared to no sheep (dotted line), indeed the lines are almost indistinguishable.

However, when 50 treated sheep per km^2 with a high tick burden are added to the model (solid line) the impact is much greater. The speed with which the grouse reach equilibrium is considerably quicker than with the low sheep tick burden model (Fig. 3.2c). The tick population reduces by 99% approximately 14 months faster than with low sheep tick burden model (Fig. 3.2a).

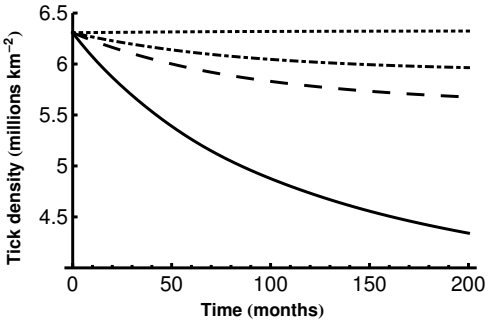
3.3.2 How do alternative hosts, such as deer, impact on the effectiveness of treated sheep?

Deer amplify the tick population due to their ability to host a large number of ticks (Gray, 1998). Therefore we used the model to predict the effect of different deer densities on the effectiveness of sheep tick mops at reducing ticks and LIV. There is the potential for large numbers of deer to render the use of sheep tick mops ineffective. The sheep flock size was kept at 50 per km^2 as in the trials conducted by the GWCT and the effect this would have on areas with different deer densities was explored.

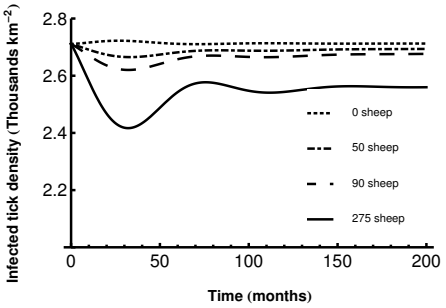
Fig. 3.3 shows that when there are 6 deer per km^2 (dot-dashed line) or fewer then the low sheep tick burden model predicts that the tick numbers are reduced and the grouse reach their carrying capacity at a slower rate than if there were no deer. If there are 7 deer per km^2 (dashed line) then the predicted tick population is much higher and causes enough LIV infection for the grouse density to drop dramatically, but not to die out. It is interesting to note that this shows a dramatic effect on the grouse population for a small change in deer density. Although we cannot predict the quantitative effect with any certainty we can be confident that this rapid change will occur for some deer density as the tick population predictions are very sensitive to reproduction host density. For high deer densities (9 per km^2 or above) the model predicts that the tick population is sufficiently large to cause enough infection for the grouse population to be significantly reduced.

This supports the preliminary key findings of the GWCT, who found that for areas of low deer density (< 5 per km^2) sheep tick mops may reduce tick burdens on grouse chicks. However, in those areas of high deer densities (> 10 per km^2) the sheep tick mops were not successful in reducing the tick burden on grouse chicks. (For their full report see Smith (c.2006))

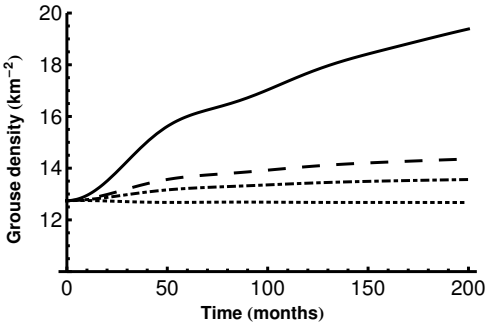
Using the high sheep tick burden model shows a similar pattern of behaviour but this occurs at



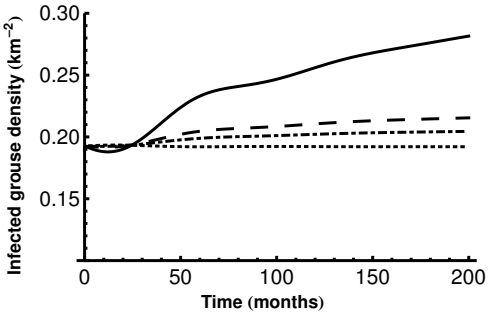
(a) Predicted tick densities



(b) Predicted infected tick densities



(c) Predicted grouse densities



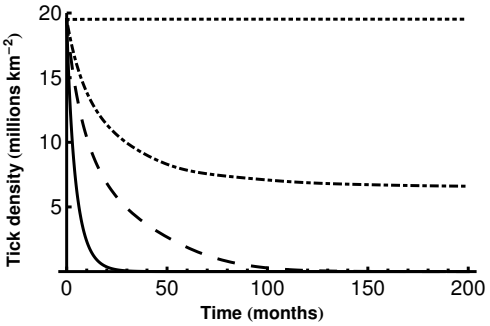
(d) Predicted infected grouse densities

Figure 3.5: The predicted effect of different sheep flock sizes treated with 100% efficacious acaricide on an area with 7 deer per km^2 on on a)tick, b)infected tick, c)grouse and d)infected grouse densities using the low sheep tick burden model. The dotted line represents no sheep. The dotdashed line represents 50 sheep. The dashed line represents 90 sheep. The solid line represents 275 sheep.

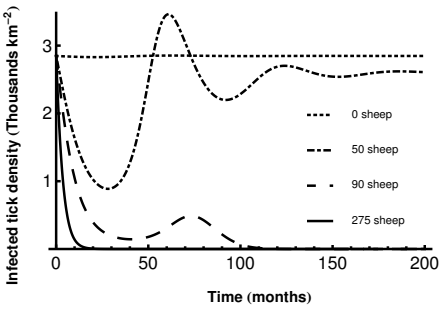
different deer densities (Fig. 3.4). The high sheep tick burden model parameter estimates are based on sheep carrying a higher relative tick burden and so one would expect them to be more effective at reducing the tick population. Although the speed of recovery slows as the deer density increases the treated sheep are now predicted by the model to be effective in an area with up to 9 deer per km^2 (dot-dashed line Fig. 3.4). With 10 deer per km^2 the model predicts an eventual recovery of the grouse population but taking many years. Above 10 deer per km^2 the grouse population declines.

3.3.3 What is the impact of different flock sizes on the effectiveness of treated sheep?

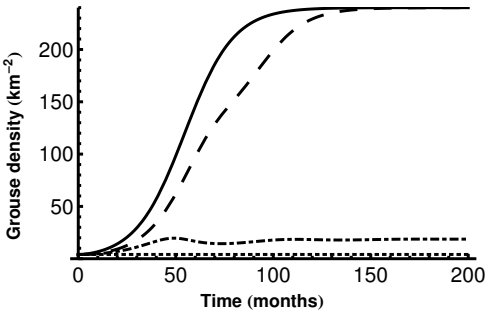
Increasing the number of treated sheep increases the number of deer the system can tolerate before the disease reduces the grouse population. The extent to which this occurs very much depends on the sheep tick burden. It can be seen from Fig. 3.1a (the low sheep tick burden model) that when there are 50 treated sheep per km^2 and < 6.5 deer per km^2 the pathogen is predicted to die out, but the pathogen is predicted to survive when there are more than 6.5 deer per km^2. This agrees with the times series plots (Fig. 3.3) of the model predictions which show grouse reaching carrying capacity for 6 deer per km^2 but not for deer densities higher than this. Below 6 deer per km^2 the pathogen will always die out irrespective of sheep numbers. The estimated line for the disease persistence threshold is almost vertical for the low sheep tick burden model, indicating that the addition of up to 300 treated sheep has little effect on how many deer the system can tolerate before the disease persists. However, Fig. 3.5 shows that the predicted tick population is reduced by the addition of increasing numbers of 100% efficacious



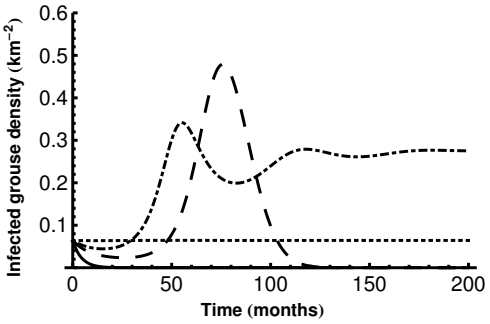
(a) Predicted tick densities



(b) Predicted infected tick densities



(c) Predicted grouse densities

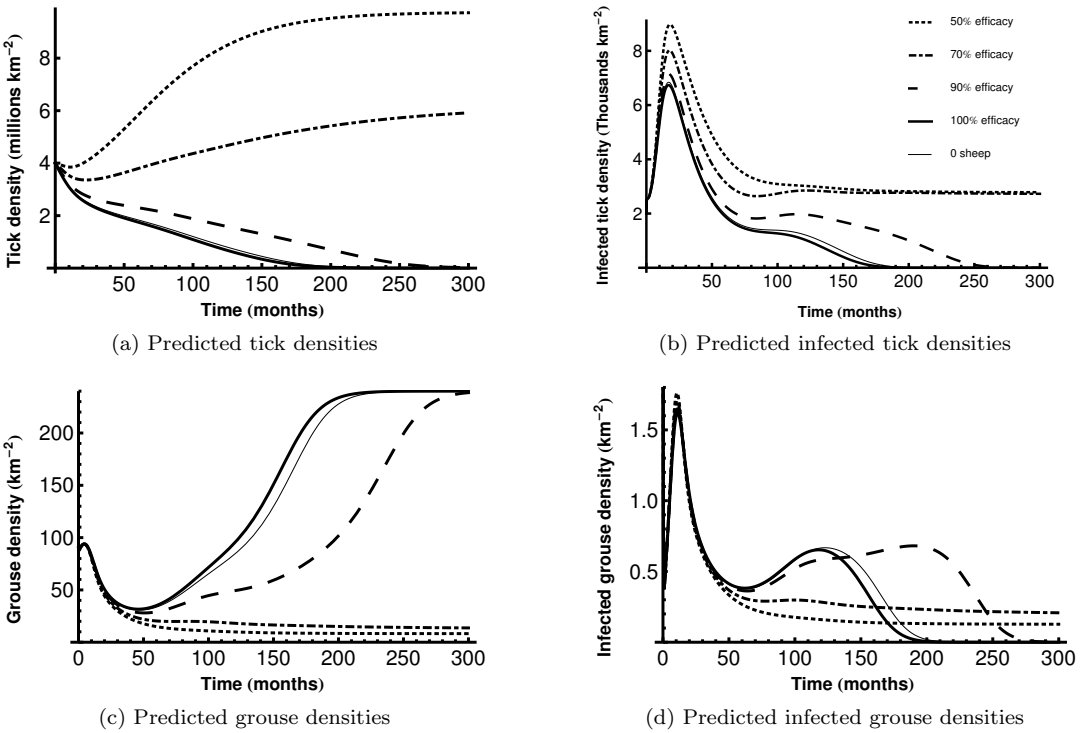


(d) Predicted infected grouse densities

Figure 3.6: The predicted effect of different sheep flock sizes treated with 100% efficacious acaricide on an area with 11 deer per km^2 on a)tick, b)infected tick, c)grouse and d)infected grouse densities using the high sheep tick burden model. The dotted line represents no sheep. The dotdashed line represents 50 sheep. The dashed line represents 90 sheep. The solid line represents 275 sheep.

treated sheep. This reduction of tick numbers reduces the opportunity for grouse to become infected and consequently the grouse population is less affected. Therefore, although the pathogen can persist the grouse population suffers lower mortality rates with treated sheep than without treated sheep. This is illustrated in Fig. 3.5 when there are 7 deer per km^2 the predicted tick population is reduced from 5.7 million per km^2 to 5.3 million per km^2 when 50 treated sheep per km^2 are included in the low sheep tick burden model. In this case the model predicts that the virus will persist in the grouse population, but the use of treated sheep allows additional grouse to survive. The predicted density of grouse per km^2 increases as the number of treated sheep increases in the model. Without treated sheep the grouse reach a predicted equilibrium of 14.2 grouse per km^2, but with 50 treated sheep per km^2 the grouse reach an equilibrium of 15.5 per km^2 this increases to 16.6 per km^2 with 90 sheep per km^2. The use of sheep tick mops also shortens the length of time the virus persists in the grouse population when there are 6 deer per km^2(Fig. 3.3) and allows the grouse to recover to their carrying capacity at a faster rate.

The effect of increasing the flock size of treated sheep is more dramatic with the high sheep tick burden model, as one would expect. Fig. 3.1b predicts that for the high sheep tick burden model increasing the sheep density to 275 per km^2(commercial stocking densities) allows over 25 deer per km^2 before LIV persists. It is unlikely that sheep would be stocked at such high density on grouse moors due to poor grazing habitat. A more realistic hill stocking density is around 90 sheep per km^2, which allows 12 deer per km^2 before disease persistence. Considering now the scenario of 11 deer per km^2, Fig 3.6a illustrates that a flock of 50 treated sheep per km^2 added to the high sheep tick burden model dramatically reduces the predicted tick population to 6.4 million per km^2 from 19.5 million per km^2 with no sheep. This allows



(a) Predicted tick densities

(b) Predicted infected tick densities

(c) Predicted grouse densities

(d) Predicted infected grouse densities

Figure 3.7: The effect of acaricide efficacy in an environment with 6 deer and 50 treated sheep with low sheep tick burden model on a)tick, b)infected tick, c)grouse and d)infected grouse densities. The dotted line represents 50% efficacy. The dotdashed line represents 70% efficacy. The dashed line represents 90% efficacy. The thick solid line represents 100% efficacy. The thin solid line represents no sheep.

the grouse to reach a higher predicted equilibrium of 19.5 per km^2 as opposed to 4.1 per km^2 without sheep. This highlights again that although the virus is still persisting in the grouse population the use of sheep tick mops is predicted to allow a greater number of grouse.

3.3.4 How does the efficacy of the acaricide impact on the effectiveness of treated sheep?

In practice it is very difficult to ensure that the acaricide applications are fully effective at preventing all ticks from attaching to all sheep. Even if initial applications are 100% efficacious the efficacy decreases over time. Therefore we used the model to predict the effect different levels of efficacy have on the tick and grouse population densities.

If a flock of 50 sheep per km^2 treated with 100% efficacious acaricide is added to the model with the low sheep tick burden model and 6 deer per km^2 then the ticks will die out and the grouse population will recover. If the acaricide efficacy is 90% then the speed of the recovery of the grouse is much slower. However Fig. 3.7 shows that if the acaricide is only 50% or 70% efficacious then the tick numbers increase and the grouse numbers are reduced. If the model is run with no sheep and 6 deer per km^2 then it is predicted that the grouse will recover as there is not a sufficient deer density to sustain the tick population. Consequently, if the efficacy cannot be maintained at a high level then no sheep at all will give a higher grouse yield than a flock of less effective sheep. This may seem counterintuitive as some intervention is surely better than none. However, the model predicts this is not the case. Introducing untreated sheep would amplify the tick population as they would be providing hosts for the adult female ticks, who could

then reproduce. In contrast, if sheep treated with 100% efficacious acaricide are introduced then they would kill these ticks. However, if the efficacy is not sufficiently high there is a fine balance between killing enough ticks to impede reproduction and allowing too many to reproduce.

3.4 Discussion

The aim of this paper was to investigate theoretically the effectiveness of controlling a vector-borne disease in one species through the management of a second species to reduce the vector population. We used the LIV system as a particular case to parameterise our model. The model was simulated over the following scenarios with a high sheep tick burden and a low sheep tick burden. 1) 50 treated sheep were added to a grouse moor with no alternative hosts, compared to no sheep. 2) 50 treated sheep were added to grouse moors with varying deer densities. 3) The treated sheep flock size was varied for a given deer density. 4) The acaricide level was varied for a given sheep flock size and deer density. This enabled us to answer the following questions; 1) How does the addition of a treated sheep flock affect ticks, LIV and grouse compared to grouse moors with no sheep at all? 2) How do alternative hosts, such as deer, impact on the effectiveness of treated sheep? 3) What is the impact of different flock sizes on the effectiveness of treated sheep? 4) How does the efficacy of the acaricide impact on the effectiveness of treated sheep?

In general the model predicted that treated sheep could speed up the decline of the tick population on a moor with no alternative tick hosts and could reduce the tick population if the density of alternative tick reproduction hosts was low. Increasing the density of treated sheep for a given deer density is predicted by the model to decrease the tick population. For a given treated sheep flock size and deer density the model predicts that decreasing the acaricide level much below 90% can actually allow the tick population to increase. The model also predicts that the effect of sheep tick mops very much depends on the sheep tick burden.

The model predicted that using acaricide treated sheep can be an effective method to reduce the tick population on a grouse moor providing there are few deer (< 6 per km^2) and efficacy levels of the acaricide are kept high ($> 90\%$). Our work supports, at least qualitatively, experimental work by the GWCT (Smith, c.2006) that also suggests that in the presence of high deer numbers the sheep tick mops will be rendered ineffective. The model predicts that not only are low efficacies less effective, but may in fact be worse than no sheep at all.

An exciting theoretical result which has emerged unexpectedly from this work is that the addition of ingestion means that R_0 no longer behaves as the threshold for disease persistence. This is a very unusual result and we believe that it is the first time that this has come to light. The formula for R_0 which can be derived in a number of different ways (ie from the Jacobian as in Norman et al. (2004) or the next generation matrix (Diekmann et al., 1990)) is given in section 3.2.2. Normally when $R_0 > 1$ the disease can persist and when $R_0 < 1$ the disease cannot persist. However, we have found here that the simulations do not agree with this threshold and in fact the disease can persist when $R_0 < 1$. This is because of the feedback mechanism that is created by the ingestion. In a totally susceptible population

grouse and ticks are at their carrying capacity, however, with ingestion the carrying capacity of ticks is lower than it would be without ingestion because the grouse are eating the ticks. If we add disease to this system then the grouse population is reduced which causes an increase in the tick population which then causes a greater decrease in the grouse population. Therefore the disease can persist more easily and calculating R_0 using the formula derived from the definition underestimates the ability of the disease to persist. This is a really interesting result and requires some further investigation to determine if there are other systems for which this is likely to be an important phenomenon and which aspects of the system are essential for it to occur.

We have not investigated the biological interaction between the sheep and deer. In nature it is possible that where sheep are removed from the moor more deer may move in to fill the void created. It may be in this case that even ineffective sheep are better than none if the alternative is an increase in deer density. We do not have any data on the relationship between deer and sheep that shows the effect the presence of sheep has on the density of deer but anecdotal evidence suggests there is a negative interaction between the two species. The segregation of wild and domestic animals has been documented (Loft et al., 1993; Acevedo et al., 2007) with Fankhauser et al. (2008) proposing that dung avoidance may explain why chamois tend to avoid domestic sheep. Due to the high tick burden of deer it is intuitive that only a few deer would be needed to feed the same number of ticks as a full flock of sheep with a low tick burden. Using the parameter values from our low sheep tick burden model we can see that the relationship between deer and sheep burdens is $S = \frac{16}{1-d}D$, where d is the acaricide efficacy. If for example the efficacy levels were only 50% and we knew that in the absence of sheep there would be 10 deer per km^2 then having up to 320 treated sheep per km^2 would be preferable to having 10 deer per km^2 and having more than 320 treated sheep per km^2 would be worse than having 10 deer per km^2. However, if we knew there would only be 5 deer per km^2 in the absence of sheep then having up to 160 treated sheep per km^2 would be preferable to the deer and having more than 160 treated sheep per km^2 would be worse than 5 deer per km^2.

Our model is very sensitive to deer density, suggesting that deer play a major role in the persistence of the tick population and LIV. Deer can carry high tick burdens, and as a result they can allow the tick population to be maintained. If the deer could be used as tick mops rather than the sheep this may, at least in theory reduce the tick population and LIV prevalence in grouse more effectively. If sheep alone are being used as tick mops but the acaricide is not highly efficacious the sheep may create more blood meals for the adult ticks and may allow ticks to reproduce at a greater rate than they are removed. Where deer are present any treatment to lower the number of ticks deer carry will be beneficial. However, treating deer in practice has many issues; legally, ethically and logistically. Acaricides are not licensed for use on wildlife. There are major difficulties with the application of acaricide to deer in practice and the dose of acaricide cannot be controlled. The percentage of deer receiving the treatment would vary as deer come and go from the treatment site. However, the '4 poster' method has been used with some success in the US (Carroll et al., 2002). There is also the problem of withdrawing the product before culling as deer are used for human consumption. The use of acaricide on deer may also increase the incidence of acquired

resistance of the ticks to the acaricide.

The model has several other limitations. It is difficult to accurately measure many of the model parameters and some have been estimated from fitting the model to achieve biologically plausible results. The sensitivity analysis indicated that the model outputs were affected most by variation in the tick parameters: tick birth and death rates and tick biting rates on deer and sheep (see Appendix C, Sec. C.2.2). For accurate quantitative predictions, therefore, it is these parameters that require the most accurate estimated values. The estimates we used for these parameters were derived from the literature and our own data, and there is considerable variation in these values between studies, depending on available hosts, time of year, region. More empirical data are needed on tick burdens of different tick stages on all the different host in the same place at the same time. We emphasise that the model outputs may not be quantitatively accurate in their predictions of grouse densities for particular sheep and deer densities. However, the models reflect the general qualitative patterns for how grouse densities may change with varying sheep and deer densities.

We have few data on the tick burdens of sheep on sites where we can make direct comparisons with other host tick burdens. Our own data includes counts of all tick life stages explicitly, but we do not have tick counts on grouse at the same site to make a direct comparison. Laurenson et al. (2003) does have red grouse tick counts but uses estimates for the larvae and nymph counts on sheep using larvae:nymph:adult ratios from Ogden et al. (1998). The ratios given are very different from the ratios we found. Ogore et al. (1999) compared the tick burden on different sheep breeds in Kenya and found that the burdens varied between breeds. It could be that different breeds in the UK display similar differences, which may help account for the differences we found. Different sites may also have different densities of alternative hosts, for example, a site with more small mammals and birds that feed larvae may result in fewer larvae on sheep. The limitations of the available relevant data make it difficult to quantitatively estimate the efficacy of sheep tick mops, although qualitative patterns still hold.

In order to validate our model we would need to be able to compare the burdens of different tick stages on all the hosts (grouse, sheep and deer) at the same site. We would then be able to improve our estimate of the tick burdens within the model and the role each host plays in the tick life cycle. Although as Laurenson et al. (2003) shows the ratios between tick stages on each host type differ from site to site. These differ again from the ratios found in Gilbert et al. (2001) from which many of our parameters are taken. The variability of nature makes it impossible to develop a quantitatively accurate mathematical model for all estates. However, we believe the qualitative results from our two models give useful insights into the dynamics of the LIV system and the use of sheep tick mops. A discussion of the sensitivity analysis is given in the Appendix C, Sec. C.2.2.

We assumed homogenous space but a grouse moor is made up of a patchwork of heather and grass areas and in reality the sheep tend to prefer the grassy areas. Consequently the sheep may be less likely to pick up the ticks questing in the heather which is the habitat the grouse prefer. We do not explicitly model the spatial heterogeneity of the distribution of the tick hosts. However, the estimation of the tick burden for each host takes this in to account and, as a result, the sheep have a lower tick burden than

the grouse in the low sheep tick burden model.

Throughout the model the life stages of the tick are combined. The effect of the different life stages in the transmission of the disease have been taken account of in the estimation of the various β_i. Future model improvements could include the stages explicitly as the different tick stages may sometimes have their peak of activity at different times of the year (Randolph et al., 2002). This would make the model much more complicated and we do not at present have the data to make this possible.

Similarly the grouse life stages are combined, but as it is only the chicks which consume the ticks in the first three weeks of life it may be appropriate to model chicks and adults separately. This would allow ticks to be ingested by the chicks for a particular three week period rather than averaging out over the year as at present.

In conclusion, our model supports the idea that controlling the vector population by managing one species can mitigate disease and enhance the population of a second target species. Specifically, our case study suggests that treating sheep with acaricide can, under certain circumstances, reduce the population of *I. ricinus*, reduce the prevalence of LIV, and increase the red grouse population. This is a more benign approach than other documented attempts at controlling disease in one species by targeting another species, such as culling badgers to control bovine tuberculosis in cattle and bison to control brucellosis in cattle. However, our study highlights the difficulties of multi-host vector-borne systems which, importantly, raises issues with this more benign method. For example, sheep tick mops are predicted to be effective only with very low densities of alternative hosts such as deer, and at very high acaricide efficacies on sheep. Such circumstances may be rarely realised in practice and there may be ethical implications with attempts to achieve them. For example, there may be health and welfare issues for farmers and livestock of increased exposure to high acaricide levels. This study exemplifies how models can be useful in predicting the effectiveness of various control strategies under different scenarios, where empirical studies are not possible. It is important, however, to consider the practical and ethical implications of implementing such methods. Modelling studies can help focus the implementation of control strategies for maximum effect under the most appropriate circumstances.

Despite the limitations of this simple model this approach can be a useful tool for predicting qualitatively the outcomes of various field scenarios. These results could help inform policy of tick and tick borne disease control. Although we focus here on the LIV system we believe that similar methods could be used to model other tick borne disease systems.

Chapter 4

Modelling the seasonal hatching of grouse chicks

4.1 Seasonal reproduction of grouse

In Chapter 2 the behaviour of grouse and ticks was assumed constant over the course of a year. However, grouse chicks hatch only in the early summer which is coincident with when ticks are generally at their most active. It is known that grouse chicks consume ticks as part of their diet for the first three weeks after hatching (Gilbert et al., 2004). Consequently grouse chicks may be more likely to pick up infection than adult grouse, either through tick bites or through ingesting infected ticks. The simple non seasonal ingestion model assumed ingestion is distributed over the course of the year and over the total grouse population. This chapter presents a more realistic model which describes the chicks and adult grouse separately with chicks hatching only at a given time of the year. It also allows just chicks to ingest ticks for the first three weeks after hatching.

The aim of this chapter is to investigate whether the incorporation of the seasonal biology of grouse alters the model predictions dramatically from the non seasonal model. After first confirming the within year predictions reflect the seasonal biology of the grouse the long term predictions will be compared to the non seasonal model to assess any difference in temporal predictions.

If the non seasonal model is already incorporating the important elements in the dynamics of the LIV system then we would expect that the long term equilibrium model predictions of the seasonal model would be similar to those of the non seasonal model. However, if averaging the behaviour of the grouse over the year means that something is lacking from the non seasonal model then the model predictions would be expected to be different. The ingestion model (Chapter 2) predicts that adding ingestion of ticks as an extra route of infection to a model without ingestion can, for low deer densities, make it more difficult for the virus to persist. This was explained by the fact that the grouse were eating a sufficient quantity of the tick population to reduce the tick population below that necessary for virus persistence. We use the predicted equilibrium densities to consider if this still occurs for the grouse pulse

hatch ingestion model or if modelling chicks separately has reduced the impact of ingestion. For higher deer densities with ingestion incorporated the non seasonal model predicts that the virus will persist more readily. We compare the grouse pulse hatch ingestion model and non seasonal ingestion model to determine the qualitative and quantitative impact of seasonal grouse biology on model predictions.

Seasonality can be added to a model in a number of ways. In this chapter we consider two; 1) grouse hatching in a single pulse; 2) grouse hatching over a period of weeks. The temporal predictions and equilibrium density predictions for different deer densities of the two models will be compared to assess which model appears to be the most realistic.

The seasonal model will then be used to assess the impact of sheep tick mops. Chapter 3 found that the sheep tick mops were effective for low deer densities but less effective for high deer densities. The seasonal model predictions will be compared to the non seasonal ingestion model to establish if the addition of seasonal grouse biology has had a significant effect on the predicted conditions for which sheep tick mops are expected to work.

4.2 The grouse pulse hatch with ingestion model

The continuous non seasonal ingestion model has been adapted to become a semi-discrete model of the seasonal biology of grouse by splitting the year in to two parts with and without ingestion occurring. Each year is assumed to begin in the spring when the chicks hatch. The grouse chicks and their ingestion of ticks are modelled explicitly and separately from the adult grouse for three weeks. After three weeks all chicks enter the adult classes corresponding to their current disease status because after this time chicks no longer ingest ticks so their virus transmission routes are the same as adults. The model allowing virus transmission from tick bites only continues to run to the end of the year. The adult and chick model then restarts with the number of grouse chicks hatching depending on the density of adult grouse at the end of the previous year. Parameter time scales are per week. Ticks are still considered to be reproducing at a rate averaged across the year. (The seasonal biology of ticks will be considered in Ch. 5.)

Although this model aims to give a more seasonally realistic representation of the grouse and tick interaction some simplifying assumptions still need to be made. In this first model it is assumed that all grouse chicks hatch instantaneously, free from infection and ingest ticks for three weeks from this date. Tick biting rates are assumed to be the same on chicks and adults and do not change throughout the year.

The seasonal model considers the grouse chicks, G_c, in three disease classes: susceptible G_{cs}, infected G_{ci} and immune G_{cz}. The adult grouse, G, and ticks, T, are similarly defined.

Equations 4.1, 4.2 and 4.3 which describe the model are given below; the terms describing the ingestion of ticks by chicks are underlined. Equations 4.1 and the ingestion terms in the tick equations (Equations 4.3) are relevant for the first three weeks of the year only. Equations 4.2 run for 52 weeks before restarting with initial conditions given by the densities of grouse and ticks at the end of the previous year. After three weeks the grouse chicks enter the adult classes corresponding to their current disease status. The

time in year n of the model simulation is denoted by t_n, with δt a small increment in time. Similarly $G(t)_n,\ T(t)_n$ refer to the grouse and tick densities at time t of year n. The number of grouse chicks entering G_{cs} at the start of each year ($t_n = 0$ is assumed to be mid May when the majority of grouse chick hatching occurs) is given by $(a_g - s_g G(52)_{n-1})G(52)_{n-1}$, where a_g is the grouse hatch rate, s_g is the density dependence rate and $G(52)_{n-1}$ is the total grouse population at the end of the previous year. The number of adult grouse and ticks in each disease class carries forward into the new year. Other parameters are defined in Table 4.1.

$$\left.\begin{aligned}
\frac{dG_{cs}}{dt} &= -b_g G_{cs} - \beta_1 T_i G_{cs} - \underline{P\beta_3 T_i G_{cs}} \\
\frac{dG_{ci}}{dt} &= \underline{P\beta_3 T_i G_{cs}} + \beta_1 T_i G_{cs} - (\alpha + b_g + \gamma)G_{ci} \\
\frac{dG_{cz}}{dt} &= \gamma G_{ci} - b_g G_{cz}
\end{aligned}\right\} \quad 0 \le t_n \le 3, \tag{4.1}$$

$$G_{cs}(0)_n = (a_g - s_g G(52)_{n-1})G(52)_{n-1}, G_{ci}(0)_n = G_{cz}(0)_n = 0,$$

$$G_j(0)_n = G_j(52)_{n-1}, T_j(0)_n = T_j(52)_{n-1},\ j = s, i, z$$

$$\left.\begin{aligned}
\frac{dG_s}{dt} &= -\beta_1 T_i G_s - b_g G_s \\
\frac{dG_i}{dt} &= \beta_1 T_i G_s - (\alpha + b_g + \gamma)G_i \\
\frac{dG_z}{dt} &= \gamma G_i - b_g G_z
\end{aligned}\right\} \quad \begin{aligned} &0 \le t_n \le 52, \\ &G_j(3 + \delta t) = G_{cj}(3) + G_j(3), j = s, i, z \end{aligned} \tag{4.2}$$

$$\left.\begin{aligned}
\frac{dT_s}{dt} &= sc.(a_t - s_t T)T\beta_5 D - b_t T_s - \beta_2 T_s G_i - \underline{\beta_3 T_s G_c} - \beta_5 D T_s \\
\frac{dT_i}{dt} &= \beta_2 T_s G_i - \underline{\beta_3 T_i G_c} - b_t T_i - \beta_5 D T_i
\end{aligned}\right\} \quad \begin{aligned} &0 \le t_n \le 52 \\ &G_c = 0, \quad t_n > 3 \end{aligned} \tag{4.3}$$

With initial conditions $G_{cs}(0)_0 = 170.523,\ G_{ci}(0)_0 = G_{cz}(0)_0 = 0,\ G_s(0)_0 = 13,\ G_i(0)_0 = 5,$
$G_z(0)_0 = 72,\ T_s(0)_0 = 3,988,000,\ G_{cs}(0)_0 = 12,000.$

For simplicity the only large mammal available for tick reproduction in this model is the deer.

Rescaling the parameters Table 4.1 gives the parameters for the seasonal model. The parameters follow from Chapter 2 but are rescaled to be weekly instead of monthly. (Table 2.1 provides references for the estimates.)

The tick reproduction function $(a_t - s_t T)T\beta_5 D$ contains two components s_t and β_5 that are affected by the rescaling of the parameters to weekly from monthly estimates. Consequently this has had to be scaled by $sc = \frac{52}{12}$ to ensure that the total yearly rate of the tick population reproduction remains consistent with the non seasonal model ingestion model (Chapter 2).

4.2.1 Re-paramaterising ingestion

The ingestion parameter had been developed using Gilbert et al. (2004) and was based on the proportion of infection due to ingestion of ticks compared to tick bites. (See Chapter 2 for further details.) It became apparent when initially running the seasonal model simulations using these parameter values the grouse were eating more ticks than is biologically plausible (predicted consumption c.1000 ticks per chick per

Param -eter	Value (unit)	Definition
a_{gs}	2 (y^{-1})	Natural hatch rate of grouse
s_{gs}	0.00128 $(g^{-1}y^{-1})$	Density dependence constraint on grouse
b_g	0.0200769 (w^{-1})	Natural death rate of grouse
α	1.15385 (w^{-1})	Disease induced death rate of grouse
γ	0.288462 (w^{-1})	Recovery rate of infected grouse
a_t	19.23 (w^{-1})	Natural birth rate of ticks
s_t	4.61×10^{-7} $(t^{-1}w^{-1})$	Density dependence constraint on ticks.
b_t	0.0191538 (w^{-1})	Natural death rate of ticks
P	0.0109	The proportion of infected ticks that infect a grouse when ingested
a	varied $(c^{-1}w^{-1})$	The number of ticks ingested by one grouse chick (Sec 4.2.1)
β_1	4.61×10^{-6} $(g^{-1}w^{-1})$	The rate at which a tick bites and infects a grouse.
β_2	$9.75\beta_1$ $(g^{-1}w^{-1})$	The rate a tick bites a grouse and becomes infected
β_3	$12\beta_1$ $(g^{-1}w^{-1})$	The rate ticks are ingested by grouse
β_5	$8.82\beta_1$ $(D^{-1}w^{-1})$	The rate an adult female tick bites a deer and reproduces
sc	$\frac{52}{12}$	A scaling constant to ensure yearly tick reproduction consistent

Table 4.1: The seasonal model parameters. For the units w denotes week, y year, g grouse, c chick, t ticks and D deer.

day depending on deer density). This is likely to be due to either the tick population being too large or the rate of ingestion too great. (This had not been noticed in the non seasonal model as the effect of ingestion cannot be teased apart from other factors contributing to tick decline as all factors are assumed constant over the year. From the within year seasonal model however it can be seen that ticks declined dramatically during the first three weeks when ingestion was occurring.)

The number of ticks in the British Isles is believed to be increasing (Kirby et al., 2004; Scharlemann et al., 2008). The field work discussed in Appendix A suggests that the tick population may often be in the tens of millions per km^2. This is the order of magnitude predicted by this grouse pulse hatch ingestion model. Consequently the parameters that govern the tick population will not be altered.

Grouse chicks are limited in the number of ticks they can physically consume regardless of the density of ticks in the environment. In this model, therefore, we limit the number of ticks that the grouse ingest rather than assume that they ingest x times as many ticks as they are bitten by. Although method 1 of Gilbert et al. (2001) suggests that the grouse were 107 times more likely to eat a tick than be bitten by one we do not know how many ticks they were exposed to. It is unlikely that the ratio of ticks ingested to ticks biting would remain constant with varying conditions. Under different circumstances (e.g., the availability of alternative invertebrate prey species such as Tipulids, Park et al. (2001)) grouse chicks may be infested with many more biting ticks but still ingest only a few a day changing the ratio of ingested ticks to biting ticks.

Methods of constraining ingestion

There are various methods by which the rate of ingestion could be constrained. In particular the following methods were considered: i)setting the rate of ingestion to a fixed value regardless of tick density, ii)making the rate of ingestion a max function, so for $\beta_3 T$ less than a given value, $\beta_3 T$ is used otherwise the maximum value is used, iii)using a saturating function.

i) A fixed rate of ingestion The findings of Gilbert et al. (2004) suggest the chicks eat approx. 5.4 ticks a day on average. However, as this was only a small scale investigation in one location involving few

grouse it does not provide evidence that the same number of ticks would be consumed on other estates with different tick densities. Using a fixed rate of ingestion may also lead to predictions of negative tick numbers if the grouse are still ingesting ticks when the predicted tick population has reduced to zero. This is obviously not biologically possible. Therefore, using a fixed rate of ingestion is not appropriate for this model.

ii) A maximum rate of ingestion In theory it would seem reasonable to model the numbers of ticks being ingested by grouse chicks as $\beta_3 T$ below a maximum value and then capping that value once the chosen maximum has been reached. In practice, however, it has been difficult to implement this accurately in Mathematica.

iii) A saturating function for the rate of ingestion. A Holling Form saturating function allows the predicted number of ticks ingested by the grouse chicks to grow with the tick population but only up to a certain point. Once this level is reached the number of ticks eaten remains constant and realistic.

The grouse pulse hatch with Holling form ingestion model

$$\left.\begin{aligned}
\frac{dG_{cs}}{dt} &= -b_g G_{cs} - \beta_1 T_i G_{cs} - P\frac{T_i}{1+\frac{1}{a}T_s}G_{cs} \\
\frac{dG_{ci}}{dt} &= P\frac{T_i}{1+\frac{1}{a}T_s}G_{cs} + \beta_1 T_i G_{cs} - (\alpha + b_g + \gamma)G_{ci} \\
\frac{dG_{cz}}{dt} &= \gamma G_{ci} - b_g G_{cz}
\end{aligned}\right\} \quad 0 \le t_n \le 3, \tag{4.4}$$

$$G_{cs}(0)_n = (a_g - s_g G(52)_{n-1})G(52)_{n-1},\ G_{ci}(0)_n = G_{cz}(0)_n = 0,\ G, T_j(0)_n = G, T_j(52)_{n-1},\ j = s, i, z$$

$$\left.\begin{aligned}
\frac{dG_s}{dt} &= -\beta_1 T_i G_s - b_g G_s \\
\frac{dG_i}{dt} &= \beta_1 T_i G_s - (\alpha + b_g + \gamma)G_i \\
\frac{dG_z}{dt} &= \gamma G_i - b_g G_z
\end{aligned}\right\} \quad \begin{aligned} &0 \le t_n \le 52, \\ &G_j(3+\delta t) = G_{cj}(3) + G_j(3), j = s, i, z \end{aligned} \tag{4.5}$$

$$\left.\begin{aligned}
\frac{dT_s}{dt} &= sc.(a_t - s_t T)T\beta_5 D - b_t T_s - \beta_2 T_s G_i - \frac{T_s}{1+\frac{1}{a}T_s}G_c - \beta_5 D T_s \\
\frac{dT_i}{dt} &= \beta_2 T_s G_i - \frac{T_i}{1+\frac{1}{a}T_s}G_c - b_t T_i - \beta_5 D T_i
\end{aligned}\right\} \quad \begin{aligned} &0 \le t_n \le 52 \\ &G_c = 0, \quad t_n > 3 \end{aligned} \tag{4.6}$$

With initial conditions $G_{cs}(0)_0 = 170.523,\ G_{ci}(0)_0 = G_{cz}(0)_0 = 0,\ G_s(0)_0 = 13,\ G_i(0)_0 = 5,$ $G_z(0)_0 = 72,\ T_s(0)_0 = 3,988,000,\ G_{cs}(0)_0 = 12,000.$

A comparison of the model predictions with different ingestion rates is discussed, including a Holling function which equates to 5.4 ticks per chick per day ($a = 37.8$) and a Holling function which equates to approx. 20 ticks per chick per day ($a = 140$). This is higher than the average found by Gilbert et al. (2004) but as they could not count larvae and larvae make up a large proportion of the tick population this does not seem unreasonable and could provide an likely estimate of the maximum predicted effect of ingestion.

Larvae emerge free from LIV infection (Gaunt, 1997) and so will not be part of the virus transmission

process. This is reflected in the model by the proportion of ticks which are infected. The ingestion of larvae is important in the model as their loss from the tick population will effect the tick population dynamics.

The exact form of the Holling Function, underlined in the model below, was chosen to ensure that the proportion of infected ticks that are ingested is the same as the proportion of infected ticks in the whole population. Hence both terms have a shared denominator, where a is the approximate number of ticks ingested per chick per week. (Parameters values are given in Table 4.1.)

4.3 The grouse pulse hatch model predictions

Using R_0, the reproductive rate of a virus, (or in the case of the ingestion model the pathogen persistence threshold, PPT) can be a useful way to compare different models of the same system (Chapter 2). These curves depicting the $R_0 = 1$ threshold allow a comparison to be made of the host densities for which the virus is predicted to persist. Comparing these curves for the non seasonal model with and without ingestion showed that in general the model with ingestion included predicted a larger area of virus persistence and hence it was possible to conclude that including ingestion as a route of infection increased the likelihood of LIV persisting.

R_0 can be found for continuous models by analysing the equations (see Chapter 2 for further details). The PPT was found by using the model predictions at different grouse carrying capacities to assess when the grouse did and did not reach that carrying capacity for different deer densities. The grouse carrying capacities for the non seasonal ingestion model could be found by using the formula

$$K_g = \frac{a_g - b_g}{s_g},$$

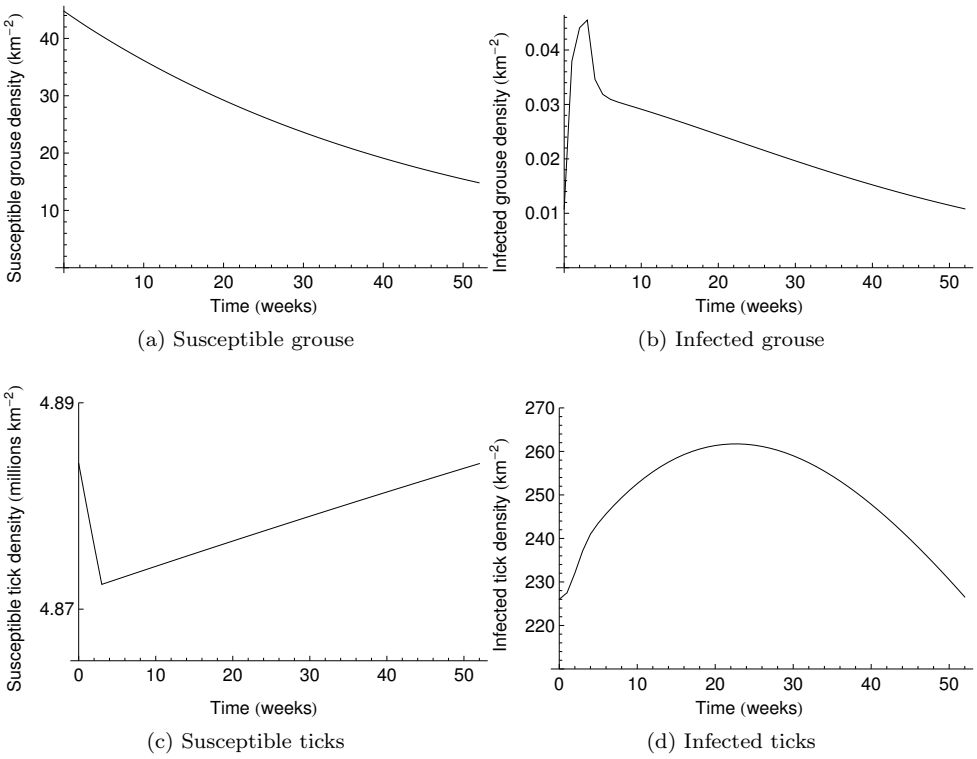
where a_g is the grouse hatch rate, b_g the grouse death rate and s_g the grouse density dependence. The density dependence parameter was varied to give different grouse carrying capacities.

However, the seasonal model cannot be analysed to find R_0 because the equations are now semi-discrete rather than continuous. Similarly the carrying capacity of grouse is no longer given by K_g. The parameters a_g and s_g are yearly estimates as the grouse now reproduce once a year, but b_g is weekly as the grouse are assumed to die naturally at a constant rate throughout the year. Consequently it is not possible to produce either $R_0 = 1$ or PPT curves to assess the range of host densities for which LIV persists. An estimate of the average yearly grouse density can be found by calculating the mean of the weekly predictions for the total grouse density over the course of the year once equilibrium has been reached. This can be used as a proxy for the carrying capacity when there is no virus present. The carrying capacity used for the ingestion model was 240 grouse per km^2 (Chapter 2). This will also be used here to allow comparison between the models. (Although it is theoretically possible to change the value of s_g in the grouse pulse hatch ingestion model to give different yearly average predictions to use in place of a carrying capacity to determine the PPT in practice this is non trivial and time consuming. An alternative method of model comparison will be used.)

In order to compare the models (seasonal and non seasonal, ingestion and no ingestion) the model predictions for grouse densities at different deer densities will be compared for the given grouse carrying capacity of 240 per km^2. The model predictions over time (for a given deer density) will be used to assess the impact of seasonality. The yearly average predictions will be used to assess the impact of ingestion at different deer densities. Grouse can only reach carrying capacity if there is no virus. We can, therefore, determine the deer densities that are predicted to allow virus persistence by considering the predicted grouse densities for different deer densities. If the grouse density is predicted to be at carrying capacity for a given deer density LIV is not persisting, if the grouse density is predicted below carrying capacity the virus is persisting.

The model predictions can determine for which deer densities the grouse reach their carrying capacity meaning virus is unable to persist and for which deer densities the predicted grouse densities are below carrying capacity meaning virus is able to persist. Comparing the predictions for the seasonal model with different levels of ingestion will show the effect of incorporating ingestion on model predictions.

