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Evaluation of approaches to control of Maedi-Visna disease of sheep using a Markov chain simulation model for a range of typical British flocks

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Abstract

An epidemiological model is described that closely mimicked results of a published serological study of natural transmission of Maedi-Visna virus in a low ground flock of sheep. We adjusted parameters in the model from this baseline to explore the possible implications for the control of Maedi-Visna virus in typical British flocks. On closed hill farms, low probability of effective contact was most critical for control. In open low ground flocks, purchasing accredited replacements eliminated disease spread, otherwise flock size was the most important factor governing flock prevalence. Results highlighted the need for more epidemiological information about Maedi-Visna, particularly whether hill farms act as a hidden reservoir of virus or reduce the impact of this disease on the industry by providing a source of clean replacements.

Keywords: Maedi-Visna; Model; Markov Chain; Sheep; Control

1. Introduction

All too frequently disease control decisions on farms must be made on the basis of incomplete and/or inaccurate information. As a result, sub-optimal control actions may be taken, damaging animal health, animal welfare and/or the efficiency of resource use. McInerney (1996) sets out a framework that identifies the disease control actions that deliver maximum economic efficiency but the data it requires are rarely available (Bennett, 2003). In this situation, simulation models of disease epidemics can aid disease control decision-making as demonstrated by Stott et al. (2003). By testing a wide range of possible assumptions, simulation models offer the best opportunity to identify key determinants of a disease outcome so providing best available estimates for the data needed to evaluate alternative control actions.

This paper provides such estimates from a model for Maedi-Visna (MV) disease in sheep. MV is of international significance, being present in most of the sheep producing nations of the world with the exceptions of Australia and New Zealand (Radostits, 2002). It is also a disease about which much is still to be discovered. MV is a 'slow' disease, with two or more years typically elapsing between infection and development of clinical signs, which include chronic pneumonia (maedi), neurological damage (visna), arthritis and indurative mastitis (Narayan et al., 1985). Disease losses can include mortality, increased culling rates and reduced lambing rates. In addition, reduced lamb output (liveweight and mortality) can arise as a result of mastitis associated with MV infection (Van der Molen et al., 1985; Dohoo et al., 1987; Houwers, 1990; Pekelder et al., 1994; Keen et al., 1997). Past experience

has shown these disease losses can be devastating to a flock (Milne et al., 1993) or, as in the case of Iceland, a country's sheep industry (Pàlsson, 1976).

Not all reported outbreaks of MV report serious losses and within flock prevalence levels vary (Spence et al., 1981; Markson et al., 1983; Narayan et al., 1985; Sihvonen et al., 1999). The extent of losses is in any case difficult to quantify as the start and end of the infection period is often indeterminate. Also, factors such as viral strain and breed susceptibility can influence the disease outcome (Houwers, 1990). Experimental studies have shown that horizontal transmission through close contact is the main route of infection. Thus flock management system (e.g. the use of housing) can be important to the rate of transmission, affecting within flock prevalence levels, as well as the level of losses (Cutlip et al., 1986; Pépin et al., 1998). However, one study records the spread of infection in an experimental flock over a period of five years following the introduction of two infected ewes. By the end of the study over 80% of ewes had become infected (Houwers and Van der Molen, 1987). With innumerable possible combinations of system factors, viral strain and breed it is unsurprising that conflicting reports on the importance of MV exist. Our model therefore helps us explore management factors that might influence a MV outbreak for a range of typical UK flock management systems and circumstances. Our study thereby provides insights into the potential relative importance of key factors that are likely to influence spread of MV and that vary considerably between farms. This helps to identify situations where risk from MV may be greatest and the strategies most likely to mitigate such

risks.

Methods

2.1 The model

We constructed a flock model using a state transition, or Markov chain, methodology within a spreadsheet program (Agrawal and Heady, 1972; Carpenter, 1988). The model encompasses a 10 year time period in annual steps, reflecting the annual production cycle of a sheep flock. A time horizon of 10 years was chosen to explore the long-term effects of the disease while being short enough to be of practical interest (Gunn et al., 2004).

