A Reed-Frost model of the spread of tuberculosis within seven Swedish extensive farmed fallow deer herds


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Abstract

The within-herd transmission of tuberculosis, after introduction of infection, was evaluated in seven Swedish herds of farmed fallow deer. The evaluation was based on a subset of data obtained from a previous epidemiological investigation, comprising 13 tuberculosis-infected deer herds, with the purpose of tracing the source of infection. A computer spreadsheet model based on the Reed-Frost method was developed to estimate the number of new infections. For each herd, a k-value (the number of effective contacts made by an individual during a time period) was estimated through fitting the model to the observed incidence in each herd. We concluded that, despite the relatively short observation periods and uncertain tuberculosis incidence estimates for the observed herds, the k’s obtained could be used to quantify the estimated spread of tuberculosis in extensive deer herds in Sweden.

1. Introduction

Tuberculosis was first diagnosed in Swedish farmed deer in 1991 after having been introduced with an imported consignment of 168 fallow deer (Dama dama) in 1987. A
total of 13 tuberculosis-infected herds have been identified (December 1997). Despite a comprehensive trace-back, all imported deer could not be traced and restrictions were imposed on all deer farms in Sweden in 1994. Movement of live animals from deer herds was allowed only from tuberculosis-free herds. A voluntary control program, aimed at declaring the Swedish deer population tuberculosis-free, was introduced in July 1994. Within the program, a herd can obtain tuberculosis-free status after three successive whole-herd tuberculin tests over a one to two year period. In addition, all animals that are killed or die of other causes have to be examined for tuberculosis by meat inspection or necropsy (Anonymous, 1994; Böliske et al., 1995). In order to maintain permission to move live animals from deer herds, all deer older than 1 year have to be identified with special ear tags and all does have to be tested biennially. By December 1997, 541 (96%) of Sweden’s 563 deer herds were affiliated with the control program. A total of 115 (20%) herds had obtained tuberculosis-free status. In addition, ≈100 (18%) herds have been depopulated and all deer subjected to meat inspection.

Deer farming in Sweden has traditionally been very extensive and individual handling of deer has not been necessary. Many Swedish deer enclosures are situated in forested and hilly areas, which do not facilitate mustering of deer for handling.

Since the program’s inception, it has become evident that it is difficult to muster all animals in certain large extensive herds. An alternative control strategy that doesn’t require individual handling of the deer may be needed for these herds. As slaughter does not require mustering of deer, meat inspection could be an alternative to the tuberculin test. In order to predict the efficacy of meat inspection as a tool to prove absence of tuberculosis at herd level, an estimate of the expected spread of tuberculosis in extensive herds is needed.

The aim of the present study was to model the within-herd spread of tuberculosis in seven Swedish deer herds and to estimate the number of effective contacts made by an individual during the time period of 1 year to be able to predict how tuberculosis would be expected to spread within an extensive Swedish deer herd.

2. The epidemiology of tuberculosis

Many species of animals can become infected with *Mycobacterium bovis*, but only few act as maintenance hosts (Morris et al., 1994). Among domesticated animals, cattle are the main reservoir, but in some countries farmed deer also act as a reservoir host (Morris et al., 1994). In the United States, transmission of tuberculosis from infected elk to cattle and also to bison herds was reported illustrating the potential role of captive cervidae in the epidemiology of tuberculosis in domestic livestock and even in wildlife (Essey, 1991).

Tuberculosis was first described in farmed deer in 1978 in New Zealand. In the UK, the disease was initially diagnosed in 1985 (Stuart et al., 1988), in Denmark in 1988 (Jørgensen et al., 1988), in Canada in 1990 (Whiting and Tessario, 1994) and in Sweden the first infected deer farm was identified in 1991 ( Böliske et al., 1995). In the United States, tuberculosis was first recognised as a problem in captive cervidae in late 1990 (Essey, 1992). All six countries have national control programs for tuberculosis in farmed deer. The programs are based on whole-herd tests with the intradermal tuberculin test.
In tuberculosis-infected deer, lesions are usually found in the retropharyngeal lymph nodes, lungs, lymph nodes of the thoracic cavity and mesenteric nodes (Livingstone, 1980; Jørgensen et al., 1988). In general, gross pathology in tuberculous deer is similar to that in cattle; however, thin-walled abscesses (usually found in the mesenteric lymph nodes) develop more-commonly in deer than in cattle (Clifton-Hadley and Wilesmith, 1991). The distribution of lesions suggests that the route of infection is both inhalation and ingestion in deer (Morris et al., 1994).