4.3.1 The within year predictions



(a) Susceptible grouse (b) Infected grouse

(c) Susceptible ticks (d) Infected ticks

Figure 4.1: The seasonal model predictions of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick densities within a year approaching equilibrium, after 600 years. (Ingestion 20 ticks per chick per day, deer density 6.5 per km^2. $G_{cs}(0)_0 = 170.523,\ G_{ci}(0)_0 = G_{cz}(0)_0 = 0,\ G_s(0)_0 = 13,\ G_i(0)_0 = 5,$
$G_z(0)_0 = 72,\ T_s(0)_0 = 3,988,000,\ G_{cs}(0)_0 = 12,000)$

Fig. 4.1 shows the model predictions for the dynamics of grouse and ticks during the course of a year after the model has run for 600 years with a deer density of 6.5 per $km^2$and ingestion at 20 ticks per chick per day, equivalent to $a = 140$ the maximum ingestion rate per week given by the Holling function. (The same pattern of behaviour occurs for different deer densities, providing ticks are able

to survive, and different levels of ingestion.) The density of infected grouse and ticks are so low that the predictions of total grouse and tick densities are almost the same as the susceptible grouse and tick densities and consequently the predictions of total grouse and tick densities have not been shown. Infection is a prerequisite for immunity in the grouse population and so it follows that the immune grouse predictions follow exactly the same pattern as the infected grouse and hence the model predictions for the immune grouse class have not been shown. Fig. 4.1a shows the model predicts the susceptible grouse begin the year with a high density because new chicks all hatch free of LIV in one pulse but then the susceptible grouse density steadily declines during the year.

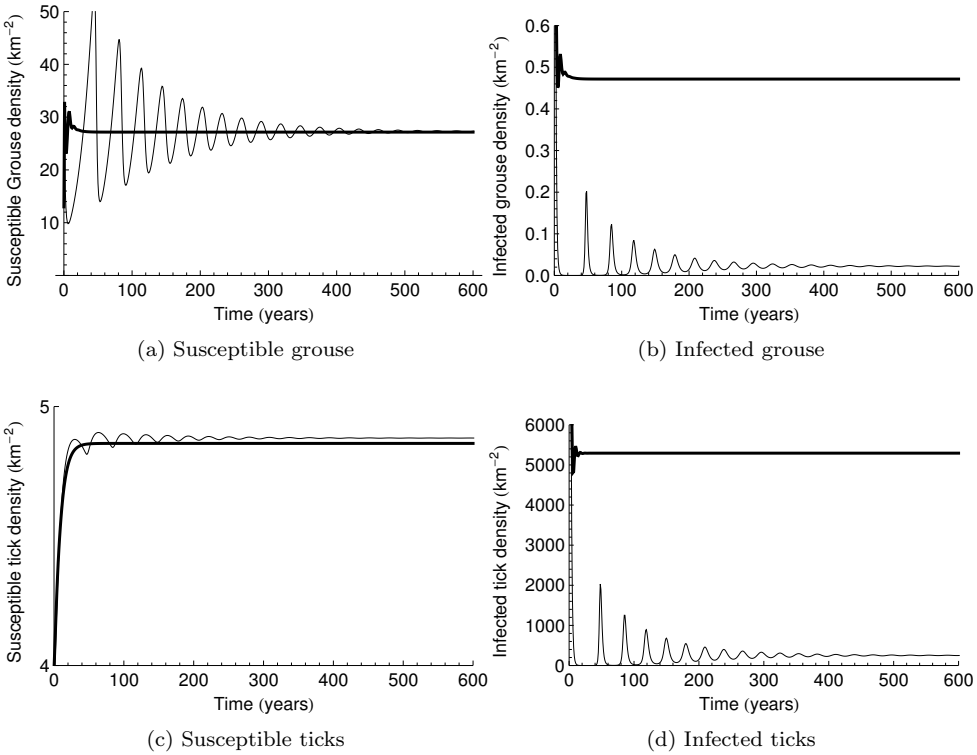
The predicted infected (and immune grouse - not shown) start the year at a low density before experiencing a sharp rise, more than tripling the predicted initial density of infected (immune) grouse, as the chicks ingest ticks and acquire the virus during the first three weeks (Fig. 4.1b). The predicted density then declines for the rest of the year. When ingestion stops after three weeks there is a short (1 week) rapid decline but the infected grouse density then follows a slower steadier decline for the rest of the year. The grouse are now only becoming infected through tick bites so the rate of new infections is slowed down and the actual density of infected grouse is falling as the grouse are dying or recovering from the disease at a faster rate than they are acquiring virus.

Plotted at a suitable scale it is possible to see a (small) decline in the predicted susceptible tick population for three weeks at the start of the year as the ticks are ingested by the grouse (Fig. 4.1c). The tick population then slowly increases for the rest of the year as more emerge but are not eaten by the grouse.

The predicted density of the infected tick population increases reaching a peak level of infection at week 24 before decreasing (Fig. 4.1d). The increase is small for the first week but then shoots up in response to the increase in infected grouse numbers. Whilst there is predicted to be a relatively high number of infected grouse the number of infected ticks continues to rise, but after week 24 the number of infected grouse is not sufficient for the number of infected ticks to keep rising and the infected tick density also declines. (Ticks are dying at a constant rate throughout the year.)

4.3.2 The long term predictions

The long term behaviour of the grouse pulse hatch ingestion model and non seasonal ingestion model predictions with deer density 6.5 per km^2 and ingestion of 20 ticks per chick per day is shown in Fig. 4.2. (The average density of grouse and ticks for each year is plotted for the seasonal model.) For all disease classes the non seasonal ingestion model (thick line) displays a short period of fluctuation in the predicted densities but then settles to an equilibrium value with 25 years. The seasonal ingestion model (thin line) however displays damped oscillations for centuries. The simulations presented here are run for 600 years. The non seasonal model does not show long term damped oscillations which suggests that the seasonality of the model must be the catalyst for this behaviour in the grouse pulse hatch ingestion model. However, it is not clear which aspect of the seasonal model is causing the oscillations. This is investigated further in Sec. 4.4.



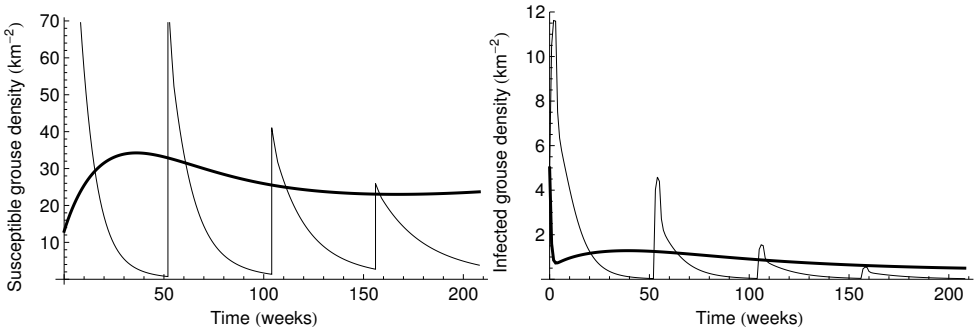
(a) Susceptible grouse (b) Infected grouse

(c) Susceptible ticks (d) Infected ticks

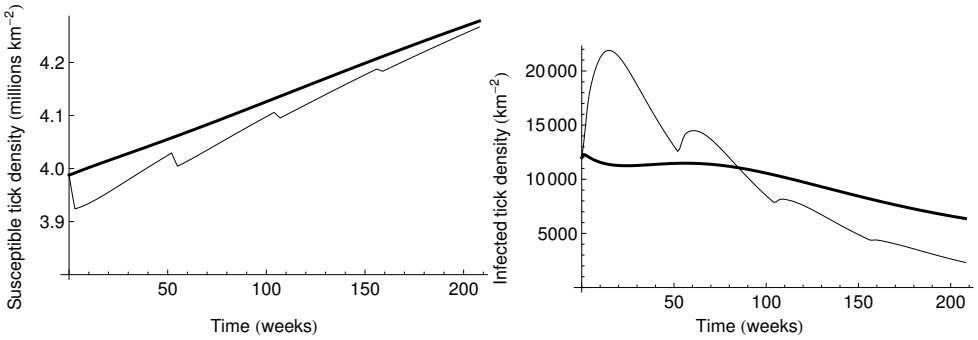
Figure 4.2: The seasonal (thin line) and non seasonal (thick line) model predictions of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick densities over time. (Ingestion 20 ticks per chick per day, deer density 6.5 per km^2).

Whilst both the grouse pulse hatch ingestion model and non seasonal ingestion model predict similar densities of susceptible grouse and ticks in the long term (Figs 4.2a and 4.2c) the density of infected grouse and ticks is much lower for the seasonal model (Figs 4.2b and 4.2d). The within year dynamics help explain the difference in the level of infection between the seasonal and non seasonal model predictions that becomes apparent in the long term. The seasonal model displays a rise and fall in the level of infection during the year with the lowest level at the end of one year and the start of the next year. It is at this time, when the density of infected grouse and ticks are at their lowest, that the grouse chicks enter the equations. Consequently there is a relatively small reservoir of infection within the grouse and tick populations so the opportunity for the grouse chicks to become infected is relatively low. Although the grouse see a sharp rise of infection as the grouse chicks ingest the ticks the actual predicted number of infected grouse is very low. In contrast the non seasonal model averages the grouse hatching and ingestion of ticks over the course of the year, so the level of infection in grouse and ticks is constant over the course of the year once equilibrium is reached. The constant reproduction of grouse also means there is a constant influx of susceptible grouse that can be infected. This means that the level of infection and opportunity for virus transmission is also constant and as a result the grouse and ticks perpetuate the transmission process thus keeping the level of infection high (approx. 20 times more than the seasonal model).

The effect of the rise and fall of the infected grouse and ticks can be seen more clearly if the model predictions are considered from the first few years of running the model (Fig. 4.3). It can be seen that the annual hatch rate of the grouse means that the susceptible grouse population is high at the start of



(a) Susceptible grouse (b) Infected grouse



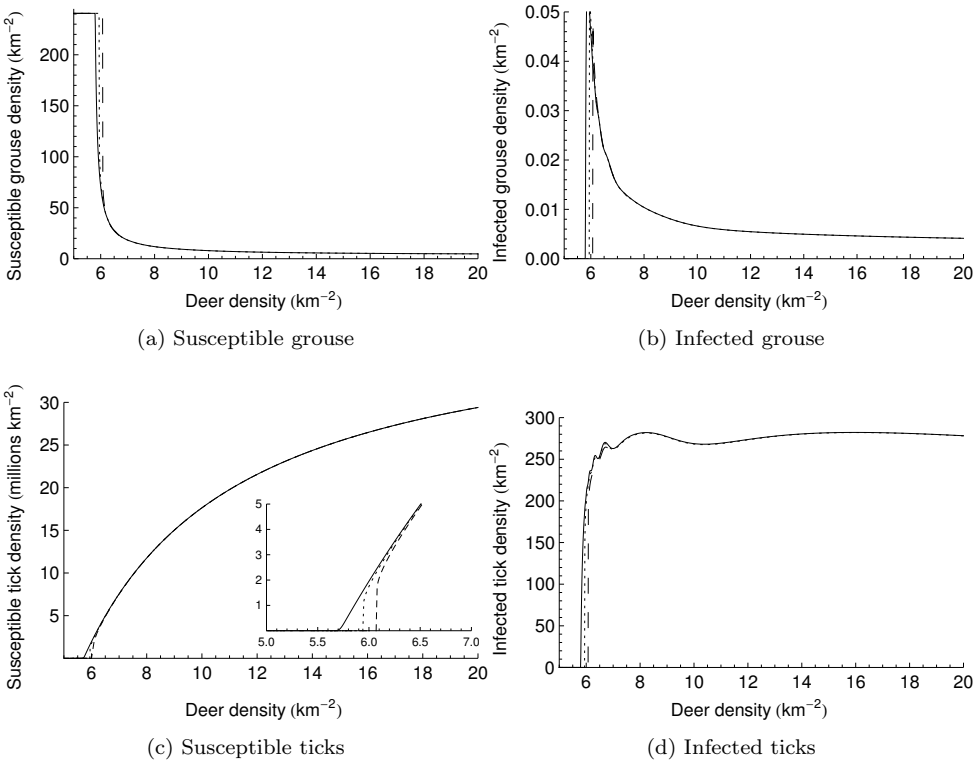
(c) Susceptible ticks (d) Infected ticks

Figure 4.3: The predictions for the seasonal (thin line) and non seasonal (thick line) model of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick densities for the first four years of the simulations. (Ingestion 20 ticks per chick per day, deer density 6.5 per km^2).

the year but falls significantly during the course of the year. This is also true for the infected grouse which means that when the grouse chicks enter the model at the start of the new year the density of infected grouse is very low (Fig. 4.3b), as is the density of infected ticks (Fig. 4.3d). Consequently the opportunity for the new influx of susceptible chicks to acquire infection is low and high levels of infection cannot be reached. The infected tick density also rises and falls throughout the year. After the first year the infected tick density returns to approximately the same density as at the very start so has the potential to rise to a high level again. It is, however, unable to reach such high levels again due to the lack of infected grouse from which the ticks acquire the infection suggesting it is the density of infected grouse that is causing the low levels of infection. Fig. 4.3b shows that even the peak number of infected grouse for the seasonal model is lower than the number in the non seasonal model after 4 years. Infection does persist in the grouse pulse hatch model but at low levels. The non seasonal model does not have the peaks and troughs of infection because grouse are being born and are able to acquire infection via ingestion and tick bites throughout the year. The perpetual influx of susceptible grouse maintains the opportunity for virus transmission and the density of infected grouse and ticks remains relatively high.

4.3.3 The effect of ingestion at different deer densities

Fig. 4.4 shows the equilibrium density predictions of the grouse pulse hatch ingestion model for the susceptible and infected grouse and ticks at different deer densities with and without ingestion at different levels. (For the seasonal model the equilibrium density is the average density over one year when the model is close to equilibrium after 600 years).



(a) Susceptible grouse

(b) Infected grouse

(c) Susceptible ticks

(d) Infected ticks

Figure 4.4: The seasonal model equilibrium density predictions of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick for different deer densities with grouse ingesting 5 (dotted line), 20 (dashed) and 0 (solid) ticks per day.

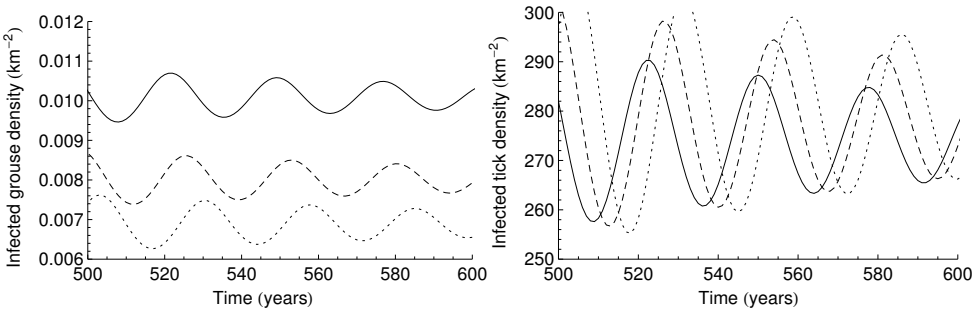
The non seasonal ingestion model (Chapter 2) predicted that for high deer densities the inclusion of ingestion in the model decreased the predicted grouse population (compared to no ingestion) as the additional route of infection made virus persistence more likely. A similar pattern of behaviour occurs for the seasonal model. For deer densities above 7 per km^2 the impact of ingestion on the model predictions of susceptible grouse is to decrease the grouse population. However the effect is slight; even at the higher rate of approx. 20 ticks ingested per chick per day the difference between model predictions cannot be discerned from Fig. 4.4a. The reduced impact of ingestion is probably a result of constraining the number of ticks that the grouse chicks can eat. If the ingestion of ticks is limited it follows that the opportunity for infection to occur in this way is also limited and hence the increase in infection from the addition of ingestion will also be lower than it would be without constraint.

The non seasonal ingestion model suggested that there were some low deer densities for which the inclusion of ingestion made it more difficult for the infection to persist (compared to no ingestion) and the grouse were able to stay at carrying capacity. The grouse chicks are able to ingest sufficient ticks to reduce the tick population below the threshold necessary for virus persistence. Fig. 4.4a indicates that this is also occurring for the seasonal model. For deer densities up to almost 6 per km^2 all the seasonal models predict grouse persisting at the carrying capacity of 240 grouse per km^2 regardless of the level of ingestion. However the inclusion of ingestion keeps the predicted grouse density at 240 per km^2 for slightly higher deer densities than without ingestion, carrying capacity is maintained for up to 6.07 deer per km^2 (20 ticks per chick per day) and 5.95 deer per km^2 (5.4 ticks per chick per day) but only 5.8 deer per km^2 without ingestion. This is reflected in Fig. 4.4c which shows the tick population is 0 up

to 5.7 deer per km^2 for the no ingestion model but until almost 6 deer per km^2 with ingestion (at both rates). The susceptible tick density increases rapidly initially which enables the efficient transmission of virus hence the grouse population is reduced. This occurs at a slightly higher deer density for the higher level of ingestion, further suggesting that the ingestion of ticks by grouse chicks is preventing the tick population from surviving.

The rapid growth of the tick population only occurs for low deer densities. This is highlighted in Fig. 4.4c. Once the deer density is such that the tick population can reproduce at a greater rate than the grouse chicks can ingest the ticks the tick population will amplify. One adult female tick is assumed to be able to reproduce 1000 ticks a year (before density dependence constraints) so the tick population will increase dramatically with every extra adult fed by the increased deer density. This will be most apparent when the tick density is small as the density dependence constraint, $s_t T$, is low. As the tick population increases (with increased deer density) the density dependence constraints will increase and the relative increase in the tick population is reduced.

It is interesting to note the effect ingestion has on the infected grouse and tick densities. For all levels of ingestion the number of infected grouse and ticks increases rapidly when the deer density is such that the tick population is able to survive. However, the density of infected grouse then decreases quickly and remains low as the overall number of grouse is reduced by the virus. For infected tick densities there is a period of fluctuation before prediction settle to an almost constant value. The fluctuations are not apparent in the infected grouse densities, although the curves describing the infected grouse densities are not perfectly smooth as the deer densities increase. The fluctuations of the predicted infected tick densities are insignificant (< 100 ticks) when compared to the size of the total tick population (tens of millions) and are probably a result of the long term dynamics of the seasonal model. The model predictions show damped oscillations and although the simulations presented here ran for 600 years oscillations are still occurring at very low amplitudes and long frequencies. Oscillations were found to occur for all deer densities (6.5-25 per km^2) and ingestion levels (0, 5.4, 20 ticks per chick per day) tested. The deer density effects the timing of the oscillation (Fig. 4.5) and the infected tick populations recorded here are at slightly different phases of this cycle. As for the susceptible grouse and tick populations the effect of ingestion is small and is only apparent at the lowest deer density where infection is able to persist.

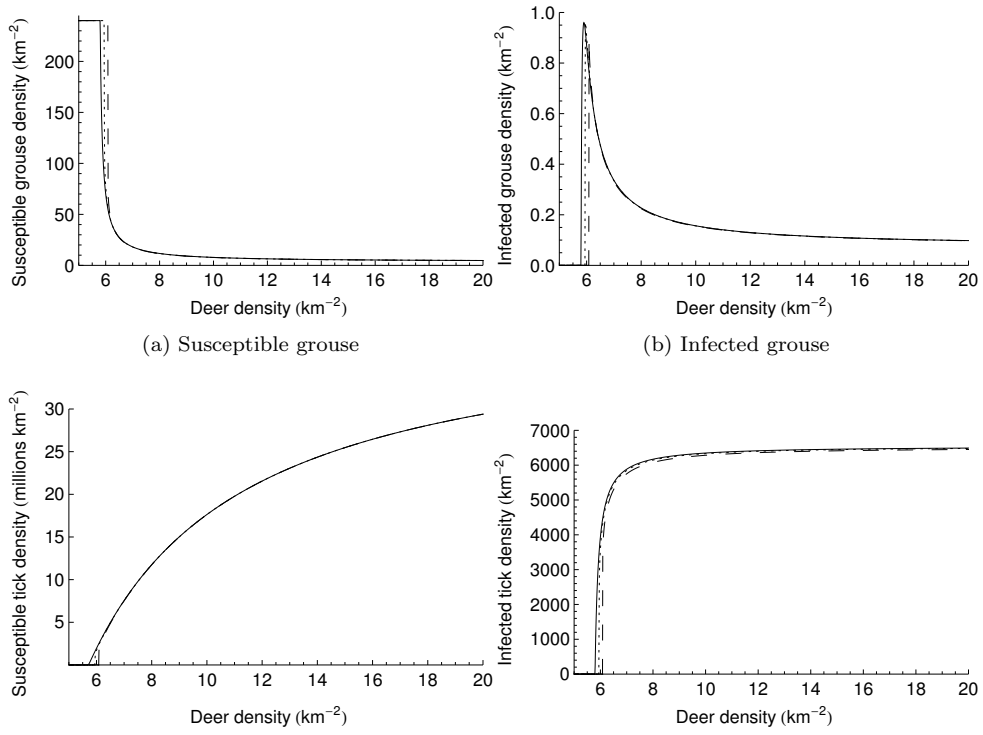


(a) Infected grouse (b) Infected ticks

Figure 4.5: The seasonal model predictions of a)infected grouse, b)infected ticks over time (between 500 and 600 years) for different deer densities of 10 (solid), 9 (dashed), 8 (solid) deer per km^2.

The change in phase of the oscillations could lead to the perceived fluctuations in the predictions of the infected tick densities for different deer densities and the lack of smoothness in the predictions of the infected grouse densities. For example Fig. 4.5b clearly shows the different phases of the oscillations of the model predictions for different deer densities. The model predictions used to compare the effect of deer are taken at 600 years, as the oscillations have become small at this point and running the simulations longer is temporally expensive. However, plotting the predictions at the above scale shows that oscillations are still occurring and using the prediction at 600 years will take the lowest point of the oscillation for 10 deer per km^2 but for 8 deer per km^2 the oscillation is almost midway. Although these differences are small for the overall tick population they are large enough to cause the apparent fluctuations of the infected ticks shown in Fig. 4.4 of Section 4.3.5. It is important to note that whilst these mathematical differences exist the differences are too small to be detectable from field data.

4.3.4 The non seasonal model with Holling form ingestion



(a) Susceptible grouse

(b) Infected grouse

(c) Susceptible ticks

(d) Infected ticks

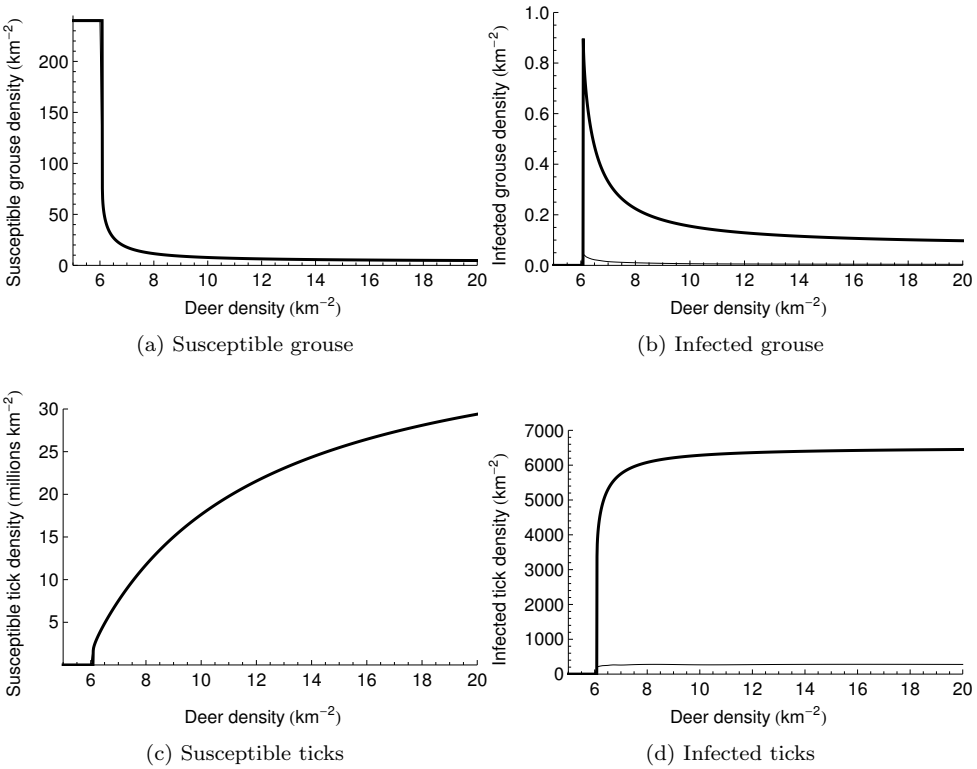
Figure 4.6: The non seasonal model predictions of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick densities for different deer densities with grouse ingesting 5 (dotted line), 20 (dashed) and 0 (solid) ticks per day.

The ingestion parameter of the seasonal model has been constrained to biologically plausible limits. In order to make meaningful comparisons between the seasonal and non seasonal models it was necessary to incorporate a Holling Form ingestion parameter in the non seasonal model. The non seasonal model averages all behaviour through the course of the year and so the three week ingestion must also be averaged throughout the year, meaning that the Holling Form parameter values had to be altered slightly for the non seasonal model. In the seasonal ingestion model the parameter a is the approximate number of ticks ingested per grouse per week (37.8 for 5.4 ticks per chick per day and 140 for 20 ticks per chick per

day) and is only in the model for three weeks. The non seasonal model needs to distribute the behaviour of these three weeks across the year. If grouse ingest 5.4 ticks per chick per day then over three weeks they ingest 113.4, averaged over a year this gives approx. 2.18 per week. A similar argument follows for 20 ticks per chick per day. The equations for the non seasonal model with a Holling Form of ingestion (the non seasonal ingestion model) are given in Appendix B.

The predictions for the non seasonal ingestion model at different deer densities are shown in Fig. 4.6. The impact of ingestion with a Holling Form on the non seasonal model is reduced from that predicted by the unlimited ingestion model presented in Chapter 2, Fig. 2.7b. Fig. 4.6 shows the predictions of the non seasonal ingestion model all follow the same pattern of behaviour regardless of the rate of ingestion and is comparable with the effect of ingestion on the grouse pulse hatch ingestion model predictions (Sec. 4.3.3).

4.3.5 The grouse pulse hatch model compared to the non seasonal model



(a) Susceptible grouse (b) Infected grouse

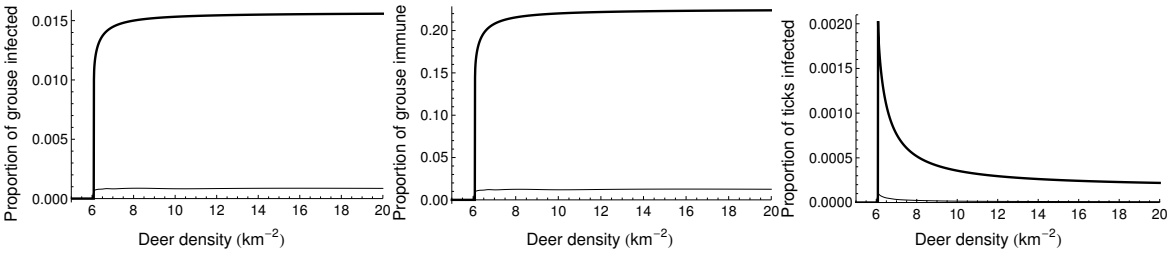
(c) Susceptible ticks (d) Infected ticks

Figure 4.7: The seasonal (thin line) and non seasonal (thick line) ingestion model predictions of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick densities for different deer densities. (Ingestion of 20 ticks per chick per day)

Fig. 4.7 shows the general pattern of behaviour of the seasonal model is the same as for the non seasonal model. The grouse population stays at carrying capacity and the tick population at 0 until approx. 6 deer per km^2 in both models. The impact of ingestion on the grouse and tick population predictions is small in both seasonal and non seasonal models. The inclusion of ingestion initially allows grouse to stay at carrying capacity for slightly higher deer densities as the tick population is kept at 0 by grouse ingesting ticks. For higher deer densities the ingestion of ticks does then become an extra route of infection causing the grouse density prediction to be slightly lower in model predictions with ingestion

(compared to without ingestion), although this cannot be distinguished from Figs. 4.4, 4.6.

The main difference between the seasonal and non seasonal model is the level of infection in grouse and ticks. Although the susceptible grouse and tick densities are virtually the same in the seasonal and non seasonal model the infected grouse and tick densities are an order of magnitude higher in the non seasonal model. This difference must be due to the seasonality of the model as all other parameters have been kept equal or chosen to be equivalent where necessary. As discussed in Sec. 4.3.2 the difference is due to the within year dynamics of the seasonal model. The non seasonal model has constant reproduction of susceptible chicks so the density of susceptible grouse is always high enough to ensure infection can occur easily. Consequently there is a relatively high level of infection. The grouse pulse hatch model however only has reproduction in one pulse when infected grouse and tick numbers are low so the chances of these grouse becoming infected and able to transmit infection is low.



(a) Proportion of infected grouse (b) Proportion of immune grouse (c) Proportion of infected ticks

Figure 4.8: The seasonal (thin line) and non seasonal (thick line) model predictions of the proportion of a)infected grouse, b)immune grouse, c)infected ticks for different deer densities.

Fig. 4.8 shows the proportion of grouse in the infected and immune classes and the proportion of ticks in the infected class for the seasonal (thin line) and non seasonal (thick line) models. The proportion of susceptible grouse and ticks show the direct opposite pattern of behaviour (not shown). For both models the infected and immune grouse classes follow exactly the same pattern: immunity can only occur following infection. The proportion of grouse in these classes rises sharply when deer densities reach the threshold required for the virus to persist. After this sudden increase the proportion of infected and immune grouse increases at a much slower rate with further deer density increase. At these deer densities the actual grouse population is already very low and affected little by the increase in the tick population caused by higher deer densities, hence the increase in the proportion of infected grouse is small. The proportion of infected ticks falls with increasing deer densities. The increase in deer density increases the opportunity for ticks to reproduce so the overall tick population increases. The actual number of ticks predicted to become infected increases very little as deer density increases (because the grouse population is so low and the opportunity for infection to be transmitted is low) but the overall tick population continues to increase as deer density increases. Consequently the proportion of ticks that are infected decreases.

Despite both models displaying the same overall pattern of behaviour in equilibrium density predictions for different deer densities Fig. 4.8 highlights the difference in the amount of infection between the seasonal and non seasonal model. The proportion of grouse infected in the non seasonal ingestion model

is over ten times more than for the seasonal ingestion model. Similarly the proportion of immune grouse is around 20 times greater in the non seasonal ingestion model compared to the seasonal ingestion model. The greatest difference is seen in the predictions for the proportion of ticks that are infected between 6 and 7 deer per km^2 although at higher deer densities the difference is reduced to be similar to the difference seen for the grouse.

4.4 The grouse hatching period model

In the field virus prevalence varies from site to site but has been recorded as 0.003 in nymphs (Gaunt, 1997). The proportion of questing ticks found during blanket dragging that are nymphs varies from site to site and on the time of year (Randolph et al. (2002), Gilbert, unpublished data). Assuming nymphs make up approx. one tenth to one half of the total tick population during the peak feeding period we can estimate that virus prevalence in the total tick population could be between 0.0003 and 0.0015. Watts et al. (2009) find higher but variable (between years and sites) prevalence in ticks ranging between 0.018 and 0.153. The grouse pulse hatch model predicts infection prevalence in ticks at around 0.00002 which is considerably lower than published estimates.

In the grouse pulse hatch ingestion model the grouse hatching happens in one pulse as a result of the initial conditions for the model at the start of each year. Although this is closer to the seasonal hatching of grouse than averaging hatching throughout the year it is still a simplification the natural hatching period of grouse chicks. Grouse chicks do not hatch simultaneously within one brood let alone across a whole estate. Grouse chicks will normally hatch over a period time on one estate and a seasonal model allowing the grouse to hatch over a number of weeks rather than simultaneously may display a more realistic pattern of behaviour over time. In order to compare this type of seasonal hatching to that described by the grouse pulse hatch model a grouse hatching period model is presented in this section.

The grouse hatching period model incorporating a period of hatching for grouse chicks is given below. (Ingestion has been removed from this model as the effect of ingestion was shown to be small when the number of ticks consumed is limited by a Holling Function. In order to make meaningful comparisons between models ingestion is not included in any of the models presented in this section.)

$$\left.\begin{aligned}
\frac{dG_s}{dt} &= (a_{gh} - s_{gh}G(0)_n)G(0)_n - b_gG_s - \beta_1T_iG_s \\
\frac{dG_i}{dt} &= \beta_1T_iG_s - (\alpha + b_g + \gamma)G_i \\
\frac{dG_z}{dt} &= \gamma G_i - b_gG_z \\
\frac{dT_s}{dt} &= (a_t - s_tT)T\beta_5D - b_tT_s - \beta_2T_sG_i - \beta_5DT_s \\
\frac{dT_i}{dt} &= \beta_2T_sG_i - b_tT_i - \beta_5DT_i
\end{aligned}\right\}
\begin{aligned}
&\\
&\\
&0 \leq t_n \leq 52, \\
&G, T_j(0)_n = G, T_j(52)_{n-1},\ j = s, i, z \\
&a_{gh}, s_{gh} = 0 \text{ for } \ w < t_n \leq 52
\end{aligned}$$

As with the grouse pulse hatch ingestion model the grouse hatching period model runs for a year before restarting with initial conditions given by the density of grouse and ticks in each disease class at

the end of the previous year. The length of the hatching period is given by w weeks. The reproduction of the grouse now occurs over the course of w weeks, rather than over year (as in the non seasonal model) or all at once (as in the grouse pulse hatch model). Consequently the parameters concerning grouse reproduction have become $a_{gh} = \frac{a_{gs}}{w}$, that is the yearly rate distributed over w weeks and the density dependence s_{gh} is estimated from the model to ensure a carrying capacity of approx. 240 grouse when there is no disease. This is part of the model only for the first w weeks of the year, after this no reproduction of grouse occurs ($a_{gh}, s_{gh} = 0 \quad \text{for } w < t \leq 52$). (All other parameters are as the grouse pulse hatch model. Table 4.1) In the non seasonal model the number of grouse hatching at time $t + \delta t$ depends on the number of grouse at time t. In effect this means that grouse that hatched at time t can contribute to the number hatching at time $t + 1$, although this is not biologically realistic neither is the assumption that grouse hatch at a constant rate throughout the year. The combined assumptions lead to a simple non seasonal model that is useful for making predictions on average grouse population behaviour. However, as the model is now being made seasonal and hatching occurs for four weeks only it does not make sense for grouse that hatched in week one, for example,to contribute to the number hatched in week two. Consequently the grouse reproduction in the weeks of hatching depend only on those grouse that were there at the start of the year in week zero.

Chicks usually hatch from late May to early June (Hudson et al., 1995), the length of the hatching period is taken as one month ($w = 4$).

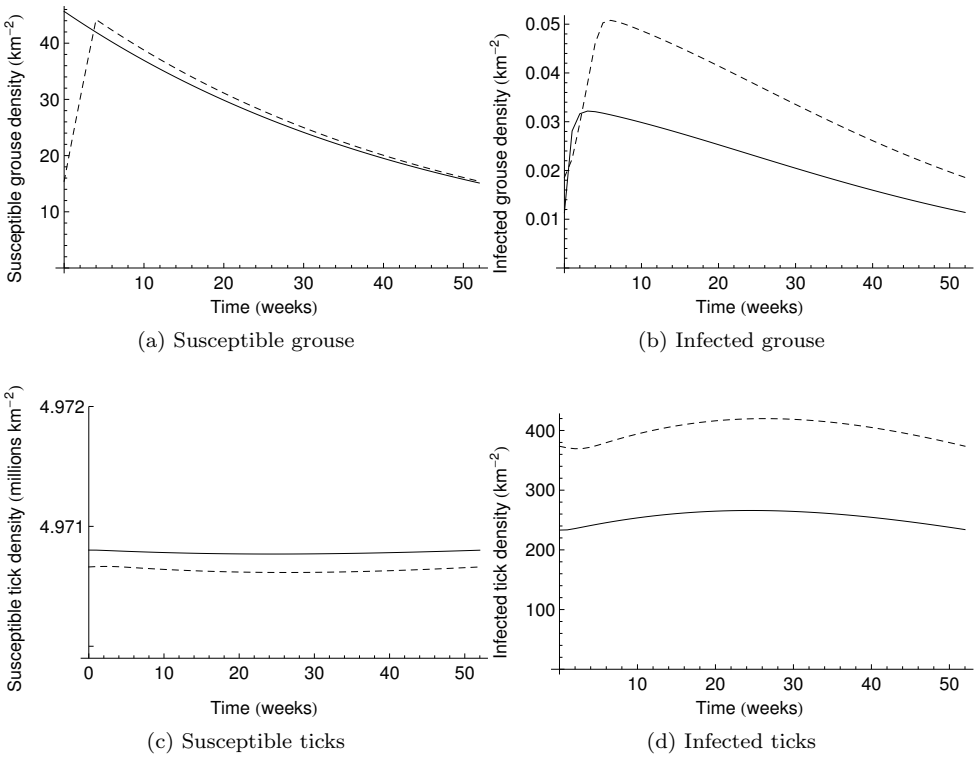
4.5 The grouse hatching period model predictions

4.5.1 The within year predictions

The model predictions within one year for the two seasonal models with a deer density of 6.5 per km^2 are shown in Fig. 4.9. The grouse hatching period of four weeks can be seen for the hatching period model (dashed line) as the susceptible grouse population grows almost linearly for four weeks (Fig. 4.9a). Although the density of the susceptible grouse at the end of the year is the same for both models the grouse hatching period model does not predict as high a peak in susceptible grouse after the hatching period as the grouse pulse hatch model this is due to the natural death rate and infection occurring within those grouse chicks born in the preceding weeks. The grouse hatching period model predicts a slightly higher susceptible grouse density for the remaining 48 weeks of the year once the peak is reached. Despite the difference in the within year dynamics of the susceptible grouse population the two models have very similar averages, 27.3 and 27.7 for the grouse hatching period model and grouse pulse hatch model respectively.

The within year predictions for the susceptible tick population is given in Fig. 4.9c. By choosing a suitable scale a very small numerical difference can be seen between the two models, with the grouse pulse hatch model predicting very slightly more ticks than the grouse hatching period model.

Fig. 4.9b shows a much greater difference in the model predictions for the infected grouse density. The infected grouse density for the grouse pulse hatch model (thin line) only increases for the first three



(a) Susceptible grouse (b) Infected grouse

(c) Susceptible ticks (d) Infected ticks

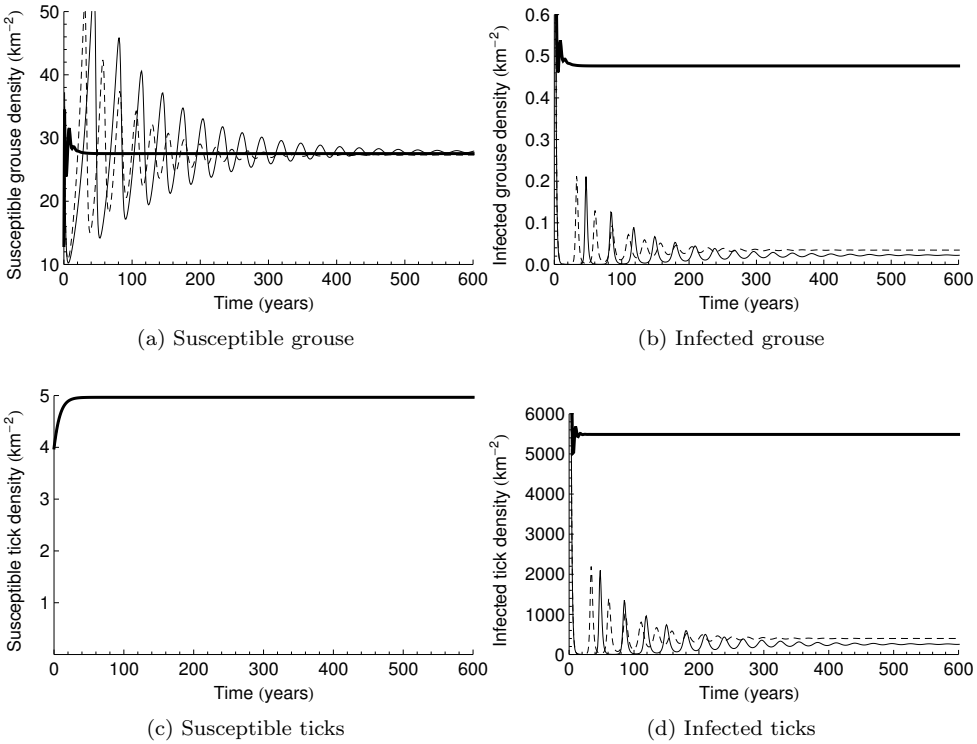
Figure 4.9: The grouse hatching period (dashed line) and pulse hatch (thin line) model predictions of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick densities during the course of a year. (Deer density 6.5 per km^2).

weeks but the grouse hatching period model (dashed line) predicts a rise for the first six weeks. This is a result of the hatching of newly susceptible chicks over four weeks. Each week more of these naive grouse are able to become infected as they interact with the ticks infected by the previously infected grouse. The grouse hatching period model has an approx. 50% higher average of 0.035 infected grouse compared with 0.021 in the grouse pulse hatch model. Although the grouse pulse hatch model has a large influx of susceptible grouse at the start of the year it occurs at one single time point so immediately all the grouse are able to become infected or die naturally and the susceptible grouse density quickly falls. This consequently means there is less chance of these grouse becoming infected and further infecting ticks. The infected tick densities also show slight differences between models, with the grouse hatching period model predicting approx. 400 infected ticks, over 50% more than the 250 predicted by the grouse pulse hatch model. Interestingly the yearly average for the total tick population is identical for both models (4971035 ticks per km^2) and the only difference in the susceptible tick population is due to the number of ticks infected.

Although the model predictions are showing numerical differences in the predictions these differences are actually slight and would not be detectable in field data.

4.5.2 The long term predictions

The long term predictions of the two seasonal models and the non seasonal model with a deer density of 6.5 per km^2 are shown in Fig. 4.10. The long term behaviour of the grouse hatching period model (dashed line) shows damped oscillations, but these oscillations are of a shorter frequency and decay away



(a) Susceptible grouse (b) Infected grouse

(c) Susceptible ticks (d) Infected ticks

Figure 4.10: The non seasonal (thick solid line), grouse pulse hatch (thin solid line) and grouse hatching period (dashed line) model predictions of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick densities over time. (Deer density 6.5 per km^2).

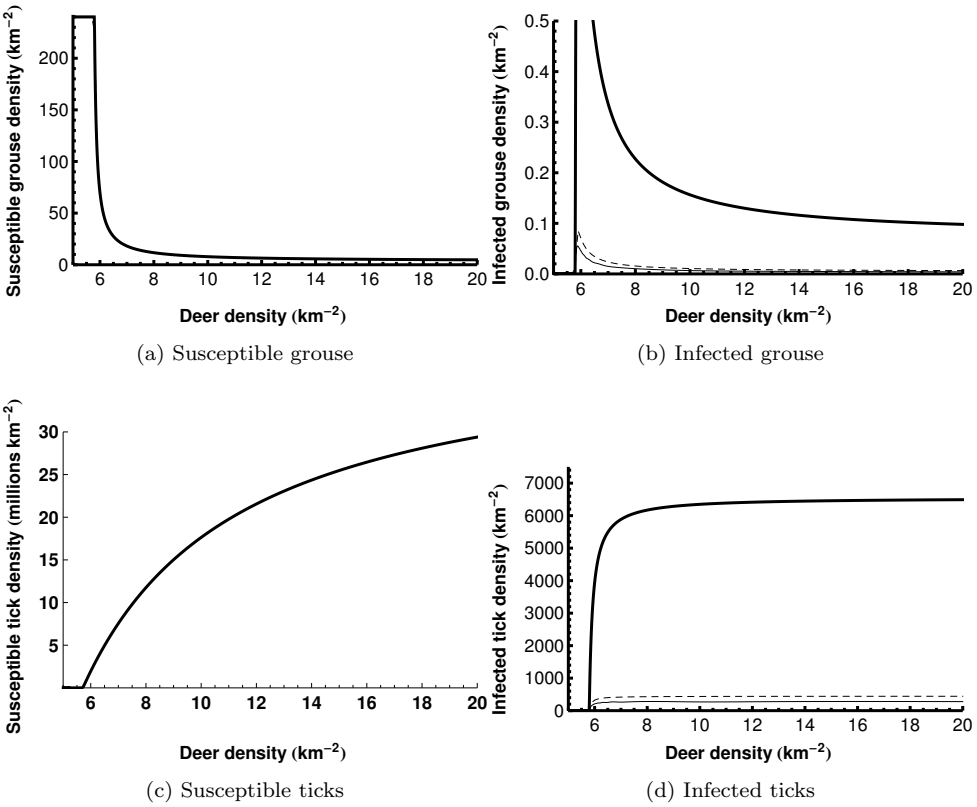
much faster than the oscillations of the grouse pulse hatch model (thin line). The non seasonal model (thick line) shows only slight initial fluctuations which have died out within 25 years. (Although the initial conditions for the models will obviously effect the numerics at the start of the oscillations the speed of decay and equilibrium density remain the same for each model.)

The predictions of the susceptible grouse densities seem to be approaching a very similar long term equilibrium for each of the models (Fig. 4.10a). The yearly average given by averaging over the within year dynamics highlighted in Sec 4.5.1 suggests that the grouse pulse hatch model predicts 27.7 grouse per km^2 with the grouse hatching period model 27.3 per km^2, but as the oscillations are still occurring all-be-it at a very low amplitude, the actual equilibrium densities remain unknown. The non seasonal model predicts a susceptible grouse density of 27.5 per km^2. Due to the sheer size of the tick population no oscillations are discernible for the susceptible tick density and all models predict densities of similar size (Fig. 4.10c).