Within the model a flock is defined in terms of size (ewe number) and parity distribution (1 to 4). Ewe and lamb mortality rates and lambing rates are then defined by parity. Replacements may be retained (homebred) or purchased. This model structure permitted alternative flock situations to be investigated, for example variable production intensity, ewe longevity and flock size to contrast typical low ground (Table 1) or hill farming (Table 2) conditions in the UK as reported in SAC (2004). For the low ground, assumptions reflected crossbred breeding ewes producing finished and store lambs on grass (page 210, SAC 2004). For the hill, the specific system chosen was Blackface ewes producing store lambs with limited pasture land, typical of the Grampians or Southern Upland regions of Scotland (page 202-203, SAC, 2004). In all cases, ewe replacement was adjusted annually according to attrition from MV

related disease so as to maintain a constant flock size throughout the time horizon. Mortality rates in ewes and lambs due to MV in the low ground situation were based on Houwers and Van der Molen (1987) and Milne and Gray (1993). In the absence of published information about the epidemiology of MV in hill farming systems, we adjusted the low ground assumptions to reflect expected differences, i.e. ewes in the hill are a year older in each parity and lambs of infected ewes are more vulnerable but under less competition from litter mates.

<Tables 1 and 2>

Within each parity, MV disease states were uninfected (U), infected (I) or dead from MV (D). The flock was therefore represented in each year (stage) by a vector of 12 states (4 parity by 3 disease). The transition probability (P) from state U in each parity at stage n to state I in the next parity at stage n+1 was governed by the number of infected ewes at stage n (Ct), according to the Reed-Frost relationship (Abbey, 1952):

$$P = (1 - Q^{Ct})$$

where Q is the probability of avoiding effective contact. This dealt with the horizontal transmission of MV. For vertical transmission, 0.8 of lambs from infected ewes were assumed to be infected based on De Boer et al. (1979).

The initial state vector was set to reflect the ewe age distribution typical of either low ground (Table 1) or hill (Table 2) flocks in the UK. These assumptions were verified using the Markov chain by testing for equivalence with the long-run steady state age distribution of a disease-free flock. This was possible because the Markov chain is stationary, i.e. all assumptions are fixed throughout the time horizon so that a steady state vector is achieved in the long run that is independent of the initial state vector and reflects the relevant assumptions used such as replacement rate.

2.2 Model Runs

Using the stated assumptions the model was first validated using published data for MV spread and then run for two situations, these were:

- A 'closed' flock where one infected animal (in parity one) is present at the start. This situation is similar to one where all replacement females are home bred but rams are purchased. As this is general practice on hill farms, this situation was associated only with hill farms (Table 2).
- An open flock where no infected animals are present at the start but some infected replacements are purchased on an annual basis. This scenario was confined to low ground flocks (Table 1), as they are more likely to be in this situation.

In the less favoured areas (LFA) of Great Britain, sheep flocks tend to be larger than in other areas (Figure 1) with a mode of over 1000 ewes/flock. For the above hill farm situation, models were therefore run with flock sizes (n) ranging from 400 to 2000 ewes in 100 ewe increments giving a total of 16 alternative flock sizes. Ten alternative values of Q were also used ranging from 0.99 to 0.999 in increments of 0.01. This gave a total of 16x10=160 alternative hill farm model runs. For the low ground situation where smaller flocks are more common (Figure 1), six alternative flock sizes were run (50 to 550 ewes/flock in 100 ewe increments). Lower values of Q were tested (0.98 to 0.996 in increments of 0.004, giving five alternatives) to reflect the closer contact between ewes in the smaller, more intensively managed low ground flocks. The proportion of MV infected replacements (r) in the open low ground flocks may reflect national prevalence of MV. Bennett et al. (1999) used a range of 0.003 to 0.005 based on a survey of seroprevalence. We therefore tested eight values from 0.002 to 0.009 in increments of 0.001, straddling the range of Bennett et al. (1999) but also going beyond this as national prevalence is uncertain and prevalence of MV in replacement ewes may not reflect that of the sheep population as a whole. A total of 240 runs (6n x 5Q x 8r) were therefore executed for the low ground situation.