Confining animals and thereby increasing the population density may facilitate the spread of the disease (Clifton-Hadley and Wilesmith, 1991; Beatson et al., 1984; Towar et al., 1965; Robinson et al., 1989; Bode, 1995). In wild deer, where the population density is low, reports indicate that the prevalence of tuberculosis is <5%, whereas, in captive deer populations, higher prevalences have been reported (Clifton-Hadley and Wilesmith, 1991). Particular grazing patterns may also exacerbate a tuberculosis problem (Anonymous, 1986 cited by Clifton-Hadley and Wilesmith, 1991). Hawden (1942) found that elk had a higher prevalence of tuberculosis compared to mule deer (*Odocoileus hemionus*) and suggested it was due to elk running in herds rather than being isolated feeders.

Deer may be more susceptible to *M. bovis* than cattle (Morris et al., 1994). Deer also appear to be more infectious to other species than cattle are (Morris and Pfeiffer, 1995). There is epidemiological evidence from New Zealand indicating that farmed deer have infected previously negative possum populations. In contrast, evidence of cattle having infected possum populations is surprisingly scarce, given the far-greater number of infected cattle that have been in contact with possums (Morris and Pfeiffer, 1995).

3. Material and methods

3.1. Epidemiological investigations of 7 tuberculosis-infected farmed fallow deer herds

A total of 13 tuberculosis-infected fallow deer herds was identified in Sweden (between May 1991 and March 1997) and all have been depopulated. In all of these herds, direct or indirect contacts had occurred with some of the 168 imported fallow deer. During the epidemiological investigations in these herds, tuberculin tests and/or necropsies were performed with the purpose of tracing the source and/or spread of infection (Böltske et al., 1995). An individual deer was considered to be infected if *M. bovis* was isolated. Based on these results (Table 1), the numbers of introduced (as well as subsequently infected) deer could be estimated in seven of the 13 herds. In herds C and D, the incidence estimates were based on a whole-herd tuberculin test. In herds A, B, E, F and G (in which a limited number of animals were examined at depopulation) the total number of infected deer was unknown. Given the objective of the epidemiological investigation, most introduced infected deer (older deer) were examined but all subsequently infected deer (younger deer) were not. In these herds, the total number of subsequently infected deer had to be estimated. We assumed that the proportion of deer infected through within-herd spread in the examined deer was representative of the whole herd and thereby, the total number of subsequently infected deer in the whole herd was estimated.
Table 1
Findings in the epidemiologic investigation and estimated number of contacts in *M. Bovis*-infected deer herds in Sweden, 1991–1996

<table>
<thead>
<tr>
<th>Herd no.</th>
<th>Time (years) between introduction of infection and depopulation (or TB-test)</th>
<th>Type of examination</th>
<th>At depopulation</th>
<th>No. of <em>M. bovis</em> infected deer detected</th>
<th>Estimated no. of subsequently infected deer</th>
<th>No. of effective contacts per year (<em>k</em>)</th>
<th>Stocking rate</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No examined</td>
<td>Total herd size</td>
<td>Introduced</td>
<td>Subsequently infected</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>4.5</td>
<td>Post-mortem</td>
<td>109</td>
<td>500</td>
<td>7 <em>c</em></td>
<td>2 <em>c</em></td>
<td>9 <em>c</em></td>
</tr>
<tr>
<td>A</td>
<td>4</td>
<td>Post-mortem</td>
<td>100 <em>d</em></td>
<td>515</td>
<td>1</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>B</td>
<td>3</td>
<td>TB-test</td>
<td>70</td>
<td>70</td>
<td>1</td>
<td>0</td>
<td>n.d.</td>
</tr>
<tr>
<td>D</td>
<td>5.5</td>
<td>TB-test</td>
<td>24</td>
<td>24</td>
<td>1</td>
<td>0</td>
<td>n.d.</td>
</tr>
<tr>
<td>E</td>
<td>6.5</td>
<td>Post-mortem</td>
<td>8</td>
<td>16</td>
<td>1</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>F</td>
<td>6</td>
<td>Post-mortem</td>
<td>25</td>
<td>94</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>G</td>
<td>6</td>
<td>Post-mortem</td>
<td>51 <em>f</em></td>
<td>107</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

n.d. = Not done since all animals were examined.