The infected grouse density shows similar oscillations but a difference in the predicted density of infected grouse for the different models can be seen quite clearly. Sec. 4.5.1 suggested that the grouse hatching period model has a higher level of infection than the grouse pulse hatch model which is apparent again in Fig. 4.10b. It also clear however that the non seasonal model predicts infected grouse equilibrium densities an order of magnitude higher than both seasonal models. This suggests that it is not purely the incorporation of the seasonal grouse hatching in one pulse that causes the low infection rates and the oscillations. (This will be explored further in Sec. 4.6.2.) The relationship between the predicted infected tick densities of the different models is similar to that of the infected grouse densities.

It is interesting to consider the proportion of infected grouse and ticks to make a more reliable comparison of the level of infection each model predicts. The level of infection in the two seasonal models is virtually the same with grouse hatching period model and grouse pulse hatch model having infected grouse proportions of 0.0012 and 0.00078 respectively and infected tick proportions 0.000081 and 0.000051. The level of infection in the non seasonal model is, not surprisingly, much higher with the proportion of infected grouse 0.014 and the proportion of infected ticks 0.0011. This indicates that the non seasonal model does indeed have a level of infection an order of magnitude higher than the seasonal models. The reasons for this will be explored further in Sec. 4.6.2.

4.5.3 The effect of different deer densities



(a) Susceptible grouse (b) Infected grouse

(c) Susceptible ticks (d) Infected ticks

Figure 4.11: The non seasonal (thick solid line), grouse pulse hatch (thin solid line) and grouse hatching period (dashed line) model predictions of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick densities for different deer densities.

Fig. 4.11 shows the model predictions of average equilibrium grouse and tick densities for the two seasonal models and the non seasonal model for different deer densities. The general trend of the predictions for all three models is the same and no discernible difference can be seen for the susceptible grouse densities (Fig. 4.11a). For low deer densities (< 6 per km^2) the grouse are able to reach carrying capacity and for higher deer densities the predicted susceptible grouse density falls dramatically. Higher deer densities allow higher tick densities and higher densities of infected ticks increase the opportunity for virus transmission (Fig. 4.11d).

Although the general trend follows for all three models the predicted average equilibrium densities of grouse and ticks at different deer densities are not the same. This is most apparent for the predicted

infected densities of grouse and ticks which follow logically on from the long term predictions of Sec. 4.5.2. The predicted equilibrium density of the infected grouse and tick populations is considerably higher for the non seasonal model whilst the two seasonal models have similar predicted equilibrium densities with the grouse hatching period model just slightly higher than the grouse pulse hatch model.

4.6 The effect of model components

The reproduction of grouse needs to be incorporated in to the model and the grouse pulse hatch model and non seasonal model are opposite extremes of doing so and give the different predictions outlined above. The grouse pulse hatch model assumes all chicks hatch simultaneously, the non seasonal model assumes chicks hatch constantly throughout the year. The grouse hatching period model forms a compromise, allowing chicks to hatch for a short period. Both seasonal models show some distinct differences to the predictions of the non seasonal model in long term predictions and for equilibrium densities of grouse and ticks at different deer densities but show similarities to each other. The seasonal models both show damped oscillations in long term dynamics. Once equilibrium is reached the seasonal models both predict a much lower level of infection than the non seasonal model for all deer densities for which the virus persists.

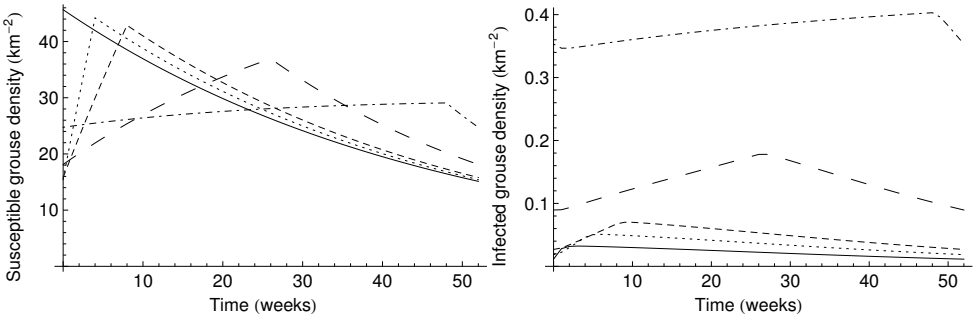
There are, however, differences in the long term and equilibrium density predictions of the grouse hatching period model and grouse pulse hatch model. The damped oscillations of the grouse hatching period model decay more rapidly than the grouse pulse hatch model. The level of infection at equilibrium is slightly higher for the grouse hatching period model than the grouse pulse hatch model for all deer densities for which virus persists. These differences are interesting and with further investigation may explain the bigger differences to the non seasonal model.

4.6.1 Changing the length of the grouse hatching period

This section considers the effect on model predictions of changing the length of the hatching period. Hatching periods of 8, 26 and 48 weeks are compared; although not biologically realistic they help to explain the pattern of model predictions.

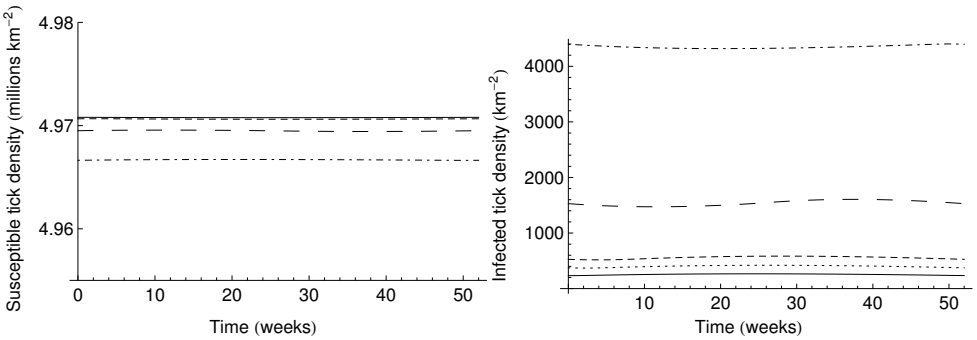
Fig. 4.12 shows that lengthening the period of hatching within the model leads to very different within year dynamics. Although all the hatching periods lead to a similar yearly average of susceptible grouse (Fig. 4.12a) and susceptible ticks (Fig. 4.12c) the infected grouse and tick densities show large differences. As the length of the hatching period is increased the density of infected grouse (Fig. 4.12b) and ticks (Fig. 4.12d) is increased.

To incorporate the extended length of hatching in to the models requires s_g the density dependence to be changed so that the grouse reach carrying capacity (240 ± 0.1) in the absence of ticks. This means that the yearly average susceptible density will be similar for all hatching periods as the model is designed so that approximately the same number of susceptible grouse hatch but over different time periods. If s_g is not decreased as the hatch period is increased the density dependence on grouse will be acting at too



(a) Susceptible grouse

(b) Infected grouse



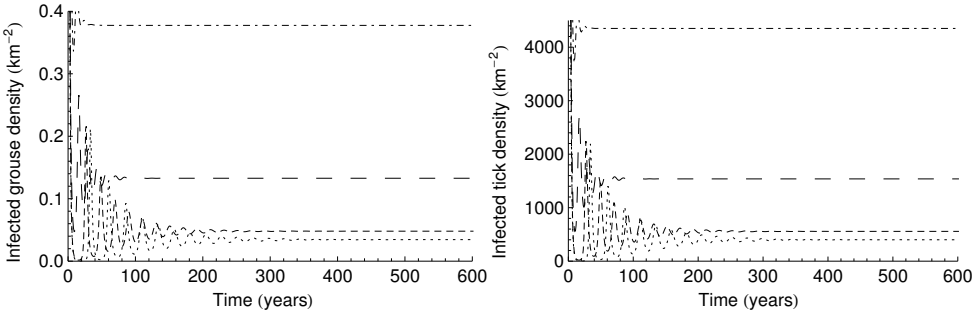
(c) Susceptible ticks

(d) Infected ticks

Figure 4.12: The grouse hatching period model predictions of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick densities during the course of a year with hatching periods of 4 (dotted), 8 (small dash), 26 (long dash) and 48 (dotdashed) weeks. The thin line represents the pulse hatch model. (Deer density 6.5 per km^2).

great a rate and consequently the grouse population prediction is unable to reach a high enough density.

A longer hatching period has a smaller hatching rate per week so consequently the susceptible grouse density is reasonably high for much of the year compared to a shorter hatching period. As a result the opportunity for susceptible grouse to interact with infected ticks is also reasonably high, leading to higher number of grouse becoming infected in models with a longer hatching period. These infected grouse can then infect a greater number of ticks and so the density of infected ticks is also higher in models with a longer hatching period.



(a) Infected grouse

(b) Infected ticks

Figure 4.13: The model predictions of a)infected grouse b)infected ticks over time. The various dashed lines represent different hatching periods of 4 (dotted), 8 (small dash), 26 (long dash) and 48 (dotdashed) weeks. (Deer density 6.5 per km^2).

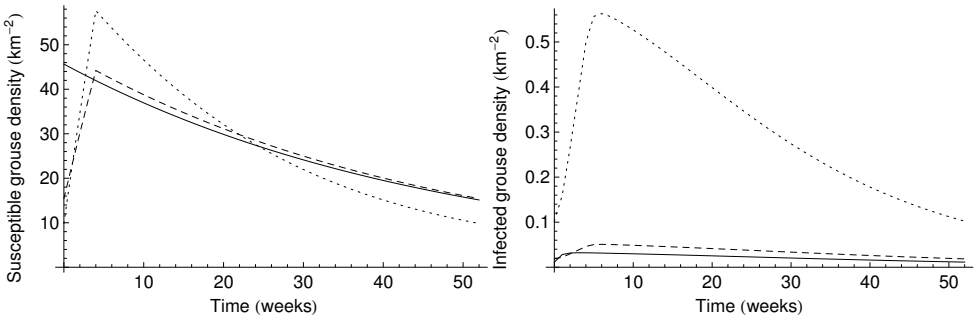
As well as affecting the within year dynamics and consequent levels of infection the length of the hatching period also affects the frequency, amplitude and decay of the long term model oscillations. Fig.

4.13 shows the long term predictions for the infected grouse and tick densities when there are 6.5 deer per km^2. (The susceptible densities are omitted because the predictions of the different models are not easily distinguished.)

Fig. 4.13a and 4.13b both show that as the hatching period increases the density of infected grouse and ticks increases and the time for the oscillations to decay decreases. This suggests that the within year dynamics of the hatching period are resonating with the underlying fluctuations at the beginning of the non seasonal model time series predictions. Long time periods show smaller variation of the grouse and tick densities within a year and consequently do not resonate to the same extent as the short time period that have larger within year variation. The same effect occurs for all deer densities for which virus persists.

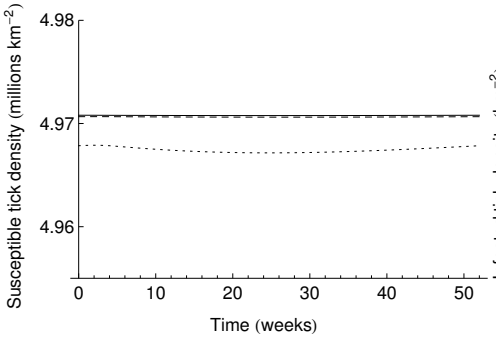
4.6.2 Changing the birth rate

Another factor that influences the within year dynamics is a_g the birth rate itself. As the hatching period was altered in the section above the birth rate was altered simultaneously so that the overall hatching rate throughout the year was consistent for all models. Each grouse is assumed to give rise to two chicks in a year, whether this is in one instant (as in pulse hatch model) or over the course of n weeks (as in grouse hatching period model and non seasonal model). To explore the model further in this section the hatching period is kept at four weeks but the birth rate is increased. The current hatch rate is 0.5 chicks per grouse per week.

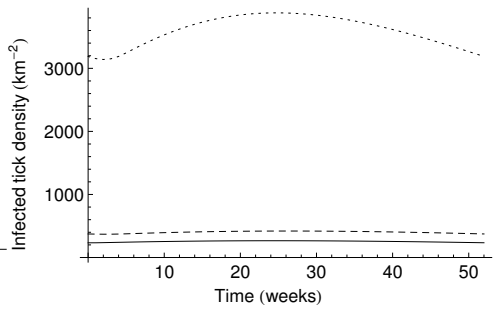


(a) Susceptible grouse

(b) Infected grouse



(c) Susceptible ticks



(d) Infected ticks

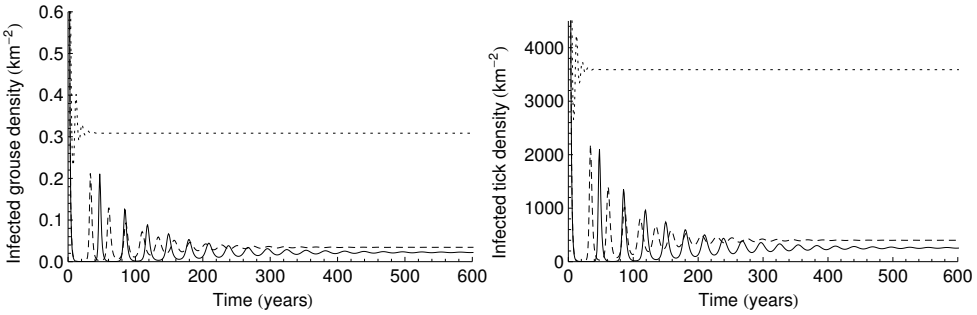
Figure 4.14: The grouse hatching period model predictions of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick densities during the course of a year with 0.5 (dashed) and 1 (dotted) eggs per grouse per week. The thin line represents the pulse hatch model. (Deer density 6.5 per km^2).

Fig. 4.14 shows the effect of doubling the hatch rate of the grouse on the within year predictions

when there are 6.5 deer per km^2. The density dependence rate is also adjusted to ensure that the grouse reach the carrying capacity of approx 240 when there are no ticks and disease. The predictions of the pulse model are also included for comparison.

Fig. 4.14a shows the susceptible grouse are predicted to reach a higher peak density when the birth rate is doubled to have a birth rate of 1 chick per grouse per week (dashed line) compared to the previous 0.5 (dotted line) chicks per grouse per week but the equilibrium predicted susceptible tick population is of a similar size for all models.

The difference in model predictions is much more apparent for the infected grouse density. In Fig. 4.14b the density of infected grouse is much higher throughout the year when the birth rate is doubled. The same effect can be seen in Fig. 4.14d for the infected tick population. Although the peak of susceptible grouse is only increased by about a quarter when the birth rate is doubled this is sufficient to increase the peak infected grouse density by an order of magnitude. A small proportional increase in susceptible grouse causes a much larger proportional increase in infected grouse because the opportunity for ticks to transmit infection to grouse is increased. One infected grouse can infect several more ticks quickly amplifying the opportunity for infection transmission as these infected ticks have the potential to each infect a grouse.



(a) Infected grouse (b) Infected ticks

Figure 4.15: The grouse hatching period model model predictions of a)infected grouse b)infected tick densities over time with 0.5 (dashed) and 1 (dotted) eggs per grouse per week. The thin line represents the pulse hatch model. (Deer density 6.5 per km^2).

Doubling the birth rate also has an effect on the long term dynamics of the model predictions. Fig. 4.15 shows the long term predictions for the infected ticks and grouse when there are 6.5 per km^2. The model reaches equilibrium at a much faster rate when the birth rate is doubled (dotted line compared to dashed line). The greater birth rate introduces a greater variation within one year so one might expect greater oscillation in the long term predictions rather than fewer, as discussed in Sec. 4.6.1. This would occur due to resonance with oscillations in the non seasonal model which also has a greater birth rate. We expect the grouse hatching period model to show more fluctuations than non seasonal model with the same parameters. Although the grouse hatching period model with the higher rate shows fewer oscillations than the grouse hatching period model with lower rate it if the non seasonal model is run with the birth rate doubled (and s_g adjusted accordingly) then the long term predictions show no fluctuations (predictions not shown) and so the grouse hatching period model with the same birth rate does in fact cause more oscillations than the non seasonal model.

The long term dynamics back up the within year predictions. The equilibrium predicted infected density of grouse and ticks for the model with the increased birth rate are much greater than the original birth rate. The same effect can be seen for all deer densities for which virus persists.

4.6.3 Changing the tick and grouse interactions

The level of infection in the grouse hatching period model is low when compared to the non seasonal model. The level of infection can be increased by increasing the birth rate or length of the hatching period but there is no biological basis for changing these assumptions, both of the original parameter values were estimated from known grouse biology. Another potential method of increasing the infection level may be to increase the interaction between grouse and ticks. This was considered, however, increasing the rate at which ticks and grouse interact (ie. β_1 and β_2) did not increase the level of infection within the model, but rather it reduces the grouse population, as too many are infected and die.

4.6.4 Fitting a seasonal model to data

The infection rates of grouse are higher in the field than predicted by the grouse pulse hatch model and no long term oscillations are shown to occur on the timescale predicted by the grouse pulse hatch model therefore the grouse pulse hatch model will be rejected.

The predictions of the grouse hatching period model both short and long term and for different deer densities are highly dependent on the length of the hatching period and on the birth rate. Increasing either the hatching period or the birth rate increases the density of infected grouse and ticks and decreases the amplitude and frequency of the damped oscillations and causes the decay to equilibrium to occur at a much faster rate. Although neither has a significant impact on total or susceptible grouse density.

The equilibrium predicted proportion of ticks infected in the seasonal models (with original parameters) is considerably lower than empirical estimates (Gaunt, 1997; Watts et al., 2009) at around 0.00002 for both the grouse pulse hatch model and grouse hatching period model for most deer densities. The predicted proportion of infected ticks varies throughout the year. If the peak proportion rather than average proportion is considered instead the model predictions are still low compared to field estimates. The peak proportion of infected ticks when there are 6.5 deer per km^2 is less than 0.0001 and decreases for higher deer densities.

The recorded virus prevalence in grouse varies dramatically. Gilbert et al. (2001) gives estimates of 0%, 7.1-26.1%, 46% and 81.8% with 75% recorded in Laurenson et al. (1997). The corresponding prevalence in the tick populations is not given. The varying prevalence in the grouse population could arise as a consequence of varying tick density on different sites all with the same prevalence in ticks, or all sites may have similar tick densities but different prevalence levels in ticks. The prevalence of LIV in grouse naturally changes through the course of the year as infected ticks bite young grouse. Empirically LIV prevalence is usually estimated from blood samples taken from grouse shot during autumn shoots (Aug-Oct) although the grouse in Laurenson et al. (1997) were sampled in the summer. The within year dynamics (Fig. 4.9) highlight that the predicted density of grouse infected with LIV also changes through

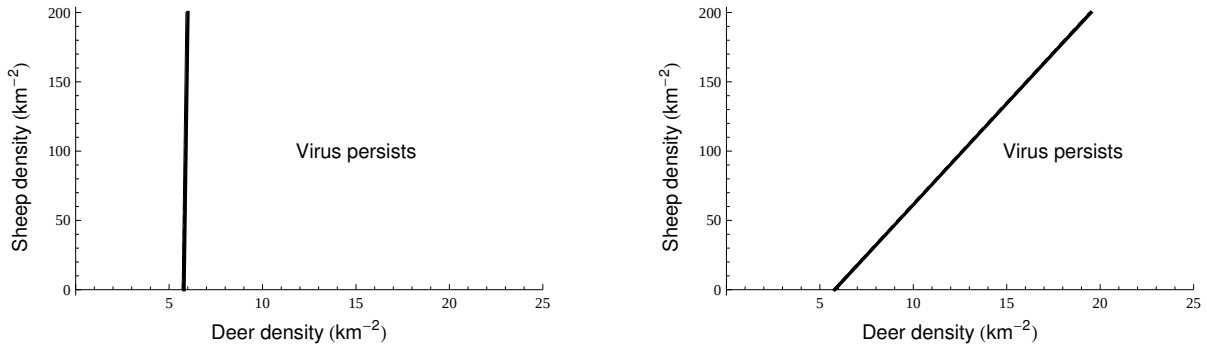
the year. If the proportion of infection is considered instead the change is slight and the difference between the average and maximum prevalence would not be detectable from field data. The peak of infection for red grouse is predicted to occur around Oct-Nov in the model and is 0.0013 when there are 6.5 deer per km^2, rising slightly to 0.0015 when there are 20 deer per km^2.

In order to achieve a higher level of infection in the model we can increase the hatching period or hatching rate, although, increasing the hatching period is clearly not realistic and the hatching rate was based on empirical evidence (four chicks per pair Hudson (1992)). However, more recent estimates suggest grouse may produce eight chicks per pair in Scotland (Thirgood et al., 2000; Park et al., 2002). The successful hatching rate of chicks in the grouse hatching period model is given by the interaction of the birth rate and the density dependence. If the birth rate is doubled from 0.5 to 1 egg per grouse per week (and density dependence changed accordingly) when there is no virus the interaction of these two parameters means that the number of successfully hatching chicks is the same as with the original birth rate and the within year predictions are indistinguishable at equilibrium. The effect of the increased birth rate only becomes apparent when virus persists and the density dependence is reduced when the grouse population is reduced by disease. If the birth rate (number of eggs laid) is doubled when there is virus more of the eggs are able to hatch and consequently there is greater influx of susceptible grouse (Fig. 4.14) although the average equilibrium number of susceptible grouse is the same. It may therefore be appropriate to re-estimate the birth rate of the grouse hatching period model to take account of extra eggs lain in areas of low density (due to virus) which were not taken account of in the original estimate of a_g. This would allow the grouse hatching period model to predict more realistic levels of infection and to reach equilibrium in a much shorter time frame.

The non seasonal model with the original birth rate (2 eggs per grouse per year) gives a good approximation of infection levels because the birth rate and density dependence are acting constantly over the year. However, in the grouse hatching period model the equivalent birth rate and density dependence does not capture a realistic level of infection because too few grouse are able to become infected in the short period of time in the year when there is a high level of susceptible grouse. Consequently in order to gain a reasonable estimate of grouse infection levels a higher birth rate is necessary. The density dependence is adjusted accordingly so that when there is no infection (i.e. for low deer densities) the reproduction of grouse occurs at exactly the same rate regardless of the given birth rate. Changing the birth rate simply allows more grouse chicks to hatch at lower grouse densities and therefore for these grouse chicks to become infected. We will therefore continue with the higher birth rate.

4.7 The seasonal grouse models with acaricide treated sheep

Chapter 3 discusses the use of acaricide treated sheep as a method of tick control. Treated sheep are put out on a grouse moor with the express purpose of killing those ticks that try to attach. This should then lead to a reduced tick population and hence a decline in LIV prevalence in red grouse. The non seasonal sheep mop model predicted that a flock of treated sheep could only be expected to be successful



(a) low sheep tick burden

(b) high sheep tick burden

Figure 4.16: The areas of virus persistence for different sheep and deer densities, with sheep having a) a low tick burden or b) a high tick burden, predicted by the non seasonal (thick solid line), grouse pulse hatch (thin solid line) and grouse hatching period (dashed line). Sheep are treated with acaricide of 100% efficacy.

in reducing tick numbers in certain situations. If the deer density on a grouse moor is too high (> 10 per km^2) then the sheep mops are ineffective. The tick burden of sheep greatly influences the effectiveness of sheep mops, with sheep that have a higher tick burden being more effective than those with a low tick burden (Porter et al., Published online 20 May 2010).

Sheep tick mops (using low and high burdens) were added to the grouse pulse hatch model and grouse hatching period model to determine whether the same qualitative predictions would be made with a seasonal model. Although differences remain between the model predictions (e.g. in long term behaviour and infection levels, as discussed in Sec. 4.5.2) the same overall conclusions were attained regarding predictions of when virus may or may not persist when treated sheep are added to different deer densities. Figure 4.16 illustrates the threshold for when virus is predicted to persist at different deer and treated sheep densities for the grouse pulse hatch model (without ingestion), grouse hatching period model and non seasonal models. It can be seen that the quantitative difference between the models is negligible and the threshold lines are virtually indistinct for both the low and high sheep tick burden. The conclusions are also unaffected by the inclusion of ingestion to the grouse pulse hatch model(not shown).

4.8 Summary

When forming a mathematical model of a real life system a compromise has to be reached between the complexity of the model, our ability to analyse it and the incorporation of realistic dynamics. Past published models of red grouse, ticks and LIV have not incorporated the seasonal reproduction of red grouse. This chapter aimed to develop a seasonal model to compare against the non seasonal model to assess whether the non seasonal model was capturing the essence of the dynamics of LIV within the tick and grouse population. The first seasonal model, the grouse pulse hatch model, incorporated the seasonal hatching of grouse occurring instantaneously as part of the initial conditions for each year of the model. This method led to unrealistic oscillations within the long term predictions. Consequently a second seasonal model, the grouse hatching period model, was developed which allowed the grouse to

'hatch' into the model over a period of weeks. This model was analysed and the factors contributing to the long term oscillations were discussed.

The effect of ingestion was considered in both the grouse pulse hatch model and non seasonal model. It became apparent that the way ingestion had originally been added to the model was leading to unrealistic numbers of ticks being eaten by the grouse. In response to this discovery the ingestion of ticks was constrained in the model using a Holling Form saturating function. This new form of ingestion was compared in the grouse pulse hatch model and non seasonal model. The effect of ingestion was the same for both models. Grouse were predicted to reach carrying capacity for very slightly higher deer densities when ingestion was included in the models. At high deer densities the inclusion of ingestion gave only a slightly higher predicted grouse density than without ingestion. However, the difference between the model predictions with and without ingestion was slight. The overall pattern of behaviour at equilibrium for different deer densities was the same with and without ingestion; grouse are predicted to reach carrying capacity for low deer densities but for high deer densities the grouse population is significantly reduced. Consequently we consider that including ingestion is an added level of complexity that is not necessary in future models.

Comparing the long term equilibrium grouse and tick density predictions at different deer densities for the two seasonal models and the non seasonal model shows they all predict the same pattern of behaviour; for low deer densities grouse reach the predicted carrying capacity, at high deer densities the grouse population is reduced due to the deer amplifying the tick population. For all the different deer densities the three models give very similar predictions for the total grouse and tick populations.

The differences between the model predictions become apparent when they are considered over time. The grouse pulse hatch model and grouse hatching period model predictions continue to show damped oscillations after several centuries, whereas the non seasonal model reaches equilibrium after a small period (c. 25 years) of fluctuation. The damped oscillations are apparent for all deer densities in the seasonal models, although the phase of the oscillation is altered by deer density. The predicted oscillations occur as a result of the within year dynamics of the seasonal models interacting with the underlying fluctuations of the non seasonal model.

The seasonal models not only display long term damped oscillations but also a much lower level of LIV infection within the grouse and tick populations. This can be explained by the within year dynamics. The seasonal models only have a short period of influx of susceptible chicks each year, which is calculated based on the number of grouse at the end of the previous year. Grouse densities are at their lowest at this point and consequently there are few grouse born. The low density of infected ticks also means the opportunity for interactions between susceptible grouse and infected ticks is relatively low. The density of infected grouse is also low so susceptible ticks have less opportunity to become infected. As a result the level of infection in both grouse and ticks is unable to reach high levels. This does not occur for the non seasonal model as this model has continuous reproduction of susceptible grouse and therefore there is always opportunity for infected ticks to contact these grouse and keep the transmission cycle going.

One aim of this thesis is to use mathematical models to consider whether management strategies will

work at different deer densities. These conclusions are drawn using the equilibrium predictions of total grouse and tick densities at different densities. The deer densities where grouse are predicted to survive at carrying capacity remain unchanged regardless of seasonality and the predicted density of grouse at higher deer densities is not very different between models. Consequently adding seasonality does not affect our predictions of management strategies. The non seasonal model can be analysed analytically unlike the seasonal models. The non seasonal model is also simpler and quicker to implement in Mathematica. However, the effect of the seasonal behaviour of ticks will be explored before deciding whether any form of seasonality will be incorporated in future work.

Chapter 5

Modelling the seasonal questing activity of ticks

5.1 Seasonal behaviour of ticks

Chapter 4 discussed the seasonal behaviour of the grouse and its incorporation in to the model. This chapter considers the seasonal dynamics of the tick population and the development of a seasonal tick model and a seasonal grouse and tick model.

The seasonal dynamics of the tick population vary between geographical locations. Scotland seems to have one major peak in the summer (Walker et al., 2001) but two peaks of activity in early and late summer have been detected in southwest England (Randolph et al., 2002) and in larvae and nymphs in SW Scotland (Walker et al., 2001). The seasonal peaks of the different tick stages may be at slightly different times (Randolph et al., 2002). Climatic conditions are changing in the UK and the periods of tick activity may change as a consequence (Gilbert, 2010). Actively questing ticks have now been found in each month of the year in the UK (Walker, 2001; Pietzsch et al., 2005) in temperatures ranging between 3.5 and 29.3°C (Walker et al., 2001). Making a universal model that takes in to account all of this variability is beyond the scope of this thesis. However, general trends still occur; ticks are rarely active in the winter months and a reasonable level of tick activity can be expected in the summer months. The model therefore assumes the ticks are either active or inactive. For grouse moors in Scotland ticks are assumed active in the model for the months of April through to October and inactive November through to March (Gilbert, unpublished data; Laurenson, unpublished data). (Although questing ticks have been found in the traditional winter periods the number is small and weather dependent. Grouse moors tend to be at a higher elevation and are therefore less likely to have suitable questing conditions during winter months. The effect of varying the tick activity season will be explored.) The model assumes that the ticks quest, transmit virus and reproduce only during periods of activity and that the ticks die naturally at a constant rate throughout the year.

To determine the effect of a seasonal tick population on the model predictions the seasonal tick

dynamics are added to the non seasonal model. The short term behaviour as well as the long term average predictions of the seasonal tick model will be compared to the model without seasonal ticks. Adding seasonal grouse reproduction caused damped oscillations not apparent in the non seasonal model. We investigate whether the seasonal tick model will also cause damped oscillations. The different seasonal grouse models also had different levels of infection, an order of magnitude lower than the non seasonal model. Here, we therefore compare the level of infection in the long term predictions for all models over a range of deer densities. We also explore the impact of the length of the seasonal tick activity period on the range of deer densities for which LIV is predicted to persist. This is relevant to current climate change scenarios and how ticks and LIV might be affected by warmer springs and autumns lengthening the ticks questing season.

The interaction of the seasonal tick activity and the hatching period of grouse will also be investigated by considering the above points in the context of comparing the seasonal tick model, grouse hatching period model and a model with both ticks and grouse seasonal.

5.2 The seasonal tick model

We have adjusted the continuous model of Chapter 2 to become a semi-discrete model with ticks actively questing, biting and reproducing in the 'spring' and 'summer' and quiescent in the 'winter', although they continue to die at the same rate. The spring/summer period is taken to be 32 weeks and the winter 20 weeks. The model restarts each year with $G, T_j(0) = G, T_j(52) \quad j = s, i, z$. Grouse are assumed to reproduce at a constant rate throughout the year. To aid understanding a time line has been given in Table 5.1.

Month	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec	Jan	Feb	Mar
Ticks	Actively questing, transmitting and reproducing							Inactive				
Grouse	Reproduction occurs throughout the year											

Table 5.1: Timeline showing the seasonal activity of grouse and ticks in the seasonal tick model.

$$\left.\begin{aligned}
\frac{dG_s}{dt} &= (a_g - s_g G)G - b_g G_s - \beta_{1t} T_i G_s \\
\frac{dG_i}{dt} &= \beta_{1t} T_i G_s - (\alpha + b_g + \gamma) G_i \\
\frac{dG_z}{dt} &= \gamma G_i - b_g G_z \\
\frac{dT_s}{dt} &= c(a_{tt} - s_{tt} T)T\beta_{5t} D - \beta_{2t} T_s G_i - \beta_{5t} D T_s - b_t T_s \\
\frac{dT_i}{dt} &= \beta_{2t} T_s G_i - \beta_{5t} D T_i - b_t T_i
\end{aligned}\right\} \begin{aligned} &0 \le t_n \le 52, \\ &G, T_j(0)_n = G, T_j(52)_{n-1},\ j = s, i, z, \\ &\beta_i = 0 \text{ for } 32 < t_n \le 52 \end{aligned} \tag{5.1}$$

With initial conditions $G_s(0)_0 = 13,\ G_i(0)_0 = 5,\ G_z(0)_0 = 72,\ T_s(0)_0 = 3,988,000,\ G_{cs}(0)_0 = 12,000.$

The questing period is now shorter so ticks are assumed to quest and attach to hosts at a greater weekly rate than in previous models, the new weekly rate $\beta_{1t} = \frac{52}{32}\beta_1$, where β_1 is the previous weekly

rate, see Sec. 4.2 of Chapter 4. The tick reproduction rate a_t has been adjusted similarly and the density dependence constraint s_t has been adjusted to give a similar tick density to previous models with reproduction occurring for 32 weeks only and are now denoted as a_{tt} and s_{tt} respectively. The remaining parameters are per week as given previously in Chapter 4. For ease of reference the table is repeated here with β_{1t}, a_{tt} and s_{tt} adjusted (Table 5.2).

For simplicity the only large mammal available for tick reproduction in this model is the deer.

Param -eter	Value (unit)	Definition
a_g	0.0385385 (w^{-1})	Natural birth rate of grouse
s_g	0.004/52 $(g^{-1}w^{-1})$	Density dependence constraint on grouse
b_g	0.0200769 (w^{-1})	Natural death rate of grouse
α	1.15385 (w^{-1})	Disease induced death rate of grouse
γ	0.288462 (w^{-1})	Recovery rate of infected grouse
a_{tt}	31.2488 (w^{-1})	Natural birth rate of ticks
s_{tt}	7.5×10^{-7} $(t^{-1}w^{-1})$	Density dependence constraint on ticks.
b_t	0.0191538 (w^{-1})	Natural death rate of ticks
β_{1t}	4.61×10^{-6} $(g^{-1}w^{-1})$	The rate at which a tick bites and infects a grouse.
β_{2t}	$9.75\beta_{1t}$ $(g^{-1}w^{-1})$	The rate a tick bites a grouse and becomes infected
β_{5t}	$8.82\beta_{1t}$ $(D^{-1}w^{-1})$	The rate an adult female tick bites a deer and reproduces
c	$\frac{32}{12}$	A scaling constant to ensure yearly tick reproduction consistent

Table 5.2: The seasonal tick model parameters. For the units w denotes week, g grouse, t ticks and D deer.

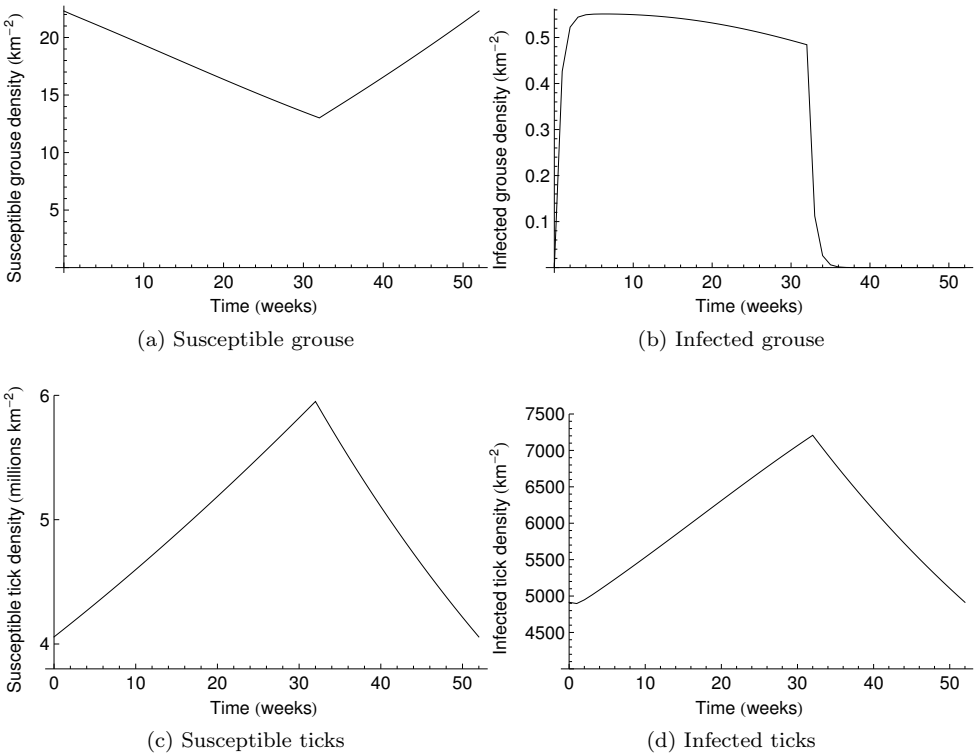
5.3 The seasonal tick model predictions

As in Chapter 4 the semi-discrete nature of the model prevents the use of the reproductive rate as a method of comparing model outputs. Instead the model predictions of the within year and long term dynamics will be compared for particular deer densities. The long term average equilibrium will be used to compare the effect of different deer densities. The average equilibrium predictions are calculated as the average density over 1 year once the long term predictions are at (or near) equilibrium.

5.3.1 The within year predictions

In order to determine the effect of seasonal ticks in this section the model has ticks acting seasonally but the grouse are non seasonal. Fig. 5.1 shows the within year predictions for the susceptible and infected grouse and tick densities at equilibrium (after 200 years) when there are 6.5 deer per km^2. (This deer density has been chosen because the virus is predicted to persist but without decimating the grouse population.) The seasonal active/inactive dynamics of the tick population can clearly be seen for the within year predictions.

The density of susceptible grouse falls during the months that the ticks are active and rises when the ticks are inactive (Fig. 5.1a). This corresponds to when the ticks are active and so can transmit the virus. After 32 weeks when ticks become inactive the number of susceptible grouse rise due to the constant birth rate of grouse in this first model. The response of the infected grouse density is shown in Fig. 5.1b, with a sudden increase in infected grouse when ticks have become active followed by a gradual decline during the period of tick activity. This slow decline is a result of decreasing susceptible grouse density as they



(a) Susceptible grouse (b) Infected grouse

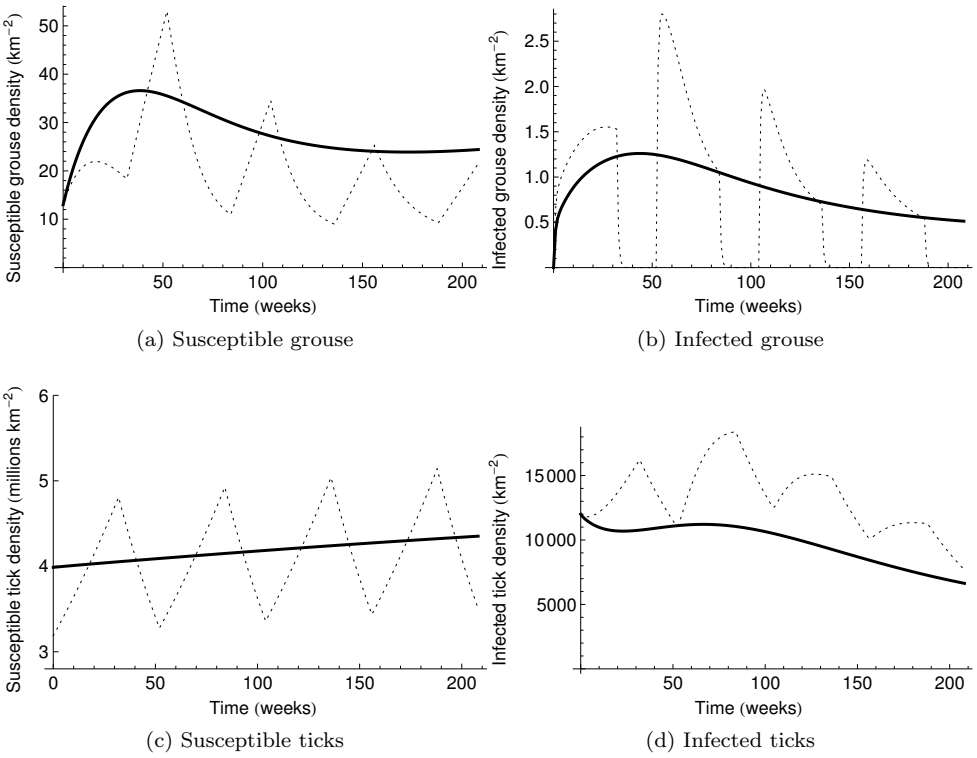
(c) Susceptible ticks (d) Infected ticks

Figure 5.1: The seasonal tick model predictions of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick densities within a year at equilibrium. (Deer density 6.5 per km^2).

become infected at a greater rate than they reproduce; if there are fewer susceptible grouse this will lead to a smaller number of infected grouse as the opportunity for susceptible grouse to become infected is reduced. The density of infected grouse quickly falls to zero when the ticks become inactive. The ticks are no longer transmitting the virus and the grouse already infected die or recover.

Fig. 5.1c shows the effect of the tick activity period on the susceptible tick population. Not surprisingly the tick population rises during the period of activity as the ticks are able to feed and reproduce during this time but falls during the period of inactivity as reproduction is no longer occurring but ticks are dying at the same rate. The infected tick population (Fig. 5.1d) shows a broadly similar pattern of behaviour but with a week delay before the infected tick density rises. At the start of the year there are no infected grouse so susceptible ticks cannot contract the virus until those ticks that are already infected have transmitted the virus and infected grouse.

The long term equilibrium is unaffected by initial (non zero) conditions. Fig. 5.2 shows how the within year dynamics for the first four years begin to affect the long term dynamics of the predictions for the seasonal tick model (dotted line) and non seasonal model (thick solid line). The shorter period of reproduction of the tick population means that for the months that the ticks are active they are at a higher density and consequently a greater number of grouse are infected and the number of susceptible grouse falls. Grouse reproduction depends on how many grouse there are, when ticks are active grouse are becoming infected at a greater rate than they can reproduce so the grouse density falls. Fewer grouse necessarily produce fewer chicks and so the grouse density falls further. The period of tick inactivity is not sufficient for the grouse to recover enough to maintain a high average density over the year. As a result although both models have the same initial density for the susceptible and infected grouse the predicted



(a) Susceptible grouse (b) Infected grouse

(c) Susceptible ticks (d) Infected ticks

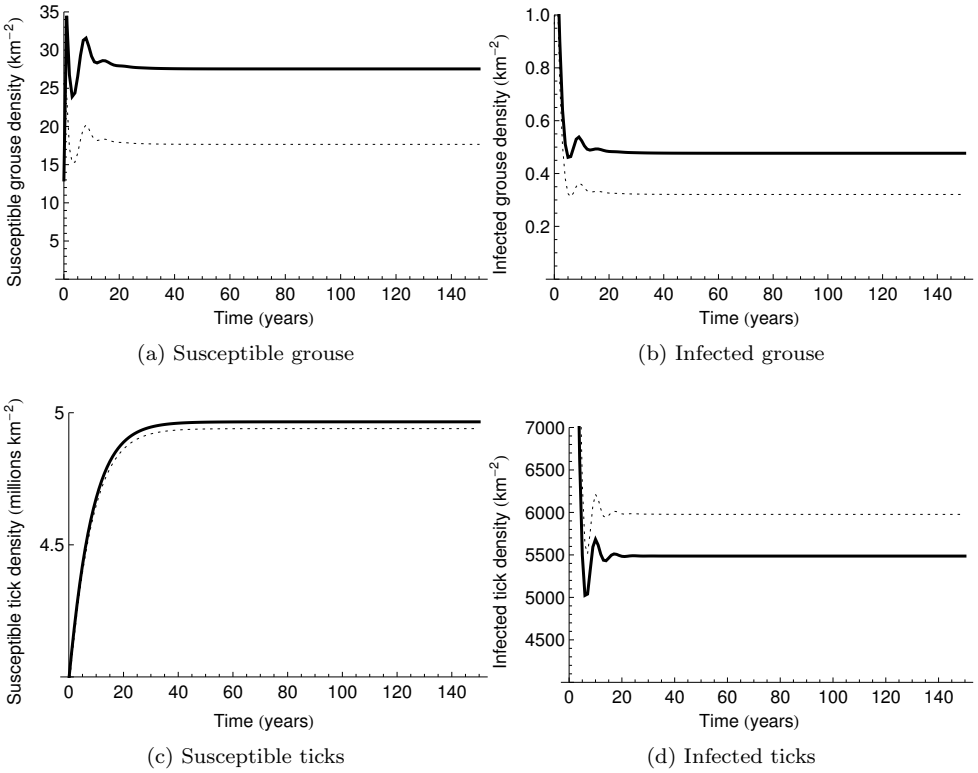
Figure 5.2: The seasonal tick (dotted line) and non seasonal (thick line) model predictions of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick densities for the first four years of the simulations. (Deer density 6.5 per km^2).

susceptible grouse in the seasonal tick model soon falls below the predicted density for the non seasonal model. It can also be seen that despite the same initial density the predicted infected tick density for the seasonal tick model is very quickly higher than the non seasonal model. Even if the initial density for the seasonal tick model is reduced to give a lower yearly average for the first year the predictions of the infected tick density soon rise above the non seasonal model and are the same as the predictions shown here after two years. The shorter period of activity results in more interactions between grouse and ticks and so the number of ticks infected increases significantly over the summer. Relatively few ticks die as a result of natural death during the winter months and unlike grouse ticks remain infected, consequently the infected tick density is higher in the seasonal tick model. The initial condition for the susceptible tick population in the seasonal tick model has been chosen so that the yearly average of the susceptible tick population in the first year is approximately the same as for the non seasonal model.

5.3.2 The long term predictions

In Chapter 4 the seasonal birth of the grouse was added to the model in two ways, in one pulse each year or over a short period of time, both of which gave rise to damped oscillations in the long term predictions. The level of infection was also much reduced in the seasonal grouse models. However, it became apparent that when grouse hatched over longer time periods the oscillations died out much faster and the level of infection increased. In the seasonal tick model the ticks are treated as either active or inactive with tick reproduction occurring during the active period of 32 weeks. I would therefore expect the long term predictions of the seasonal tick model to settle to an average equilibrium in a similar time frame and

have a similar level of infection to the non seasonal model because the period of tick activity is long and the within year variation is small.