The average annual prevalence (pr) of MV (proportion of flock in states I) across the 10 year simulated epidemic was used as the output variable for each model run. Relationships between the three parameters (n, Q, r) and pr were explored graphically. Where appropriate, pr was used as the dependent variable and n, Q and r as the independent variables in a multiple linear regression analysis conducted using the Genstat statistical package (Lawes Agricultural Trust, 2005). Using the multiple regression equation, the predicted partial impact over the ranges of n, Q and r on pr was used as a measure of the relative importance of each parameter for the development of the simulated MV epidemic under the different circumstances investigated.

2. Results

3.1 Validation

Figure 2 shows the comparison between the output from our low ground flock model and the data from a serological study of natural transmission of MV in a low ground flock reported by Houwers and Van der Molen (1987). The proportion of infected ewes in our model in year one was adjusted to 0.1 in line with Houwers and Van der Molen (1987) and the Q value adjusted to 0.98 to give the best possible fit (lowest standard error of differences). Otherwise all assumptions in the model were as reported above.

<Figure 1>

3.2 Low ground flocks

The lowest value of r (0.002) produced negligible values of pr for all values of the other parameters tested. Associated data were therefore dropped from subsequent analysis, reducing the data set from 240 to 210 records. The best fit multiple linear regression equation for low ground flocks was then as follows:

$$pr = 9.24 + 0.00059n - 9.52Q + 19.03r$$
(0.71) (0.000024) (0.72) (2.04)

Standard errors for the intercept and each regression coefficient are shown in brackets underneath their corresponding estimate. All estimates were significantly different from zero (p<0.001). The adjusted R^2 was 0.81.

However, there was a tendency for the linear model to predict less well at very high and very low values of pr. This was attributed mainly to flock size. Plotting flock size against the mean values of pr produced a quadratic relationship (R^2 =0.97) suggesting a maximum pr of 0.41 at 917 ewes/flock and a pr of 0.0 at 92 ewes/flock. As such large flocks are rare in the low ground (Figure 1) and as adding appropriate additional terms to the linear regression model gave no appreciable improvement in model fit to the original data, the above linear model was used to test the relative impact of the parameters on pr. The results of this test are in Table 3. They indicated that given the range of parameter values (chosen to reflect the likely range in the field), variation in flock size tends to be a more important influence on MV than variations in Q and r for low ground flocks.

3.3 Hill flocks

In contrast to the low ground flocks, the relationships between pr and n and Q for hill flocks were non-linear, each was best represented by a quadratic curve. The best fit equation was as shown below with standard errors given in brackets:

$$Pr = -7964 + 0.000352n - 9.1E-08n^{2} + 16069Q - 8105Q^{2}$$
(412) (0000346) (1.45E-08) (829) (417)

As for the low ground model, all estimates were significantly different from

zero (p<0.001). The adjusted R^2 statistic in this case was 0.96. Predictions over the range of each parameter at the mean of all other parameters is given in Table 4. Compared to the low ground flock, flock size had relatively less effect (0.014 increase in pr per 100 ewes versus 0.12 for low ground flocks) while Q had more effect on pr (0.52 per 0.01 Q vs 0.09 for low ground flocks) over the range of parameter values tested. Beyond this range, the quadratic relationship between the mean value of pr at each value of n implied a maximum value of pr of 0.56 at a flock size of 2222, with pr still at 0.16 with flock sizes as small as 100. The corresponding relationship between Q and mean pr gave a maximum pr of 0.53 at a Q value of 0.991, with pr falling to 0.0 at a Q value of 0.999.

3. Discussion

The reasonably close agreement between our model's output and the results of an experimental MV epidemic are encouraging (Figure 2). This provided a means to establish an appropriate value for Q and a plausible range for sensitivity analysis. Such an approach was not possible when modelling spread of bovine viral diarrhoea (BVD) in cattle where the Reed-Frost equation was also used. In that case no empirical data were available and a wide range of Q values appeared in the literature (Gunn et al., 2004).