*a* At depopulation.

*b* Purchased animals and their first calves.

*c* Two deer (date of infection unknown) assumed to be infected before introduction.

*d* Purchased animals (*n*=73) and born in the herd (*n*=27).

*e* All adult deer in the herd.

*f* All deer older than two years.
3.2. The model assumptions

A Reed-Frost model equation

\[ C_{t+1} = S_t \times (1 - q^{C_t}) \]  \hspace{1cm} (1)

was constructed, where \( t \) is the time period, \( C_{t+1} \) is the number of newly infected cases in time period \( t+1 \), \( S_t \) is the number of susceptible individuals in time period \( t \) and \( q = 1 - p \), \( p \) is the probability of one specific individual making effective contact with another given individual, in the specified time interval, which would result in an infection if one were infectious and the other susceptible (Abbey, 1952). The probability of an individual avoiding such a contact is \( q \).

\( p \) is calculated as

\[ p = k/(N - 1) \]  \hspace{1cm} (2)

where \( k \) is the number of effective contacts made by an individual during time period \( t \) and \( N \) is the population size.

The Reed-Frost model was the basis for our calculation of disease transmission (Abbey, 1952). In order to better simulate our situation some of the standard assumptions were modified. The following assumptions were made: (1) A time interval of one year, reflecting an average length of the incubation period was used. (2) After infection a deer would become infectious in the following time period. As there is no immune state (Blood and Radostitis, 1989), the deer will remain infectious for life. The total number of infectious animals in the next time period was, therefore, calculated as:

\[ CC_{t+1} = CC_t + C_{t+1} \]  \hspace{1cm} (3)

The Reed-Frost equation was subsequently formulated as

\[ C_{t+1} = S_t \times (1 - q^{CC_t}) \]  \hspace{1cm} (4)

where \( C_t \) is substituted by \( CC_t \), the total number of infectious animals in the previous time period. (3) The number of effective contacts \( (k) \) made by an individual during a time period was independent of herd size. (4) The population was allowed to increase over time. As information on the number of annually born/slaughtered animals was not available, these numbers were estimated based on the observed increase in herd size from time of infection to depopulation and herd size was assumed to increase linearly. (5) These assumptions remained constant over time. (6) Before tuberculosis was detected in the herd, it was assumed that only non-infected animals were slaughtered.

Information obtained from the 7 herds was incorporated into the Reed-Frost model (a simple deterministic model) using techniques described by Carpenter, 1984. To describe the within-herd incidence, a model was constructed as a spreadsheet matrix in Excel (Microsoft, Redmond, WA) (Table 2). It consisted of two states: infectious \( (I) \) and susceptible \( (S) \) and one probability: probability of effective contact \( (p) \). In addition, six other variables were included: population size \( (N) \), number of effective contacts \( (k) \), number of calves born \( (B) \), number of slaughtered animals \( (L) \), total number of infectious animals \( (CC) \) and newly infected animals \( (C) \) in period \( t \). The \( k \) that optimised the fit between the predicted and observed incidence in each herd, was obtained by using the Solver add-in in Excel. In herd A where two estimates of the within-herd spread had been obtained (Table 1), the model was run twice. In herds where no transmission of disease
Table 2
Equations used in the spreadsheet matrix

<table>
<thead>
<tr>
<th>Time period ($t$)</th>
<th>No. of newly infected deer per year ($C_t$)</th>
<th>No. slaughtered per year ($L_t$)</th>
<th>No. born per year ($B_t$)</th>
<th>Population size ($N_t$)</th>
<th>No. of susceptible individuals ($S_t$)</th>
<th>Probability of one specific individual making effective contact per year ($p_t$)</th>
<th>$q_t$</th>
<th>Total no. of infected deer ($CC_t$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$t$</td>
<td>$C_t=$ Introduced infected deer</td>
<td>0</td>
<td>0</td>
<td>$N_t$</td>
<td>$S_t$</td>
<td>$p_t = k/(N_t - 1)$</td>
<td>$q_t = 1 - p_t$</td>
<td>$CC_t=$ Introduced infected deer</td>
</tr>
<tr>
<td>$t+1$</td>
<td>$C_{t+1} = S_t \times (1 - q_t CC_t)$</td>
<td>$L_{t+1} =$</td>
<td>$B_{t+1} =$</td>
<td>$N_{t+1} =$</td>
<td>$S_{t+1} =$</td>
<td>$p_{t+1} =$</td>
<td>$q_{t+1} =$</td>
<td>$CC_{t+1} =$</td>
</tr>
<tr>
<td></td>
<td>$= N_t + B_{t+1} - L_{t+1}$</td>
<td>$= N_t + B_{t+1} - L_{t+1}$</td>
<td>$= N_t + B_{t+1} - L_{t+1}$</td>
<td>$= N_t + B_{t+1} - L_{t+1} + B_{t+1}$</td>
<td>$= k(N_{t+1} - 1)$</td>
<td>$= 1 - p_{t+1}$</td>
<td></td>
<td>$= CC_t + C_{t+1}$</td>
</tr>
</tbody>
</table>