(a) Susceptible grouse (b) Infected grouse

(c) Susceptible ticks (d) Infected ticks

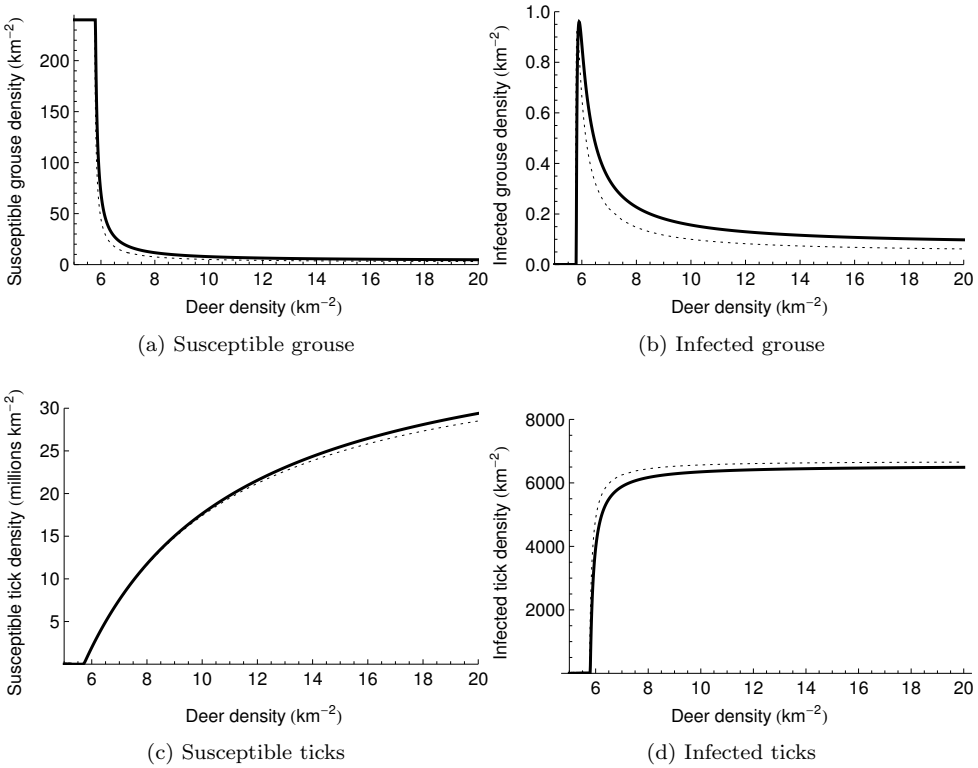
Figure 5.3: The seasonal tick (dotted line) and non seasonal (thick line) model predictions of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick densities over time. (Deer density 6.5 per km^2).

Fig. 5.3 shows that the seasonal tick model does predict the system to reach equilibrium in the same time as the non seasonal model when there are 6.5 deer per km^2. However, the density of grouse and ticks are not the same. The most noticeable difference is for the susceptible grouse density shown in Fig. 5.3a. The seasonal tick model predicts approx 18 susceptible grouse per km^2 at equilibrium but the non seasonal model predicts approx 28 susceptible grouse per km^2. This can be explained by considering how the seasonal behaviour affects the predictions during each year (Sec. 5.3.1). Briefly, the tick population is higher during the period of tick activity so more grouse are infected and hence there are fewer grouse to reproduce. The infected grouse density (Fig. 5.3b) is also lower as a result of there being fewer susceptible grouse to become infected.

The susceptible tick population is slightly lower in the seasonal tick model compared to the non seasonal model. Although the tick reproduction parameters have been adjusted to take account of the shorter period of reproduction (Fig. 5.3c) the linear scaling of s_t, the density dependence parameter, may have a disproportionate effect on model predictions. See Sec. 5.5 for more details. Interestingly the density of infected ticks is higher in the seasonal tick model with almost 6000 infected ticks per km^2; 500 more than the non seasonal model. This can be explained by the within year dynamics of the tick and grouse populations, i.e. the increase in tick infections over the summer and the ability of the ticks to retain infection (Sec. 5.3.1).

5.3.3 The effect of different deer densities

To compare the seasonal tick model and non seasonal model for different deer densities the average yearly predictions of the seasonal tick model are taken at equilibrium (after 200 years). The general pattern of the seasonal tick model is the same as for the non seasonal model with the tick population increasing with the deer density (Fig. 5.4). As a consequence of an increasing tick density the grouse population plummets and is low for all deer densities above 6 per km^2. However, the average equilibrium predicted densities of grouse and ticks show some small differences for the two models.



(a) Susceptible grouse (b) Infected grouse

(c) Susceptible ticks (d) Infected ticks

Figure 5.4: The seasonal tick (dotted line) and non seasonal (thick line) model predictions of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick densities for different deer densities.

Fig. 5.4 shows that the seasonal tick model does not affect the deer densities for which the equilibrium densities of the grouse population are predicted to reach carrying capacity compared to the non seasonal model. However, there are slight differences between models in the numerical predictions of the grouse and tick densities. The seasonal tick model consistently predicts a slightly lower density of susceptible grouse and ticks than the non seasonal model as explained by the within year dynamics (Sec. 5.3.1).

The equilibrium infected grouse density is also consistently predicted to be lower in the seasonal tick model than in the non seasonal model, but the infected tick density is predicted to be higher. Fig. 5.4b shows that this difference is most marked for lower deer densities (between 6 and 10 per km^2) but becomes smaller as the deer densities increase. The effect of the seasonal dynamics is amplified by the larger grouse population at lower deer densities when the tick population is not enough to spread the virus to the extent that the grouse population is decimated. Consequently the difference in model predictions appears relatively large compared to when the grouse density is much reduced by the disease at larger deer and tick densities.

5.4 The seasonal tick with grouse hatching period model predictions

Chapter 4 outlines the effect on model predictions of seasonal behaviour within the grouse population. Although the predicted level of infection appears to be unrealistically low with seasonal grouse it is interesting to explore the interaction of seasonal grouse and ticks. It also makes more sense biologically to include the seasonal behaviour of both species. This section considers the effect of adding seasonal tick behaviour to the grouse hatching period model with the grouse birth rate occurring over four weeks at the rate of 0.5 chicks per grouse per week. This model shall be referred to as the combined seasonal model.

The combined seasonal model allows the ticks to be active or inactive as in the seasonal tick model with the grouse hatching period occurring six weeks after the ticks become active. The model nominally begins in April with ticks actively questing. After six weeks in mid May the grouse hatch over a period of four weeks (ticks still active). After this time the grouse stop reproducing but ticks remain active for a further 22 weeks, until 1st November. The ticks are then inactive for 20 weeks over the winter. In this way the ticks are active for 32 weeks and inactive for 20 weeks as in the seasonal tick model. The full model is given in Appendix B but a time line is given in Table 5.3 to aid the understanding of the periods of different behaviour in the model.

Month	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec	Jan	Feb	Mar
Weeks	0	6	10					32				
Ticks	Actively questing, transmitting and reproducing							Inactive				
Grouse		Hatch										

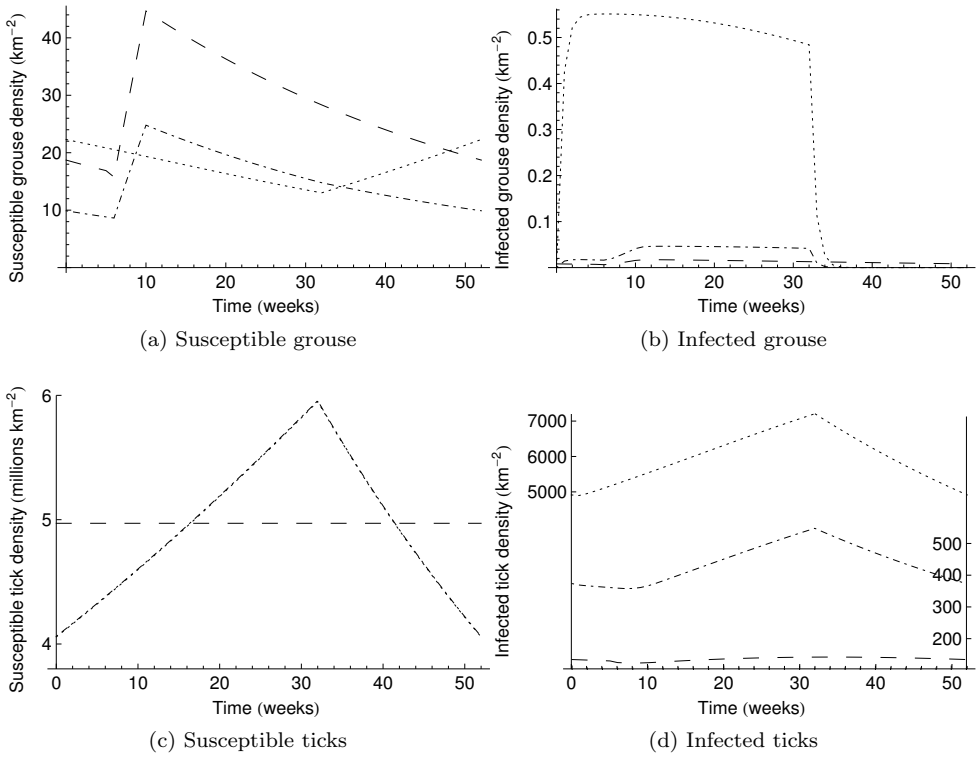
Table 5.3: Timeline showing the seasonal activity of grouse and ticks in the combined model.

5.4.1 The within year predictions

To allow for a better understanding of the different contributory factors within the combined model the plots in this section include the seasonal tick model (dotted line), the grouse hatching period model (dashed line) and the combined seasonal model (dotdashed line).

The within year dynamics of the combined model can easily be seen from Fig. 5.5. Comparing the predicted susceptible grouse densities over the year (Fig. 5.5a) for the seasonal tick model, grouse hatching period model and combined seasonal model shows that combining the seasonal behaviour of the grouse and ticks prevents the susceptible grouse reaching as high a density as in the grouse hatching period model. This is a result of the concentrated tick activity over the summer months. The increased tick density in the first weeks of spring in the combined seasonal model compared to the grouse hatching period model means more grouse become infected and die reducing the grouse population that is able to reproduce. Consequently fewer chicks are born and the density of susceptible grouse is lower. More of the newly hatched grouse chicks will also become infected by the greater number of infected ticks.

The infected grouse density (Fig. 5.5b) changes throughout the year for all the models which reflects



(a) Susceptible grouse (b) Infected grouse

(c) Susceptible ticks (d) Infected ticks

Figure 5.5: The seasonal tick (dotted line), grouse hatching period (dashed line) and combined (dot-dashed) model predictions of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick densities within a year at equilibrium. (Deer density 6.5 per km^2).

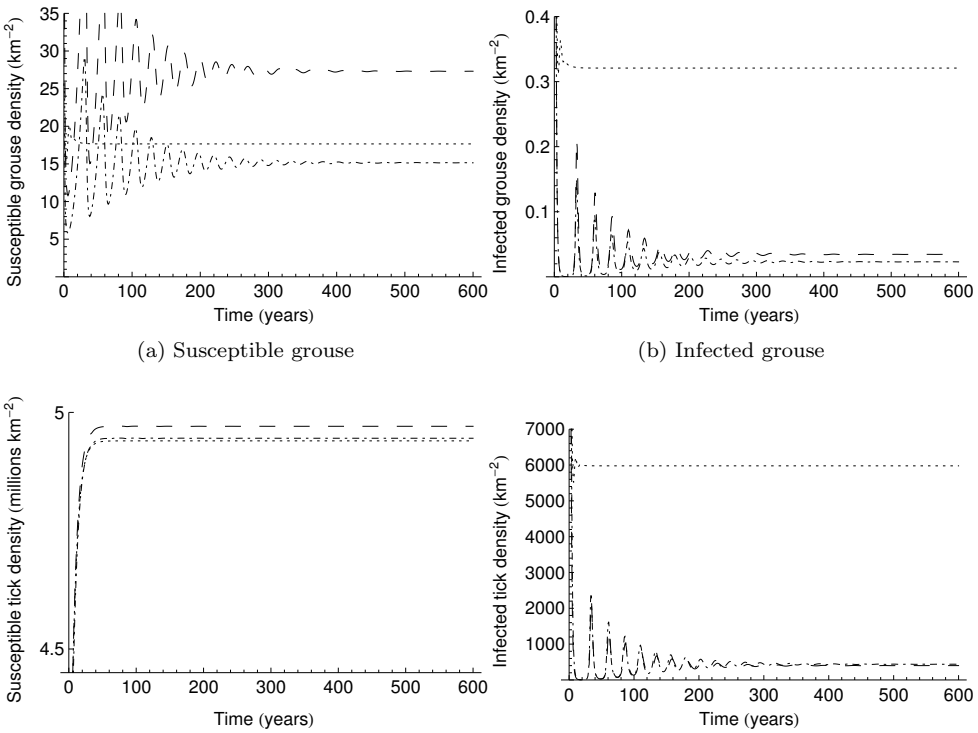
the seasonal biology. The combined model has a higher density of infected grouse throughout the summer than the grouse hatching period model as the ticks are only active during the summer when the grouse density is at its highest. Consequently there are more grouse able to become infected and therefore a higher infected grouse density. Both the combined model and grouse hatching period model have much lower infection rates than the seasonal tick model. Sec. 4.6.1 of Ch. 4 explains the low infection rates in the grouse hatching period model. Briefly, the susceptible grouse density is high as susceptible chicks hatch but this short lived period provides little opportunity for virus transmission. The combined seasonal model predicts a higher density of infected grouse than the grouse hatching period model because the tick activity is concentrated in the summer months and so the opportunity for ticks to interact with grouse is increased during this time, which coincides with when the grouse reproduce and consequently the new susceptible grouse are more likely to become infected.

The tick population is unaffected by the grouse population so the predicted densities of the susceptible tick population throughout the year is the same for the seasonal tick model and combined seasonal model showing an increase during the periods of activity as ticks are reproducing and decrease during inactivity (Fig. 5.5c). The grouse hatching period model has no tick seasonality and consequently the total tick density is constant throughout the year. (As explained in Chapter 4 there is a slight change in the susceptible tick density as infection occurs but this is not apparent on this scale).

The infected tick density changes according to the opportunity for infection transmission to occur. Fig. 5.5d shows that when the grouse hatch over a short period the infected tick density rises as a result of an increased opportunity for virus transmission. (Note the axis for the seasonal grouse models is a

different scale, on the right of the plot.) Newly susceptible grouse are infected by the infected ticks and these newly infected grouse transmit virus back to the susceptible ticks.

5.4.2 The long term predictions



(a) Susceptible grouse (b) Infected grouse

(c) Susceptible ticks (d) Infected ticks

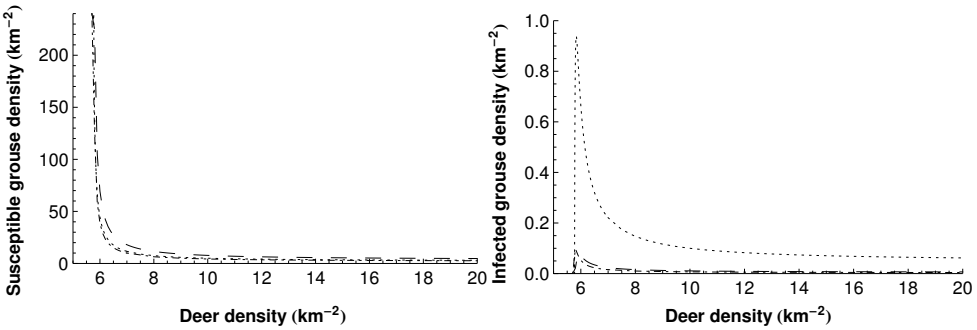
Figure 5.6: The seasonal tick (dotted line), grouse hatching period (dashed line) and combined (dot-dashed) model predictions of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick densities over time. (Deer density 6.5 per km^2).

Fig. 5.6 shows that in general the pattern of the combined seasonal model long term predictions most closely matches that of the grouse hatching period model, suggesting that the seasonal behaviour of the grouse has more influence on long term predictions than the seasonal tick activity. The seasonal tick model reaches equilibrium in a much shorter time scale (c. 25years) than the models with seasonal grouse which take centuries. The long term dynamics of the grouse hatching period model was discussed in Sec 4.5.2 of Chapter 4. Briefly, the within year dynamics interacts with the underlying fluctuations of the non seasonal model creating long term damped oscillations. This still occurs for the combined seasonal model. The level of infection in both grouse and ticks (Figs 5.6b,5.6d) is approx. 10 times higher in the seasonal tick model than the grouse hatching period model and combined seasonal model. This follows from the within year predictions (Sec. 5.3.1).

The effect of the seasonal tick activity in the combined model can be seen in the predictions of the susceptible tick density (Fig. 5.6c). The susceptible tick population is predicted to be slightly lower for the combined seasonal model and seasonal tick model compared to the grouse hatching period model. (This will be discussed further in Sec. 5.5 of this chapter). All three models predict the susceptible tick density to be of the same order of magnitude and the difference is relatively small ($< 1\%$).

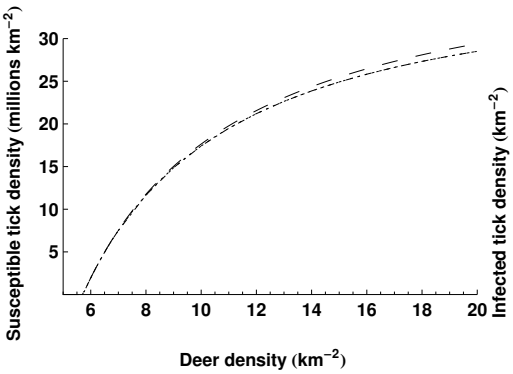
5.4.3 The effect of different deer densities

The combined seasonal model does not change the range of deer densities over which the virus is predicted to reduce the grouse density when compared to the other seasonal models. The predicted grouse and densities at different deer densities are changed however.

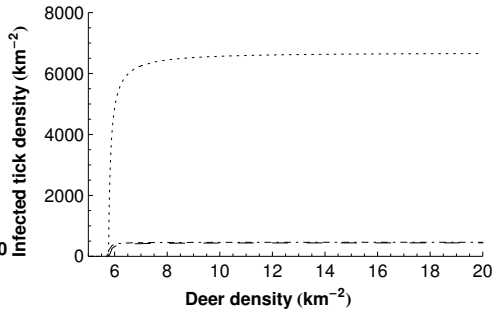


(a) Susceptible grouse

(b) Infected grouse



(c) Susceptible ticks



(d) Infected ticks

Figure 5.7: The seasonal tick (dotted line), grouse hatching period (dashed line) and combined (dot-dashed) model predictions of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick densities for different deer densities.

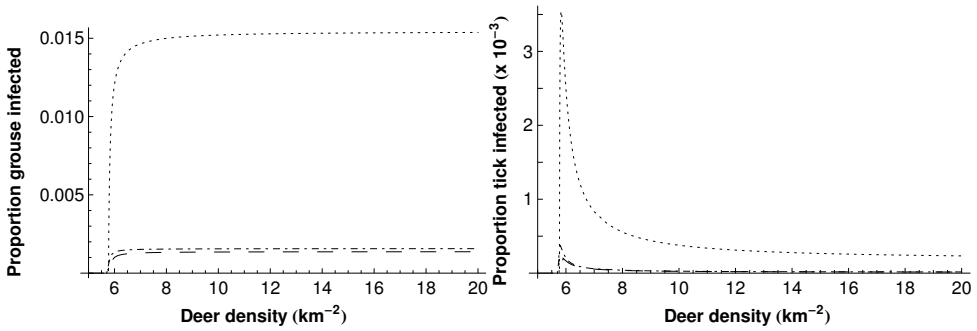
The grouse are predicted to reach carrying capacity at equilibrium until approx 5.7 deer per km^2 for all models after which the susceptible grouse density falls as a result of the deer carrying sufficient ticks to sustain the virus (Fig. 5.7a). The models predict different average equilibrium densities of susceptible grouse with the grouse hatching period model having the highest density, the combined seasonal model the lowest and the seasonal tick model in between for all densities above approx 5.7 deer per km^2. The difference in model predictions decreases as the deer density increases. At very high deer densities the grouse population is so low that actual differences are minimal. The difference between the combined seasonal model and seasonal tick model is small for all deer densities, suggesting that the seasonal tick behaviour has a smaller effect on the predictions of grouse density than the seasonal grouse behaviour.

The equilibrium susceptible tick density is similar for all the models at most deer densities but at higher deer densities (> 12) the grouse hatching period model begins to show a noticeably higher tick density (Fig. 5.7c). (Please refer to Sec. 5.5 for a discussion of this).

Fig. 5.7b shows the predicted equilibrium densities of the infected grouse population, which shows that the seasonal tick model is consistently predicted to have a higher density of infected grouse for all deer densities. The combined seasonal model consistently has the lowest density of infected grouse but

the difference between this and the grouse hatching period model is very small.

Although the equilibrium susceptible tick density is predicted to continue to increase as the deer density increases the infected tick density saturates so that for deer densities above 8 per km^2 there is very little increase. Both the susceptible and infected grouse densities are predicted to decline and then plateau above 8 deer per km^2. For higher deer densities the tick population is so high that the grouse population is significantly reduced by disease. An increase in the tick population due to more deer will have little effect as there are so few grouse that the chance of a susceptible tick finding an infected grouse and becoming infected is very small. Similarly the chances of an infected tick finding and infecting a susceptible grouse is also low.



(a) Proportion infected grouse (b) Proportion infected ticks

Figure 5.8: The seasonal tick (dotted line), grouse hatching period (dashed line) and combined (dot-dashed) model predictions of the proportion of a)infected grouse b)infected tick densities for different deer densities.

Considering the proportion of grouse and ticks infected at equilibrium for different deer densities shows that as the deer density increases the proportion of infected grouse increases and the proportion of infected ticks decreases (Fig. 5.8). However, the proportion saturates so that for deer densities above 8 per km^2 there is little effect of further increases in deer density, particularly for grouse. This can be explained as above.

It is interesting to note that the comparative differences between models remains for all deer densities. The seasonal tick model consistently has a much higher level of infection than the grouse hatching period model and combined seasonal model, which have similar levels of infection.

Although there has been discussion of numerical differences between the model predictions it should be noted that the differences are small and would not necessarily be detectable in the field. The models have been built with the aid of various assumptions and quantitative predictions may not be accurate, although the qualitative predictions are robust to parameter changes. (Please see Appendix C for sensitivity analysis.)

5.5 The effect of scaling the parameters

To incorporate tick seasonality in to the model the tick activity that had been averaged over the course of the year in previous models (with non seasonal ticks) was condensed in to 32 weeks of activity. As a consequence the tick reproduction parameters were adjusted to be $\frac{52}{32}$ of their previous values. The birth

s_{tt}	6.5 deer per km^2							10 deer per km^2						
	Grouse				Tick			Grouse				Tick		
($\times 10^{-7}$)	Sus.	Inf.	Imm.	Tot.	Sus.	Inf.	Tot.	Sus.	Inf.	Imm.	Tot.	Sus.	Inf.	Tot.
7.5	17.66	0.32	4.68	22.66	4.94	5977	4.95	5.02	0.10	1.46	6.58	17.4	6566	17.4
7.47	17.57	0.32	4.66	22.56	4.96	5980	4.97	4.99	0.10	1.45	6.54	17.5	6567	17.5
7.43	17.49	0.31	4.65	22.46	4.99	5984	5	4.96	0.10	1.45	6.51	17.6	6568	17.6
ns	27.5	0.48	6.85	34.86	4.97	5486	4.97	7.81	0.16	2.25	10.22	17.6	6348	17.6

Table 5.4: The predicted densities of the seasonal tick model with different values of s_{tt} compared with the non seasonal model (ns) for different deer densities. The susceptible and total tick populations are given in millions.

rate, a_{tt} and contact rates, β_i, of ticks with hosts occur as coefficients of linear forms of the T_j, $j = s, i$, within the tick equations (Eqn. 5.1). The term that governs tick reproduction is $c(a_{tt} - s_{tt}T)T\beta_{5t}D$, with c chosen to cancel the effect of scaling β_{5t}. The density dependence s_{tt} is a coefficient of T^2. Changing s_{tt} therefore has a disproportional effect on model outputs. There is not an easy way to describe the relationship between s_{tt} and the tick density now that the model is seasonal; the algebraic manipulation used in non seasonal models can no longer be used. In the grouse hatching period model the grouse density dependence s_g was changed by using the model to estimate s_g so that the density of grouse was approximately 240 per km^2 in the absence of ticks. The tick population cannot be estimated in quite the same way, as it varies with deer density. Estimating s_{tt} so that the predicted average equilibrium tick density is equivalent with and without seasonality for a particular deer density does not mean that the tick densities will be comparable for other deer densities. The model uses the value 7.500×10^{-7} for s_{tt} because this is $\frac{32}{52}$ of the previous s_t, however, this leads to a slight disparity in model predictions. The seasonal tick model and non seasonal model predictions of the tick population when there are 6.5 deer per km^2 show slight discrepancy. This difference is increased when there are 10 deer per km^2 but is still comparatively small, a difference of 0.2 million in approx. 17.5 million (Table 5.4). If $s_{tt} = 7.465 \times 10^{-7}$ is used instead then the model predictions match when there are 6.5 deer per km^2 but there is still a small difference when there are 10 deer per km^2, showing that it is not possible to choose a value of s_{tt} so that the seasonal tick model predictions of the tick density always match the non seasonal model predictions. Changing the value of s_{tt} within these limits has a negligible effect on infected tick densities and grouse densities and given that estimates of tick density vary from site to site using $s_{tt} = 7.5 \times 10^{-7}$ is sufficient. Although these differences are small for 6.5 and 10 deer per km^2 they may go some way to explaining the difference in model predictions at higher deer densities. It should also be noted that small differences would not be detected from field data.

5.6 The effect of the varying the length of the summer

The period of tick activity is taken to be 32 weeks as current research indicates that this reflects the period of greatest activity, although there are peaks of more intense activity within that time. Research also indicates that the ticks are becoming more active throughout the year (Scharlemann et al., 2008; Pietzsch et al., 2005). Climate change may cause the activity patterns of ticks to change. It is likely that the length of the tick activity season will increase although we cannot be certain by how much. In this

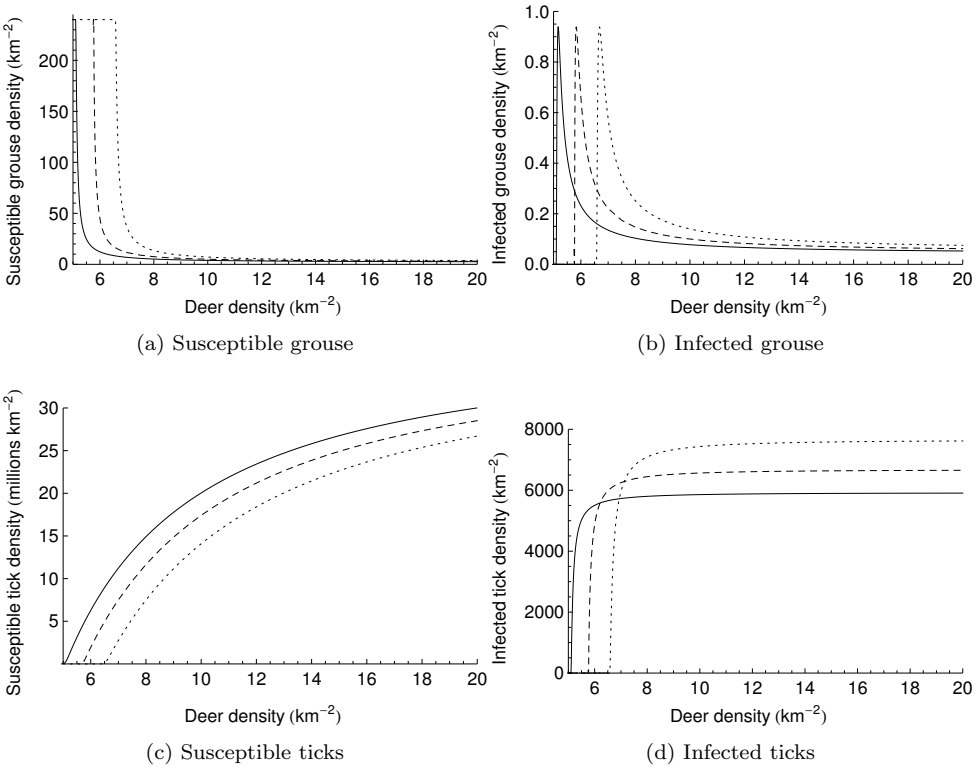
section the length of the summer is altered to consider the potential effect of a longer or shorter period of activity.

In order to incorporate the seasonal activity of ticks in the seasonal tick model but still have a similar tick density the tick reproduction parameters, the tick birth rate a_{tt} and the corresponding density dependence s_{tt}, needed to be altered from the non seasonal model to take in to account the length of the tick activity season. The rates at which ticks attach to hosts in the non seasonal model were assumed to occur all year round and give a reasonable level of interaction. To account for the shorter questing period of the seasonal tick model the rate at which infected ticks attach to grouse β_1 was also increased so that a similar total rate of interaction may occur over the shorter time period. (As all host interactions relate to β_1 all interactions will be increased simultaneously.)

Adult female ticks can only reproduce after a meal, so tick reproduction depends on the availability of hosts. It is assumed that each female lays approx. 1000 eggs, over 52 weeks in the non seasonal model or over 32 weeks in the seasonal tick model. If the length of the tick activity season is changed the birth rate should be changed accordingly so that ticks are 'born' at the rate of $\frac{1000}{su}$ per week for a tick activity season of length su, i.e. $a_{tt} = \frac{12}{su}a_t$, where a_t is the original monthly rate. The density dependence will change similarly so that $s_{tt} = \frac{12}{su}s_t$, where s_t is the monthly rate. The rate at which ticks quest will remain unchanged from the seasonal tick model with 32 weeks, i.e. $\beta_{1t} = \frac{12}{32}\beta_1$, where β_1 is the monthly biting rate, will not change, as we are assuming that an increase (or decrease) in tick activity season length will extend (or reduce) the period of questing but not the rate. The scaling parameter c is also changed in accordance with the length of the tick activity season so that $c = \frac{su}{12}$. The term describing tick reproduction thus becomes $\frac{su}{12}(\frac{12}{su}a_t - \frac{12}{su}s_tT)T\frac{12}{32}\beta_4 D \equiv (a_t - s_tT)T\frac{12}{32}\beta_4 D$ and acts in the equations for su weeks. Each adult female tick will therefore give rise to 1000 eggs as required but the rate at which they attach to deer stills occurs at the same rate as in the tick activity season of length 32 weeks .

The deer density for which the tick population (and hence virus) can persist changes with summer length. Fig. 5.9c shows the tick population survives at lower deer densities for longer summer lengths with the grouse population reducing accordingly once the tick population exceeds approx. 300,000. An increase of only four weeks from 32 to 36 weeks shows the grouse population crashing when there are 5.1 deer per km^2 as opposed to 5.8 deer when the summer is 32 weeks (Fig. 5.9a). A summer of length 28 weeks predicts the grouse will reach carrying capacity with up to 6.6 deer per km^2. Although the differences are quite small and it would be impossible to count deer to that degree of accuracy it is interesting to see that the model does predict a potential impact of summer length for relatively small changes.

It interesting to note that the density of infected ticks and grouse is increased slightly for shorter summers, despite a lower susceptible and total tick density (Fig. 5.9d, 5.9b). Shorter summers allow more time for the grouse to recover over the winter and so there is greater density of susceptible grouse to infect, leading to a higher infected average equilibrium grouse density and therefore higher infected average equilibrium tick density.



(a) Susceptible grouse (b) Infected grouse

(c) Susceptible ticks (d) Infected ticks

Figure 5.9: The seasonal tick model predictions of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick densities for different deer densities for summer lengths of 28 weeks (dotted), 32 weeks (dashed), 36 weeks (solid).

5.7 Summary

Various models with and without different elements of seasonality have now been compared. Although differences in the within year dynamics and long term predictions for the different seasonal models can be seen the grouse hatching period model, seasonal tick model and non seasonal model all make broadly similar predictions of equilibrium grouse and tick densities for different deer densities. All predict that the grouse density plummets below economically viable densities for a sustainable grouse moor when the deer density exceeds 6 deer per km^2, although the level of infection differs slightly between models.

Many of the model parameters have uncertainty within the estimation and so all model predictions need to be interpreted with caution. Adding seasonality brings further assumptions and complexity to the model but does not seem to make major qualitative differences in predictions or bring any significant further insight. The length of the ticks active season has been explored and some slight impact has been noted. However, the addition of seasonal ticks is quite crude as all tick stages are assumed to act equally, but there is some evidence (Randolph et al., 2002) that different tick stages may display questing peaks at different times. It may therefore make more sense to develop a model with independent tick stages if the full effect of the tick season is to be explored. This would require a better understanding of the different seasonality of each tick stage on Scottish grouse moors and further data would need to be collected.

It is assumed that ticks die at the same rate throughout the year but the death rate over winter may be different to that over the summer, depending on climate and potential saturation deficit. An alternative may be to model only the summer activity and then to assume that a proportion of the ticks

(and grouse) die over the winter. (See Ghosh and Pugliese (2004) for a more in depth model of the seasonal tick activity in Italy.)

Estimating the density dependence of the ticks is another source of potential error. Although small differences in s_t only make small differences to the equilibrium tick population predictions, especially at higher deer densities.

The grouse seasonality was initially added as a pulse birth in Ch. 4. Although ticks are not born in one pulse but emerge throughout the summer months it is interesting mathematically to consider the effect of adding a tick pulse birth. The pulse birth of grouse caused damped oscillations which may occur with the addition of a tick pulse birth. However, the investigation of a tick pulse birth model revealed that adding ticks in one pulse each year does not cause damped oscillations. This may be because in comparison to the tick population as a whole the addition of new ticks each year is small and has little effect on the dynamics.

The aim of this thesis is to consider the impact of management strategies on grouse populations at different deer densities. The seasonal models were developed to ascertain if a more realistic description of grouse and tick seasonal behaviour affected the model predictions of when grouse reach carrying capacity or are reduced by disease. All the seasonal models predict that grouse population remains at carrying capacity if the deer density is below approx. 6 per km^2 and is substantially reduced for deer densities greater than approx. 6 per km^2. This is not affected by the different level of infection predicted by the different models. The amount of infection in grouse found in the field varies greatly (Gilbert et al. (2001) gives estimates between 0 and 81.8%) and it is not possible to make a simple universal model that reflects the the infection levels in all cases. Consequently we do not believe that the addition of seasonal biology will change the predictions of when management strategies are likely to work. (Using a seasonal grouse model did not affect the predictions of when acaricide treated sheep might work Ch. 3, Sec. 4.7.) Adding seasonality makes the models temporally more expensive to run and they can no longer be analysed algebraically. We therefore consider that the added complexity of incorporating seasonality outweighs the benefits, and in order to answer questions regarding potential management strategies, the seasonal factors will not be included.

Chapter 6

The potential role of acaricide treated deer as a management strategy for ticks and LIV

6.1 Introduction

Practical trials of the use of sheep treated with acaricide in an attempt to reduce the tick population have shown limited success in the presence of deer and this is also supported by theoretical models (Porter et al., Published online 20 May 2010). Adding acaricide treated sheep to a grouse moor has the disadvantage that when the acaricide efficacy wanes the sheep become an extra source for adult ticks to achieve a blood meal that would otherwise not have been there. Consequently if the acaricide levels on the sheep cannot be maintained at a sufficiently high level the sheep can increase the potential for LIV persistence by enabling a greater number of adult ticks to reproduce. A greater immature tick population feeding on red grouse, a LIV transmission host, means LIV is more likely to persist. Many estates managed for grouse shooting also have deer. It is known that deer carry a high tick burden and may be the major tick reproduction host in many areas (Gray, 1998).

Taking into account the fact that deer are already present on almost all shooting estates and that they are known to carry a high tick burden some moorland managers are keen to ask whether ticks and LIV could be controlled by treating the deer with acaricide. In the US there have been studies using a topical treatment of white-tailed deer (*Odocoileus virginianus*) with acaricide that shows this can be effective. One such study Carroll et al. (2002) found that after four years the percentage mortality for *Ixodes scapularis* (a hard tick similar to *Ixodes ricinus*) in treated areas reached as much as 80%. The Northeast Area-wide Tick Control Project that took place in the US from 1997 to 2004 showed that by the 6th treatment year the relative density of the nymphal stage of *I. scapularis* had reduced by 71% on some sites (Brei et al., 2009).

The model presented in this chapter is used to predict the effect of applying an acaricide treatment

with varying efficacy levels to deer populations of different densities. The practical methods of employing such a treatment strategy are not modelled explicitly but are discussed. The model does not distinguish between how a certain level of efficacy is achieved in a practical situation; treating a high number of deer with low efficacy treatment and treating a low number of deer with a high efficacy treatment result in the same average level of efficacy within the herd. In other words, only the average level of herd efficacy is modelled.

Many estates routinely cull deer as part of habitat management and tick control and deer stalking is also an important source of revenue for many estates. Although deer culling may not occur at the same rates as potential acaricide use it is interesting theoretically to compare the model predictions for the effect of culling at an equivalent rate to acaricide treatment. The effectiveness of culling deer is compared to the effectiveness of acaricide treatment at equivalent levels for different deer densities.

The effectiveness of a combined treatment strategy is also considered. Culling deer to reduce the density before treating deer with acaricide may provide an alternative method of treatment and is compared to the single treatments for different deer densities.

6.2 The acaricide treated deer model

The model presented here is an extension of that presented in Gilbert et al. (2001). As discussed in previous chapters the addition of seasonality does not alter the deer densities for which the grouse are able to survive at sustainable levels and hence seasonality has not been included in this model. In previous models only the attachment of adult ticks to deer had been modelled as deer do not transmit the virus and the only role deer played was to facilitate tick reproduction. However, the acaricide treatment of deer will affect all ticks trying to attach to deer including immature stages. The model has been adapted to take this in to account with β_4 and β_5 representing the rate at which immature and adult females ticks attach to deer respectively. The acaricide efficacy (proportion of ticks killed by the acaricide) is given by d, so that the proportion of adults that try and attach to deer that can reproduce is $(1-d)$ (all adult ticks die either from acaricide contact or as a result of reproduction) and the proportion of immature ticks trying to attach to deer that are killed is d. Deer may be culled at rate c so that the proportion of deer that remains is $(1-c)$.

$$
\begin{aligned}
\frac{dG_s}{dt} &= (a_g - s_g G)G - b_g G_s - \beta_1 T_i G_s \\
\frac{dG_i}{dt} &= \beta_1 T_i G_s - \Gamma G_i \\
\frac{dG_z}{dt} &= \gamma G_i - b_g G_z \\
\frac{dT_s}{dt} &= (a_t - s_t T)T(1-d)\beta_5(1-c)D - b_t T_s - \beta_2 T_s G_i - (d\beta_4 + \beta_5)(1-c)DT_s \\
\frac{dT_i}{dt} &= \beta_2 T_s G_i - b_t T_i - (d\beta_4 + \beta_5)(1-c)DT_i
\end{aligned}
$$

where $\Gamma = \alpha + b_g + \gamma$.

All other parameters are as previously. For ease of reference the table of parameter values is repeated here.

Param -eter	Value (unit)	Reasoning
a_g	0.167 (m^{-1})	Natural birth rate of grouse (Hudson, 1992)
s_g	$0.000\dot{3}$ $(g^{-1}m^{-1})$	Density dependence constraint on grouse (Ch. 2)
b_g	0.087 (m^{-1})	Natural death rate of grouse (Hudson, 1992)
α	5 (m^{-1})	Disease induced death rate of grouse (Reid, 1975)
γ	1.25 (m^{-1})	Recovery rate of infected grouse (Reid, 1975)
a_t	83.33 (m^{-1})	Natural birth rate of ticks (Gilbert et al., 2001)
s_t	0.000002 $(t^{-1}m^{-1})$	Density dependence constraint on ticks (Ch. 2)
b_t	0.083 (m^{-1})	Natural death rate of ticks (Gilbert et al., 2001)
β_1	0.00002 $(g^{-1}m^{-1})$	The rate at which a tick bites and infects a grouse (Ch. 2)
β_2	$9.75\beta_1$ $(g^{-1}m^{-1})$	The rate a tick bites a grouse and becomes infected (Gilbert et al., 2001)
β_4	$37.1\beta_1$ $(D^{-1}m^{-1})$	The rate an immature tick bites a deer (Gilbert et al., 2001)
β_5	$8.82\beta_1$ $(D^{-1}m^{-1})$	The rate an adult female tick bites a deer and reproduces (Gilbert et al., 2001)
d	varied $(D^{-1}m^{-1})$	The efficacy of acaricide. Varied for comparison.

Table 6.1: The acaricide treated deer model parameters. For the units m denotes month, g grouse, t ticks and D deer.

The reproductive rate of the virus, R_0

The reproductive number of a virus, R_0 is classically defined as the number of new infected individuals caused by the introduction of one infected individual to a totally susceptible population. The reproductive rate of a virus can be used to predict whether the virus is able to establish in a population. If $R_0 > 1$ the virus can persist, if $R_0 < 1$ the virus will die out. The same principle applies for tick-borne infections but a state of endemic infection may be started in a wholly susceptible population by the addition of either an infected tick or an infected host; R_0 combines the number of infected ticks created by an infectious host and the number of infected hosts created by an infected tick. Considering the equations we can see that one infected grouse lives for $(\alpha+b_g+\gamma)^{-1}$ units of time and infects $\beta_2 K_t(\alpha+b_g+\gamma)^{-1}$ ticks; similarly an infected tick lives for $((1-c)(d\beta_4+\beta_5)D+b_t)^{-1}$ units of time and infects $\beta_1 K_g((1-c)(d\beta_4+\beta_5)D+b_t)^{-1}$ grouse. Combining these gives,

$$R_0 = \frac{\beta_1\beta_2 K_g K_t}{(\alpha+b_g+\gamma)((1-c)(d\beta_4+\beta_5)D+b_t)},$$

where K_g and K_t are the carrying capacity of grouse and ticks respectively and are given by,

$$K_g = \frac{a_g - b_g}{s_g} \qquad \text{and} \qquad K_t = \frac{a_t(1-c)(1-d)\beta_5 D - ((1-c)(d\beta_4+\beta_5)D+b_t)}{s_t(1-c)(1-d)\beta_5 D}.$$

The threshold conditions for virus persistence can be determined by considering $R_0 = 1$. If the curve given by $R_0 = 1$ is plotted over different deer densities for different treatment regimes the level of treatment required to reduce R_0 below one can be determined for a given deer density.

Time series predictions

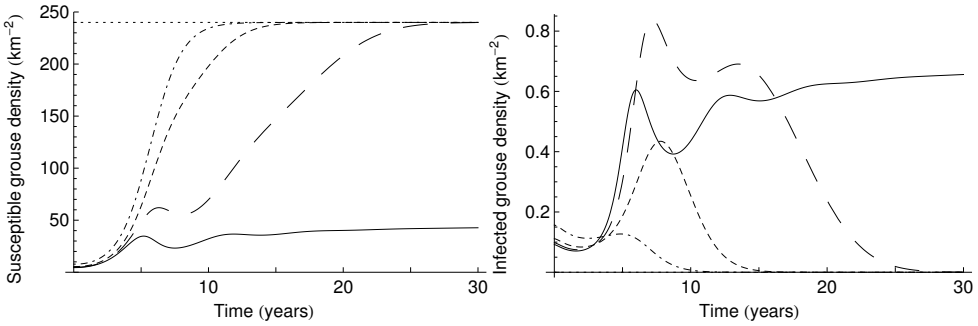
In order to assess the impact of treating deer with acaricide in a system that is already at LIV-induced equilibrium we first run the model without treatment for 100 years at each given deer density to ensure

equilibrium is reached. We then run the model with treatment using the predicted equilibrium densities of grouse and ticks as the initial conditions. The grouse equilibrium density is predicted to be at carrying capacity for deer densities of approx. 5.7 and below. The grouse equilibrium density is reduced below that necessary for a commercial estate for all deer densities above 6 per km^2. Driven shooting needs a grouse density > 60 per km^2 in order to be economically viable (Hudson, 1992).

6.3 The predicted effect of acaricide treated deer

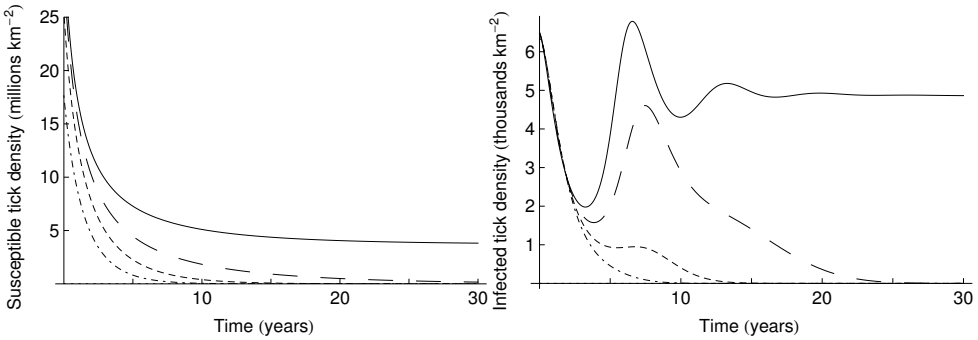
6.3.1 The effect of treating deer with acaricide at different deer densities

As stated previously the practical application of acaricide to deer is not considered in the model and the efficacy level given in the model is assumed to be for the deer herd as a whole and occurs as a result of the combined effect of individual deer. It is likely that there will be some deer with high levels of acaricide and some with low levels, the net effect will be somewhere in between. The practical trials in the US found that acaricide treated deer reduced tick density by approx. 70-80% (Carroll et al., 2002). Consequently the acaricide efficacy in the plots presented here is chosen to be 0.7 corresponding to acaricide efficacy of 70%, as the evidence from the US suggests this may be achievable. The efficacy level will be altered in future sections to assess the impact this has.



(a) Susceptible grouse

(b) Infected grouse



(c) Susceptible ticks

(d) Infected ticks

Figure 6.1: The treated deer model predictions of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick densities for different deer densities. The deer densities (per km^2) are 5 (dotted), 10 (dotdashed), 15 (short dashed), 20 (long dashed) and 25 (solid). Acaricide efficacy (d) is 0.7.