In the study of Houwers and Van der Molen (1987) a small flock of 19 ewes ranging in age from 1 to 9 years and free of MV were combined with two MV seropositive ewes. The flock was maintained on a small pasture (1.0 or 1.5

Ha) and housed during the night, at lambing time and during periods of cold or wet weather. Ewes and lambs were kept together for 5 years. We would expect the Q value appropriate for commercial flocks in the UK to be higher than this especially on hill farms where ewes rarely come into such close contact with one another in an enclosed space. Our lowest Q value was therefore 0.98 and all sensitivity analysis was conducted using higher Q values than this. Houwers and Van der Molen (1987) acknowledge that the incidence of horizontal transmission in their experiment was exceptionally high due to the particular management of their flock

If MV Q values are very high, our results demonstrate that even if infected ewes are introduced into an MV free flock the disease may not spread or may do so only very slowly. This outcome is even more likely if the flock is small, open and buys in replacements with very low MV prevalence. These results are in line with two surveys in the 1990's that indicated a low national MV flock prevalence level of around 1.5% (VLA, 2002). Also it may be necessary for the flock to suffer stress before MV takes a strong hold and serious losses occur as was observed by Milne and Gray (1993) and alluded to by Houwers and Van der Molen (1987). There may also be other factors that predispose certain flocks to MV such as particularly susceptible genotypes or a more virulent strain of the virus. In all these cases the value of Q may be lowered and the modelled consequences of MV much more serious due to the high sensitivity of the model outcomes to Q.

The above arguments imply that the economic importance of MV at national level may be relatively small compared to other endemic diseases of sheep.

Bennett et al. (1999) put the annual output loss/resource wastage due to MV in Great Britain at about £1m (1996 values), which was lower than the other sheep diseases investigated (Blow-fly strike, Enzootic abortion, Orf and Toxoplasmosis). However, the impact of MV on an individual farm where clinical disease has become established may be extremely serious and distressing as shown by Milne and Gray (1993). A model such as ours may help to prevent such occurrences by identifying those practices most likely to mitigate the risks. For example, our results suggest that buying in replacement ewes with low or zero MV prevalence may be a good precaution.

Our results suggested that the relative importance of n and Q may be quite different in hill compared to low ground sheep flocks. These results reflect the different circumstances and management practices operating. For example, closed extensive hill farms could be particularly dependent on maintaining a low probability of MV transmission between individuals. However, values of Q are not precisely known. We therefore tested a wide range for hill farms with some very close to 1.0 (certain to avoid transmission of MV). This gave a wide range for pr creating an impression of greater relative importance of Q in the hill farm simulations. In practice Q values will be higher in hill flocks than in low ground flocks due to the reduced opportunity for horizontal transmission of MV but their range may not be as great as we used here to reflect uncertainty over the values likely to apply in practice. Despite this, hill farm simulations were more sensitivity to marginal variations in Q below a very high threshold of 0.999. This is likely to be important. If Q is greater than 0.999 in hill flocks MV will not spread within hill farms and they are therefore unlikely to be a

reservoir of MV virus, passing it down to low ground flocks through the sale of breeding stock. Most reported outbreaks of MV have been in low ground flocks, which lends some weight to this hypothesis and suggests an important attribute of the stratified system that traditionally operates in the UK. In the stratified system, hill farms sell on pure-bred ewes to upland farmers who sell the cross-bred progeny in turn to low ground farmers. However, given the 'slow' nature of MV, symptoms of the disease may not be obvious in this system until infected ewes reach the low ground farms. This provides another explanation for the predominance of recorded outbreaks in low ground flocks and suggests that a study of MV prevalence in hill farms may be warranted. Given the role of hill farms in the stratification system and the likely absence of MV symptoms there, freedom from MV at the top of the pyramid is particularly important. However, reform of the Common Agricultural Policy in Europe (European Union, 2005) may threaten the stratified sheep farming system, altering the way in which sheep are traded thus influencing the epidemiological factors that govern MV spread and perhaps leading to a higher prevalence of MV. Even if the stratification system persists, increased commercial pressures post CAP reform may encourage bigger hill farms. If this is combined with more intensive operations that reduce Q then our results suggest that the combination of greater n, lower Q and high sensitivity to Q may increase the risk of MV spread in hill flocks and hence increase the risk of MV across the whole sheep farming system of Great Britain.