$k =$ No. of effective contacts per year.
was observed, the \( k \) that corresponded to transmission of disease to one deer at the time of depopulation was calculated.

To evaluate the effect of imperfect tests on the obtained \( k \)-values, the true number of newly infected deer at depopulation was calculated in herds A, B and E (where spread of tuberculosis was found). The sensitivity of necropsy or tuberculin testing was assumed to be 0.7 (Anonymous, 1990; Jørgensen et al., 1988; Pers. comm. Schaap, P., 1997) and the specificity was set to 1 (as a deer was considered infected only if \( M. \) bovis was isolated). The true number of newly infected deer was calculated by using the formula (Thrusfield, 1986)

\[
\text{True prevalence} = \text{AP} + \text{Se} - 1/\text{Se} + \text{Sp} - 1
\]

where AP=apparent prevalence, Se=sensitivity and Sp=specificity.

The effect of increasing the lag period (the time from infection of an animal until it becomes infectious) was evaluated by assuming that newly infected deer (in time period \( t \)) would not become infectious until time period \( t+2 \).

4. Results and discussion

Results of the epidemiological investigations, incidence estimates and obtained \( k \)’s for the seven deer herds were summarised in Table 1. The \( k \)’s that were obtained when using estimates of true incidence and/or increasing the latency period were summarised in Table 3.

<table>
<thead>
<tr>
<th>Herd</th>
<th>Test sensitivity</th>
<th>Estimated no. of infected deer at depopulation</th>
<th>Estimate for ( k )</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>One year lag period</td>
</tr>
<tr>
<td>A(^d)</td>
<td>1</td>
<td>16</td>
<td>0.24</td>
</tr>
<tr>
<td>&quot;</td>
<td>0.7</td>
<td>20</td>
<td>0.32</td>
</tr>
<tr>
<td>A</td>
<td>1</td>
<td>23</td>
<td>0.49</td>
</tr>
<tr>
<td>&quot;</td>
<td>0.7</td>
<td>31</td>
<td>0.62</td>
</tr>
<tr>
<td>B</td>
<td>1</td>
<td>6</td>
<td>0.57</td>
</tr>
<tr>
<td>&quot;</td>
<td>0.7</td>
<td>8</td>
<td>0.69</td>
</tr>
<tr>
<td>C</td>
<td>1</td>
<td>2</td>
<td>&lt;0.26</td>
</tr>
<tr>
<td>D</td>
<td>1</td>
<td>2</td>
<td>&lt;0.15</td>
</tr>
<tr>
<td>E</td>
<td>1</td>
<td>9</td>
<td>0.60</td>
</tr>
<tr>
<td>&quot;</td>
<td>0.7</td>
<td>12</td>
<td>0.88</td>
</tr>
<tr>
<td>F</td>
<td>1</td>
<td>2</td>
<td>&lt;0.12</td>
</tr>
<tr>
<td>G</td>
<td>1</td>
<td>3</td>
<td>&lt;0.07</td>
</tr>
</tbody>
</table>

\(^a\) Given assumption that tuberculosis was spread to one deer (in herds C, D, F and G).

\(^b\) Deer infected in time period \( t \) become infectious in time period \( t+1 \).

\(^c\) Deer infected in time period \( t \) don’t become infectious until time period \( t+2 \).