The equilibrium predictions for the susceptible grouse when the model is run without deer treatment suggest the grouse will already be at carrying capacity when there are 5 deer per km^2 but not for the higher deer densities. (This was shown in previous chapters.) However, the model predictions of the

susceptible grouse when treatment is included (Fig 6.1a) suggest that the grouse are able to recover to carrying capacity for intermediate deer densities (10-20 per km^2) if the deer are treated with acaricide with an efficacy of 0.7. (The grouse density at $t = 0$ is the predicted equilibrium density without treatment.) The recovery is quickest for lower deer densities because fewer deer will carry fewer ticks and eradicating (or reducing) these ticks will be easier. Although the same proportion of ticks will be killed for all deer densities the actual number of adult ticks that survive will be greater for higher deer densities (Fig. 6.1c). Any surviving adults ticks are then able to reproduce and the model assumes each one gives rise to 1000 larvae (although density dependence may reduce this number in model simulations), which will lead to a large increase in the tick population making it much harder to eradicate the ticks. The grouse are not predicted to reach carrying capacity when there are 25 deer per km^2. The model predictions are given for 30 years, which is longer than most experiments would run for. The predicted density of susceptible grouse is still rising slightly when there are 25 deer per km^2 so it is possible that given long enough the grouse may recover to carrying capacity. However, for practical purposes a treatment that has not worked within 30 years would be regarded as a failure.

The predicted infected grouse and tick densities (Figs 6.1b, 6.1d) are reduced to 0 when the grouse population reaches carrying capacity, suggesting that grouse will only reach carrying capacity in the absence of virus. However, when there are 25 deer per km^2 the grouse density is reduced by the virus but not eradicated so there are infected grouse and ticks surviving.

When there are 25 deer per km^2 the model predicts that the tick population is reduced but persisting and the grouse are able to persist at a higher density than without treatment but the grouse density is still lower than that required for a viable grouse moor. This suggests there may be a potential deer density for which grouse and ticks may coexist with the virus but given the limitations of the model a numerical estimate of this value would not be useful.

6.3.2 The effect of acaricide efficacy on treatment effectiveness

Although acaricide efficacy of 70% may be feasible it is likely that should deer treatment become part of a tick management regime that there will be variation in the efficacy achieved. This could be due to the method of application, type of acaricide, location, deer behaviour etc. We therefore consider how acaricide efficacy influences the predicted effectiveness of treatment.

Fig. 6.2 shows the deer densities and efficacy levels for which grouse are predicted to reach carrying capacity (240 per km^2, above the solid curve), a economically viable density (≥ 60 per km^2, between the solid and dashed curves) and fail to be sustained at a viable level (< 60 per km^2, below the dashed curve) when equilibrium is obtained. The plot suggests that there is a deer density below which the grouse will always reach carrying capacity as there are not enough deer to sustain the tick population. The plot also indicates that there is an upper level of acaricide efficacy above which the grouse can reach carrying capacity regardless of deer density (within biologically reasonable limits). Using the parameter estimates presented here suggests that the grouse will reach carrying capacity if there are fewer than approx. 5.7 deer per km^2, which agrees with previous chapters. If the efficacy level is kept above approx. 73% then

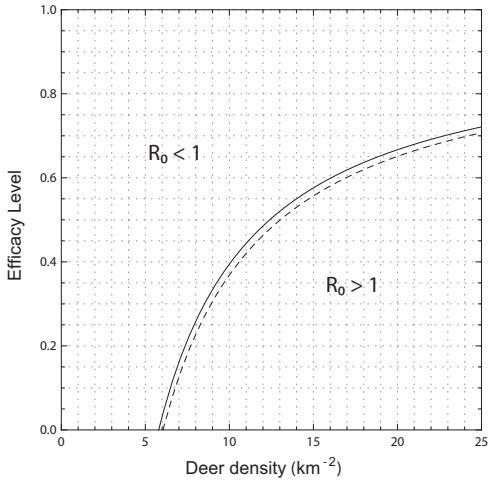


Figure 6.2: The curve described by $R_0 = 1$ for different deer densities and acaricide efficacies. Above and to the left of the curve the virus dies out, below the curve virus persists. The solid line denotes when grouse population reaches carrying capacity (240 per km^2). The dashed line denotes when the grouse population reaches a viable density (60 per km^2).

the grouse are predicted to reach carrying capacity for all deer densities below 25 per km^2. (It is unlikely that there will be a much greater density of deer on a grouse moor as they are often kept below this level as part of the moor management. It appears that the efficacy level would not need to be increased much further for higher deer densities should they be achieved.) There are few combinations of deer and efficacy levels for which the grouse are predicted to reach a sustainable density but not carrying capacity. This is further suggestion of the importance of deer as tick hosts and the influence this can have on the grouse population, as also predicted in previous chapters. The predicted grouse density is very sensitive to deer density, with a slight increase in deer density causing a catastrophic fall in the grouse density. (Especially at low deer densities and acaricide efficacies.)

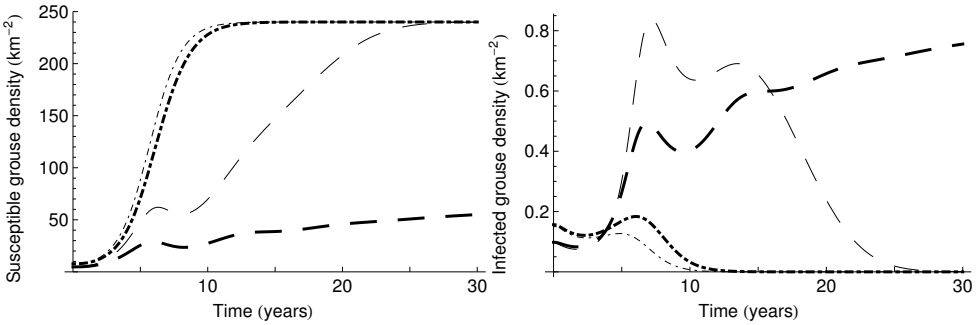
At intermediate deer densities (12-18 per km^2) intermediate acaricide levels (0.45-0.65) are required to decrease the predicted tick population sufficiently to allow grouse to reach sustainable levels. This suggests that if the deer density can be kept at intermediate densities a lower dose of acaricide would still be effective.

6.4 The predicted effect of culling

6.4.1 The effect of culling deer at different deer densities

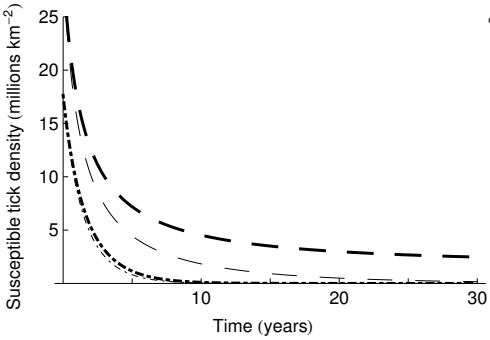
Acaricide treatment of deer may be beneficial on a grouse moor as it reduces the tick population density by killing ticks that attach to deer. Another method of reducing the tick population could be to reduce the deer population and hence reduce the opportunity for adult ticks to feed and reproduce. The predicted effect of culling deer at a given rate for a given deer density will be compared to the predicted effect of treating deer with acaricide of equivalent efficacy. The deer density is reduced by a given factor to model the effect of deer culling, e.g. if we wish to consider the effect of culling at a rate of 70% when there are 20 deer the predicted equilibrium grouse and tick densities are first found by running the model with 20 deer per km^2 with no treatment regime. These values are used as the initial densities of grouse and ticks

and the model is rerun but with 6 deer per km^2 (i.e. 70% fewer deer).

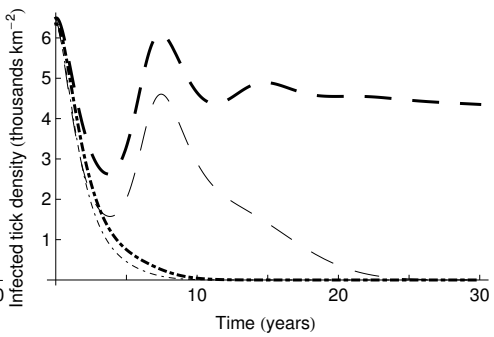


(a) Susceptible grouse

(b) Infected grouse



(c) Susceptible ticks



(d) Infected ticks

Figure 6.3: The model predictions of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick densities for different deer densities. Acaricide treatment is denoted by thin lines. Culling is denoted by thick lines. The initial deer densities (per km^2) are 10 (dotdashed) and 20 (long dashed). After culling deer densities (per km^2) are 3 (thick dotdashed) and 6 (thick long dashed). Treatment efficacy is 0.7 (70%).

Fig. 6.3 compares the predicted effect of treating deer with acaricide of efficacy 0.7 (thin lines) and culling 70% of deer (thick lines). The model predicts that, in general, treating deer with acaricide allows grouse to reach carrying capacity in a quicker time frame than culling and for some deer densities that culling does not. To aid reading the plot only deer densities of 10 and 20 per km^2 are shown. It can be seen that treating deer with acaricide is predicted to allow grouse to reach carrying capacity quicker than culling at an equivalent rate when there are 10 deer per km^2 (dotdashed lines Fig. 6.3a). When there are 20 deer per km^2 (dashed lines) treating deer with acaricide of 70% allows grouse to reach carrying capacity when culling 70% of deer does not because the tick population is eradicated by acaricide use but is not by culling (Fig. 6.3c).

Infected grouse are predicted to be reduced to 0 for both acaricide use and culling when there are 10 deer per km^2 (Fig. 6.3b). When there are 20 deer per km^2 although acaricide treatment is predicted to cause an initial increase in infected grouse (as the overall population rises) this is shortlived and the infected grouse density is predicted to reach 0. However, the infected grouse density is predicted to continue to rise for deer culling. A similar effect is seen for the infected tick densities (Fig. 6.3d).

Both methods of deer management reduce the opportunity for ticks to feed, acaricide by killing ticks and culling by removing potential hosts. However, acaricide use also kills ticks that try to attach removing these from the tick population and preventing reproduction. Although culling removes some hosts and thus potential meals some ticks that would have fed on these removed hosts will be able to feed on the

remaining hosts and continue to reproduce. The effect of culling deer on the tick population is therefore going to be less than treating deer with acaricide.

6.4.2 The effect of culling rates on culling effectiveness

Whilst it is interesting theoretically to directly compare culling with acaricide treatment it is unlikely that a cull of 70% would be implemented. Culling 70% of a deer herd would be a large undertaking and could be difficult to attain, particularly for large herds. Culling a large proportion of deer could also have impacts on habitat and bio-diversity and may cause conflicts of interest with neighbouring land holding units.

Lower culling rates may still be beneficial, especially for smaller deer populations. Fig. 6.4 shows a contour plot of the predicted total grouse and tick densities for different culling rates and deer densities.

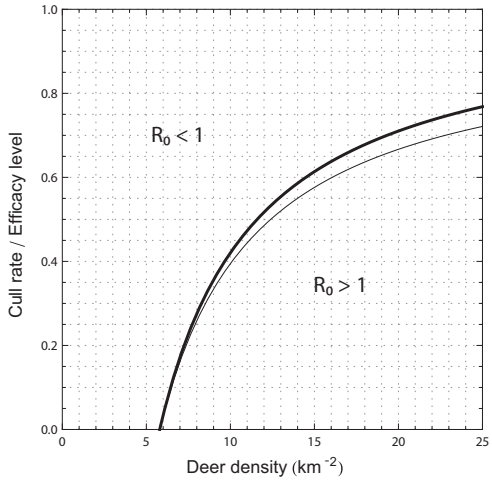


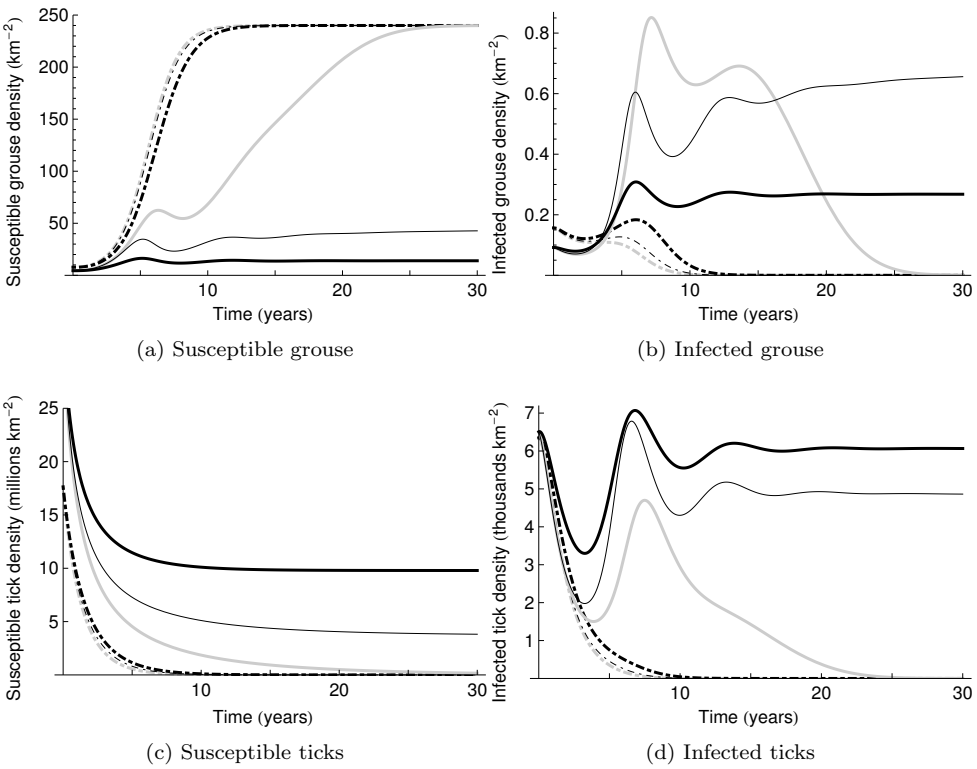
Figure 6.4: The curve described by $R_0 = 1$ for different deer densities, culling rates and efficacy levels. Above and to the left of the curve the virus dies out, below the curve virus persists, upon the curve the grouse reach carrying capacity (240 per km^2). The thin line denotes deer acaricide treatment. The thick line denotes deer culling.

Fig. 6.4 shows that there is a culling rate above which grouse are always predicted to reach carrying capacity. This is slightly higher than the acaricide efficacy for treating deer. Approx. 77% of deer would need to be culled when there are 25 deer per km^2 but acaricide efficacy need only be at approx. 72%. This difference can be explained by the added effect acaricide treatment has on the tick population compared to culling, as explained above. Fig. 6.4 suggests that low culling rates may be effective at reducing the tick population and allowing grouse survival at lower host densities. As for acaricide treatment there are very few combinations of deer and culling levels that are predicted to allow grouse to reach viable densities without reaching carrying capacity.

In practice deer culling does not at present generally occur at high levels. Reducing deer densities dramatically may have significant effects on habitat. It may, therefore, be beneficial to explore the effect of a combined treatment regime, where the deer are first culled at a low perentage level such as is already occurring and then acaricide treatment given.

6.5 The predicted effect of a combined treatment approach

6.5.1 The effect of combined treatment on different deer densities



(a) Susceptible grouse (b) Infected grouse

(c) Susceptible ticks (d) Infected ticks

Figure 6.5: The model predictions of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick densities for different initial deer densities. Combined treatment is denoted by grey lines. Acaricide treatment (70%) is denoted by thin lines. Culling (70%) is denoted by thick lines. The deer densities (per km^2) are 10 (dotdashed) and 25 (solid).

Culling rates in practice depend on the management aims of the estate. Estates managed for conservation purposes have different requirements from those managed for sport. As a generic guide it is recommended that approx. $1/6^{th}$ of the deer population is culled (Clutton-Brock and Albon, 1989) although estimates of actual cull rates vary. Here, for illustration purposes the figures show a culling rate of 20% followed by acaricide treatment of 0.7 efficacy.

The initial grouse and tick densities are given by the equilibrium predictions without treatment for a given deer density. The deer population is then reduced by 20% and treated with acaricide of 70% efficacy.

To allow comparisons between the treatments to be seen only the model predictions for 10 (dotdashed) and 25 (solid) deer per km^2 have been drawn. It can be seen from Fig. 6.5a that the combined treatment strategy of culling first and then treating with acaricide is predicted to speed up the recovery of susceptible grouse to carrying capacity when compared to acaricide treatment or culling alone when there are 10 deer per km^2 as a result of the increased decline in the tick population (Fig. 6.5c). When there are 25 deer per km^2 the combined treatment is predicted to allow the susceptible grouse density to reach carrying capacity but it does not for acaricide treatment or culling alone. Correspondingly the infected grouse and ticks are predicted to be reach 0 for all methods when there are 10 deer per km^2 but only for the

combined treatment when there are 25 deer per km^2 (Figs 6.5b, 6.5d).

6.5.2 The effect of combined treatment on acaricide efficacy

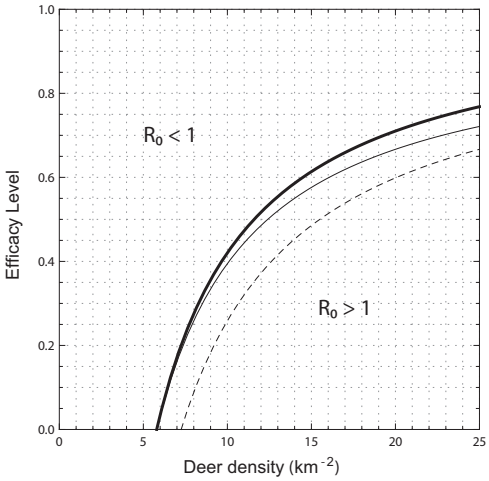


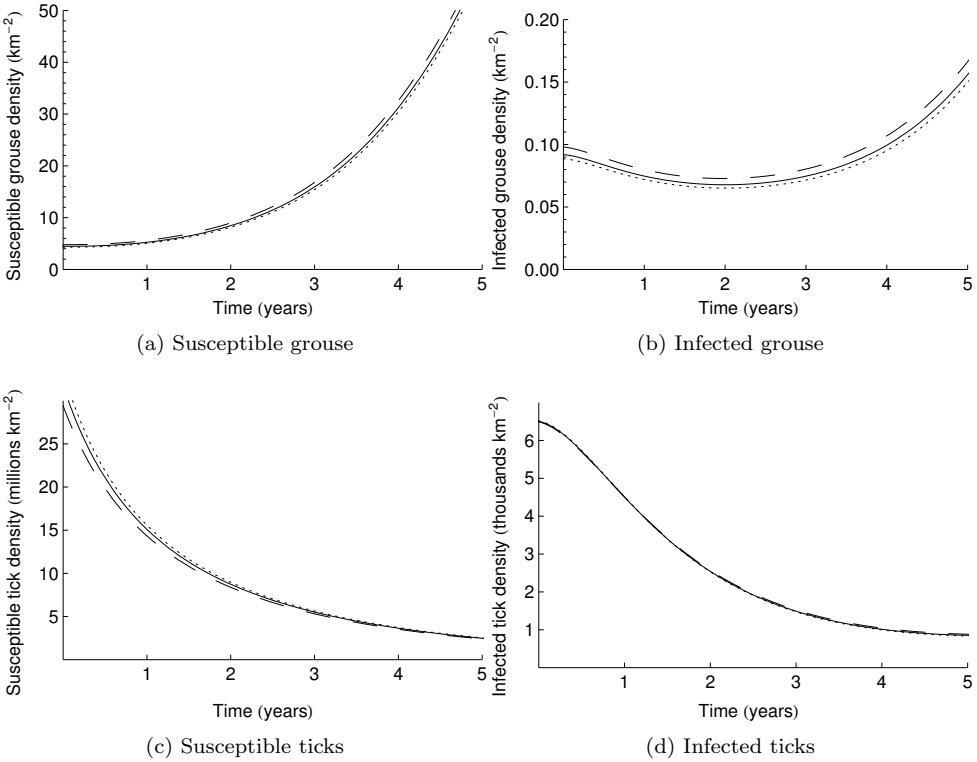
Figure 6.6: The curve described by $R_0 = 1$ for different deer densities and treatment rates. Above and to the left of the curve the virus dies out, below the curve virus persists, upon the curve the grouse reach carrying capacity (240 per km^2). The thin line denotes deer acaricide treatment. The thick line denotes deer culling. The dashed line denotes a deer cull of 20% followed by acaricide treatment.

Fig. 6.6 shows that virus is predicted die out for higher initial deer densities and lower acaricide efficacies if the deer are first culled by 20% compared to culling or acaricide treatment alone. For example, an initial deer density of 15 deer per km^2 is predicted to require acaricide efficacy of approx. 0.58 without culling (thin line), however, if the deer are first reduced by 20% then the predicted acaricide efficacy is reduced to approx. 0.49 (dashed line). This is intuitive as Fig. 6.2 showed that lower deer densities required lower efficacy acaricide to eradicate the virus so reducing a higher deer density before beginning acaricide treatment would be expected to be more effective.

In general the combined treatment is predicted to speed the recovery of the grouse to carrying capacity and allow recovery for deer densities that the single treatments do not. This is not surprising as culling the deer before treatment reduces the opportunity for ticks to find a host to feed on and more importantly reduces the opportunity for adults to reproduce. It should be noted that culling from say 25 or 30 deer to 15 deer per km^2 and then treating with acaricide is not quite the same as treating an area that had only 15 deer per km^2 initially. Although ultimately the end result is the same the speed at which this occurs is reduced slightly if the deer were first culled. This is because 25 deer per km^2 allow a greater number of ticks to reproduce and consequently the tick population will be higher, there will be a short time lag whilst this larger tick population decreases being no longer able to find enough meals.

Fig. 6.7a shows that the grouse density is predicted to increase at a similar rate for the different initial deer densities and it takes 5 years for the grouse to reach viable densities. Although differences this small would not be detectable in the field for these densities it is interesting to consider them mathematically.

The difference between the tick densities is more noticeable (Fig. 6.7c) , with an initial deer density of 15 and 25 deer per km^2 predicted to have approx. 14.3 million and 15.6 million after one year, respectively. These differences have little effect on the grouse densities because the infected tick density



(a) Susceptible grouse (b) Infected grouse

(c) Susceptible ticks (d) Infected ticks

Figure 6.7: The model predictions of a)susceptible grouse b)infected grouse c)susceptible tick d)infected tick densities for different deer densities. The initial deer densities before culling (per km^2) are 20 (long dashed), 25 (solid) and 30 (dotted). After culling to 15 deer per km^2 acaricide efficacy is 0.7.

is virtually the same for each of them (Fig. 6.7d). Consequently the infected grouse densities are similar for each initial deer density (Fig. 6.7b), falling initially as the infected tick densities fall but then rising as the grouse population as a whole increases.

6.6 Summary

We have investigated the potential role of treating deer for the management of ticks and LIV in red grouse. Acaricide efficacy of 80% is predicted to be effective at allowing grouse to reach carrying capacity for all deer densities of 25 per km^2 and below. Intermediate acaricide efficacy is effective at allowing grouse to achieve carrying capacity for intermediate deer densities but there are very few combinations of deer densities and acaricide efficacy for which intermediate sustainable grouse densities are achieved. If there are approx. 5 deer per km^2 or fewer then the tick population is unable to survive as there are too few meals for adult ticks.

The model predictions suggest that a combined treatment of culling followed by acaricide use may be the most effective tick management strategy. Combined treatment is predicted to have a quicker effect on the tick and grouse populations. Combined treatment may also allow grouse to reach sustainable levels when the deer density is too high for one treatment method alone.

Whilst we are confident in the broad predictions of the model it has its limitations. In order to parameterise a model assumptions need to be made which may effect the numerical predictions. The model assumes that deer are at a constant density because deer numbers are usually controlled on grouse

moors. However this does not take in to account that the actual deer density will vary over the course of the year as calves are born in the spring and again as older deer are shot during the hunting season. The deer density will therefore be highest when ticks are questing during the summer months which could affect the impact of any acaricide treatment. Although there may be more deer to pick up acaricide from a feeding station natural food is more abundant in summer which may reduce the number of deer using feeding stations. Also the acaricide would need to be withdrawn for a period as deer meat is used for human consumption. Seasonal application of acaricide may affect the predicted efficacy required to achieve sufficient tick reduction to allow economically sustainable grouse levels for commercial driven shooting.

How acaricide efficacy is achieved is not considered in the model. Treating 100% of deer with acaricide of 70% efficacy, 70% of deer are with acaricide of 100% efficacy or 78% of deer with acaricide of 90% efficacy would all lead to a herd efficacy of approx. 70%. Mathematical models by their nature need to be a simplification of real life and an explicit model of individual deer efficacies is not necessary. However, if a spatial model were to be developed then individual deer may need to be modelled explicitly.

The commercial acaricides for use on livestock in the UK are not currently licensed for use on wildlife, including deer, and there are ethical issues of treating wild animals that need to be considered. The methods found successful in the US may not necessarily be applicable to the UK. The US has different species of tick and deer from the UK and the habitat where trials in the US have been successful are fenced woodland rather than open heath. White-tailed deer are not herd animals like red deer, where dominant individuals may prevent the rest from accessing the food/salt lick. In Carroll et al. (2002) the deer were attracted to '4-poster' feeding station where the action of feeding caused them to rub against one of four paint rollers impregnated with acaricide. The white tailed deer of the US carry almost 90% of the *I. scapularis* ticks on the head, ears, neck and brisket (Carroll et al. (2002) and references therein) and so the rubbing of this area against the treated rollers at the feeding station would seem likely to be effective. However, the deer of the UK may not necessarily carry the majority of their tick burden in this area and the efficacy of the treatment may be reduced. Tick counts from a small sample of deer on a Scottish estate suggests that the majority of nymphal ticks are found on the deer head whilst the adult ticks were mostly found on the inguinal areas (Gilbert, unpublished data). Similarly the sheep tick species of the UK, *I. ricinus*, although the same genus as *I. scapularis* does have a different life cycle and genetic differences may cause it to react differently to the treatment.

The treatment of deer with acaricide may not yet be possible in the UK but this chapter provides some theoretical evidence of the circumstances under which acaricide treated deer may or may not be beneficial to grouse on moors with deer and LIV.

Chapter 7

The potential role of acaricidal leg bands on female grouse as a management strategy for ticks and LIV

7.1 Acaricidal Leg Bands

Previous chapters have considered the potential effect that acaricide use on sheep (Ch. 3) and deer (Ch. 6) might have on LIV prevalence and grouse densities. The presence of deer was shown to reduce the effectiveness of treating sheep and although theoretically the treatment of deer may be of benefit to the grouse population it is not yet possible in the UK. This chapter therefore considers the potential effect of directly treating the target species, red grouse, with acaricidal leg bands. Grouse transmit and receive the virus through tick bites (as well as ingesting ticks) so a direct treatment that reduces the number of tick bites on grouse may help reduce virus prevalence.

Empirical studies have shown a degree of success in reducing tick burdens on grouse with direct acaricidal treatment on grouse (Laurenson et al., 1997; Mougeot et al., 2008). Laurenson et al. (1997) tested the use of slow release acaricidal wing tags on either hen grouse or chicks and a one-off pour-on acaricide treatment on chicks. Although wing tags on both hens and chicks reduced tick burdens on grouse they were not found to increase survival or reduce LIV prevalence in grouse. There were problems with attaching the tags to chicks as broods dispersed when located and could not all be found. The wing tags also caused inflammation on some chicks and may restrict wing growth, consequently the authors did not recommend this method of attachment for tags. The direct application of pour on acaricide was more effective at reducing tick burden initially but the effects quickly diminished. Although LIV prevalence was reduced in these chicks the reduction may not be enough to make this treatment cost effective.

Mougeot et al. (2008) tested the effectiveness of acaricide (permethrin) impregnated leg bands attached to female grouse before the breeding season with untreated leg bands attached to control hens. The chicks from treated broods had much lower tick infestations after 1 month than control chicks with approx. 1.5 ticks per chick compared to approx. 13 ticks per chick in the controls. It is likely that some of the acaricide from the hen's leg band is rubbed upon the chicks during brooding and thus reduces the attachment rate of ticks. Upon capture for treatment (Mar-Apr) approx. 12% of hens had ticks whereas upon recapture in October no treated hens had ticks but 13.3% of control females had ticks. No negative impacts of the bands themselves on hen survival or breeding were found but some swelling had occurred when the band was tight or had tangled with the metal ID band. These problems should be overcome by careful attachment and using ID wing tags instead. Consequently here we develop a model to explore the potential effect of acaricidal leg bands on a wider scale with different alternate host (deer) densities, not just on the individuals treated but the grouse population as a whole. In particular we consider how varying the number of hens treated affects model predictions of the grouse population if the acaricide on chicks lasts both all season and only 12 weeks. We also consider the effect of varying the acaricide efficacy on chicks for a given number of treated hens, as well as the effect of varying the length of time the acaricide remains effective on the chicks.

7.2 Acaricidal leg band model

Assumptions The application of acaricide to grouse will be seasonal (as described above). As such the model presented here describes the seasonal behaviour of grouse and ticks so that the timing of acaricide treatment can be modelled explicitly. To avoid the long term oscillations seen in previous models (Ch. 4 and 5) the number of eggs laid per adult pair has been doubled although the period of hatching remains at four weeks. This equates to 8 chicks per pair (before density dependence constraints act) which is in line with estimates of the number of chicks that hatched on different estates in Scotland during the mid 1990s (Thirgood et al., 2000; Park et al., 2002). Ticks are assumed to be active for 32 weeks over the 'summer' and inactive over the 'winter' for 20 weeks. Female grouse are assumed to be treated before the ticks become active. The number of hens h treated per km^2 is assumed consistent each year (once there are sufficient grouse if initial densities are too low). Warren and Baines (2007) caught 61 grouse over 3 km^2 which equates to approx. 20 birds per km^2. We vary the number of hens treated from zero up to a max of 60 per km^2. The leg bands are assumed 100% efficacious on the females all summer (no ticks were found on treated females in October (Mougeot et al., 2008)). It is not known how long the residual acaricide lasts on the chicks. Although treated chicks had reduced tick burdens compared to control chicks after one month (Mougeot et al., 2008) no counts have been recorded on older chicks. The efficacy of the acaricide on chicks does not appear to be 100% (Mougeot et al. (2008) found approx. 1.5 ticks per treated chick). The proportion of ticks killed by the acaricide on chicks is given by d and is varied for comparison. Although the acaricide efficacy on chicks is likely to decrease over time (Laurenson et al. (1997) found that pour-on acaricide waned over time) we do not have sufficient data to estimate the decay

function, the model instead assumes the acaricide on chicks acquired from the leg band on the mother is of a given efficacy d for w weeks before becoming completely ineffective. Grouse begin to hatch at week 6 of the 'summer' and hatching occurs over four weeks. All newly hatched chicks are susceptible. Only female grouse are treated with leg bands as it is usually hens rather than cocks that brood the chicks. To account for the fact the males are not treated but are part of the reproducing population the number of grouse chicks that are treated are from $2h$ adults (i.e. the treated females that have mated with untreated males). The number of grouse chicks that are not treated is therefore from $G(6) - 2h$ adults, i.e. the total density of grouse at week 6 when reproduction begins minus treated pairs. To avoid the biological impossibility of treating a negative number of grouse within the theoretical model an "IF" constraint is applied in the numerical simulation. At week 0, h grouse are treated if $h > G(0)/2$, otherwise $G(0)/2$ are treated, i.e. if there are h or more females at the start of the year h are treated, otherwise all females are treated. It is assumed that sex ratio of adults is 50:50 so only half the adult population (females) will be treated. Grouse are either susceptible or immune to LIV at the start of each 'summer' as LIV infection is short lived. The susceptible and immune grouse will not be distinguished upon treatment so we assume that they are treated equally and the proportion of susceptible and immune grouse are kept consistent in treated and non treated birds.

The model

$$T_j(0)_n = T_j(52)_{n-1} \quad j = s, i,$$

$$G_{st}(0)_n = If[\frac{G(52)_{n-1}}{2} > h, h\frac{G_s(52)_{n-1}}{G(52)_{n-1}}, \frac{G_s(52)}{2}], \quad G_{zt}(0)_n = If[\frac{G(52)_{n-1}}{2} > h, h\frac{G_z(52)_{n-1}}{G(52)_{n-1}}, \frac{G_z(52)}{2}],$$

$$G_s(0)_n = G_s(52)_{n-1} + G_{st}(52)_{n-1} - G_{st}(0)_n, \quad G_z(0)_n = G_z(52)_{n-1} + G_{zt}(52)_{n-1} - G_{zt}(0)_n, \quad G_i(0) = 0$$

$$\left.\begin{aligned}
\frac{dG_{st}}{dt} &= -b_g G_{st} \\
\frac{dG_{zt}}{dt} &= -b_g G_{zt} \\
\frac{dG_{sct}}{dt} &= 2h(a_g - s_g G(6)) - (1-d)\beta_1 T_i G_{sct} - b_g G_{sct} \\
\frac{dG_{ict}}{dt} &= (1-d)\beta_1 T_i G_{sct} - (\alpha + b_g + \gamma) G_{ict} \\
\frac{dG_{zct}}{dt} &= \gamma G_{ict} - b_g G_{zct}
\end{aligned}\right\} \quad 6 \le t_n \le 10 + w,$$

$$\left.\begin{aligned}
\frac{dG_s}{dt} &= (G(6) - 2h)(a_g - s_g G(6)) - \beta_1 T_i G_s - b_g G_s \\
\frac{dG_i}{dt} &= \beta_1 T_i G_s - (\alpha + b_g + \gamma) G_i \\
\frac{dG_z}{dt} &= \gamma G_i - b_g G_z
\end{aligned}\right\} \quad G_j(10 + w + \delta) = G_j(10 + w) + G_{jct}(10 + w), \quad j = s, i, z$$

$$\frac{dT_s}{dt} = (a_t - s_t T)T\beta_5 D - \beta_2 T_s(G_i + (1-d)G_{ict}) - \beta_2 T_s(G_t + dG_{ct}) - T_s\beta_5 D - b_t T_s$$

$$\frac{dT_i}{dt} = \beta_2 T_s(G_i + (1-d)G_{ict}) - \beta_2 T_i(G_t + dG_{ct}) - T_i\beta_5 D - b_t T_i$$

$$a_g = s_g = 0, \quad t_n < 6, t_n > 10, \qquad \beta_i = 0, \quad t_n > 32$$

Using the assumptions outlined above a set of 10 coupled differential equations have been formed for treated grouse which are either susceptible G_{st} or immune G_{zt}, chicks of treated grouse which are susceptible G_{sct}, infected G_{ict} or immune G_{zct} , untreated grouse (including untreated chicks) which are either susceptible G_s, infected G_i or immune G_z and ticks which are either susceptible T_s or infected T_i. The overall model is semi-discrete as not all equations and parameters are valid for 52 weeks of the year and each year begins afresh using densities from the end of the previous year. The time in year n is given by t_n and $G(t)_n, T(t)_n$ are the corresponding grouse and tick densities at time t_n. Time $t_n = 0$ denotes the start of each year, taken to be March, the start of tick activity. The treated chick equations are only relevant from week 6 when chicks begin to hatch to week $10 + w$ when the acaricide has stopped working, at which point the treated chicks become untreated and as such move in to the corresponding untreated grouse classes. The ticks are assumed to be active for 32 weeks of the year, hence the $\beta_i = 0, i = 1, 2, 5$ for $t_n > 32$. δ denotes a small increment in time. Other model parameters and definitions are as previously but are also included in Table 7.1. For justifications not given in the above text please refer to Ch. 5.

The model equations are given with determining conditions for the densities in each class at the start of each year.

Param -eter	Value (unit)	Definition
a_g	1 (y^{-1})	Natural birth rate of grouse
s_g	0.0039 $(g^{-1}y^{-1})$	Density dependence constraint on grouse
b_g	0.0200769 (w^{-1})	Natural death rate of grouse
α	1.15385 (w^{-1})	Disease induced death rate of grouse
γ	0.288462(w^{-1})	Recovery rate of infected grouse
a_t	83.33 (w^{-1})	Natural birth rate of ticks
s_t	2×10^{-6} $(t^{-1}w^{-1})$	Density dependence constraint on ticks.
b_t	0.0191538 (w^{-1})	Natural death rate of ticks
β_1	7.5×10^{-6} $(g^{-1}w^{-1})$	The rate at which a tick bites and infects a grouse.
β_2	$9.75\beta_{1t}$ $(g^{-1}w^{-1})$	The rate a tick bites a grouse and becomes infected
β_5	$8.82\beta_{1t}$ $(D^{-1}w^{-1})$	The rate an adult female tick bites a deer and reproduces
h	varied (w^{-1})	The number of hens treated
d	varied $(c^{-1}w^{-1})$	The efficacy of acaricide on treated chicks
w	varied	The period for which acaricide is effective on chicks

Table 7.1: The acaricidal leg band model parameters. For the units w denotes week, y year, g grouse, t ticks and D deer.

The semi-discrete nature of this model means that the reproductive rate of the virus cannot be found and used to show when the virus is predicted to persist. Instead the model predictions of grouse (tick) densities were used to draw contour plots of the densities of grouse (tick) densities. The model was run repeatedly, each time varying deer density and the component of interest, for example, the number of hens treated to gain estimates of grouse (or tick) densities in different scenarios. If grouse are predicted to reach carrying capacity (240 per km^2) the virus is eradicated, for lower grouse densities the virus persists although the grouse population may be at higher density than without treatment. Grouse need to be at a density of at least 60 per km^2 in order for an estate to be commercially viable (Hudson, 1992). Contours are drawn connecting points where the grouse density is predicted to be at 60, 100, 150, 200, 240 per km^2 to give an indication of how effective the treatment is predicted to be at different deer densities.

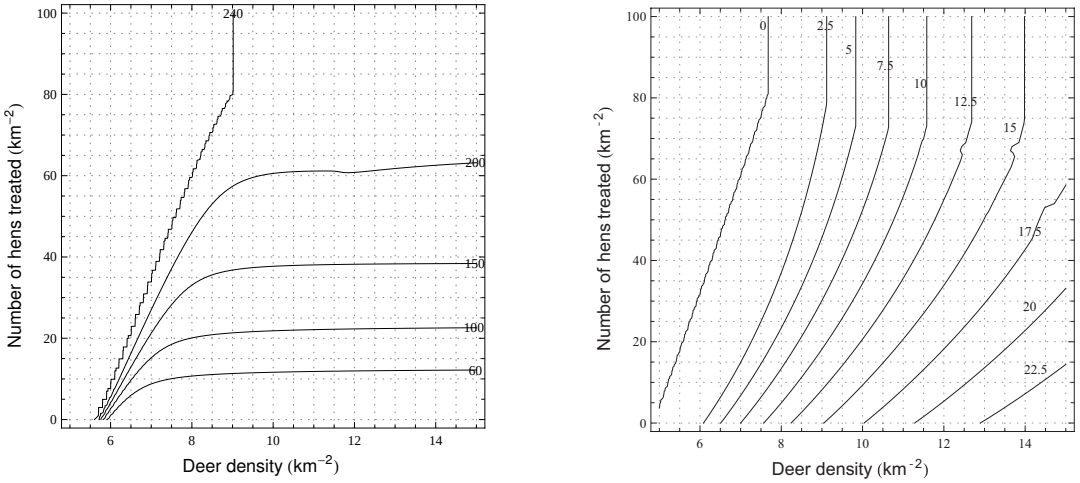
The corresponding tick densities are similarly shown in Section 7.3.1 to highlight the effect the treatment has on the tick population. The tick densities have been omitted from subsequent sections because

the tick density is affected relatively little (compared to other treatments, Ch. 3 and 6) by the treatment and it is the grouse density that is of most significance to estate managers.

7.3 Model predictions

7.3.1 The effect of varying the number of hens treated

Assuming 100% acaricide efficacy on chicks for full season



(a) Total grouse density

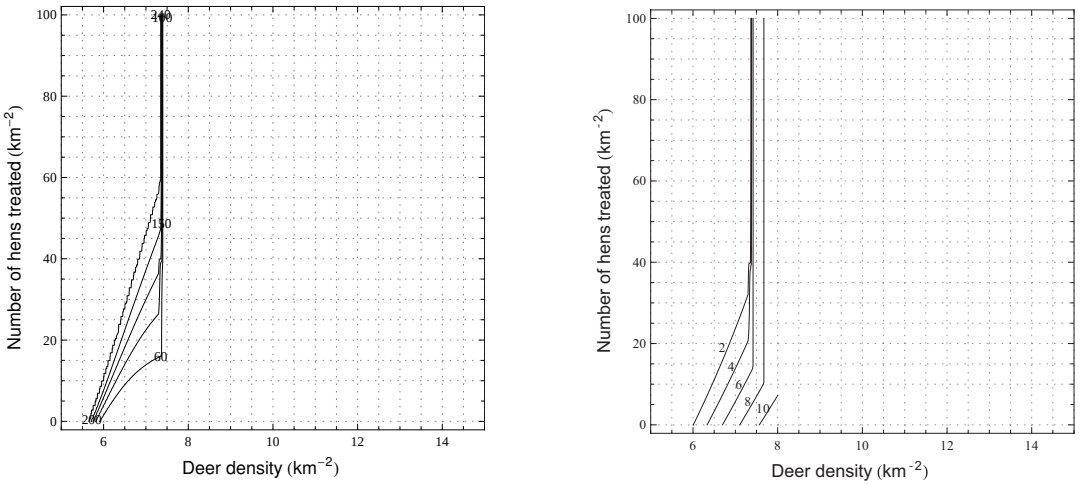
(b) Total tick density

Figure 7.1: A contour plot of model predictions of a)total grouse b)total tick densities at different deer densities for varied numbers of hens treated. Contours at 60, 100, 150, 200, 240 grouse per km^2 and 0, 2.5, 5, 7.5, 10, 12.5, 15, 17.5, 20 million ticks per km^2. Acaricide efficacy on chicks is assumed to be 100% for whole season ($w = 22$).

The acaricide efficacy and period of waning on chicks are unknown variables. The 'best case scenario' would be for the acaricide to be 100% effective for the whole summer after hatching (i.e. $w = 22$). Although this is unlikely to be achievable this gives us a limit to the maximum treatment effectiveness that may be expected. Fig. 7.1 shows that the model predicts treating as few as approx. 12 hens will increase the grouse population to a commercially viable density (> 60 per km^2) but more than 20 hens per km^2 will need to be treated to increase the grouse population above 100 per km^2 for most deer densities. As for previous chapters, if there are fewer than approx. 5.7 deer per km^2 the grouse always reach carrying capacity because there are too few deer to sustain the tick population. For deer densities above approx. 9 per km^2 the model predicts grouse never reach carrying capacity irrespective of how many hens are treated. When the deer density is above approx. 9 per km^2 there are not enough hens at the start of the year to treat more than 80 hens per km^2. Fig. 7.1b shows that the tick population is predicted to be reduced by treating grouse but is only eradicated for low deer densities when the tick population is already low due to few reproduction hosts. Grouse carry few ticks compared to deer so even if all the female grouse were treated it is likely that the tick population would always survive for higher deer densities. The tick population would serve as a reservoir for the virus with ticks infected in one year transmitting it to untreated male grouse and chicks after the acaricide has waned when they feed again in the following year. The model predictions suggest that if the deer density is high using acaricidal leg

bands on hens will not eradicate ticks in the environment and LIV if used as the only control method (ticks are still persisting when there are more than 9 deer per km^2 present even with 100 hens per km^2 treated).

Assuming 100% acaricide efficacy on chicks for 12 weeks



(a) Total grouse density

(b) Total tick density

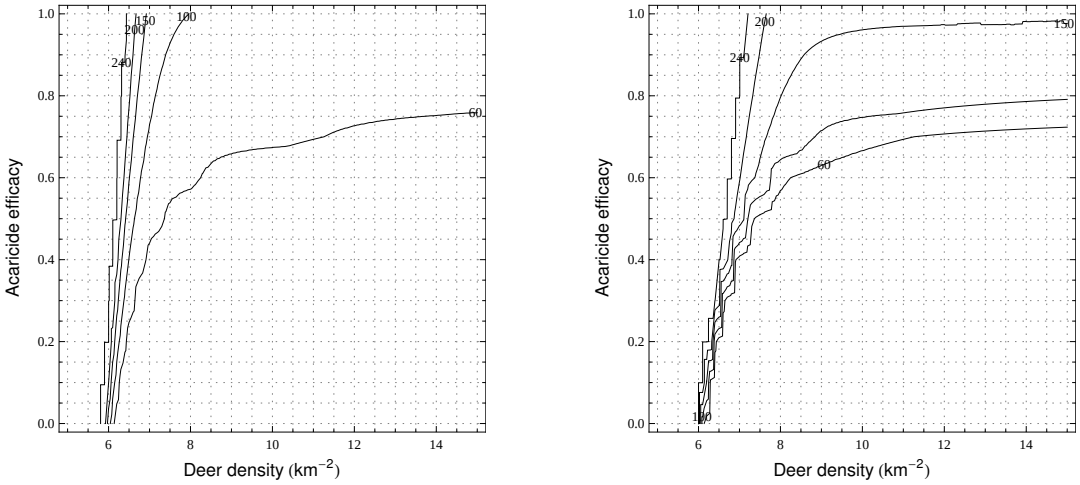
Figure 7.2: A contour plot of model predictions of a)total grouse b)total tick densities at different deer densities for varied numbers of hens treated. Contours at 60, 100, 150, 200, 240 grouse per km^2 and 2.5, 5, 7.5, 10, 12.5, 15, 17.5, 20 million ticks per km^2. Acaricide efficacy on chicks is assumed to be 100% for 12 weeks.

It is extremely unlikely that the residual acaricide on chicks will last for the whole season, consequently here the effect of varying the number of hens treated is considered for acaricide waning on chicks after 12 weeks. This may still be an overestimate of the waning period; Laurenson et al. (1997) found pour-on acaricide had reduced in strength by 50% after 8 days and the pour-on acaricide applied to sheep used in the tick reduction trial is reapplied after approx. 6 weeks. Fig. 7.2 suggests that the number of hens treated makes little difference compared to deer density. If the deer density is above approx. 7.5 per km^2 the grouse are unable to reach a viable density (> 60 per km^2), although increasing the number of grouse treated does appear to increase the grouse population to a viable density for deer densities between 6 and 7.5 per km^2. If the acaricide efficacy on chicks does not last long the model predicts that the benefit to the grouse population as a whole is limited, although the individual hens treated will always benefit.