The dominant factor in low ground farms seems to be flock size. At the mean value of the other parameters, small flock size was sufficient to prevent

spread of MV (Table 3) with a suggestion from the quadratic curve fitting that about 90 ewes/flock represents a threshold. This appears to contradict the empirical study of Houwers and Van de Molen (1987) who clearly established a very serious MV epidemic in a very small flock. However, their flock was closed. We assumed that low ground flocks would be open i.e. would replenish their flock each year with replacements drawn from a pool with very low prevalence of MV, reflecting the assumed low level of MV in the national flock. This would appear to be sufficient to ensure that small flocks at least remain MV free. However, in practice n, Q and r are unlikely to be independent. Small flocks may keep animals more closely confined decreasing Q. They may source replacements from fewer sources thus affecting r. Our results suggest that a threshold level of r exists within the current range of national prevalence estimates that is sufficient to maintain MV-free low ground flocks regardless of the level of the other parameters tested. It is therefore important to more precisely establish national prevalence. For the individual flock it is clearly important to ensure that replacements are MV free. If these things can be done then there can be less concern for the implications of any trends towards larger more intensive flocks that might otherwise increase the risks from MV. However, if MV prevalence is increasing in replacement ewes from hill farms for the reason discussed above, then this may combine with increased risk from structural change on low ground farms until thresholds are exceeded and more MV epidemics appear in low ground flocks.

We have shown that in the absence of much empirical information or

complete understanding of MV it is still possible to use a simplified model of disease spread to explore priorities for prevention and control and to estimate the relative value of missing information. As more information becomes available, the model could be used to explore the economic impact of the disease as advocated by McInerney (1996). However, it is of course no substitute for scientific proof and our results are therefore put forward as an encouragement to further experimental research on this important disease.

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References

- Abbey, H., 1952. An examination of the Reed-Frost theory of epidemics. Human Biology 3, 201-233.
- Agrawal, R.C., Heady, E.O., 1972. Markov Chain Processes. Methods for Agricultural Decisions, 179-194.
- Bennett, R. M., 2003. The 'direct' costs of livestock disease: the development of a system of models for the analysis of 30 endemic livestock diseases in Great Britain. Journal of Agricultural Economics 54, 55-72.
- Bennett, R. M., Christiansen, K., Clifton-Hadley, R., 1999. Preliminary estimates of the direct costs associated with endemic diseases of

livestock in Great Britain. Preventive Veterinary Medicine 39, 155-171.

- Carpenter, T.E., 1988. Microcomputer programs for Markov and modified Markov Chain disease models. Preventive Veterinary Medicine 5, 169-179.
- Cutlip, R.C., Lehmkuhl, H.D., Brogden, K.A., Sacks, J.M., 1986. Breed Susceptibility to Ovine Progressive Pneumonia (Maedi-Visna) Virus. Veterinary Microbiology 12, 283-288.
- De Boer, G. F., Terpstra, C., Houwers, D. J., 1979. Studies in epidemiology of maedi/visna in sheep. Research in Veterinary Science 26, 202-208.
- Dohoo, I.R., Heaney, D.P., Stevenson, R.G., Samagh, B.S., Rhodes, C.S.,
 1987. The effects of Maedi-Visna virus infection on productivity in ewes. Preventative Veterinary Medicine 4, 471-484.
- European Union, 2005. The Common Agricultural Policy A policy evolving with the times. European Union, Brussels. http://europa.eu.int/comm/agriculture/public/capleaflet/cap_en.pdf
- Gunn, G.J, Stott, A.W, Humphry, R.W., 2004. Modelling and costing BVD outbreaks in beef herds. The Veterinary Journal 167, 143-149.
- Houwers, D.J., 1990. Economic importance, epidemiology and control In: Pètursson,G., and Hoft-Jorgensen,R. (Eds.), . Maedi-Visna and related diseases. Springer, Berlin, pp. 90-114.

Houwers, D.J., Van der Molen, E.J., 1987. A 5-Year serological study of

natural transmission of Maedi-Visna virus in a flock of sheep, completed with postmortem investigation. Journal of Veterinary Medicine Series B-Zentralblatt Fur Veterinarmedizin Reihe B-Infectious Diseases and Veterinary Public Health 34, 421-431.