\(^d\) Two deer with unknown date of infection assumed to be infected before introduction.
By assuming a test sensitivity of 0.7, the estimated $k$’s increased only slightly. As the calculation of true prevalence of newly infected deer was only relevant in the 3 herds where transmission of tuberculosis was observed (herd A, B and E) and as the changes in obtained $k$’s were small we decided to assume a test sensitivity of 1.0. The increase of the lag period from 1 to 2 years did have a greater effect on the obtained $k$’s (Table 3). Based on the limited information obtained from the seven herds it was not possible to estimate the optimal length of the lag period. We decided to use an incubation period of one year (with no additional lag period) which is similar to the lag period used by Barlow et al., 1997. This might be an underestimation as indicated in herd G where no spread of disease could be found more than 5 years after introduction of 2 infected deer. An underestimation of the lag period would however be compensated by a lower $k$-value.

The obtained $k$’s were low, indicating a slow spread of disease. In a Reed-Frost model of Johne’s disease in cattle, a $k$ (no. of effective cow–calf contacts per year) ≥ 1 was used (Collins and Morgan, 1991a). Barlow et al., 1997, modelled the within-herd spread of tuberculosis in intensively managed cattle herds and obtained an estimate of the number of potentially infectious contacts made per infectious cow per day of 0.0073. This equals approximately two potentially infectious contacts per infectious cow per year. When modelling tuberculosis in New Zealand red deer herds with the same model, a lag period of 6 months and 16 infectious contacts per year were used (pers. comm. Livingstone P., 1997). This is consistent with the opinion of Morris et al., 1994 and Morris and Pfeiffer, 1995 who states that deer seem to be more infectious and more susceptible compared to cattle. The reason for the observed difference in estimated number of infectious contacts between Swedish fallow deer herds and New Zealand red deer herds is unknown. Possible explanations are different management systems, stocking densities and/or difference in disease transmission among red and fallow deer (pers. comm. Livingstone P., 1997). Generally, deer farming in Sweden is less intensive compared to New Zealand.

Information from farmed fallow deer herds in New Zealand indicates that tuberculosis does not seem to spread as rapidly in those populations compared to reed deer herds (pers. comm. Livingstone P., 1997). Our findings are more consistent with the described within-herd spread of tuberculosis in cattle (Barlow et al., 1997).

For herd A two different values for $k$ (0.24 and 0.49) were obtained due to uncertainties in farm records and identification of two deer (Table 1). The highest $k$-value was obtained in herd E ($k=0.6$) where repeated confinement of deer to smaller areas for feeding occurred, as pasture in the deer enclosures was scarce. This is in accordance with Clifton-Hadley and Wilesmith, 1991 who report that particular grazing patterns can exacerbate a tuberculosis problem. In this respect herd E was not representative of a typical extensive deer herd, but as this type of management can occur in Swedish extensive deer herds, this herd was not excluded. In herds C, D, F and G (where no spread of infection was found for 3, 5.5, 6 and 6 years, respectively) the $k$’s were less than 0.26, 0.15, 0.12 and 0.07, respectively (Table 1). Since tuberculosis is a progressive disease, it is probable that the infected deer would have developed more extensive lesions if the observation period had been longer. In that case, the calculated probability of disease spread would be expected to increase.

No correlation was observed between how infectious the infected animal(s) was/were at depopulation and the value of $k$ (unpublished data). This might be due to the fact that
the expected increase in disease transmission from deer with generalised tuberculosis had not yet resulted in lesions detectable at post-mortem examination in newly infected deer. It seems probable that if depopulation had not been ordered, an increased prevalence of infection would have been expected especially in herds A and F where deer with generalised lesions were found at depopulation. The spread of tuberculosis did not seem to be higher in deer herds with higher stocking densities, probably because all stocking densities were low. Despite an increase in average population density to 4 deer/ha at the time of depopulation, the observed population densities were in the lower range of densities recommended in other countries (Reinken et al., 1990; Alexander and Buxton, 1994).

In most of the infected herds, farm records were incomplete and deficiencies occurred in identification of individual deer. Due to these deficiencies, only seven of the thirteen M. bovis-infected herds could be included in the model. In the 6 herds excluded, neither the number of introduced, nor the subsequently infected animals could be determined. To clarify if any systematic difference existed, the herds included in the model were compared with the excluded herds. The total numbers of infected deer at depopulation were equal in the two groups (\(\bar{x} = 3\) deer per herd) but the average proportion of examined deer that was infected was lower in the included (\(\bar{x} = 12\%\)) than in the excluded herds (\(\bar{x} = 22\%\)). This difference was mainly due to 2 excluded herds: herd 1 where management factors probably caused a high prevalence, and herd 2 where many infected deer were introduced (unpublished data). The mean area of the deer enclosures were larger in the included (44 ha) than in the excluded herds (19 ha) and the mean stocking density at depopulation were lower (\(\bar{x} = 4\) deer/ha and \(\bar{x} = 8\) deer/ha, respectively). The differences were mainly due to herd 1 (unpublished data). We concluded that despite existing differences no selection bias was introduced by selecting only 7 of the 13 herds.