7.3.2 The effect of varying the efficacy of acaricide on chicks of treated hens

The work of Warren and Baines (2007) suggests that treating 20 hens per km^2 is a viable target to treat, consequently we consider the treatment of 20 hens per km^2 with varying efficacy on chicks (d). This is compared to treating 40 hens per km^2, which may still be attainable with extra manpower. As explained above the length of time the acaricide remains effective on chicks is unknown but is assumed here to last the whole season.

Fig. 7.3a shows that although an efficacy above 75% is predicted to allow a viable grouse density (> 60 per km^2) for all deer densities shown the grouse are not predicted to exceed 100 per km^2 except



(a) 20 hens per km^2 treated

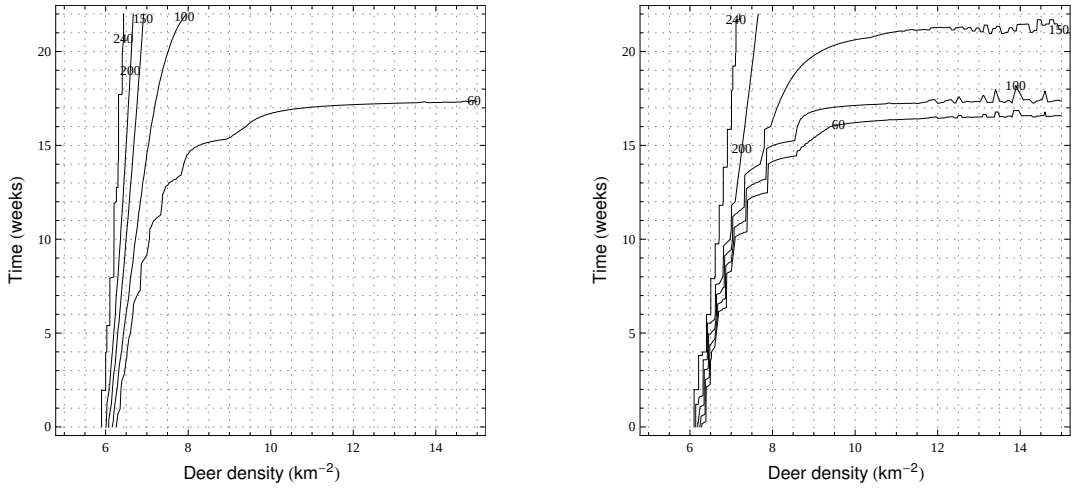
(b) 40 hens per km^2 treated

Figure 7.3: A contour plot of model predictions of total grouse densities at different deer densities for varied acaricide efficacy on chicks when a) 20 hens and b) 40 hens are treated per km^2. Contours at 60, 100, 150, 200, 240 grouse per km^2. Acaricide efficacy on chicks (d) is assumed to last the whole season.

at high acaricide efficacy and low deer densities if 20 hens per km^2 are treated. This assumes the efficacy on chicks is maintained all season which, as mentioned previously, is unlikely to be realistic.

If 40 hens per km^2 are treated the model predicts that a slightly lower efficacy (approx. 72%) may allow a viable grouse density (> 60 per km^2) and only a small increase in efficacy to 0.8 is predicted to allow the grouse density to reach 100 per km^2 (Fig. 7.3b). It is interesting that when 40 hens per km^2 are treated a small increase in the acaricide is predicted to have a much greater effect than when only 20 hens per km^2 are treated. Indeed if efficacy were 100% then the grouse are predicted to reach over 150 per km^2 for all deer densities but 100% efficacy for 20 treated hens per km^2 predicts grouse densities between 60 and 100 per km^2 for deer densities exceeding 8 per km^2.

7.3.3 The effect of varying the length of acaricide waning on chicks



(a) 20 hens per km^2 treated

(b) 40 hens per km^2 treated

Figure 7.4: A contour plot of model predictions of total grouse densities at different deer densities for varied acaricide waning time on chicks when a) 20 hens and b) 40 hens are treated per km^2. Contours at 60, 100, 150, 200, 240 grouse per km^2. Acaricide efficacy on chicks is assumed to be 100% until waning.

It is unlikely that the acaricide on chicks will last all season. Here we consider the impact of varying

the time for which acaricide is active. The acaricide efficacy is assumed constant (100%) for w weeks after hatching.

Fig. 7.4a shows the model predicts that the grouse reach a viable density if the acaricide lasts for 17 or more weeks for all deer densities shown when 20 hens per km^2 are treated. However, if the acaricide on chicks lasts for fewer than 17 weeks treating 20 hens per km^2 has a minimal effect on the grouse population. If 40 hens are treated per km^2 similar length treatments allow grouse to reach higher densities as one may expect. However, the model predicts that the acaricide needs to last for over 16 weeks to allow grouse to reach a viable density if deer densities are greater than 10 per km^2.

7.4 Summary

This chapter has considered the potential impact on the whole grouse population of treating individual females with acaricidal leg bands. The model predicts that treating individual female grouse will increase the grouse population only in limited circumstances. Treating 40 hens rather than 20 hens per km^2 increases the range of deer densities, acaricide efficacies and waning periods that allow the grouse population to reach an economically viable density for driven shooting. Treating a greater number of grouse is always more effective but the effect appears to be reduced when the acaricide efficacy on chicks is reduced or the period for which the acaricide remains effective is shortened. The current studies (Laurenson et al., 1997; Mougeot et al., 2008) do not provide the necessary information to make an estimate for either the acaricide efficacy on chicks or the length of time this lasts. Treatment of individuals will always have a protective effect on the hen and her young for the period the acaricide lasts. However, the effect on the whole population of treating individuals will be small because grouse feed few ticks relative to deer and will therefore be unable to kill many ticks. The effect will be reduced for short-lived and low-efficacy acaricide but may be increased to some extent by treating a greater number of birds. To make better predictions of how effective treatment can be expected to be with varying deer densities and number of females treated we need more information on the acaricide on the chicks.

The model predictions suggest that direct treatment of female grouse will always be of benefit to individual broods. However, unless acaricide on chicks is long lasting and of high efficacy treating individual hens is unlikely to be of benefit to the whole grouse population in the presence of deer. This could point towards the need to develop alternative methods of application of acaricide to grouse chicks to ensure a greater efficacy for longer.

However, we need to interpret the model predictions with caution. Mathematical models by their very nature are simplifications of real life based on various assumptions. The model presented here takes account of the seasonal variation in grouse and tick biology but in a simple way. Ticks are assumed to be either active (summer) or inactive (winter) but there is no variation in how active they are during the summer. The reproduction of ticks is assumed to occur at the same rate during the active period which causes the tick population to rise steadily to a peak at the end of the activity period. There is so much conflicting data in the literature about the periods of peak tick activity which may vary according

to location and year that there would be no benefit in fitting tick activity peaks to any particular set. The length of the tick activity season chosen here reflects the general trend of tick activity during the summer months (Randolph et al., 2002; Walker, 2001; Pietzsch et al., 2005).

The ingestion of ticks by young grouse chicks has also been neglected in this model. Although Chapter 4 suggested that the ingestion of ticks was of limited consequence it may have more significance here because although a tick may be killed by the acaricide and so no longer able to infect a chick through a tick bite it may still be eaten and cause infection that way. Grouse chicks may also eat ticks questing on the heather that have not been in contact with the acaricide. The ingestion of infected ticks may lessen the effectiveness of acaricidal treatment of grouse at reducing LIV prevalence.

The direct treatment of grouse will benefit those individual grouse that are treated. However, as the tick population is not eradicated the tick population will remain a reservoir of the virus over the winter and may infect untreated grouse the following summer. Even if all females are treated there will be untreated males and susceptible chicks once the acaricide has waned that could receive infection.

Mountain hares and non-viraemic transmission were not included in this model but may be an important route of transmission in areas where hares are abundant, enabling the virus to persist in the tick population even in the event of all grouse being treated. This would mean that even if LIV was eradicated in the grouse population after a few years of total treatment LIV treatment would need to be continued indefinitely whilst the virus remains in the tick population.

The model presented here is not without limitations but provides useful insight in to the possible effect of treating female grouse with acaricidal leg bands. Whilst treating individual hen grouse will benefit herself and her brood it is unlikely that the treatment will be of benefit to the grouse population as a whole. However, if high levels of efficacy can be achieved and a large number of grouse are treated then the grouse population may be increased.

There are practical as well as theoretical issues that need to be considered. The use of acaricide would need to be licensed for use on game birds to ensure safety of meat used for human consumption. The grouse need to be captured individually to receive treatment but as grouse are routinely caught for anthelmintic treatment the attachment of an acaricidal tag may not require much extra man power. The type of tag needs to be chosen to ensure no damage is caused to the bird and a long lasting acaricide on chicks may be more effective. If these issues are resolved the model presented here suggests that the treatment of individual grouse broods has the potential to be a useful method of reducing LIV in red grouse.

Chapter 8

Discussion

The aim of this thesis was to explore potential tick and tick-borne disease management strategies using LIV as a case study. In the light of new empirical evidence we extended the previous models of the LIV-red grouse-tick disease system to include an additional route of infection (ingestion). We also considered the impact on model predictions of the seasonal dynamics of grouse and ticks. We used models to investigate how effective treating sheep, deer and grouse with acaricide might be at reducing ticks and LIV. It became apparent in initial investigative work that including mountain hares as tick reproduction hosts and a source of virus transmission brought additional complexity in model analysis and potential error from unknown parameters. Consequently mountain hares were removed from the models and the findings of this thesis are applicable only to hare free environments. However, we can tentatively predict that the management strategies will be of reduced effectiveness in the presence of mountain hares since it is known that hares feed all stages of ticks and allow non-viraemic transmission (Gaunt, 1997).

8.1 Model development

Ingestion as a route of infection

The first aim of this thesis was to develop the model to include a route of infection that had not been incorporated in previous models (e.g. Gilbert et al. (2001); Laurenson et al. (2003); Norman et al. (2004)). Chapter 2 investigated the role of the ingestion of ticks by young grouse chicks as a route of infection. Ingestion was initially added directly to the model of Gilbert et al. (2001) but it became apparent that the model needed to be reparameterised so that a more realistic tick density was predicted. After including new parameters estimates (App. A) the model predictions suggested that ingestion may be an important route of infection with virus persisting for a greater range of host densities when ingestion is included. However, at high grouse densities and low deer densities the model predicted that the consumption of ticks by grouse may reduce the tick population below the threshold required for virus persistence.

An interesting mathematical concept was discovered during the development of the ingestion model. The reproductive rate of the virus R_0 is often used to develop a threshold for different host densities for virus persistence. However the ingestion of ticks by grouse leads to an interesting feedback mechanism

that causes the traditional form of R_0 to persistently underestimate the persistence of the virus. The ingestion of ticks by grouse causes infection which reduces the grouse population. The reduced grouse population eats fewer ticks so the tick population grows. A greater tick population leads to an increased likelihood of infection reducing the grouse population further. Thus the tick population is able to increase further and so on until equilibrium is reached.

R_0 is defined as the number of new infections created when one infected individual is added to a wholly susceptible population and is given by

$$R_0 = \frac{\beta_2(\beta_1 + P\beta_3)K_g K_t + \Gamma\theta K_t H}{\Gamma(b_t + \beta_3 K_g + \beta_4 H + \beta_5 D)}$$

with K_t and K_g the carrying capacities of ticks and grouse without disease. R_0 and K_t both assume that the number of grouse ingesting ticks is always K_g but once the grouse density is reduced by disease the density of grouse eating ticks will be lower and hence the tick population will be higher. Consequently R_0 always underestimates the persistence of the virus and is no longer a useful aid in determining the host densities required for virus persistence. However, a second pathogen persistence threshold (PPT) value can be found using the estimated densities of tick and grouse densities at disease induced equilibrium for different deer densities in the R_0 formula (i.e. replace K_t with T^* and K_g with G^*) to estimate when the virus does and does not persist. This threshold is in agreement with the model predictions; the grouse are predicted to reach carrying capacity when the PPT curve shows the virus not to persist (and vice versa). This phenomena has not been recorded elsewhere as far as we are aware and highlights the dual impact that this unusual route of infection has on both the grouse and tick population predictions.

Seasonal dynamics

The seasonal biology of the grouse and the ticks were considered in Chapter 4 and Chapter 5 respectively. Previously published models have not incorporated the seasonal dynamics of the system which are known to occur and may affect transmission. The ingestion of ticks in particular occurs for the first three weeks after a grouse chick hatches (Gilbert et al., 2004). The seasonal hatching of grouse was incorporated in to the model in two ways; firstly as a single pulse, secondly as a period of hatching. The advantage of the pulse hatch model was that the ingestion of ticks could easily be incorporated for the first three weeks after hatching, however, the grouse pulse hatch model predicted damped oscillations occurring in the long term, i.e. for centuries, which is not realistic. The grouse hatching period model incorporated a more realistic hatching period which decreased the long term oscillations but did not incorporate the ingestion of ticks by chicks. Both models predicted a much lower level of infection in grouse and ticks compared to the non seasonal model.

The explicit modelling of chicks in the grouse pulse hatch model suggested that using the assumption that grouse are 5.25 times more likely to get infected through ingestion than by being bitten (as in Ch. 2) lead to unrealistically high numbers of ticks being consumed. Once the rate had been reduced to restrict ingestion to 20 ticks or fewer per chick per day the inclusion of ingestion no longer made significant

differences to model predictions of virus persistence. The sites used to estimate the effect of ingestion on LIV prevalence in grouse in Gilbert et al. (2004) had very low biting rates compared to ingestion leading to the conclusion that ingestion may be the primary source of infection. The tick biting rates of the model are high (approx. 9 ticks per chick at any one time, or 1.8 ticks per day) in comparison to the findings of Gilbert et al. (2004) (0.255 ticks per chick at any one time, or 0.051 ticks per day) hence the model will give more importance to the tick bite route of infection when compared to the empirical data. The model could use the tick burdens on chicks from Gilbert et al. (2004) which may increase the influence of ingestion. However, given that there is great variation in tick burdens found on hosts (Laurenson et al., 2003) it would be difficult to extrapolate the findings to other areas, especially as tick burdens on chicks appear to be rising, e.g. Kirby et al. (2004) found that the average tick load had increased to 12.71 ± 1.44 in 2003 from 2.60 ± 1.12 in 1985 on 13 estates in Scotland.

The hatching period and hatch rate of the grouse hatching period model were investigated further to determine the cause of the long term oscillations. It appeared that the within year variation of the seasonal models interacted with the underlying fluctuations of the model predictions. Greater variation within the year lead to increased oscillations. Both seasonal grouse models predicted much lower levels of infection than have been estimated from field data because the short time interval in which susceptible grouse chicks hatch does not allow enough opportunity for infection to occur unless the number of chicks that hatch is increased.

The seasonal biology of ticks and the effect this had on model predictions was discussed in Chapter 5. Ticks are known to have peak periods of activity during the year although the size and timing may vary due to location and climatic conditions (Randolph et al., 2002; Gilbert, 2010). However, in general ticks are more active over the summer and quiescent during colder weather. The ticks were therefore assumed to be active for 32 weeks (Apr-Oct) and inactive for 20 weeks (Nov-Mar) each year (Gilbert, unpublished data). The inclusion of seasonal tick biology had less of an effect on model predictions than the seasonal grouse biology had. The long term predictions showed no additional fluctuations compared to the non seasonal model and the predicted level of infection was much closer to the non seasonal model prediction. The seasonal activity of ticks occurs for over half the year and the within year variation of the tick population is relatively small compared to the within year variation of the grouse population with seasonal grouse biology. The grouse hatching period model was combined with the seasonal tick model which caused damped oscillations to occur in the long term predictions and also reduced the infection prevalence.

8.2 Model limitations

The length of the tick activity season was varied to simulate the possible effect of climate change. The model predicted that a longer questing period may increase the tick population and hence the incidence of LIV in chicks. However, the model does not take account of the fact that different tick stages may have different peak questing periods (Randolph et al., 2002) which may affect virus transmission. A more

detailed model that could take account of the different tick stages may provide a more reliable indication of climate change effects.

There is so much variation in empirical data which may be due to factors that have not been considered in this and related LIV studies. Red grouse populations suffer from parasitism of the intestinal strongyle worm *Trichostrongylus tenuis*, which can cause population cycles seen in natural populations (Hudson, 1992; Hudson et al., 2002). Grouse populations are assumed to be at equilibrium in Gilbert et al. (2001) in order to make comparisons with model predictions but it may be that the natural populations were at a point of a cycle and the prevalence of LIV may be affected by the presence or absence of worms. Although Hudson et al. (2002) propose that LIV infection dampens cycles caused by *T. tenuis* trying to fit model predictions with field data may always be difficult.

Given the complexities and variation of the natural world it is not possible to accurately predict levels of infection at a given place at a given time. Although the different seasonal models predict different infection levels they all make the same predictions of the deer densities for which virus is predicted to occur. Therefore although mathematically interesting in terms of answering questions regarding when virus persists and management strategies might be successful the infection levels are not important.

The temporal model predictions showed damped oscillations when seasonal grouse biology was included but not when only seasonal tick biology was considered. However, there was no difference between model predictions of the deer densities for which grouse were predicted to reach carrying capacity and virus to die out. Although there were slight numerical differences in the predicted grouse and tick densities for higher deer densities the overall pattern of the predictions was the same; large deer densities allow large tick populations which infect the grouse and drastically reduce the grouse density. All the seasonal models predicted grouse numbers were significantly reduced when deer densities exceeded 6 deer per km^2, which agrees with the non seasonal predictions. Consequently the seasonal dynamics of the grouse and tick populations do not seem to be important when considering the likely effect of management strategies at different deer densities. Incorporating seasonal dynamics adds complexity to a model making it impossible to analysis it algebraically and there is no longer a way of determining the reproductive rate of the virus. Seasonal dynamics may be important if temporal predictions are required but this would require a much better knowledge of the model parameters and the aim of this thesis was to consider the potential effect of management strategies at different deer densities not temporal dynamics. Given that all the seasonal models make broadly similar predictions for virus persistence at different deer densities it seems the added complications of a seasonal model are not outweighed by any further insight. Therefore seasonal biology was not included in the models of Chapters 3 or 6.

Although the use of seasonal models may be limited in answering questions regarding potential management strategies the grouse pulse hatch model was useful in determining the effect of the ingestion of ticks by grouse chicks. Modelling chicks explicitly highlighted the fact that the chicks were consuming unrealistically high numbers of ticks in the non seasonal model. We were then able to modify the model to incorporate a more realistic level of ingestion.

Ghosh and Pugliese (2004) developed a semi-discrete model of tick dynamics and tick-borne encephali-

tis virus (TBEv) in Italy. The model of Ghosh and Pugliese (2004) includes more detail of the tick biology than this thesis with different stages modelled separately and an assumption that a proportion of eggs laid by an adult tick do not hatch and begin to quest until the following season. The number of ticks in each stage are given by integrals based on the variables from the previous year taking into account the likelihood of successfully moulting and surviving the winter. This model is compared to a similar non seasonal model of TBEv (Rosa et al., 2003) and Ghosh and Pugliese (2004) suggest the seasonal model predicts that the virus may die out for lower host densities than predicted by the seasonal model. However, the model of Ghosh and Pugliese (2004) assumes that the hosts are unaffected by the virus but the hosts in Rosa et al. (2003) suffer virus induced mortality. It is, therefore, impossible to tell if the prediction that the virus may die out at lower host densities is due to the seasonality of the model or if it is the lack of virus induced mortality. There is currently insufficient data on ticks in Britain to design a model with the detail of Ghosh and Pugliese (2004). New data on the peak questing periods of different tick stages on grouse moorland habitat would allow a more detailed seasonal model to be developed including the different tick stages and their different transmission potentials. This would be time consuming and expensive. If the difference in model predictions for Ghosh and Pugliese (2004) (compared to Rosa et al. (2003)) could be attributed solely to the seasonal dynamics then this may suggest that it is worthwhile to spend time and resources developing a seasonal model of LIV. However, there is insufficient evidence from Ghosh and Pugliese (2004) to predict whether an improved seasonal model of LIV might be of benefit to understanding the dynamics of the LIV system.

8.3 Management strategies

The main aim of this thesis was to consider potential management strategies to reduce tick abundance and LIV in red grouse. The strategies under consideration are 1) using acaricide treated sheep to kill ticks, 2) treating deer with acaricide to kill ticks and 3) direct treatment of female grouse with acaricidal leg bands to prevent ticks biting the hen and her chicks. The use of acaricide treated sheep is currently being trialled by the Game and Wildlife Conservation Trust (GWCT) with preliminary results available online (Smith, c.2006). Acaricide is not currently licensed for use on deer but as deer are known to be important tick reproduction hosts it is interesting to consider theoretically the potential of treating deer. Direct acaricide treatment of grouse has been trialled in different ways and to varying degrees of success but has the potential to be a practical method to reduce LIV in red grouse (Laurenson et al., 1997; Mougeot et al., 2008).

Acaricide treated sheep

The findings of the GWCT (Smith, c.2006) suggest that the use of acaricide treated sheep put on to grouse moors with the purpose of killing ticks that tried to attach has limited success and the treatment was found to be ineffective when deer densities are high (> 10 per km^2). Chapter 3 uses a mathematical model to explore the role of acaricide treated sheep in different scenarios, which is rarely possible in

field experiments. In order to model the effect of sheep we had to estimate the attachment rate of ticks on sheep. We were fortunate to be allowed access to a farm to count ticks attached to treated and untreated sheep. It became apparent that attachment rates can vary widely with some data suggesting tick burdens on sheep to be considerably higher than the tick burdens we encountered. To overcome the discrepancy in the data we used two different estimates to simulate an area with a high tick burden on sheep and an area with a low tick burden on sheep. Not surprisingly the treated sheep with a high tick burden were predicted to be more effective at reducing tick density and hence virus in red grouse than the treated sheep with a low burden. However, the model predicted that the effectiveness of treated sheep at reducing ticks and LIV was significantly reduced in the presence of high deer densities (> 10 per km^2 for the low burden model) in agreement with the field trials by the GWCT. The model also predicted that higher sheep densities were more effective and that even if virus is persisting the grouse densities can be increased by the addition of acaricide treated sheep.

The efficacy level of the acaricide was altered to determine the impact efficacy has on the grouse population. Higher efficacy was predicted to increase the grouse population quicker for a given deer density as one might expect. However, it also became apparent that a low efficacy reduced the grouse population compared to no sheep at all. This unexpected effect stems from the fact that untreated sheep allow adult ticks to feed and reproduce, if the acaricide on sheep is of low efficacy so that the sheep are allowing tick reproduction to occur at a greater rate than the acaricide is killing ticks then the tick population will grow and the grouse population decrease. If the sheep were absent the ticks would not be able to feed upon the sheep and the tick population would be lower. This highlights the importance of keeping acaricide efficacy high if sheep are to be used to reduce the tick population. However, anecdotal evidence suggests that the deer density may be reduced by the presence of sheep so even low efficacy sheep may be of benefit if they are a deterrent to deer.

The use of mathematical models to explore the use of acaricide treated sheep has brought to light the difficulties in determining the potential effect of treating hosts with variable tick burdens but by varying the parameters models can be used to predict the potential effect of treating sheep under different circumstances that cannot be easily tried in field experiments. Large-scale, replicated, controlled field trials for all scenarios would be extremely difficult, if not impossible, to conduct. The model highlights the differences between sheep with high and low burdens but also the similarities in that both are rendered ineffective with high deer densities present. The effect of acaricide efficacy can be explored with a model without putting livestock and farmers at risk as a field trial would. This exploration lead to the unexpected but important prediction that sheep treated with low efficacy acaricide may be detrimental to grouse populations due to an increase in ticks and LIV.

Acaricide treated deer

Chapter 6 follows on from the implications of Chapter 3. If acaricide treated sheep are less effective (if at all) in the presence of deer then it seems logical to use a model to explore the effect of treating deer, especially as practical trials of treating deer with acaricide in the UK are not currently possible. Trials

in the US have found that the treatment of white tailed deer when they visit feeding stations may reduce the questing tick population by approx. 70% (Carroll et al., 2002).

The model presented in Chapter 6 predicts that treating deer with acaricide may be an effective method of reducing the tick population, especially if used in combination with culling. Culling already forms part of estate practice for many landowners as part of habitat management and also as an additional source of income through deer stalking. The model predicted that a lower deer population requires a lower efficacy of acaricide to achieve tick and LIV eradication. Although the US studies (Carroll et al., 2002; Brei et al., 2009) suggest a high efficacy of acaricide can be attained the conditions of the UK are not comparable to those in the US and the efficacy that could be achieved in the UK may well be lower. The red deer of the UK have a different social structure to the white tailed deer of the US. The hierarchy of red deer means dominant individuals may prevent subordinate deer accessing the acaricide. Although supplementary feeding of red deer occurs on some Scottish estates this is only over winter when natural food is scarce and not during the summer when ticks are active reducing the likelihood of success.

Acaricide treated grouse

The third management strategy considered was that of directly treating grouse with acaricide. Chapter 7 used a model to explore the potential impact of treating female grouse with an acaricidal leg band on the grouse population as a whole. Mougeot et al. (2008) found that a leg band may benefit an individual female and her brood by reducing tick attachment but the wider effects of treatment remain unknown. Herd immunity is a well known concept in studies of vaccination; only a proportion of a population needs to be vaccinated to reduce the chance of infection for the whole population. Using a mathematical model we were able to test whether a similar theory may apply to treating grouse with acaricide; i.e. can treating a proportion of the grouse population with acaricide benefit the whole population?

The model of Chapter 7 was chosen to be seasonal to allow explicit modelling of the indirect treatment of grouse chicks after rubbing against the acaricide on the mother's leg band. The acaricide on chicks is likely to be less effective than on the mother and once brooding is over the acaricide on chicks is likely to wane. Although this was modelled explicitly there are no published estimates of the efficacy of acaricide on chicks or the length of time that it remains effective. However, we were able to vary these parameters to estimate the likely effect of acaricide strength and the waning period.

Using the 'best case scenario' (i.e. that chicks were 100% efficacious for the whole summer) the model predicted that treating only 20 female grouse could increase the grouse density to above 100 per km^2 for all deer densities tested. The grouse population did not reach carrying capacity unless high numbers of grouse (> 60) were treated, and then only for low (< 8 per km^2) deer densities. The treatment of grouse will kill relatively few ticks compared to treating sheep or deer and is therefore likely to be of less benefit to the whole grouse population. Unlike the treatment of sheep and deer the treatment of grouse can be effective without eradicating the tick population. Ticks do provide protein for birds (including grouse chicks, Park et al. (2001)) and it may not be desirable to be rid of them completely. When the efficacy of the acaricide was reduced or the time of efficacy shortened the grouse population was less able to reach

a viable density even if high numbers of grouse were treated. This suggests that unless the acaricide efficacy on chicks can be maintained at a high level treatment of females is likely to only benefit the individual broods and not the population. If the efficacy on chicks is likely to be low and/or for a short time a greater number of females will need to be treated in order to bring the grouse population to an economically viable density for driven shooting.

The seasonal acaricidal leg band model of Chapter 7 assumes the activity of the ticks is constant during the summer months and therefore may miss particular peaks that may affect how effective the treatment of grouse actually is. For example, if ticks are most active when chicks are first exposed to acaricide and therefore are highly effective at killing ticks the effect of the treatment will be greater than if tick activity peaks after the acaricide has started to wane. The model is therefore likely to be underestimating the effectiveness of the treatment as the model predicts the peak in the tick population occurs at the end of the activity period when tick reproduction has ceased.

The model assumes that adult grouse and chicks are equally likely to be bitten and infected by a tick but this may not be the case. Although the immune class contains the adults that have previously been infected and are no longer susceptible even those adult grouse that are susceptible to LIV may have developed some immunity to tick bites and therefore be less likely to be bitten. Hudson (1986a) found a higher level of tick attachment on grouse chicks aged 10 weeks compared to adult grouse. The model also overlooks the ingestion of ticks by chicks. Although this was found to be of limited consequence in Chapter 4 it may be more important here as grouse chicks that are protected by the acaricide from tick bites may still be infected after eating ticks that are questing on the heather or preened from siblings.

What we have not been able to show using all these models is how the practical needs and costs of these strategies may be met. Acaricide and applicators will have a financial cost as well as the cost of labour to implement the treatment. Sheep may well be treated with acaricide for their own benefit to reduce tick and mite infestation so the additional cost of using acaricide treated sheep may be in collecting them from and returning them to the moor rather than the treatment itself.

Grouse on moors with high incidence of the gut nematode *T. tenuis* are often routinely caught and treated with an anthelmintic to reduce worm burdens early in spring so an acaricidal tag could be fitted at the same time with little additional cost in man hours. The acaricide treatment itself is inexpensive so this may be an effective strategy to adopt if reasonable numbers of grouse can be caught and efficacy on chicks maintained for long enough.

The acaricidal treatment of deer although theoretically of great benefit may be least practicable. At present acaricide is not licensed for use on deer and trials would need to be undertaken to ensure the acaricide could be applied and withdrawn safely before deer are shot for human consumption. If feeding stations are to be used as the method of acaricide application the stations would need to be purchased, taken up on to the hill and assembled, which would entail a large set up cost. A lot of stations would be necessary and the stations would also require frequent replenishing and maintenance.

All of the methods presented in this thesis have their limitations and we acknowledge that the numerical predictions of the models cannot be made with any certainty. However, this thesis has been able to

address important issues regarding the management of LIV in red grouse and the models provide a useful aid in determining the general pattern of virus persistence and likely outcome of treatment success.

In particular, we have found that deer are the most significant factor in the likely success of a treatment strategy. If deer densities are low using treated sheep may be effective but we suggest that acaricide treatment would need to be reapplied frequently to ensure the presence of treated sheep does not have a detrimental effect. If deer densities are high an alternative or additional method may be more successful if deer densities cannot be reduced.

If deer densities are high acaricidal leg bands on female grouse may be a useful strategy as this treatment will always be of some benefit to individual broods. Individual treatment is unlikely to be of benefit to the whole grouse population. If treatment efficacy and longevity on chicks is high the benefit to the whole population may be greater than for lower/short lived treatment.

If deer densities are high reducing densities by culling can improve the success of all treatments. Indeed reducing the deer population sufficiently may reduce the tick population sufficiently for the virus to die out (if no other large hosts are present). However, the treatment of deer with acaricide may provide a more benign solution, allowing higher deer densities to remain whilst conserving the grouse population. This may be desirable for conservation purposes or for sporting estates that also shoot deer.

Ticks and tick-borne pathogens are found throughout the world. Although we focus on LIV, the western variant of the TBE complex of viruses, in this thesis the models and methods discussed here could be applied to other tick-borne disease systems. In particular, TBEv may benefit from similar management strategies because deer are also incompetent hosts for TBEv. There is a great deal of data on TBEv which could be used to parameterise models similar to those presented here for TBEv endemic areas to predict the potential effect on the tick population and virus prevalence.

8.4 Future work

Mountain hares are known to be significant in the transmission of LIV as they both host ticks and transmit LIV through non-viraemic transmission. Most estates that use tick control strategies have heavily culled their mountain hare populations. Hares have not been included in the models of this thesis because we do not have any estimates of the rate at which non-viraemic transmission is thought to occur. Including hares brings further uncertainty to models that already have unknown parameters. However, it would be useful to develop the models of management strategies to include mountain hares as some estates do still have mountain hares.

The models could all be improved by accurate up-to-date estimates of tick attachment rates on different hosts on the same site at the same time. More detailed information on different tick stages (i.e. peak periods of questing, how long each stage remains attached, aggregation of stages on/between hosts) could enable more detailed models like those of Rosa et al. (2003); Ghosh and Pugliese (2004); Rosa and Pugliese (2007) to be developed. Modelling the different periods of questing and attachment may give greater insight in to the importance of different tick stages and interventions could be tailored to target

specific stages. The data of Laurenson et al. (2003) shows there is variation in tick burdens on hosts between sites so no one model would be an accurate representation of different sites. However, models of this type are a useful predictor of general trends.

The density dependence of ticks is not fully understood but likely to act on moulting and development through life stages (Rosa et al., 2003). Rosa et al. (2003) experimented with different density dependence functions that may be a more realistic representation than the function used throughout this thesis. It would be interesting to explore similar functions (i.e. negative exponential function depending on total tick population or host tick burden) within the models of this thesis. Rosa et al. (2003) found that the tick population showed a different response to increasing host densities with the different density dependence functions. Rosa et al. (2003) favour the function depending on host tick burden but acknowledge that real life may be somewhere between the two. To make conclusions for the LIV system we would need data on relative tick abundance at different host densities in order to gauge if model predictions of tick abundance are correlated to host densities in the same way as empirical evidence suggests.

The model of Gilbert et al. (2001) on which this thesis is based has already been extended to cover the spatial spread of LIV through the movement of ticks on deer (Watts et al., 2009; Jones et al., 2010). The success of any treatment strategy is dependent on deer density and it would be interesting to consider how the movement of deer between sites might affect treatment. Using acaricide directly applied to grouse has the potential for LIV to be eradicated without removing all ticks, however, LIV may be reintroduced by infected ticks on mountain hares or on deer from neighbouring estates. This could be explored with a spatial model.

At present we assume that the hosts are evenly mixed in homogeneous space. However, treating sheep or deer may be less effective at reducing LIV prevalence in grouse if they are not reaching the ticks that feed on grouse preferring to feed in different areas. This could be incorporated into a spatial model by manipulating host densities in/between different patches.

Appendix A

Field Work

A.1 Estimating tick density

To develop the ingestion model (Chapter 2) it was necessary to estimate the number of ticks available for the grouse to eat. This may include ticks that are not actively questing on the tips of the heather but are on the lower growth. There are no available published data on the actual density of ticks found on a grouse moor. Although many studies have collected questing ticks using the blanket drag method (Gray and Lohan, 1982) it is not known what proportion of the actual tick population is collected in this way. Clearly this method will not collect those ticks that are hiding within the matt layer or questing on the lower reaches of the undergrowth at the time of the drag. In order to estimate the true density of ticks on heather moorland from existing blanket drag data we needed to approximate the proportion of ticks that are picked up by the blanket drag method in heather moorland. To achieve this a simple experiment was conducted during the summer of 2008.

In order to make an estimate of the density dependence of ticks it is necessary to have an estimate of the total tick population so that the model predicts a biologically realistic density of ticks.

Blanket dragging is a well established standard method of collecting and counting questing ticks (Gray and Lohan, 1982). Although blanket dragging is a useful aid in giving an index of relative tick abundance between different areas of similar habitat it does not provide an estimate of the density of the tick population.

To gain an index of the relative questing tick abundance by blanket dragging a 1 metre square pale woolen blanket, weighted with a wooden pole at the front, is dragged for a distance of 10 metres. After the drag is completed the blanket is overturned and any ticks that have attached are collected and counted.

The obvious limitation of this collection method, particularly in dense vegetation such as heather, is that only ticks that are actively questing at the top of the vegetation will be picked up by the blanket. The method can be enhanced by prodding the blanket into the heather at the beginning, middle and end of the drag but there will still be questing ticks not captured. Furthermore, ticks that are dormant in the matt layer beneath the vegetation will always be missed. Although a useful aid in determining which areas have a higher/lower tick population blanket dragging is not in itself a sufficient resource for making

a prediction of the actual total tick population.

Studies have been conducted on the efficiency of blanket drag methods in deciduous woodland in the US which concluded that on average 6.3% of the tick population was collected per drag (Daniels et al., 2000). Although this may lead us to believe a similarly low proportion may be collected in British drags the moorland of upland Britain has quite different characteristics and dragging a blanket above heather is unlikely to compare to grass/bare earth on a woodland floor. The material of the blanket and the length of drag are not consistent with those typically used in the UK. The methods and location of Daniels et al. (2000) are not sufficiently similar to those of Scottish moorland and cannot be used to predict the proportion of ticks would might expect to collect on a blanket drag of a heather moorland in the UK. Instead we conducted our own experiments to make an estimate of proportion of ticks that are collected by the method of blanket dragging on heather moorland in the UK. We could then apply this "efficiency index" to existing blanket drag data for typical Scottish grouse moors to obtain an estimate of a typical tick population density.

Methods In order to estimate the density of ticks per km^2 we combined data from $10m$ blanket drag surveys with the blanket drag efficiency determined from the experiment outlined below. The number of ticks per km^2 was calculated using,

$$\text{ticks per } km^2 = \frac{\text{ticks per } 10m \text{ blanket drag}}{\text{percentage of tick population caught}} \times 100,000.$$

In this experiment a known number of ticks were added to four one metre square patches of heather known not to contain ticks previously. The metre squares patches were isolated from the surrounding vegetation by means of a trench dug around them. This was to minimise the chances of ticks escaping. The plots were then left for 8-9 days to allow the ticks to acclimatise and begin questing. The plots were then blanket dragged and the number of ticks captured were counted. Caught ticks were replaced at the base of the vegetation before subsequent drags were made. This process was repeated four times in total; one afternoon, the morning and afternoon of the following day and again the next morning. (Dates were 13-15 August 08).

Due to time constraints on the first afternoon each plot was dragged four times, on subsequent days each plot was dragged until two consecutive drags contained no ticks. On subsequent days drags were also made around the plots to check for escapees.

The dragging was attempted again a week later but progress was hindered by heavy rain and the number of escapees found rendered the results unusable so the experiment was curtailed.

Results When $10m$ blanket drags are used as a standard method of calculating relative tick burdens only one drag is performed on each strip of land. Therefore in order to estimate the proportion of the tick population picked up by a standard blanket drag only the first drag for each plot in each sampling session will be included. Subsequent drags were taken out of interest to see how many ticks would be collected in total. It is possible that the movement of the heather caused by the first drag may encourage

Table A.1: Nymphs collected on the first drag from each plot for each sampling session. The actual number and percentage caught of nymphs are both given with the percentage in bold.

	Nymphs added	Nymphs caught (Actual **Percentage**)							
		Session 1		Session 2		Session 3		Session 4	
Plot 1	158	5	**3.3**	1	**0.7**	0	**0**	3	**1.9**
Plot 2	150	2	**1.3**	0	**0**	0	**0**	5	**3.3**
Plot 3	150	0	**0**	3	**2**	4	**2.7**	4	**2.7**
Plot 4	150	0	**0**	0	**0**	1	**0.7**	3	**2**

ticks further down in the heather to climb to the higher reaches of the heather which would allow them to be collected on subsequent drags. It could also be a feature of the natural variation in the way the blanket passes over the heather that allows some ticks to be collected whilst others are missed.

Unfortunately some escaped ticks were found, probably due to the release of the ticks at the base edges of the plot after capture. Although this meant that we no longer knew the exact number of ticks still available on each plot the experiment could still give a useful insight into the efficacy of blanket dragging in making estimates of total tick populations. The percentage of ticks lost was low with a maximum of 2.6% from plot four.

To calculate the percentage of ticks caught only the first drag on each plot for each sampling session was used. Only the number of nymphs could be used as too few of the other stages were caught. These data are summarised in Table A.1. (The complete raw data for the number of larvae, nymph and adult ticks added to each $1 \times 1m$ plot and the numbers collected are given in Tables A.5, A.6, A.7, A.8 at the end of this appendix.) Using the first drag data only we calculated that on average $1.28 \pm 0.30\%$ (mean $\pm$ s.e.) of nymphal ticks were caught.

Altitude	# drags	Mean	S.E.
500	118	1.54	0.44
550	118	0.45	0.10
600	118	0.34	0.08
650	118	0.08	0.03
700	118	0.05	0.02

Table A.2: A summary of nymphs caught per blanket drag for each altitudinal gradient on 9 hills in eastern Scotland.

# drags	Mean	SE
15	0.27	0.15
21	1.05	0.57
20	0.30	0.16
14	0.29	0.16
15	0.07	0.07
20	0.05	0.05
16	0.13	0.09

Table A.3: A summary of nymphs caught per blanket drag for 7 grouse moors in central and eastern Scotland.

Blanket drags are not used to determine actual tick densities but are a useful aid to determining relative differences between sites as it is acknowledged that they will not pick up ticks that are not questing or that are low in the heather. However, given an estimate of the proportion of ticks that may be caught on an average drag it is possible to make a prediction of the total tick density. For example on average 1.54 nymphs were caught per blanket drag at $500m$ altitude (Table A.2) thought to be approx 1.3% of the tick population, hence one can estimate that the tick population may actually be in the region of 118.5 per 10 m^2, by extrapolation this gives approx. 11.85 million per km^2. Tables A.2 and A.3 provide a summary of blanket drags on heather moorland managed as grouse moors across central and eastern Scotland. Although ticks per blanket drag vary from site to site a rough average of ticks

found on altitudes typical of a grouse moor suggests that tick populations on a grouse moor may be of the order of magnitude of 10 million. In addition Gilbert (2010) nymph counts vary between 0 and 11 but are much lower in Ruiz-Fons and Gilbert (2010) with the highest mean value less than 2 per drag.

A.2 Estimating tick burdens on sheep

The model of sheep tick mops presented in Chapter 3 required parameter estimates for the tick burden on sheep. I was fortunate to be able to assist in the counting of ticks on sheep at a farm in Scotland taking part in the GWCT trials of sheep tick mops in the summer of 2008. These counts were then used to form the basis of the low burden model presented in Chapter 3.

Chapter 3 discusses a model of sheep 'tick mops': acaricide treated sheep are put out on the hill with the intention that the acaricide kills the ticks that try to attach, thus reducing the tick population and potentially the number of ticks biting grouse. Some estates in Scotland are currently trialling the use of sheep as 'tick mops' in an effort to reduce the tick burdens on red grouse. The trials apply acaricide to the 'sheep mops' every six weeks (or as close as possible given weather and other practical constraints). As a control a number of sentinel (untreated) sheep are put out on the moor alongside the treated sheep. The number of ticks attaching to the treated and untreated sheep are counted and compared to assess the efficacy of the sheep tick mops.

We are grateful to the Game and Wildlife Conservation Trust for allowing access to a farm taking part in the sheep mop trials to count the tick burdens on both treated and untreated sheep. This allowed us to make an estimate of the tick burdens on sheep and to use this to model the potential impact of using sheep tick mops under various scenarios.

Sheep were hefted on different areas of the estate, with and without deer. The table below summarises the data for untreated sheep on the whole estate and the areas with and without deer.

	Untreated sheep					Treated sheep				
Area	Ave	SE	Min	Max	n	Ave	SE	Min	Max	n
Whole estate	4.27	1.25	0	11	11	0.38	0.24	0	6	26
Deer	6.4	2.11	0	11	5	1.5	0.96	0	6	6
No deer	2.5	1.18	0	7	6	0.05	0.05	0	1	20

Table A.4: Total tick burdens counted on untreated and treated sheep on a moorland in Scotland, both from an area with no deer and an area with deer present. Counts were taken in the summer of 2008.

In addition tick burdens on treated sheep were also counted, 1 (of 20) treated sheep from the no deer area had one tick. On the area with deer 3 (of 6) treated sheep had one or more ticks. On average 83% of ticks on sheep were adults (combining all sheep burdens on treated/untreated throughout the estate), this reduced to approx. 70% on untreated sheep. Deer are usually found on grouse moors so we used the average estimate for the areas with deer in the model, i.e. 6.4 ($\pm$2.11) ticks per sheep. However, altering the tick burden on sheep within the limits we found empirically had little effect on model predictions. For the low burden model (Ch. 3) we assumed that the tick burden on sheep was approx. 75% of that found on grouse (grouse are assumed to host 9.2 ticks on average (Gilbert et al., 2001)) and that 80% of the ticks on sheep are adults.

Plot 1		Larvae	Nymph	Adult F	Adult M
Number added		0	158	0	0
Date and time	Drag #	Number of ticks collected			
		Larvae	Nymph	Adult F	Adult M
13/8/08 pm	1	0	5	0	0
	2		8		
	3		0		
	4		2		
14/8/08 am	1	0	1	0	0
	2		1		
	3		1		
14/8/08 pm	1	0	0	0	0
	2		0		
	3		1		
15/8/08 am	1	0	3	0	0
	2		2		
	3		0		
	4		1		
	5		1		
	6		0		
	7		2		

Table A.5: Ticks counts from repeated blanket drags on plot 1. The two consecutive zero drags before stopping are not reported. (F-female, M-male)

Plot 2		Larvae	Nymph	Adult F	Adult M
Number added		62	150	5	5
Date and time	Drag #	Number of ticks collected			
		Larvae	Nymph	Adult F	Adult M
13/8/08 pm	1	0	2	0	0
	2		2		
	3		1		
	4		0		
14/8/08 am	1	0	0	0	0
	2		1		
	3		0		
14/8/08 pm	1	0	0	0	0
	2		0		
15/8/08 am	1	0	5	0	0
	2		5		
	3		0		

Table A.6: Ticks counts from repeated blanket drags on plot 2. The two consecutive zero drags before stopping are not reported. (F-female, M-male)

Plot 3		Larvae	Nymph	Adult F	Adult M
Number added		49	150	5	5
Date and time	Drag #	Number of ticks collected			
		Larvae	Nymph	Adult F	Adult M
13/8/08 pm	1	0	0	0	0
	2		4		
	3		1		
	4		0		
14/8/08 am	1	0	3	0	1
	2		4		
	3		3		
	4		2		
	5		3		
	6		2		
	7		2		
	8		2		
	9		3		1
	10	1	3		
	11		0		
	12		2		
14/8/08 pm	1	0	4	0	0
	2		2		
	3		0		
15/8/08 am	1	0	4	0	0
	2		6		
	3		2		
	4		1		
	5		1		
	6		0		
	7		1		
	8		4		

Table A.7: Ticks counts from repeated blanket drags on plot 3. The two consecutive zero drags before stopping are not reported. (F-female, M-male)

Plot 4		Larvae	Nymph	Adult F	Adult M
Number added		105	150	5	5
Date and time	Drag #	Number of ticks collected			
		Larvae	Nymph	Adult F	Adult M
13/8/08 pm	1	0	0	0	0
	2		5		
	3		2		
	4		0		
14/8/08 am	1	0	0	0	0
	2		2		
	3		1		
	4		3		
	5		2		
	6		2		
	7		2		
	8		0		
	9		2		1
	10		1		
	11		1		
14/8/08 pm	1	0	1	0	0
	2		0		
	3		0		
	4		1		
15/8/08 am	1	0	3	1	1
	2		3	1	
	3		3		
	4		2		

Table A.8: Ticks counts from repeated blanket drags on plot 4. The two consecutive zero drags before stopping are not reported. (F-female, M-male)

Appendix B

The models

The models cannot be solved analytically and numerical solutions are obtained from Mathematica. The Mathematica function '`NDSolve`' uses stepwise functions to find numerical solutions to differential equations. The '`NDSolve`' function varies the numerical methods depending on the stepsize, speed of method and if stiffness is detected to use the most efficient method for that system of equations.