- Keen, J.E., Hungerford, L.L., Littledike, E.T., Wittum, T.E., Kwang, J., 1997. Effect of ewe ovine lentivirus infection on ewe and lamb productivity. Preventive Veterinary Medicine, 30, 155-169.
- Lawes Agricultural Trust (Rothamsted Experimental Station), 2005. GenStat Release 8.1 (PC/Windows 2000).
- Markson, L.M., Spence, J.B., Dawson, M., 1983. Investigations of a flock heavily infected with Maedi-Visna virus. Veterinary Record 112, 267-271.
- McInerney, J., 1996. Old economics for new problems livestock disease: Presidential address. Journal of Agricultural Economics 47, 295-314.
- Milne, C.E., Gray, D., 1993. Maedi-Visna: The disease, its potential economic impact on the UK sheep industry and a cost:benefit appraisal of control strategies. SAC,.Edinburgh.
- Narayan, O., Cork, L.C., 1985. Lentiviral diseases of sheep and goats: Chronic pneumonia leukoencephalomyelitis and arthritis. Reviews of Infectious Diseases 7, 89-98.
- Pàlsson, P.A., 1976. Maedi-Visna in sheep. In: Kimberlin, R.H. (Ed.) Slow virus diseases of animals and man., North Holland Publishing Co.**

- Pekelder, J.J., Veenink, G.J., Akkermans, J.P.W.M., van Eldik, P., Elving, L., Houwers, D.J., 1994. Ovine lentivirus induced indurative lymphocytic mastitis and its effect on the growth of lambs. The Veterinary Record 134, 348-350.
- Pépin, M., Vitu, C., Russo, P., Mornex, J.F., Peterhans, E., 1998. Maedi-Visna virus infection in sheep: a review. Veterinary Research 29, 341367.
- Radostits, O. M., Gay, C. C., Blood, D. C., Hinchcliff, K. W., 2000. Diseases caused by viruses and chlamydia - II: Ovine Progressive Pneumonia (Maedi , Maedi-Visna). In Veterinary Medicine. W B Saunders, London.
- SAC, 2004. Farm Management Handbook 2004/5. L. Chadwick (Ed.), SAC, Edinburgh.
- Sihvonen, L., Hirvelä-Koski, V., Nuotio, L., Kokkonen, U.-M., 1999. Serological survey and epidemiological investigation of Maedi-Visna in sheep in Finland. Veterinary Microbiology 65, 265-279.
- Spence, J.B., Dawson, M., Markson, L.M., 1981. Maedi-Visna in Great-Britain. Veterinary Record 108, 466-466.
- Stott, A. W., Lloyd, J., Humphry, R. W., Gunn, G. J., 2003. A linear programming approach to estimate the economic impact of bovine viral diarrhoea (BVD) at the whole-farm level in Scotland. Preventive Veterinary Medicine 59, 51-66.

Van der Molen, E.J., Vecht, U., Houwers, D.J., 1985. A chronic indurative mastitis in sheep, associated with Maedi-Visna virus-infection. Veterinary Quarterly 7,112-119.

VLA, 2002. Surveillance report small ruminants. 6, 8.

Table 1: Assumptions used in the Markov chain model to reflect typical low ground flock performance based on SAC (2004)

| Assumption | Parity | | | | |
|-----------------------------------|--------|--------|--------|--------|--|
| | 1 | 2 | 3 | 4 | |
| Proportion of ewes | 0.27 | 0.26 | 0.24 | 0.23 | |
| Lambing rate (lambs/ewe) | 1.4 | 1.7 | 1.7 | 1.62 | |
| Ewe replacement rate | 0.04 | 0.08 | 0.04 | 1.00 | |
| Lamb mortality rate (dams | 0.0125 | 0.0125 | 0.0125 | 0.0125 | |
| uninfected) | | | | | |
| Ewe mortality from MV if infected | 0.0 | 0.0 | 0.3 | 0.5 | |
| Lamb mortality from infected dams | 0.05 | 0.05 | 0.05 | 0.05 | |