Ch. 2: Non seasonal ingestion model

$$\begin{aligned}
\frac{dG_s}{dt} &= A + (a_g - s_g G)G - b_g G_s - \beta_1 T_i G_s - P\beta_3 T_i G_s \\
\frac{dG_i}{dt} &= P\beta_3 T_i G_s + \beta_1 T_i G_s - (\alpha + b_g + \gamma)G_i \\
\frac{dG_z}{dt} &= \gamma G_i - b_g G_z \\
\frac{dT_s}{dt} &= (a_t - s_t T)T(\beta_4 H + \beta_5 D) - b_t T_s - \beta_2 T_s G_i - \beta_3 T_s G - (\beta_4 H + \beta_5 D)T_s - \theta T_s T_i H \\
\frac{dT_i}{dt} &= \theta T_s T_i H + \beta_2 T_s G_i - \beta_3 T_i G - b_t T_i - (\beta_4 H + \beta_5 D)T_i
\end{aligned}$$

The ingestion model investigated in Section 2.3.1 assumes that there is no immigration ($A = 0$) and mountain hares are assumed absent ($H = 0$).

Ch. 3: Non seasonal acaricide treated sheep model

$$\begin{aligned}
\frac{dG_s}{dt} &= (a_g - s_g G)G - b_g G_s - \beta_1 T_i G_s - P\beta_3 T_i G_s \\
\frac{dG_i}{dt} &= P\beta_3 T_i G_s + \beta_1 T_i G_s - (\alpha + b_g + \gamma)G_i \\
\frac{dG_z}{dt} &= \gamma G_i - b_g G_z \\
\frac{dT_s}{dt} &= (a_t - s_t T)T(\beta_5 D + \beta_6(1-d)S) - b_t T_s - \beta_2 T_s G_i - \beta_3 T_s G - (\beta_5 D + \beta_6 S + d\beta_7 S)T_s \\
\frac{dT_i}{dt} &= \beta_2 T_s G_i - \beta_3 T_i G - b_t T_i - (\beta_5 D + \beta_6 S + d\beta_7 S)T_i
\end{aligned}$$

Ch. 4: Seasonal grouse behaviour models

t_n refers to the time in year n of the model simulation. Similarly $G(t)_n,\ T(t)_n$ refer to the grouse and tick densities at time t of year n. δt is a small increment in time.

The grouse pulse hatch model with ingestion

$$\left.\begin{aligned}\frac{dG_{cs}}{dt} &= -b_g G_{cs} - \beta_1 T_i G_{cs} - P\beta_3 T_i G_{cs}\\ \frac{dG_{ci}}{dt} &= P\beta_3 T_i G_{cs} + \beta_1 T_i G_{cs} - (\alpha + b_g + \gamma)G_{ci}\\ \frac{dG_{cz}}{dt} &= \gamma G_{ci} - b_g G_{cz}\end{aligned}\right\}\ 0 \le t_n \le 3,$$

$$G_{cs}(0)_n = (a_g - s_g G(52)_{n-1})G(52)_{n-1}, G_{ci}(0)_n = G_{cz}(0)_n = 0, G, T_j(0)_n = G, T_j(52)_{n-1},\ j = s, i, z$$

$$\left.\begin{aligned}\frac{dG_s}{dt} &= -\beta_1 T_i G_s - b_g G_s\\ \frac{dG_i}{dt} &= \beta_1 T_i G_s - (\alpha + b_g + \gamma)G_i\\ \frac{dG_z}{dt} &= \gamma G_i - b_g G_z\end{aligned}\right\}\ \begin{aligned}&0 \le t_n \le 52,\\ &G_j(3+\delta t) = G_{cj}(3) + G_j(3), j = s, i, z\end{aligned}$$

$$\left.\begin{aligned}\frac{dT_s}{dt} &= sc.(a_t - s_t T)T\beta_5 D - b_t T_s - \beta_2 T_s G_i - \beta_3 T_s G_c - \beta_5 D T_s\\ \frac{dT_i}{dt} &= \beta_2 T_s G_i - \beta_3 T_i G_c - b_t T_i - \beta_5 D T_i\end{aligned}\right\}\ 0 \le t_n \le 52$$

The grouse pulse hatch model with a Holling Form of ingestion

$$\left.\begin{aligned}\frac{dG_{cs}}{dt} &= -b_g G_{cs} - \beta_1 T_i G_{cs} - P\frac{T_i}{1+\frac{1}{a}T_s}G_{cs}\\ \frac{dG_{ci}}{dt} &= P\frac{T_i}{1+\frac{1}{a}T_s}G_{cs} + \beta_1 T_i G_{cs} - (\alpha + b_g + \gamma)G_{ci}\\ \frac{dG_{cz}}{dt} &= \gamma G_{ci} - b_g G_{cz}\end{aligned}\right\}\ 0 \le t_n \le 3,$$

$$G_{cs}(0)_n = (a_g - s_g G(52)_{n-1})G(52)_{n-1},\ G_{ci}(0)_n = G_{cz}(0)_n = 0,\ G, T_j(0)_n = G, T_j(52)_{n-1},\ j = s, i, z$$

$$\left.\begin{aligned}\frac{dG_s}{dt} &= -\beta_1 T_i G_s - b_g G_s\\ \frac{dG_i}{dt} &= \beta_1 T_i G_s - (\alpha + b_g + \gamma)G_i\\ \frac{dG_z}{dt} &= \gamma G_i - b_g G_z\end{aligned}\right\}\ \begin{aligned}&0 \le t_n \le 52,\\ &G_j(3+\delta t) = G_{cj}(3) + G_j(3), j = s, i, z\end{aligned}$$

$$\left.\begin{aligned}\frac{dT_s}{dt} &= sc.(a_t - s_t T)T\beta_5 D - b_t T_s - \beta_2 T_s G_i - \frac{T_s}{1+\frac{1}{a}T_s}G_c - \beta_5 D T_s\\ \frac{dT_i}{dt} &= \beta_2 T_s G_i - \frac{T_i}{1+\frac{1}{a}T_s}G_c - b_t T_i - \beta_5 D T_i\end{aligned}\right\}\ 0 \le t_n \le 52$$

The non seasonal model with Holling Form ingestion

$$
\begin{aligned}
\frac{dG_s}{dt} &= (a_g - s_g G)G - b_g G_s - \beta_1 T_i G_s - P\frac{T_i}{1+\frac{1}{a}T_s}G_s \\
\frac{dG_i}{dt} &= P\frac{T_i}{1+\frac{1}{a}T_s}G_s + \beta_1 T_i G_s - (\alpha + b_g + \gamma)G_i \\
\frac{dG_z}{dt} &= \gamma G_i - b_g G_z \\
\frac{dT_s}{dt} &= (a_t - s_t T)T\beta_5 D - b_t T_s - \beta_2 T_s G_i - \frac{T_s}{1+\frac{1}{a}T_s}G - \beta_5 D T_s \\
\frac{dT_i}{dt} &= \beta_2 T_s G_i - \frac{T_i}{1+\frac{1}{a}T_s}G - b_t T_i - \beta_5 D T_i
\end{aligned}
$$

The grouse pulse birth model without ingestion

$$
\left.
\begin{aligned}
\frac{dG_s}{dt} &= -\beta_1 T_i G_s - b_g G_s \\
\frac{dG_i}{dt} &= \beta_1 T_i G_s - (\alpha + b_g + \gamma)G_i \\
\frac{dG_z}{dt} &= \gamma G_i - b_g G_z \\
\frac{dT_s}{dt} &= sc.(a_t - s_t T)T\beta_5 D - b_t T_s - \beta_2 T_s G_i - \beta_5 D T_s \\
\frac{dT_i}{dt} &= \beta_2 T_s G_i - b_t T_i - \beta_5 D T_i
\end{aligned}
\right\}
\begin{aligned}
&0 \le t_n \le 52, \\
&G_s(0)_n = (a_g - s_g G(52)_{n-1})G(52)_{n-1}, \\
&G_j, T_k(0)_n = G_j, T_k(52)_{n-1}, \\
&j = i, z,\ k = s, i
\end{aligned}
$$

The grouse hatching period model

$$
\left.
\begin{aligned}
\frac{dG_s}{dt} &= (a_{gh} - s_{gh} G(0)_n)G(0)_n - b_g G_s - \beta_1 T_i G_s \\
\frac{dG_i}{dt} &= \beta_1 T_i G_s - (\alpha + b_g + \gamma)G_i \\
\frac{dG_z}{dt} &= \gamma G_i - b_g G_z \\
\frac{dT_s}{dt} &= (a_t - s_t T)T\beta_5 D - b_t T_s - \beta_2 T_s G_i - \beta_5 D T_s \\
\frac{dT_i}{dt} &= \beta_2 T_s G_i - b_t T_i - \beta_5 D T_i
\end{aligned}
\right\}
\begin{aligned}
&0 \le t_n \le 52, \\
&G, T_j(0)_n = G, T_j(52)_{n-1},\ j = s, i, z \\
&a_{gh}, s_{gh} = 0 \text{ for } \ w < t_n \le 52
\end{aligned}
$$

Ch. 5: Seasonal tick behaviour models

Seasonal tick activity model

$$
\left.
\begin{aligned}
\frac{dG_s}{dt} &= (a_g - s_g G)G - b_g G_s - \beta_{1t} T_i G_s \\
\frac{dG_i}{dt} &= \beta_{1t} T_i G_s - (\alpha + b_g + \gamma)G_i \\
\frac{dG_z}{dt} &= \gamma G_i - b_g G_z \\
\frac{dT_s}{dt} &= c(a_{tt} - s_{tt} T)T\beta_5 D - \beta_2 T_s G_i - \beta_5 D T_s - b_t T_s \\
\frac{dT_i}{dt} &= \beta_2 T_s G_i - \beta_5 D T_i - b_t T_i
\end{aligned}
\right\}
\begin{aligned}
&0 \le t_n \le 52, \\
&G, T_j(0)_n = G, T_j(52)_{n-1},\ j = s, i, z, \\
&\beta_i = 0 \text{ for } 32 < t_n \le 52
\end{aligned}
$$

Seasonal tick activity with grouse hatching period model

$$
\left.
\begin{aligned}
\frac{dG_{sp}}{dt} &= -b_g G_{sp} - \beta_{1t} T_{ip} G_{sp} \\
\frac{dG_{ip}}{dt} &= \beta_{1t} T_{ip} G_{sp} - (\alpha + b_g + \gamma) G_{ip} \\
\frac{dG_{zp}}{dt} &= \gamma G_{ip} - b_g G_{zp} \\
\frac{dT_{sp}}{dt} &= c(a_{tt} - s_{tt} T_p) T_p \beta_5 D - \beta_2 T_{sp} G_{ip} - \beta_5 D T_{sp} - b_t T_{sp} \\
\frac{dT_{ip}}{dt} &= \beta_2 T_{sp} G_{ip} - \beta_5 D T_{ip} - b_t T_{ip}
\end{aligned}
\right\}
\begin{aligned}
&0 \leq t_n \leq 6, \\
&G_{jp}(0)_n = G_{jw}(52)_{n-1}, j = s, i, z \\
&T_{jp}(0)_n = T_{jw}(52)_{n-1}, j = s, i,
\end{aligned}
$$

$$
\left.
\begin{aligned}
\frac{dG_{ss}}{dt} &= (a_g - s_g G_p(6) n) G_p(6)_n - b_g G_{ss} - \beta_{1t} T_{is} G_{ss} \\
\frac{dG_{is}}{dt} &= \beta_{1t} T_{is} G_{ss} - (\alpha + b_g + \gamma) G_{is} \\
\frac{dG_{zs}}{dt} &= \gamma G_{is} - b_g G_{zs} \\
\frac{dT_{ss}}{dt} &= c(a_{tt} - s_{tt} T) T \beta_5 D - \beta_2 T_{ss} G_{is} - \beta_5 D T_{ss} - b_t T_{ss} \\
\frac{dT_{is}}{dt} &= \beta_2 T_{ss} G_{is} - \beta_5 D T_{is} - b_t T_{is}
\end{aligned}
\right\}
\begin{aligned}
&6 < t \leq 10, \\
&G_{js}(6)_n = G_{jp}(6)_n, j = s, i, z, \\
&T_{js}(6)_n = T_{jp}(6)_n, j = s, i,
\end{aligned}
$$

$$
\left.
\begin{aligned}
\frac{dG_{sa}}{dt} &= -b_g G_{sa} - \beta_{1t} T_{ia} G_{sa} \\
\frac{dG_{ia}}{dt} &= \beta_{1t} T_{ia} G_{sa} - (\alpha + b_g + \gamma) G_{ia} \\
\frac{dG_{za}}{dt} &= \gamma G_{ia} - b_g G_{za} \\
\frac{dT_{sa}}{dt} &= c(a_{tt} - s_{tt} T_a) T_a \beta_5 D - \beta_2 T_{sa} G_{ia} - \beta_5 D T_{sa} - b_t T_{sa} \\
\frac{dT_{ia}}{dt} &= \beta_2 T_{sa} G_{ia} - \beta_5 D T_{ia} - b_t T_{ia}
\end{aligned}
\right\}
\begin{aligned}
&10 < t_n \leq 32, \\
&G_{ja}(10)_n = G_{js}(10)_n, j = s, i, z \\
&T_{ja}(10)_n = T_{js}(10)_n, j = s, i,
\end{aligned}
$$

$$
\left.
\begin{aligned}
\frac{dG_{sw}}{dt} &= -b_g G_{sw} \\
\frac{dG_{iw}}{dt} &= -(\alpha + b_g + \gamma) G_{iw} \\
\frac{dG_{zw}}{dt} &= \gamma G_{iw} - b_g G_{zw} \\
\frac{dT_{sw}}{dt} &= -b_t T_{sw} \\
\frac{dT_{iw}}{dt} &= -b_t T_{iw}
\end{aligned}
\right\}
\begin{aligned}
&32 < t_n \leq 52, \\
&G_{jw}(32)_n = G_{ja}(32)_n, j = s, i, z \\
&T_{jw}(32)_n = T_{ja}(32)_n, j = s, i,
\end{aligned}
$$

The additional subscripts denote the time of year for which the equations are relevant so that for example G_{jp} denotes a grouse with infection state j in the spring (p), s denotes summer, a autumn and w winter.

Ch. 6: Non seasonal acaricide treated deer model

$$
\begin{aligned}
\frac{dG_s}{dt} &= (a_g - s_g G)G - b_g G_s - \beta_1 T_i G_s \\
\frac{dG_i}{dt} &= \beta_1 T_i G_s - (\alpha + b_g + \gamma) G_i \\
\frac{dG_z}{dt} &= \gamma G_i - b_g G_z \\
\frac{dT_s}{dt} &= (a_t - s_t T)T((1-c)(1-d)\beta_5 D) - b_t T_s - \beta_2 T_s G_i - (1-c)(d\beta_4 + \beta_5) D T_s \\
\frac{dT_i}{dt} &= \beta_2 T_s G_i - b_t T_i - (1-c)(d\beta_4 + \beta_5) D T_i
\end{aligned}
$$

Ch.7: Acaricidal leg band model

$T_j(0)_n = T_j(52)_{n-1} \quad j = s, i,$

$G_{st}(0)_n = If[\frac{G(52)_{n-1}}{2} > h, h\frac{G_s(52)_{n-1}}{G(52)_{n-1}}, \frac{G_s(52)}{2}], \quad G_{zt}(0)_n = If[\frac{G(52)_{n-1}}{2} > h, h\frac{G_z(52)_{n-1}}{G(52)_{n-1}}, \frac{G_z(52)}{2}],$

$G_s(0)_n = G_s(52)_{n-1} + G_{st}(52)_{n-1} - G_{st}(0)_n, \quad G_z(0)_n = G_z(52)_{n-1} + G_{zt}(52)_{n-1} - G_{zt}(0)_n, \quad G_i(0) = 0$

$$
\begin{aligned}
\frac{dG_{st}}{dt} &= -b_g G_{st} \\
\frac{dG_{zt}}{dt} &= -b_g G_{zt} \\
\left.\begin{aligned}
\frac{dG_{sct}}{dt} &= 2h(a_g - s_g G(6)) - (1-d)\beta_1 T_i G_{sct} - b_g G_{sct} \\
\frac{dG_{ict}}{dt} &= (1-d)\beta_1 T_i G_{sct} - (\alpha + b_g + \gamma) G_{ict} \\
\frac{dG_{zct}}{dt} &= \gamma G_{ict} - b_g G_{zct}
\end{aligned}\right\} & \quad 6 \le t_n \le 10 + w, \\
\left.\begin{aligned}
\frac{dG_s}{dt} &= (G(6) - 2h)(a_g - s_g G(6)) - \beta_1 T_i G_s - b_g G_s \\
\frac{dG_i}{dt} &= \beta_1 T_i G_s - (\alpha + b_g + \gamma) G_i \\
\frac{dG_z}{dt} &= \gamma G_i - b_g G_z
\end{aligned}\right\} & \quad G_j(10 + w + \delta) = G_j(10 + w) + G_{jct}(10 + w), \\
& \quad j = s, i, z \\
\frac{dT_s}{dt} &= (a_t - s_t T)T\beta_5 D - \beta_2 T_s(G_i + (1-d)G_{ict}) - \beta_2 T_s(G_t + dG_{ct}) - T_s\beta_5 D - b_t T_s \\
\frac{dT_i}{dt} &= \beta_2 T_s(G_i + (1-d)G_{ict}) - \beta_2 T_i(G_t + dG_{ct}) - T_i\beta_5 D - b_t T_i
\end{aligned}
$$

$a_g = s_g = 0, \quad t_n < 6, t_n > 10, \qquad \beta_i = 0, \quad t_n > 32$

Appendix C

Model Analysis

C.1 Algebraic Analysis

The non seasonal models can be analysed algebraically to find the model equilibria and assess the stability of the model predictions at the given equilibria (Anderson and May, 1981). The methods are discussed in detail for the ingestion model and results are given for the acaricide treated sheep model and the acaricide treated deer model.

C.1.1 The ingestion model

The ingestion model can be written as

$$\begin{aligned}
\frac{dG_s}{dt} &= (a_g - s_g G)G - b_g G_s - \rho T_i G_s \\
\frac{dG_i}{dt} &= \rho T_i G_s - \Gamma G_i \\
\frac{dG_z}{dt} &= \gamma G_i - b_g G_z \\
\frac{dT_s}{dt} &= (a_t - s_t T)TX - \beta_2 T_s G_i - \beta_3 T_s G - Y T_s - \theta T_s T_i H \\
\frac{dT_i}{dt} &= \theta T_s T_i H + \beta_2 T_s G_i - \beta_3 T_i G - Y T_i
\end{aligned}$$

where $X = \beta_4 H + \beta_5 D,\ Y = \beta_4 H + \beta_5 D + b_t,\ \rho = \beta_1 + P\beta_3$ and $\Gamma = \alpha + b_g + \gamma$.

Equilibrium Analysis

Setting the system equations equal to zero and solving for G_j and T_j gives the equilibria of the system. The biologically relevant equilibria, written as $(G_s, G_i, G_z, T_s, T_i)$, are now given.

1. (0,0,0,0,0) The trivial equilibrium with no grouse, no ticks and no disease.

2. (K_g,0,0,0,0) Grouse present at their carrying capacity, K_g, with no ticks or disease, where

$$K_g = \frac{a_g - b_g}{s_g}$$

To be biologically relevant this must be positive. Hence, we require $a_g > b_g$.

3. (0,0,0,K_t,0) Ticks present at their carrying capacity, K_t, with no grouse or disease, where

$$K_t = \frac{a_t X - Y}{s_t X}$$

For this to be biologically relevant we require $a_t X > Y$.

4. (K_g,0,0,K_t^g,0) Both grouse and ticks present, with no disease. Grouse at their carrying capacity, K_g but the tick density is reduced to K_t^g with the presence of grouse and their consequent consumption of the ticks.

$$K_t^g = \frac{a_t X - (Y + \beta_3 K_g)}{s_t X}$$

For this to be biologically relevant we require both K_g and K_t^g to be positive. i.e. $a_g > b_g$ and $a_t X > Y + \beta_3 K_g$

5. $(0, 0, 0, T_s^+, T_i^+)$ No grouse are present, but ticks and disease are. The carrying capacity of the ticks K_t is unchanged by the presence of disease. The equilibrium densities of susceptible ticks, T_s^+, and infected ticks, T_i^+ are given by the following formulae.

$$T_s^+ = \frac{Y}{\theta H} \quad \text{and} \quad T_i^+ = K_t - \frac{Y}{\theta H}$$

T_s^+ is always positive and biologically relevant but for T_i^+ this only occurs when $K_t \theta H > Y$.

6. $(G_s^*, G_i^*, G_z^*, T_s^*, T_i^*)$ Grouse, ticks and disease are all present. This equilibrium is more complicated and is described by the following formulae:

$$\begin{aligned}
G_s^* &= \frac{b_g \Gamma}{s_g} \left(\frac{a_g (b_g \Gamma + (b_g + \gamma)\rho T_i^*) - b_g \Gamma (b_g + \rho T_i^*)}{(b_g \Gamma + (b_g + \gamma)\rho T_i^*)^2} \right) \\
G_i^* &= \frac{\rho T_i^* G_s^*}{\Gamma} \\
G_z^* &= \frac{\gamma}{b_g} G_i^* \\
G^* &= G_s^* + G_i^* + G_z^*
\end{aligned}$$

The total tick carrying capacity is given by

$$\begin{aligned}
K_t^{g*} &= \frac{a_t X - (Y + \beta_3 G^*)}{s_t X}, \\
T_s^* &= K_t^{g*} - T_i^*,
\end{aligned}$$

and T_i^* is given by $\qquad f(T_i^*) = A(T_i^*)^4 + B(T_i^*)^3 + C(T_i^*)^2 + DT_i^* + E$

where

$$
\begin{aligned}
A &= -\theta H s_t s_g^2 \rho^3 \sigma^3 X \\
B &= s_g s_t \rho^3 \sigma (s_g \sigma^2 Y + v\tau) X + \theta H s_g \rho^2 \sigma^2 (\rho\sigma\omega - s_g s_t b_g \Gamma X - \rho\tau\beta_3) \\
C &= s_g s_t b_g \Gamma \rho^2 X \left[3 s_g \sigma^2 Y + v(\tau + r_g \sigma)\right] - s_g b_g \rho^2 \sigma\tau\phi + b_g \rho^3 \tau^2 \beta_2 \beta_3 \\
&\quad - \theta H s_g b_g \rho\sigma\Gamma \left[3 s_g (\rho\sigma\omega + s_t b_g \Gamma X) + \rho\beta_3 (r_g \rho - 2\tau)\right] \\
D &= 3 s_g^2 s_t b_g \Gamma \rho^2 \sigma^2 XY + s_g s_t b_g^2 \Gamma r_g \rho v X + 2 b_g^2 r_g \Gamma \rho^2 \tau \beta_2 \beta_3 - s_g b_g^2 \Gamma \phi \rho (\tau + \sigma r_g) \\
&\quad - \theta H b_g^2 s_g \Gamma^2 \left[3 s_g \rho (b_g + \gamma)\omega + s_g s_t b_g \Gamma X + \rho\tau\beta_3 + 2 r_g \rho (b_g + \gamma)\beta_3\right] \\
E &= s_g^2 s_t b_g^3 \Gamma^3 XY + r_g^2 b_g^3 \Gamma^2 \rho \beta_2 \beta_3 - s_g r_g b_g^3 \Gamma^2 \phi + \theta H s_g b_g^3 \Gamma^3 (s_g \omega - r_g \beta_3)
\end{aligned}
$$

and

$r_g = a_g - b_g,\ \sigma = b_g + \gamma,\ \tau = r_g\sigma - \alpha b_g,\ \upsilon = \beta_2 b_g + \beta_3 \sigma,\ \omega = a_t X - Y,$ and $\phi = \beta_2 \rho\omega - s_t X \beta_3 \Gamma.$

Stability Analysis

To assess local stability the standard methods of analysis Anderson and May (1981) were followed. If the system is locally stable then a small perturbation from the equilibrium should return to the equilibrium point under certain conditions. The eigenvalues of the Jacobian matrix were evaluated for each of the equilibrium values. If the eigenvalues have negative real parts then the equilibrium is stable. The general form of the Jacobian is given by:

$$
\begin{pmatrix}
a_g - 2s_g G - b_g - \rho T_i & a_g - 2s_g G & a_g - 2s_g G & 0 & -\rho G_s \\
\rho T_i & -\Gamma & 0 & 0 & \rho G_s \\
0 & \gamma & -b_g & 0 & 0 \\
-\beta_3 T_s & -\beta_3 T_s - \beta_2 T_s & -\beta_3 T_s & a_t X - 2 s_t T X - \beta_3 G & a_t X - 2 s_t T X \\
 & & & -\beta_2 G_i - Y - \theta T_i H & -\theta T_s H \\
-\beta_3 T_i & -\beta_3 T_i + \beta_2 T_s & -\beta_3 T_i & \beta_2 G_i + \theta T_i H & \theta T_s H - Y - \beta_3 G
\end{pmatrix}
$$

1. The eigenvalues of the Jacobian at (0,0,0,0,0) are given by

$$
\begin{vmatrix}
a_g - b_g - \lambda & a_g & a_g & 0 & 0 \\
0 & -\Gamma - \lambda & 0 & 0 & 0 \\
0 & \gamma & -b_g - \lambda & 0 & 0 \\
0 & 0 & 0 & a_t X - Y - \lambda & a_t X \\
0 & 0 & 0 & 0 & -Y - \lambda
\end{vmatrix} = 0
$$

The eigenvalues are $a_g - b_g$, $-\Gamma$, $-b_g$, $a_t X - Y$ and $-Y$. For these to be negative and give a stable equilibrium $b_g > a_g$ and $Y > a_t X$. That is we require the death rates to exceed birth rates for both

the grouse and the ticks.

2. The eigenvalues of the Jacobian at (K_g,0,0,0,0) are given by

$$\begin{vmatrix} a_g - 2s_gK_g - b_g - \lambda & a_g - 2s_gK - g & a_g - 2s_gK_g & 0 & -\beta_1 K - g - P\beta_3 K_g \\ 0 & -\Gamma - \lambda & 0 & 0 & \beta_1 K_g + P\beta_3 K_g \\ 0 & \gamma & -b_g - \lambda & 0 & 0 \\ 0 & 0 & 0 & a_t X - Y & a_t X \\ & & & -\beta_3 K_g - \lambda & \\ 0 & 0 & 0 & 0 & -\beta_3 K_g - Y - \lambda \end{vmatrix} = 0$$

The eigenvalues are $a_g - 2s_gK_g - b_g$, $-\Gamma$, $-b_g$, $a_tX - Y - \beta_3K_g$ and $-\beta_3K_g - Y$. These give a stable equilibrium when $a_g > b_g$ (as $s_g = \frac{a_g - b_g}{K_g}$) and $Y + \beta_3K_g > a_tX$. That is the birth rate exceeds the death rate for grouse, but the birth rate is lower than the death rate for ticks.

3. The eigenvalues of the Jacobian at (0,0,0,K_t,0) are given by

$$\begin{vmatrix} a_g - b_g & a_g & a_g & 0 & 0 \\ 0 & -\Gamma - \lambda & 0 & 0 & 0 \\ 0 & \gamma & -b_g - \lambda & 0 & 0 \\ -\beta_3 K_t & -\beta_3 K_t - \beta_2 K_t & -\beta_3 K_t & a_t X - 2s_t K_t X & a_t X - 2s_t K_t X \\ & & & -Y - \lambda & -\theta K_t H \\ 0 & \beta_2 K_t & 0 & 0 & \theta K_t H \\ & & & & -Y - \lambda \end{vmatrix} = 0$$

The eigenvalues are $-\Gamma$, $a_g - b_g$, $-b_g$, $a_tX - 2s_tK_tX - Y \equiv -s_tK_tX$ and $\theta K_tH - Y$. To give stability $b_g > a_g$, $K_t > 0$ and $Y > \theta K_tH$. In other words for grouse the death rate needs to exceed the birth rate, for ticks the death rate needs to be lower than the birth rate and ticks need to die at a rate quicker than they can spread the disease through non-viraemic transmission.

4. The eigenvalues of the Jacobian at (K_g,0,0,K_t^g,0) are given by

$$\begin{vmatrix} a_g - 2s_gK_g - b_g - \lambda & a_g - 2s_gK_g & a_g - 2s_gK_g & 0 & -\rho K_g \\ 0 & -\Gamma - \lambda & 0 & 0 & \rho K_g \\ 0 & \gamma & -b_g - \lambda & 0 & 0 \\ -\beta_3 K_t^g & -\beta_3 K_t^g - \beta_2 K_t & -\beta_3 K_t^g & a_t X - 2s_t K_t^g X & a_t X - \theta K_t^g H \\ & & & -\beta_3 K_g - Y - \lambda & -2s_t K_t^g X \\ 0 & \beta_2 K_t^g & 0 & 0 & \theta K_t^g H - \beta_3 K_g \\ & & & & -Y - \lambda \end{vmatrix} = 0$$

Three eigenvalues are $a_g - 2s_gK_g - b_g$, $-b_g$ and $a_tX - 2s_tK_t^gX - \beta_3K_g - Y$. For these to be negative we require $b_g < a_g$ and $Y + \beta_3K_g < a_tX$. That is we require the death rates to be lower than the birth rates for both the grouse and the ticks.

The remaining eigenvalues are given by the quadratic,

$$\lambda^2 + (\Gamma - \theta K_t^g H + \beta_3 K_g + Y)\lambda - \beta_2 \rho K_g K_t^g - \Gamma(\theta K_t^g H - \beta_3 K_g - Y) = 0$$

The Routh Hurwitz conditions state that a quadratic of the form $x^2 + Ax + B = 0$ has negative real parts iff $A, B > 0$. So for this equilibrium to be stable we require:

$$\Gamma - \theta K_t^g H + \beta_3 K_g + Y > 0 \quad \text{and} \quad -\beta_2 \rho K_g K_t^g - \Gamma(\theta K_t^g H - \beta_3 K_g - Y) > 0.$$

The first of these rearranges to give $\Gamma + \beta_3 K_g + Y > \theta K_t^g H$. Which requires grouse and ticks to die (or recover) quicker than the disease can be passed on through non-viraemic transmission.

The second of these is equivalent to $\Gamma(\beta_3 K_g + Y) > \beta_2 \rho K_g K_t^g + \Gamma \theta K_t^g H$. Which is precisely the condition required to ensure $R_{0,virus} < 1$.

5. The eigenvalues of the Jacobian at $(0, 0, 0, T_s^+, T_i^+)$ are given by

$$\begin{vmatrix} a_g - b_g - \rho T_i^+ - \lambda & a_g & a_g & 0 & 0 \\ \rho T_i^+ & -\Gamma - \lambda & 0 & 0 & 0 \\ 0 & \gamma & -b_g - \lambda & 0 & 0 \\ -\beta_3 T_s^+ & -\beta_3 T_s^+ - \beta_2 T_s^+ & -\beta_3 T_s^+ & a_t X - 2 s_t T^+ X & a_t X - \theta T_s^+ H \\ & & & -Y - \theta T_i^+ H - \lambda & -2 s_t T^+ X \\ -\beta_3 T_i^+ & -\beta_3 T_i^+ + \beta_2 T_s^+ & -\beta_3 T_i^+ & \theta T_i^+ H & \theta T_s^+ H - Y - \lambda \end{vmatrix} = 0$$

For block matrices

$$\det \begin{pmatrix} A & 0 \\ C & D \end{pmatrix} = \det A \det D.$$

Hence the above determinate can be considered in two separate parts. Firstly

$$\begin{vmatrix} a_g - b_g - \rho T_i^+ - \lambda & a_g & a_g \\ \rho T_i^+ & -\Gamma - \lambda & 0 \\ 0 & \gamma & -b_g - \lambda \end{vmatrix} = 0$$

This has eigenvalues given by the cubic,

$$\lambda^3 - (a_g - b_g - \rho T_i^* - (\Gamma + b_g))\lambda^2 - [(a_g - b_g - \rho T_i^*)(\Gamma + b_g) - \Gamma b_g + a_g \rho T_i^*]\lambda$$

$$-b_g \Gamma(a_g - b_g - \rho T_i^*) - a_g(b_g + \gamma)\rho T_i^* = 0.$$

The Routh Hurwitz conditions state a cubic of the form $x^3 + Ax^2 + Bx + C = 0$ has negative real parts iff $A, B, C > 0$ and $AB > C$.

All the coefficients are positive when $a_g < b_g$, that is when grouse die quicker than they are born. (This is equivalent to $r_g < 0$, where $r_g = a_g - b_g$ is the intrinsic growth rate of the grouse population.)

It can also be shown that the condition $AB > C$ holds when $r_g < 0$.

Secondly,

$$\begin{vmatrix} a_tX - 2s_tT^+X - Y - \theta T_i^+H - \lambda & a_tX - \theta T_s^+H - 2s_tT^+X \\ \theta T_i^+H & \theta T_s^+H - Y - \lambda \end{vmatrix} = 0.$$

This has solutions $-Y - \theta H(T_i^+ - T_s^+)$ and $-Y + (a_t - 2s_t)X$.

Substituting for K_t and T_i^+ shows that for these solutions to be negative we require $Y < \theta HK_t$ and $Y < a_tX$. These are precisely the requirements that the disease can spread quicker than the ticks die and that the ticks die quicker than they are born.

6. The eigenvalues of the Jacobian at $(G_s^*, G_i^*, G_z^*, T_s^*, T_i^*)$ are given by

$$\begin{vmatrix} a_g - 2s_gG^* - b_g - \rho T_i^* - \lambda & a_g - 2s_gG^* & a_g - 2s_gG^* & 0 & -\beta_1 G_s^* - P\beta_3 G_s^* \\ \rho T_i^* & -\Gamma - \lambda & 0 & 0 & \rho G_s^* \\ 0 & \gamma & -b_g - \lambda & 0 & 0 \\ -\beta_3 T_s^* & -\beta_3 T_s^* - \beta_2 T_s^* & -\beta_3 T_s^* & a_tX - 2s_tT^*X - \beta_3 G^* & a_tX - \theta T_s^* H \\ & & & -\beta_2 G_i^* - Y - \theta T_i^* H - \lambda & -2s_tT^*X \\ -\beta_3 T_i^* & -\beta_3 T_i^* + \beta_2 T_s^* & -\beta_3 T_i^* & \beta_2 G_i^* + \theta T_i^* H & \theta T_s^* H - \beta_3 G^* \\ & & & & -Y - \lambda \end{vmatrix} = 0$$

The algebraic analysis is very complicated at this stage. It is therefore assumed that this equilibrium is stable (or bounded by stable limit cycles) when the others are not. This is supported by numerical simulations.

C.1.2 The acaricide treated sheep model

The acaricide treated sheep model is a special case of the general ingestion model (Ch. 2) with $\theta = 0$ and can be written as

$$\begin{aligned} \frac{dG_s}{dt} &= (a_g - s_gG)G - b_gG_s - \rho T_iG_s \\ \frac{dG_i}{dt} &= \rho T_iG_s - \Gamma G_i \\ \frac{dG_z}{dt} &= \gamma G_i - b_gG_z \\ \frac{dT_s}{dt} &= (a_t - s_tT)TX - \beta_2T_sG_i - \beta_3T_sG - YT_s \\ \frac{dT_i}{dt} &= \beta_2T_sG_i - \beta_3T_iG - YT_i \end{aligned}$$

where $X = \beta_5D + (1-d)\beta_6S$, $Y = \beta_5D + \beta_6S + d\beta_7S + b_t$, $\rho = \beta_1 + P\beta_3$ and $\Gamma = \alpha + b_g + \gamma$.

Equilibrium Analysis

There are now five possible biologically relevant equilibria within this system: the trivial case where nothing exists, grouse only, tick only, grouse and tick only and finally all present, i.e. grouse, tick and

disease all coexisting. The equilibria are described below and written in the form $(G_s, G_i, G_z, T_s, T_i)$.

1. $(0,0,0,0,0)$ No grouse, ticks or disease present.

2. $(K_g,0,0,0,0)$ Grouse are present at their carrying capacity, K_g, given by $K_g = \frac{a_g - b_g}{s_g}$. For this to be biologically relevant K_g needs to be positive, so we require $a_g > b_g$.

3. $(0,0,0,K_t,0)$ Ticks are present at their carrying capacity, K_t, given by $K_t = \frac{a_t X - Y}{s_t X}$. For this to be biologically relevant K_t needs to be positive, so we require $a_t X > Y$.

4. $(K_g,0,0,K_t^g,0)$ Grouse are present at their carrying capacity, K_g as above, but the tick carrying capacity is lower as a result of their consumption by the grouse, and is now given by, $K_t^g = \frac{a_t X - (Y + \beta_3 K_g)}{s_t X}$. For this to biologically relevant we need both K_g and K_t^g to be positive, that is, $a_g > b_g$ and $a_t X > Y + \beta_3 K_g$.

5. $(G_s^*, G_i^*, G_z^*, T_s^*, T_i^*)$ The grouse and ticks coexist with the disease, but at disease induced reduced densities. The grouse population now consists of susceptibles, G_s^*, infecteds, G_i^*, and immune, G_z^*, and the ticks of susceptibles, T_i^*, and infecteds, T_s^*. These densities are given by the following formulae.

$$
\begin{aligned}
G_s^* &= \frac{b_g \Gamma}{s_g}\left(\frac{a_g(b_g\Gamma + (b_g+\gamma)\rho T_i^*) - b_g\Gamma(b_g + \rho T_i^*)}{(b_g\Gamma + (b_g+\gamma)\rho T_i^*)^2}\right)\\
G_i^* &= \frac{\rho T_i^* G_s^*}{\Gamma}\\
G_z^* &= \frac{\gamma}{b_g} G_i^*\\
G^* &= G_s^* + G_i^* + G_z^*
\end{aligned}
$$

The total tick carrying capacity is given by

$$
\begin{aligned}
K_t^{g*} &= \frac{a_t X - (Y + \beta_3 G^*)}{s_t X},\\
T_s^* &= K_t^{g*} - T_i^*,
\end{aligned}
$$

and T_i^* is given by $\quad f(T_i^*) = A(T_i^*)^3 + B(T_i^*)^2 + CT_i^* + D,$

with
$$
\begin{aligned}
A &= s_t s_g^2 XY\sigma^3\rho^3 + s_t s_g X\rho^3\sigma\upsilon\tau\\
B &= \beta_2\beta_3\rho^3 b_g\tau^2 + 3s_t s_g^2 XY b_g\Gamma\rho^2\sigma^2 + s_g s_t\rho^2 X b_g\Gamma\upsilon\tau - s_g\rho\sigma(b_g\phi\tau - s_t X\rho r_g b_g\Gamma\upsilon)\\
C &= 2\beta_2\beta_3\rho^2 b_g^2 r_g\Gamma\tau - s_g b_g^2\Gamma(\phi\tau - s_t X\rho r_g\Gamma\upsilon) - s_g r_g b_g^2\Gamma\rho\sigma\phi + 3s_t s_g^2 XY b_g\Gamma\rho^2\sigma^2\\
D &= \beta_2\beta_3\rho b_g^3 r_g^2\Gamma^2 + s_t s_g^2 XY b_g^3\Gamma^3 - s_g r_g b_g^3\Gamma^2\phi
\end{aligned}
$$

and $r_g = a_g - b_g,\ \sigma = b_g + \gamma, \tau = r_g\sigma - \alpha b_g,\ \upsilon = \beta_2 b_g + \beta_3\sigma,\ \phi = \beta_2\rho(a_t X - Y) - s_t X\beta_3\Gamma.$

Stability Analysis

The general form of the Jacobian is given by:

$$\begin{pmatrix} a_g - 2s_gG - b_g - \rho T_i & a_g - 2s_gG & a_g - 2s_gG & 0 & -\rho G_s \\ \rho T_i & -\Gamma & 0 & 0 & \rho G_s \\ 0 & \gamma & -b_g & 0 & 0 \\ -\beta_3 T_s & -\beta_3 T_s - \beta_2 T_s & -\beta_3 T_s & (a_t - 2s_tT)X - \beta_2 G_i - \beta_3 G - Y & (a_t - 2s_tT)X \\ -\beta_3 T_i & -\beta_3 T_i + \beta_2 T_s & -\beta_3 T_i & \beta_2 G_i & -Y - \beta_3 G \end{pmatrix}$$

1. The trivial equilibrium has eigenvalues $-\Gamma, a_g - b_g, -b_g, -Y, a_tX - Y$. All model parameters are positive and consequently many of the eigenvalues will be negative. However, we also require, $a_g < b_g$ and $a_tX < Y + \beta_3 K_g$. In this case we require the grouse and ticks to both die quicker than they can reproduce.

2. Grouse only equilibrium has eigenvalues $-\Gamma, -b_g, a_g - b_g - 2s_gK_g, -Y - \beta_3 K_g, a_tX - Y$. In order for these to be negative we require $a_g > b_g$ and $a_tX < Y$. In other words the grouse need to reproduce quicker than they die and the ticks need to die quicker than they reproduce.

3. Tick only equilibrium has eigenvalues $-\Gamma, a_g - b_g, -b_g, -Y, (a_t - 2s_tK_t)X - Y$. Hence the conditions for stability are $a_g < b_g$ and $a_tX > Y$. The two conditions are now the opposite of the above as we wish the tick to persist and the grouse to die out.

4. Grouse and tick only, no disease equilibrium has eigenvalues $-b_g, a_g - b_g - 2s_gK_g, (a_t - 2s_tK_t^g)X - Y - \beta_3 K_g$ and the solutions to $\lambda^2 - \lambda(-Y - \beta_3 K_g) - \Gamma(-Y - \beta_3 K_g) - \beta_2 \rho K_g K_t^g = 0$. For these to be negative we require, $a_g > b_g$ and $a_tX > Y + \beta_3 K_g$. Also, $\beta_3 K_g + Y + \Gamma > 0$ and $R_{0,virus} < 1$ where $R_{0,virus} = \frac{\Gamma(Y + \beta_3 K_g)}{\beta_2 \rho K_g K_t^g}$. For this equilibrium to be stable we require both the grouse and tick to survive and so reproduce quicker than they die. The third condition requiring the sum of the death rates to be positive will always be true given that the death rates themselves must be positive. We also require the reproductive value of the virus ($R_{0,virus}$) to be less than one to prevent the disease from persisting.

5. Grouse, tick and disease all coexisting equilibrium has the Jacobian

$$\begin{pmatrix} a_g - 2s_gG - b_g - \rho T_i & a_g - 2s_gG & a_g - 2s_gG & 0 & -\rho G_s \\ \rho T_i & -\Gamma & 0 & 0 & \rho G_s \\ 0 & \gamma & -b_g & 0 & 0 \\ -\beta_3 T_s & -\beta_3 T_s - \beta_2 T_s & -\beta_3 T_s & (a_t - 2s_tT)X - Y - \beta_2 G_i - \beta_3 G & (a_t - 2s_tT)X \\ -\beta_3 T_i & -\beta_3 T_i + \beta_2 T_s & -\beta_3 T_i & \beta_2 G_i & -Y - \beta_3 G \end{pmatrix}$$

Due to the complexity of the algebra it is assumed that this equilibrium is stable (or replaced by stable limit cycles) when the others are not. Numerical simulations back up this assumption.

C.1.3 The acaricide treated deer model

The deer model can be written as

$$\begin{aligned}
\frac{dG_s}{dt} &= (a_g - s_g G)G - b_g G_s - \beta_1 T_i G_s \\
\frac{dG_i}{dt} &= \beta_1 T_i G_s - \Gamma G_i \\
\frac{dG_z}{dt} &= \gamma G_i - b_g G_z \\
\frac{dT_s}{dt} &= (a_t - s_t T)TX - \beta_2 T_s G_i - YT_s \\
\frac{dT_i}{dt} &= \beta_2 T_s G_i - YT_i
\end{aligned}$$

where $X = (1-d)\beta_5 D,\ Y = d\beta_4 D + \beta_5 D + b_t$ and $\Gamma = \alpha + b_g + \gamma$.

Equilibrium Analysis

There are five biologically relevant equilibria: the trivial equilibrium, grouse alone, ticks alone, grouse and ticks without disease and grouse and ticks with disease. The equilibria are described below and written in the form $(G_s, G_i, G_z, T_s, T_i)$.

1. $(0,0,0,0,0)$ No grouse, ticks or disease present.

2. $(K_g,0,0,0,0)$ Grouse are present at their carrying capacity, K_g, given by $K_g = \frac{a_g - b_g}{s_g}$. For this to be biologically relevant K_g needs to be positive, hence we require $a_g > b_g$.

3. $(0,0,0,K_t,0)$ Ticks are present at their carrying capacity, K_t, given by $K_t = \frac{a_t X - Y}{s_t X}$. In order for K_t to be positive we require $a_t X > Y$.

4. $(K_g,0,0,K_t,0)$ Grouse and ticks are both present at their carrying capacity, K_g and K_t as given above. Both need to be positive.

5. $(G_s^*, G_i^*, G_z^*, T_s^*, T_i^*)$ Grouse are present at densities reduced by the disease and are susceptible G_s^*, infected G_i^* or immune G_z^* and the tick population is comprised of susceptible T_s^* and infected ticks T_i^*. Densities are given by the following equations;

$$\begin{aligned}
G_s^* &= \frac{b_g\Gamma(b_g\Gamma(a_g - b_g) + \beta_1(a_g(b_g+\gamma) - b_g\Gamma)T_i^*)}{s_g(b_g\Gamma + \beta_1(b_g+\gamma)T_i^*)^2} \\
G_i^* &= \frac{\beta_1 T_i^*}{\beta_2 T_s^* + \Gamma} G_s^* \\
G_z^* &= \frac{\gamma}{b_g} G_s^* \\
T_s^* &= K_t - T_i^*
\end{aligned}$$

K_t is as before, but T_i^* is given by $\quad f(T_i^*) = A(T_i^*)^3 + B(T_i^*)^2 + CT_i^* + D.$

$$
\begin{aligned}
\text{with} \quad A &= s_g s_t \beta_1^3 \sigma (s_g \sigma^2 Y + b_g \beta_2 \tau) X \\
B &= s_g s_t b_g \Gamma \beta_1^2 X \left[3 s_g \sigma^2 Y + \beta_2 b_g (\tau + r_g \sigma) \right] - s_g b_g \beta_1^3 \beta_2 \sigma \omega \tau \\
C &= 3 s_g^2 s_t b_g \Gamma \beta_1^2 \sigma^2 XY + s_g s_t b_g^3 \Gamma r_g \beta_1 \beta_2 X - s_g b_g^2 \Gamma \beta_1^2 \beta_2 \omega (\tau + \sigma r_g) \\
D &= s_g^2 s_t b_g^3 \Gamma^3 XY - s_g r_g b_g^3 \Gamma^2 \beta_1 \beta_2 \omega
\end{aligned}
$$

and $r_g = a_g - b_g,\ \sigma = b_g + \gamma,\ \tau = r_g \sigma - \alpha b_g$ and $\omega = a_t X - Y$.

Stability Analysis

The general form of the Jacobian is given by:

$$
\begin{pmatrix}
a_g - 2s_g G - b_g - \beta_1 T_i & a_g - 2s_g G & a_g - 2s_g G & 0 & -\beta_1 G_s \\
\beta_1 T_i & -\Gamma & 0 & 0 & \beta_1 G_s \\
0 & \gamma & -b_g & 0 & 0 \\
0 & -\beta_2 T_s & 0 & a_t X - 2 s_t T X - \beta_2 G_i - Y & a_t X - 2 s_t T X \\
0 & \beta_2 T_s & 0 & \beta_2 G_i & -Y
\end{pmatrix}
$$

1. The eigenvalues of the Jacobian at (0,0,0,0,0) are $a_g - b_g$, $-\Gamma$, $-b_g$, $a_t X - Y$ and $-Y$. For these to be negative and give a stable equilibrium $b_g > a_g$ and $Y > a_t X$. That is we require the death rates to exceed birth rates for both the grouse and the ticks.

2. The eigenvalues of the Jacobian at (K_g,0,0,0,0) are $a_g - 2 s_g K_g - b_g$, $-\Gamma$, $-b_g$, $a_t X - Y$ and $-Y$. These are negative when $a_g > b_g$ and $Y > a_t X$. That is the birth rate exceeds the death rate for grouse, but the birth rate is lower than the death rate for ticks.

3. The eigenvalues of the Jacobian at (0,0,0,K_t,0) are $-\Gamma$, $a_g - b_g$, $-b_g$, $a_t X - 2 s_t K_t X - Y$ and $-Y$. For stability we require $b_g > a_g$ and $K_t > 0$. In other words, the grouse death rate needs to exceed the birth rate and the tick death rate needs to be lower than the birth rate.

4. The eigenvalues of the Jacobian at (K_g,0,0,K_t,0) are $a_g - 2 s_g K_g - b_g$, $-b_g$, $a_t X - 2 s_t K_t X - Y$ and $\Gamma Y > \beta_2 \beta_1 K_g K_t$. For these to be negative we require $b_g < a_g$, $Y < a_t X$ and $R_{0,virus} < 1$. That is we require the death rates to be lower than the birth rates for both the grouse and the ticks and the reproductive rate of the virus to be below unity.

5. The eigenvalues of the Jacobian at $(G_s^*, G_i^*, G_z^*, T_s^*, T_i^*)$ are given by

$$
\begin{vmatrix}
a_g - 2 s_g G^* - b_g - \beta_1 T_i^* - \lambda & a_g - 2 s_g G^* & a_g - 2 s_g G^* & 0 & -\beta_1 G_s^* \\
\beta_1 T_i^* & -\Gamma - \lambda & 0 & 0 & \beta_1 G_s^* \\
0 & \gamma & -b_g - \lambda & 0 & 0 \\
0 & -\beta_2 T_s^* & 0 & a_t X - 2 s_t T^* X - \beta_2 G_i^* - Y - \lambda & a_t X - 2 s_t T^* X \\
0 & \beta_2 T_s^* & 0 & \beta_2 G_i^* & -Y - \lambda
\end{vmatrix} = 0
$$

Due to the complex algebra for this solution it is assumed that this equilibrium is stable (or replaced by stable limit cycles) when the others are not. This is supported by numerical simulations.

C.2 Sensitivity analysis

The sensitivity of each model to parameter choices can be assessed using the methods of Watts et al. (2009).

1. **The correlation between model parameters and model predictions** The first method considers the correlation between a given parameter and the model predictions. The model is run 1000 times with each parameter value taken at random from a range of ±1% of the estimated value. The parameters are grouped as grouse demography, tick demography and virus transmission parameters. The individual parameter (and first order interactions within groups) are then correlated against the model predictions of grouse density and virus prevalence in the grouse population. Significant (at the 5% level) correlations from this analysis are given in Table C.1. The numbers in bold represent those parameters that explain more than 20% of the variation in the model output. From this analysis we can determine the parameters which the model is sensitive to. This method also determines the sensitivity of the model to interactions between parameters that may be important but neglected if only individual parameters are considered.

2. **The magnitude of individual parameter effects** The second approach determines the magnitude of the effect of individual parameters on model outputs by varying each by ±10% and recording the percentage change of the model outputs. For each parameter the percentage change is given in Table C.2 where it is greater than 10%. Using this analysis we can determine whether a small change made to an individual parameter has a disproportionate effect on model predictions. If we can identify the parameters that have a disproportionate effect on model predictions we can aim to improve estimates from new data where possible or if this is not possible we can identify the weaknesses in the model and explain the likely consequences of alternative estimates.

C.2.1 The ingestion model

The ingestion model developed in Chapter 2 did not include mountain hares and non viraemic transmission and consequently the following analysis considers the ingestion model with deer as the only adult tick host.

Parameter choice effects on predicted grouse density

Not surprisingly the grouse demography parameters show the highest levels of correlation with the predicted density of the grouse population. The parameter that shows the largest correlation with model output is b_g the natural death rate of grouse. Increasing the death rate of grouse has a negative effect on the predicted grouse density for all deer densities tested, but it is most apparent for 4 deer and 10 deer per km^2. When the deer density is low (4 per km^2) there is no virus and so it makes sense that the greatest influence on the size of the grouse population comes from the natural death rate. When the deer density is high (10 deer per km^2) the grouse population is much reduced by the virus and an increase in the natural death rate will have a large effect on the grouse population. However, at intermediate

deer densities (6.5 per km^2) the effect of changing the natural death rate may be lower because the virus has a larger relative effect at this point. The model predictions for grouse density show a weaker but still statistically significant positive correlation to a_g, the natural birth rate of grouse. The correlation is strongest when the deer density is low as there is no disease.

When the deer density is such that the tick population can survive the model predictions show significant but relatively small correlation with the parameters governing tick demography. When a_t the tick birth rate is increased the predicted grouse density falls and when b_t the tick death rate is increased the predicted grouse density rises. The density dependence constraint on the tick population s_t shows a weak positive correlation with the grouse population prediction with the high deer density. These correlations are all intuitive as any increase in the tick population will increase the opportunity for virus transmission and hence reduce the grouse population.

The parameters governing the viral dynamics only show weak negative correlation and predominantly for the high deer density when the virus is most prevalent. Interestingly though there appears to be no correlation with β_2 the rate at which ticks are infected by grouse and the model predictions of grouse density. This could be due to the high numbers of infected ticks compared to grouse. There are so many infected ticks already that a slight increase or decrease in infected tick numbers will make little difference to the number of grouse that become infected (and consequently die). It is also interesting to note that β_5 the tick biting rate on deer appears to have a very small positive correlation with the predicted grouse density when the deer density is low. This does not make any biological sense. The apparent correlation is very small and although significant at the 5% level I would suggest it is a statistical quirk and not a true reflection of the system. (Similarly, the very small weak negative correlation of the model to s_t at low deer density.)

The only interactions that show any significant correlation to the model predictions of the grouse density are those that involve significant individual parameters. In the main the effect of the interactions is smaller than that of the individual parameter, although the interactions of the other grouse demography parameters with the natural death rate of grouse do show higher correlation when there are 6.5 deer per km^2. This may be because 6.5 deer per km^2 is on the cusp of there being no virus with grouse at carrying capacity and the virus reducing the grouse population significantly.

The interactions of p, the proportion of infected ticks ingested that cause infection in grouse, and the other virus parameters also show higher levels of correlation than just p alone when there are 10 deer per km^2.

Although the grouse demography parameters showed some strong correlation with the model out puts for predicted grouse density they do not have a disproportionate effect on the model predictions when altered individually. The tick demography parameters do however have a disproportionate effect on the grouse density predictions. When the parameters are altered in such a way as to decrease the tick population (i.e. a_t- and b_t+) the predicted grouse population shows an increase of around 30%. (Table C.2) If the parameters are altered to increase the tick population (i.e. a_t+ and b_t-) the grouse population shows a decrease of around 20%. This disproportionate effect can be explained by the sheer magnitude

of the tick population, a small proportional change will lead to a large actual change in density. Similarly β_5 the rate adult ticks bite deer and reproduce shows a disproportionate effect on the grouse predictions.

Of the virus dynamic parameters only β_2 showed no correlation with the grouse density predictions but here shows a slightly disproportionate response to a 10% decrease. Decreasing the number of ticks infected per grouse will lead to fewer ticks and hence fewer grouse being infected, leading to a slightly higher grouse population.

Parameter choice effects on grouse virus prevalence

Although some correlation was found between many model parameters and the model predictions of the grouse population there is very little correlation shown between model parameters and the predictions of virus prevalence in grouse. The natural death rate of grouse, b_g, seems to have the largest influence on virus prevalence. With only small effects apparent for a_t, b_t and p when there are 6.5 deer per km^2 and a_g when there are 10 deer per km^2. These effects may again be due to deer density being on the brink of when the virus is able to persist so small changes have a big effect. The influence of a_g the natural grouse birth rate on virus prevalence for 10 deer per km^2 may be due to the small grouse density at equilibrium. If the grouse density is increased then there is the potential for the virus to transmit to a greater number of grouse which may infect a greater number of ticks and thus increase the proportion of grouse infected. There is an apparent but very small effect of α and β_3 on the predicted virus prevalence when there are 10 deer per km^2. This may be due to the low grouse density at high deer densities but may also be a result of the particular random choices of the model parameters in these simulations.

The interactions of parameters only show correlation when one of the individual parameters has and all correlations are lower, suggesting that the interactions are not significant to the virus prevalence in grouse.

Only the grouse demography parameters have a disproportionate effect on the model predictions of the infection prevalence. Decreasing the grouse birth rate a_g decreases grouse density and so the opportunity for virus transmission from grouse to ticks is reduced and the lower level of infection in ticks will lead to a smaller proportion of grouse being infected. The opposite argument can be made for increasing the grouse density. A similar argument can be made for increasing or decreasing the natural grouse death rate b_g. Decreasing α the disease induced death rate causes a slightly disproportional increase in the virus prevalence in grouse. This can be explained by the same reasoning as above, a larger grouse population will have a larger proportion infected as the virus has more opportunity to transmit from grouse to ticks and then back again.

C.2.2 The acaricide treated sheep model

Correlation Effects In general the grouse dynamic parameters (birth and death rates) and corresponding interactions show high correlation with the predicted grouse density only for low deer density (Table C.3). This is the same for both models and can be explained by the lack of ticks at this deer density. At low deer densities the tick population cannot be maintained at a high enough density to

allow LIV to persist so the disease dynamics are not important. As a consequence the grouse population dynamics are governed by the natural birth and death rates.

The grouse dynamics parameters show high correlation with the virus prevalence for high deer densities. At high deer densities the tick population is large enough to allow disease persistence and the grouse population is regulated by the disease. If the natural death rate is increased this will reduce the grouse population already at a low density, which reduces the proportion of infected grouse.

The tick dynamic parameters do not show a high correlation with any of the model outputs.

Of the viral dynamics β_3, the rate at which grouse ingest ticks, has the highest correlation both with the grouse density and virus prevalence at higher deer densities. At the intermediate deer density the ingestion of ticks shows a positive effect on the grouse population. This seems counterintuitive as ingestion is another route of infection but here the tick population is small enough for the grouse to be able to consume a sufficient quantity that the overall effect is to reduce the tick population and thus the virus prevalence. However, at higher deer densities the tick population is large and the ingestion of ticks has a limited effect on the tick population and the overall effect on the grouse population is negative; now ingestion is essentially just another route of infection.

β_7, the rate at which immature ticks attach to sheep, also shows a positive correlation with the grouse population for higher deer densities. At low deer densities the tick population is already small. At higher deer densities the increased attachment, and subsequent death, of ticks that attach to sheep would be expected to have a positive effect on the grouse population as the tick population is reduced and with it the virus prevalence.

Individual Parameters The parameters that have the largest disproportionate effect on the model outputs are the same for both the high and low sheep tick burden models (Table C.4). Where there are differences the effect is only just beyond what would be expected and may be simply due to the effect of the particular deer density. Section 3.3.2 discusses the impact of different deer densities on the model predictions.

The tick parameters show a highly disproportionate effect on the model predictions for the grouse density. In particular decreasing a_t the tick birth rate, increasing b_t the death rate and decreasing β_5 the tick biting rate on deer which will all reduce the tick population have a huge positive effect on the predicted grouse population. Although a positive effect would be expected as a reduction in the tick population will reduce the virus prevalence the magnitude of the effect is an order of magnitude higher than expected. This can be explained by the sheer size of the tick population and a small relative change can be a large change in terms of actual numbers. We are at a point where a small change in the tick population has a large effect on the grouse population and so the effect is disproportionate. Consequently the model is sensitive to the estimates of these parameters.

Although a change to those parameters increasing the tick population does have a negative effect on the grouse population predictions the magnitude is not as extreme.

In the high sheep tick burden model a change to β_7 the rate at which immature ticks attach to sheep

has a disproportionate effect on the grouse population predictions. The explanation for this may be due to the effect this parameter in the high sheep tick burden model has on the tick population and small relative changes now make sufficiently large numerical changes to show a large effect on the grouse population.

A smaller, but still disproportionate, effect can be seen by changing a_g the grouse birth rate, b_g the natural grouse death rate and β_2 the rate at which infected ticks bite grouse.

Interestingly although β_3 the rate at which ticks are ingested by grouse showed high correlation for both models it does not have a disproportionate effect on model outputs.

C.2.3 The seasonal models

Sensitivity analysis has been performed on the grouse pulse hatch model, grouse hatching period model, seasonal tick model and combined model.

Parameter choice effects on predicted grouse density

The natural death rate of grouse b_g remains a significant parameter for the seasonal models as it was for the ingestion model. However, it is interesting to note that it is not significant for the grouse pulse hatch model when the virus is able to persist (compare Tables C.5 and C.6 with Table C.1). This may be because the grouse population is so low that a change in the natural death rate makes little difference compared to other parameters.

The tick reproduction parameters show more correlation with predictions for the grouse pulse hatch model and seasonal tick model, but less so for the other seasonal models. A large tick population when the grouse hatch in one pulse has the potential to infect many of these chicks and will have a significant impact on the grouse population. The seasonal tick model concentrates the tick reproduction in a shorter time period and increasing the reproduction during this time will have more influence than a similar proportional increase over a longer time.

The virus transmission parameters are more influential for the grouse pulse hatch model and seasonal tick model when there are 10 deer. This may be because the opportunity for infection is concentrated into a shorter time period either because the influx of susceptible grouse occurs in one instant (grouse pulse hatch model) or because the ticks are only active for a short time when the grouse population is reproducing susceptible grouse all year (seasonal tick model).

Not surprisingly for the seasonal grouse models the grouse reproduction parameters have a disproportionate effect on model predictions of the grouse population (Table C.7). However, for the grouse pulse hatch model this only occurs when the grouse population is less able to reproduce and consequently dies out. The effect of the tick demography parameters for the seasonal grouse models is comparable with the ingestion model. The virus transmission parameters have a slightly greater effect for the grouse hatching period model.

For the seasonal tick model the effect of varying parameters on the grouse population predictions is very similar to the ingestion model. The combined model is also similar but the grouse demography

parameters match more closely to the grouse hatching period model.

Parameter choice effects on predicted virus prevalence

The birth rate of grouse a_g shows less correlation than the ingestion model with the seasonal grouse model predictions of virus prevalence but is more important in the seasonal tick models than found with the ingestion model (compare Tables C.5 and C.6 with Table C.1). The natural death rate b_g has a greater correlation for 6.5 per km^2 for all the seasonal models compared to the ingestion model.

The tick and virus parameters are less influential for all the seasonal models than for the ingestion model.

The parameters that have a disproportionate effect on the model predictions for virus prevalence for the grouse pulse hatch model are similar to the ingestion model but a_g and b_g have a greater impact (compare Table C.7 with Table C.2). The grouse hatching period model however shows an even greater sensitivity to a_g and b_g as well as the recovery rate and the tick reproduction parameters.

The seasonal tick model shows similar sensitivity to the model parameters as the ingestion model. The combined model however shows greater sensitivity to the grouse demography parameters. It is interesting that the seasonal tick models are not sensitive to changes in the tick or virus parameters.

C.2.4 The acaricide treated deer model

Parameter choice effects on predicted grouse density

The acaricide treated deer model is a very similar model to the ingestion model with the deer model including the effect of acaricide on deer and $\beta_3 = 0$ (compare Table C.8 with Table C.1). It is therefore not surprising that the parameters of the acaricide treated deer model show very similar correlation to that found for the ingestion model. The parameters that have a disproportionate effect also remain similar (compare Table C.9 with Table C.2) but the effect of increasing β_5 is greater because the acaricide on deer kills ticks which try to attach so increasing the tick attachment rate on deer will cause a greater number of ticks to be killed and fewer can reproduce. The opposite will occur if β_5 is decreased.

Parameter choice effects on predicted infection prevalence in grouse

Compared to the ingestion model the tick reproduction parameters a_t, b_t and β_5 have a slightly more disproportionate effect on virus prevalence so that reducing the tick population reduces the virus prevalence further. This is a consequence of the acaricide on deer reducing the ability of the tick population to reproduce.

C.2.5 The acaricidal leg band model

Parameter choice effects on predicted grouse density

The correlation of b_g with model predictions is comparable with other models but a_g and s_g show a slighter greater correlation (Table C.10). Increasing the reproductive ability of grouse will now increase

the number of grouse that are protected from infection through treatment so will have a greater effect than in previous models.

The tick reproductive parameters a_t, b_t, β_5 only show correlation when there are 6.5 deer per km^2 as this is when the size of the tick population has the most effect with the grouse hovering between carrying capacity and severe depletion by the virus. Other models showed a greater sensitivity when there were 10 deer per km^2 but now the grouse are directly treated this is less important.

The grouse reproduction parameters are now the only parameters that cause a disproportionate response in model predictions when increased or decreased (Table C.11) because the treatment of grouse amplifies the relative important of grouse reproduction. Treating grouse prevents the grouse contracting infection so even if only 20 hens are treated increasing the number of chicks they produce will increase the number of chicks being treated and unable to contract the virus. (The opposite will occur if reproduction is decreased.) As a result of the treatment of grouse the effect of other parameters is lessened as the grouse will remain protected from virus even if transmission rates are increased or the tick population increased. It should be noted that the model currently assumes the efficacy of the acaricide on chicks is 100% all summer. The effect of the grouse reproduction parameters may be lessened if these assumptions are relaxed.

Parameter choice effects on predicted infection prevalence in grouse

The model predictions of infection prevalence show a similar pattern of sensitivity to the grouse parameters but the extent of the correlation is reduced for 6.5 deer per km^2. The tick parameters show a slightly increased correlation to the infected grouse prevalence predictions, perhaps because an increased tick population can increase the proportion of grouse that are infected.

The grouse reproduction parameters have had a disproportionate effect on the infection levels for most model predictions in this thesis, so it is no surprise that changing the grouse reproduction parameters has a disproportionate effect for the acaricidal leg band model. However, as explained above the effect of increasing/decreasing grouse density is amplified by the direct treatment of grouse.

C.2.6 Summary

The sensitivity analysis indicates that we need to be cautious when interpreting the model predictions. Small changes in the parameter choices can lead to larger changes within the model predictions. In particular the grouse and tick demography parameters have a disproportional effect on model predictions. A better estimate of tick demography parameters, including the attachment rate of ticks on deer (in relation to tick attachment on grouse), will help improve the accuracy of model predictions of the grouse density, which is the easiest/most useful measure for grouse moor managers to use. The grouse demography parameters become more important for the seasonal models and a better estimate may be required to improve seasonal models.

The numerical model predictions may be affected by the parameter choices but I believe the general conclusions for the overall effect of different deer densities is valid regardless of the parameter choices

within the given limits. In general increasing the deer density decreases the grouse density as the deer increase the tick density. Although the model cannot be used to accurately predict the exact deer densities where this occurs I am confident that this will always occur.

Table C.1: **Ingestion** Significant correlation (Pearson's correlation coefficient, $P < 0.05$) between model parameters and the ingestion model predictions of grouse density (per km^2) and grouse infection prevalence respectively. Non-significant correlations are not shown. The model was run for 4, 6.5 and 10 deer per km^2. The parameters are grouped as grouse, tick and viral dynamic parameters and their corresponding interactions. Numbers in bold represent parameters (or interactions) that account for more than 20% of the variation in the model outputs.

	Grouse density per km^2			Grouse Infection prevalence		
	4 deer	6.5 deer	10 deer	4 deer	6.5 deer	10 deer
a_g	0.15	0.10	0.08	No Virus		0.20
b_g	**-0.98**	**-0.52**	**-0.84**	No Virus	0.13	**-0.98**
s_g	-0.07	-0.09		No Virus		
α				No Virus		-0.08
γ				No Virus		
a_t		-0.20	-0.36	No Virus	0.22	
b_t		0.21	0.30	No Virus	-0.22	
s_t	-0.09		0.18	No Virus		
β_1			-0.09	No Virus		
β_2			-0.08	No Virus		
β_3			-0.09	No Virus		0.06
β_5	0.07		-0.11	No Virus		
p	-0.07	-0.16	-0.16	No Virus	0.19	
$a_g * b_g$	**-0.96**	-0.51	**-0.83**	No Virus	0.12	**-0.95**
$a_g * s_g$				No Virus		0.12
$a_g * \alpha$	0.09	0.08	0.10	No Virus		0.08
$a_g * \gamma$	0.08	0.07	0.09	No Virus		0.12
$b_g * s_g$	**-0.98**	**-0.53**	**-0.84**	No Virus	0.13	**-0.97**
$b_g * \alpha$	**-0.97**	**-0.52**	**-0.83**	No Virus	0.12	**-0.98**
$b_g * \gamma$	**-0.97**	**-0.52**	**-0.83**	No Virus	0.13	**-0.97**
$s_g * \alpha$	-0.06	-0.06		No Virus		-0.08
$s_g * \gamma$	-0.08	-0.07		No Virus		
$\alpha * \gamma$			0.07	No Virus		-0.08
$a_t * b_t$				No Virus		
$a_t * s_t$	-0.09	-0.10	-0.12	No Virus	0.11	
$b_t * s_t$	-0.07	0.18	0.34	No Virus	-0.19	
$\beta_1 * \beta_2$			-0.12	No Virus		
$\beta_1 * \beta_3$			-0.13	No Virus		
$\beta_1 * \beta_5$			-0.14	No Virus		
$\beta_1 * p$	-0.06	-0.13	-0.18	No Virus	0.15	
$\beta_2 * \beta_3$			-0.13	No Virus		
$\beta_2 * \beta_5$			-0.14	No Virus		
$\beta_2 * p$	-0.07	-0.11	-0.18	No Virus	0.12	
$\beta_3 * \beta_5$			-0.15	No Virus		
$\beta_3 * p$		-0.12	-0.18	No Virus	0.14	
$\beta_5 * p$		-0.08	-0.19	No Virus	0.10	

Table C.2: **Ingestion** The percentage change (not shown if $< 10\%$) of the model predictions of total grouse density and infection prevalence in the grouse population after changing each parameter individually by $\pm 10\%$ for the ingestion model. (10 deer per km^2.)

	Grouse density per km^2	Grouse Infection prevalence
a_g-		-21
a_g+		21
b_g-		11
b_g+		-11
s_g-		
s_g+		
$\alpha-$		11
$\alpha+$		
$\gamma-$		
$\gamma+$		
a_t-	33	
a_t+	-20	
b_t-	-21	
b_t+	28	
s_t-	-10	
s_t+	10	
β_1-		
β_1+		
β_2-	11	
β_2+		
β_3-		
β_3+		
β_5-	17	
β_5+	-11	
$p-$		
$p+$		

Table C.3: **Sheep** Significant correlation (Pearson's correlation coefficient, $P < 0.05$) between model parameters and the model predictions of grouse density (per km^2) and virus prevalence within the grouse population respectively. Non-significant correlations are not shown. The model was run for 4, 7 and 15 deer per km^2, with(w) and without(wo) 50 sheep per km^2 treated with acaricide of 100% efficacy. The parameters are grouped as grouse, tick and viral dynamic parameters and their corresponding interactions.

	Grouse density per km^2												Infection prevalence											
	Low						High						Low						High					
	4w	4wo	7w	7wo	15w	15wo	4w	4wo	7w	7wo	15w	15wo	4w	4wo	7w	7wo	15w	15wo	4w	4wo	7w	7wo	15w	15wo
a_g	0.22	0.22	0.10	0.10			0.19	0.23									0.26	0.20					0.20	0.22
b_g	**-0.98**	**-0.98**	-0.40	-0.10			**-0.98**	**-0.98**	-0.31	-0.24							**-0.89**	**-0.76**		0.11			**-0.76**	**-0.88**
s_g	-0.11	-0.17						-0.17																
α							0.12																	
γ																							0.09	
$a_g \cdot b_g$	**0.96**	**0.96**	-0.39				**-0.95**	**-0.95**	-0.30	-0.24							**-0.87**	**-0.74**		0.11			**-0.74**	**-0.85**
$a_g \cdot s_g$				0.09													0.18	0.14					0.19	0.15
$a_g \cdot \alpha$	0.14	0.13					0.22	0.16									0.13	0.13					0.09	0.17
$a_g \cdot \gamma$	0.14	0.14					0.15	0.18									0.18	0.15					0.21	0.16
$b_g \cdot s_g$	**-0.98**	**-0.98**	-0.40	-0.10			**-0.98**	**-0.98**	-0.30	-0.23							**-0.89**	**-0.77**		0.11			**-0.75**	**-0.87**
$b_g \cdot \alpha$	**-0.97**	**-0.97**	-0.41	-0.10			**-0.97**	**-0.97**	-0.30	-0.24							**-0.89**	**-0.77**		0.11			**-0.76**	**-0.88**
$b_g \cdot \gamma$	**-0.97**	**-0.97**	-0.40	-0.10			**-0.97**	**-0.97**	-0.30	-0.24							**-0.89**	**-0.76**		0.10			**-0.75**	**-0.87**
$\alpha \cdot s_g$	-0.10	-0.14						-0.12																
$\alpha \cdot \gamma$							0.10																	
$\gamma \cdot s_g$	-0.10	-0.13						-0.10															0.11	
a_t												-0.12		0.10										
b_t	-0.10																0.10		-0.11					
s_t				-0.10						-0.10						0.10						0.09		
$a_t \cdot b_t$	-0.10											-0.10							-0.12					
$a_t \cdot s_t$																								
$b_t \cdot s_t$	-0.11																0.09						0.10	
p	0.09										0.13											0.12		
β_1																								
β_2															-0.11									0.10
β_3			0.35	**0.87**	**-0.81**	**-0.88**			0.35	**0.85**	**-0.89**	**-0.80**	-0.16	-0.37	-0.33	**-0.85**	-0.16	-0.40	-0.19	-0.34	-0.34	**-0.84**	**-0.48**	-0.11
β_5																					0.09			
β_6				0.10	0.12											-0.11								
β_7			0.33		0.40				0.33			0.34	-0.13		-0.30				-0.15		-0.32			0.11
$\beta_1 \cdot \beta_2$									-0.09				0.10											
$\beta_1 \cdot \beta_3$			0.35	**0.87**	**-0.81**	**-0.88**			0.35	**0.85**	**0.89**	**-0.80**	-0.16	-0.37	-0.33	**-0.85**	-0.16	-0.40	-0.19	-0.34	-0.34	**-0.84**	**-0.48**	-0.11
$\beta_1 \cdot \beta_5$																					0.09			
$\beta_1 \cdot \beta_6$				0.09	0.13											-0.11								
$\beta_1 \cdot \beta_7$			0.33		0.40				0.33			0.34	-0.13		-0.30		0.10		-0.15		-0.32			0.11
$\beta_2 \cdot \beta_3$			0.35	**0.87**	**-0.81**	**-0.88**			0.35	**0.85**	**-0.89**	**-0.80**	-0.16	-0.37	-0.33	**-0.85**	-0.16	-0.40	-0.19	-0.34	-0.34	**-0.84**	**-0.48**	-0.11
$\beta_2 \cdot \beta_5$									-0.11												0.10			0.10
$\beta_2 \cdot \beta_6$				0.10	0.12											-0.11								
$\beta_2 \cdot \beta_7$			0.33		0.40				0.33			0.34	-0.13		-0.30		0.10		-0.15		-0.32			0.11
$\beta_3 \cdot \beta_5$			0.35	**0.87**	**-0.81**	**-0.88**			0.35	**0.85**	**-0.89**	**-0.80**	-0.16	-0.37	-0.33	**-0.85**	-0.16	-0.40	-0.19	-0.34	-0.34	**-0.84**	**-0.48**	-0.11
$\beta_3 \cdot \beta_6$			0.28	**0.70**	**-0.59**	**-0.66**			0.29	**0.64**	**-0.67**	**-0.59**	-0.13	-0.27	-0.25	**-0.68**	-0.16	-0.28	-0.14	-0.26	-0.28	**-0.64**	-0.40	
$\beta_3 \cdot \beta_7$			0.35	**0.87**	**-0.81**	**-0.88**			0.35	**0.85**	**-0.89**	**-0.80**	-0.16	-0.37	-0.33	**-0.85**	-0.16	-0.40	-0.19	-0.34	-0.34	**-0.84**	**-0.48**	-0.11
$\beta_5 \cdot \beta_6$				0.10	0.12											-0.11								
$\beta_5 \cdot \beta_7$			0.32		0.40				0.33			0.34	-0.13		-0.30		0.10		-0.15		-0.32			0.11
$\beta_6 \cdot \beta_7$			0.26		0.41				0.28			0.31	-0.11		-0.24				-0.11		-0.26			

Table C.4: **Sheep** The percentage change (not shown if $< 10\%$) of the model predictions of total grouse density and infection prevalence in the grouse population after changing each parameter individually by $\pm 10\%$. The results are given for both the high and low sheep tick burden models run with 11 and 7 deer per km^2 respectively.

	Grouse density per km^2		Infection prevalence	
	Low	High	Low	High
a_g+	13	11	21	22
a_g-	-12		-21	-22
b_g+	-11		-12	-11
b_g-	16	12	11	11
s_g+				
s_g-				
$\alpha+$				
α-			12	12
$\gamma+$				
γ-				
a_t+	-51	-45		
a_t-	297	553	-37	-71
b_t+	248	113	-31	-13
b_t-	-51	-35		
s_t+		12		
s_t-		-11		
$p+$				
p-				
β_1+				
β_1-				
β_2+	-12	-12		
β_2-	17	15		
β_3+				
β_3-				
β_5+	-45	-39		
β_5-	247	424	-32	-56
β_6+				
β_6-				
β_7+		39		
β_7-		-22		

Table C.5: **Seasonal grouse** Significant correlation (Pearson's correlation coefficient, $P < 0.05$) between parameters and the seasonal grouse model predictions of grouse density (per km^2) and grouse infection prevalence respectively. Non-significant correlations are not shown. The model was run for 4, 6.5 and 10 deer per km^2. The parameters are grouped as grouse, tick and viral dynamic parameters and their corresponding interactions. Numbers in bold represent parameters (or interactions) that account for more than 20% of the variation in the model outputs. "-" indicates that parameter is not relevant in that model.

	Grouse density per km^2						Grouse Infection prevalence					
	Grouse pulse hatch			Grouse hatch period			Grouse pulse hatch			Grouse hatch period		
	4 deer	6.5 deer	10 deer	4 deer	6.5 deer	10 deer	4 deer	6.5 deer	10 deer	4 deer	6.5 deer	10 deer
a_g				0.09	0.07	0.11	no virus		0.07	no virus		
b_g	**-1**			**-0.99**	**-0.59**	**-0.51**	no virus	**-0.98**	**-0.94**	no virus	**-0.92**	**-0.79**
s_g							no virus			no virus		-0.07
α			0.18				no virus			no virus		-0.09
γ							no virus			no virus		
a_t		**-0.56**	**-0.51**		-0.07		no virus	0.11		no virus		
b_t		**0.56**	**0.52**				no virus	-0.06		no virus		
s_t		0.08	0.13				no virus			no virus		
β_1		-0.11	-0.20			-0.07	no virus			no virus		-0.07
β_2			-0.21	-0.06			no virus			no virus		-0.06
a				-	-	-	no virus	0.13		-	-	-
β_5			-0.13		-0.08		no virus			no virus		
$a_g * b_g$	**-0.70**			**-0.98**	**-0.58**	**-0.50**	no virus	**-0.71**	**-0.65**	no virus	**-0.91**	**-0.78**
$a_g * s_g$				0.08		0.06	no virus		0.07	no virus		
$a_g * \alpha$			0.13	0.07		0.12	no virus			no virus		
$a_g * \gamma$						0.07	no virus			no virus		
$b_g * s_g$	**-0.71**			**-0.99**	**-0.58**	**-0.51**	no virus	**-0.71**	**-0.68**	no virus	**-0.97**	**-0.97**
$b_g * \alpha$	**-0.70**		0.09	**-0.99**	**-0.59**	**-0.50**	no virus	**-0.73**	**-0.70**	no virus	**-0.98**	**-0.98**
$b_g * \gamma$	**-0.70**			**-0.99**	**-0.59**	**-0.51**	no virus	**-0.70**	**-0.66**	no virus	**-0.92**	**-0.79**
$s_g * \alpha$			0.12				no virus			no virus		
$s_g * \gamma$							no virus			no virus		
$\alpha * \gamma$			0.15				no virus			no virus		
$a_t * b_t$							no virus	0.08		no virus		
$a_t * s_t$		-0.34	-0.26		-0.09		no virus			no virus		
$b_t * s_t$		**0.45**	**0.48**				no virus			no virus		
$\beta_1 * \beta_2$		-0.08	-0.30	-0.06			no virus			no virus		-0.09
$\beta_1 * \beta_5$			-0.12		-0.08	-0.09	no virus	0.13		no virus		-0.08
$\beta_2 * \beta_5$		-0.11	-0.20		-0.07		no virus			no virus		-0.08

Table C.6: **Seasonal tick** Significant correlation (Pearson's correlation coefficient, $P < 0.05$) between parameters and the seasonal tick models predictions of grouse density (per km^2) and grouse infection prevalence respectively. Non-significant correlations are not shown. The model was run for 4, 6.5 and 10 deer per km^2. The parameters are grouped as grouse, tick and viral dynamic parameters and their corresponding interactions. Numbers in bold represent parameters (or interactions) that account for more than 20% of the variation in the model outputs.

	Grouse density per km^2						Grouse Infection prevalence					
	Seasonal tick			Combined			Seasonal tick			Combined		
	4 deer	6.5 deer	10 deer	4 deer	6.5 deer	10 deer	4 deer	6.5 deer	10 deer	4 deer	6.5 deer	10 deer
a_g	0.12		0.11	0.10			No virus	0.21	0.19	No virus	0.09	
b_g	**-0.98**	**-0.44**	**-0.86**	-1	**-0.55**	**-0.53**	No virus	**-0.97**	**-0.97**	No virus	**-0.99**	**-0.99**
s_g	-0.06		-0.08				No virus			No virus		
α		0.07					No virus		-0.15	No virus		
γ					0.09		No virus			No virus		
a_t		**-0.53**	-0.29		-0.17		No virus			No virus		
b_t		**0.53**	0.29		0.13		No virus	-0.11		No virus		
s_t							No virus		-0.09	No virus		
β_1			-0.14			-0.08	No virus			No virus		-0.07
β_2		-0.07	-0.16				No virus		-0.06	No virus		
β_5		**-0.43**	-0.13		-0.11		No virus			No virus		
$a_g * b_g$	**-0.95**	**-0.44**	**-0.84**	**-0.98**	**-0.55**	**-0.52**	No virus	**-0.94**	**-0.95**	No virus	**-0.98**	**-0.98**
$a_g * s_g$							No virus	0.11	0.10	No virus		0.08
$a_g * \alpha$		0.07	0.09	0.12			No virus	0.11		No virus		
$a_g * \gamma$	0.07		0.12	0.06	0.10		No virus	0.14	0.14	No virus	0.10	
$b_g * s_g$	**-0.98**	**-0.44**	**-0.86**	**-0.99**	**-0.55**	**-0.53**	No virus	**-0.96**	**-0.97**	No virus	**-0.99**	**-0.98**
$b_g * \alpha$	**-0.97**	**-0.44**	**-0.85**	**-0.99**	**-0.55**	**-0.52**	No virus	**-0.97**	**-0.98**	No virus	**-0.99**	**-0.98**
$b_g * \gamma$	**-0.97**	**-0.44**	**-0.85**	**-0.99**	**-0.54**	**-0.53**	No virus	**-0.96**	**-0.97**	No virus	**-0.99**	**-0.98**
$s_g * \alpha$	-0.09						No virus	-0.07	-0.14	No virus		
$s_g * \gamma$	-0.06				0.07		No virus			No virus		
$\alpha * \gamma$		0.09					No virus		-0.10	No virus		
$a_t * b_t$							No virus			No virus		
$a_t * s_t$		-0.35	-0.19		-0.09		No virus		-0.07	No virus		
$b_t * s_t$		0.40	0.23		0.12		No virus	-0.10		No virus		
$\beta_1 * \beta_2$		-0.07	-0.16				No virus		-0.06	No virus		
$\beta_1 * \beta_5$	0.08	-0.34	-0.19		-0.11	-0.06	No virus			No virus		
$\beta_2 * \beta_5$		-0.35	-0.20		-0.08		No virus			No virus		

Table C.7: **Seasonal** The percentage change (not shown if $< 10\%$) of the model predictions of total grouse density and infection prevalence in the grouse population after changing each parameter individually by $\pm 10\%$ for the grouse pulse hatch model (Pulse), grouse hatching period model (Period), seasonal tick model (Tick) and combined model (Comb). (10 deer per km^2.)

	Grouse density per km^2				Grouse Infection prevalence			
	Pulse	Period	Tick	Comb	Pulse	Period	Tick	Comb
a_g-	-100	-35		-35	-100	-100	-21	-78
a_g+		-13			133	257	21	83
b_g-		-11			213	456		126
b_g+	-100	-96		-100	no grouse	-100		-100
s_g-							11	
s_g+							-11	
$\alpha-$	-10	-11		11		11		11
$\alpha+$	10	11						
$\gamma-$						-24		
$\gamma+$						30		
a_t-	31	37	31	31		56		
a_t+	-20	-26	-19	-19		-27		
b_t-	-22	-48	-20	-21	11	34		
b_t+	27	19		26	26	298		
s_t-				-10				
s_t+				10				
β_1-	11	12	11	11				
β_1+		-10						
β_2-	11	12	11	11				
β_2+		-10						
β_5-	18	24	17	17		72		
β_5+	-11	-17	-11	-11		-25		

Table C.8: **Deer** Significant correlation (Pearson's correlation coefficient, $P < 0.05$) between model parameters and the acaricide treated deer model predictions of grouse density (per km^2) and grouse infection prevalence respectively. Non-significant correlations are not shown. The model was run for 6.5, 10 and 15 deer per km^2. The parameters are grouped as grouse, tick and viral dynamic parameters and their corresponding interactions. Numbers in bold represent parameters (or interactions) that account for more than 20% of the variation in the model outputs. Acaricide efficacy 50% throughout.

	Grouse density per km^2			Grouse Infection prevalence		
	6.5 deer	10 deer	15 deer	6.5 deer	10 deer	15 deer
a_g	0.14	0.20	0.08	No virus	No virus	0.21
b_g	**-0.98**	**-0.98**	**-0.62**	No virus	No virus	**-0.98**
s_g	-0.16	-0.12		No virus	No virus	
α				No virus	No virus	-0.16
γ				No virus	No virus	
a_t			-0.35	No virus	No virus	0.10
b_t			0.41	No virus	No virus	
s_t			0.07	No virus	No virus	
β_1			-0.09	No virus	No virus	
β_2			-0.08	No virus	No virus	
β_4				No virus	No virus	
β_5			-0.29	No virus	No virus	0.09
$a_g * b_g$	**-0.95**	**-0.95**	**-0.61**	No virus	No virus	**-0.95**
$a_g * s_g$		0.06		No virus	No virus	0.11
$a_g * \alpha$	0.10	0.16	0.07	No virus	No virus	
$a_g * \gamma$	0.12	0.13	0.09	No virus	No virus	0.14
$b_g * sg$	**-0.98**	**-0.98**	**-0.62**	No virus	No virus	**-0.97**
$b_g * \alpha$	**-0.97**	**-0.97**	**-0.61**	No virus	No virus	**-0.98**
$b_g * \gamma$	**-0.97**	**-0.97**	**-0.61**	No virus	No virus	**-0.97**
$s_g * \alpha$	-0.12	-0.06		No virus	No virus	-0.15
$s_g * \gamma$	-0.10	-0.09		No virus	No virus	
$\alpha * \gamma$				No virus	No virus	-0.11
$a_t * b_t$				No virus	No virus	0.07
$a_t * s_t$			-0.18	No virus	No virus	0.07
$b_t * s_t$			0.34	No virus	No virus	
$\beta_1 * \beta_2$			-0.12	No virus	No virus	
$\beta_1 * \beta_5$				No virus	No virus	
$\beta_2 * \beta_5$			-0.26	No virus	No virus	0.09

Table C.9: **Deer** The percentage change (not shown if < 10%) of the model predictions of total grouse density and infection prevalence in the grouse population after changing each parameter individually by ±10% for the acaricide treated deer model. (15 deer per km^2, acaricide 50% efficacy.)

	Grouse density per km^2	Grouse Infection prevalence
a_g-		-23
a_g+		23
b_g-		11
b_g+		-12
s_g-		
$_g+$		
$\alpha-$		12
$\alpha+$		-10
$\gamma-$		
$\gamma+$		
a_t-	128	-16
a_t+	-36	
b_t-	-36	
b_t+	88	-11
s_t-		
s_t+		
β_1-	11	
β_1+		
β_2-	11	
β_2+		
β_4-		
β_4+		
β_5-	100	-12
β_5+	-29	

Table C.10: **Acaricidal leg band** Significant correlation (Pearson's correlation coefficient, $P < 0.05$) between model parameters and the acaricidal leg band model predictions of grouse density (per km^2) and grouse infection prevalence respectively. Non-significant correlations are not shown. The model was run for 4, 6.5 and 10 deer per km^2. The parameters are grouped as grouse, tick and viral dynamic parameters and their corresponding interactions. Numbers in bold represent parameters (or interactions) that account for more than 20% of the variation in the model outputs. Acaricide on chicks 100% efficacy all summer, 20 hens treated.

	Grouse density per km^2			Grouse Infection prevalence		
	4 deer	6.5 deer	10 deer	4 deer	6.5 deer	10 deer
a_g	0.27	0.21	0.18	No virus		0.11
b_g	**-0.95**	**-0.53**	**-0.98**	No virus	**-0.44**	**-0.99**
s_g	-0.12	-0.15	-0.07	No virus		-0.06
α				No virus		
γ				No virus		
a_t		**-0.45**		No virus	**0.47**	
b_t		0.35		No virus	-0.41	
s_t				No virus		
β_1		-0.07		No virus		
β_2				No virus		
β_5		**-0.45**		No virus	**0.46**	
$a_g * b_g$	**-0.92**	**-0.51**	**-0.96**	No virus	-0.43	**-0.98**
$a_g * s_g$	0.10		0.08	No virus		
$a_g * \alpha$	0.15	0.14	0.14	No virus		0.07
$a_g * \gamma$	0.17	0.14	0.12	No virus		0.06
$b_g * s_g$	**-0.96**	**-0.54**	**-0.98**	No virus	-0.43	**-0.99**
$b_g * \alpha$	**-0.95**	**-0.53**	**-0.98**	No virus	-0.43	**-0.99**
$b_g * \gamma$	**-0.95**	**-0.53**	**-0.98**	No virus	**-0.44**	**-0.99**
$s_g * \alpha$	-0.12	-0.11		No virus		
$s_g * \gamma$	-0.10	-0.11		No virus		
$\alpha * \gamma$				No virus		
$a_t * b_t$		-0.07		No virus		
$a_t * s_t$		-0.29		No virus	0.31	
$b_t * s_t$		0.28		No virus	-0.31	
$\beta_1 * \beta_2$				No virus		
$\beta_1 * \beta_5$		-0.36		No virus	0.34	
$\beta_2 * \beta_5$		-0.28		No virus	0.29	

Table C.11: **Acaricidal leg band** The percentage change (not shown if $< 10\%$) of the model predictions of total grouse density and infection prevalence in the grouse population after changing each parameter individually by $\pm 10\%$ for the acaricidal leg band model. (10 deer per km^2, acaricide on chicks 100% efficacy all summer, 20 hens treated.)

	Grouse density per km^2	Grouse Infection prevalence
a_g-	-11	-18
a_g+	11	16
b_g-		19
b_g+		-20
s_g-		
s_g+		
$\alpha-$		
$\alpha+$		
$\gamma-$		
$\gamma+$		
a_t-		
a_t+		
b_t-		
b_t+		
s_t-		
s_t+		
β_1-		
β_1+		
β_2-		
β_2+		
β_5-		
β_5+		

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