Table 2: Assumptions used in the Markov chain model to reflect typical hill flock performance based on SAC (2004)

| Assumption | | | Parity | | |
|-----------------------------------|-------|-------|--------|-------|--|
| | 1 | 2 | 3 | 4 | |
| Proportion of ewes | 0.28 | 0.26 | 0.24 | 0.22 | |
| Lambing rate (lambs/ewe) | 0.95 | 0.95 | 0.95 | 0.95 | |
| Ewe replacement rate | 0.07 | 0.08 | 0.08 | 1.00 | |
| Lamb mortality rate (dams | 0.032 | 0.032 | 0.032 | 0.032 | |
| uninfected) | | | | | |
| Ewe mortality from MV if infected | 0.0 | 0.0 | 0.3 | 0.5 | |
| Lamb mortality from infected dams | 0.1 | 0.1 | 0.1 | 0.1 | |
| | | | | | |

Table 3: Predicted impact (average prevalence of Maedi-Visna in a ten-year simulated epidemic) of flock size (n), probability of avoiding effective contact between infected and susceptible ewes (Q) and proportion of replacements infected (r) for an open low ground flock over the range of parameter values tested (all other parameters at their mean values)

| Range of parameter values | | | | | |
|---------------------------|---------|---------|------------|--------|--|
| Parameter | Minimum | Maximum | Difference | SE | |
| Ν | -0.02 | 0.28 | 0.30 | 0.0072 | |
| Q | 0.21 | 0.06 | -0.15 | 0.0071 | |
| R | 0.07 | 0.19 | 0.12 | 0.0074 | |

Table 4: Predicted impact (average prevalence of Maedi-Visna in a ten-year simulated epidemic) of flock size (n) and probability of avoiding effective contact between infected and susceptible ewes (Q) for a closed hill flock over the range of parameter values tested (all other parameters at their mean values)

| Range of parameter values | | | | | |
|---------------------------|---------|---------|------------|--------|--|
| Parameter | Minimum | Maximum | Difference | SE | |
| Ν | 0.24 | 0.46 | 0.22 | 0.0394 | |
| Q | 0.52 | 0.05 | -0.47 | 0.0390 | |

Figure 1: Distribution of sheep by holding size and region in Great Britain at the June Census 2004. Farms in the "Less Favoured Areas" (shaded bars), sheep in other areas (unshaded bars). Data supplied by Defra, Seerad and Welsh Office February 2006.

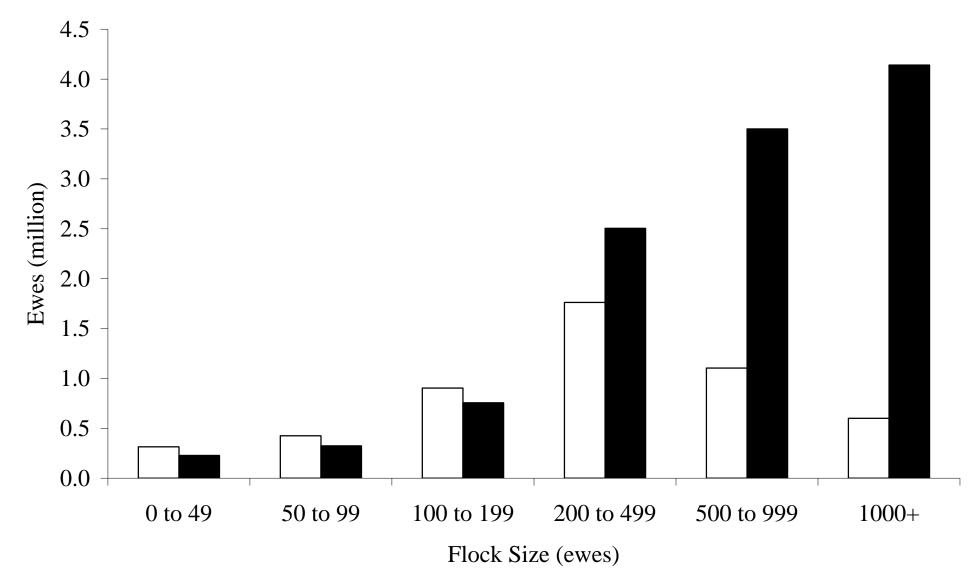


Figure 2: Comparison between the output of our low ground Markov chain model (light shaded bars) and the results of a serological study of natural transmission of Maedi-Visna virus in a flock of sheep by Houwers and Van der Molen (1987) (dark shaded bars).