The estimates of tuberculosis incidence were based on a whole-herd tuberculin test in herds C and D (Table 1). Despite the fact that only one test was performed before depopulation, we concluded that spread of infection had not occurred. This was based on the facts that the infected deer only had small tuberculous lesions in the lymph nodes (submandibular lymphnode in herd C and mesenteric lymphnodes in herd D) and that the epidemiological investigation indicated that these deer were the sources of infection. In the remaining herds, the estimates of tuberculosis incidence were based on post-mortem examinations. Animals born in the herd (younger deer) were less likely to be necropsied, as the purpose of the investigations was to identify all introduced infected deer (the source of infection). Although examined deer were not selected at random, the observed prevalence of subsequently infected deer among the examined animals was assumed to be representative of the whole herd.

As (non-examined) young animals had a shorter time of exposure than older animals, their probability of becoming infected might be lower (Whiting and Tessario, 1994) indicating that our estimate of \(k\) might be too high. However, should the spread of infection be more frequent between doe and fawn, \(k\) might be too low. The incidence of subsequently infected animals may also be underestimated by the fact that the newly infected animals probably have fewer and smaller lesions that are more easily missed at meat inspection.
Several authors have described modelling of tuberculosis infections. In New Zealand, modelling has contributed importantly to understanding and controlling tuberculosis in brushtail possums (*Trichosurus vulpecula*), which are a reservoir of tuberculosis for cattle (Barlow, 1994). Modelling has also focused on the dynamics of tuberculosis in badger (*Meles meles*) populations (mainly in England) and their role as reservoir hosts (Bentil and Murray, 1993; Clifton-Hadley and Wilesmith, 1991; White and Harris, 1995a, b). Only recently a model of the within-herd spread of tuberculosis in cattle has been published (Barlow et al., 1997). Models describing the agent–host–environment complex including farmed deer are more scarce (Ryan, 1995; Ryan et al., 1995) and, to our knowledge, no estimate of within-herd spread of infection in farmed deer herds has been published.

Deer farming in Sweden is different from that seen in the above mentioned countries as animal movements from deer herds are prohibited, deer farming is usually more extensive, the major part of deer herds consists of fallow deer, no known wildlife reservoirs exist, and the cattle population is free from tuberculosis. Thus, none of the previous models were applicable to the current Swedish situation and a simpler model describing only the within-herd spread of infection, was preferred.

In the present study, a simple Reed-Frost model was chosen to describe the spread of infection, as it is usually preferable to start with a simple model and only build a more complex one if necessary (Martin et al., 1987). This model has earlier been used to simulate mycobacterial infections (*M. Paratuberculosis*) in cattle (Collins and Morgan, 1991a, b).

Our model differed from the classical Reed-Frost model principally in two ways: (i) Infectiousness was lifelong. Similar modifications have been made in modelling paratuberculosis (Collins and Morgan, 1991a) and brucellosis (Carpenter et al., 1987). (ii) The time period was not the average incubation period. The time period from the infected but non-infectious status to the infectious status varies greatly (Blood and Radostitis, 1989). In cattle, the dose of infection is inversely related to the delay before excretion begins (Neill et al., 1991). The time interval is also affected by the susceptibility of the host (Bruner and Gillespie, 1973) and by environmental factors (Blood and Radostitis, 1989). In natural bovine infections occurring via the respiratory route, *M. bovis* can be detected in nasal mucus as early as 80–100 days after infection. Griffin and Dolan, 1995 concluded that cattle can remain free from tuberculosis for prolonged periods even though they have been in contact with tuberculous cattle. Similar findings have been reported from Australia, where it was believed that an infected cow had been resident in a herd for nearly 17 years and, when detected at post-mortem, she had only recently spread the infection to other cattle in the herd (Tolson and Jervois, 1990). Under experimental conditions the duration of the incubation period in cattle was found to lie within the range of 87–226 days (Neill et al., 1992), a realistic range for most New Zealand cattle was estimated to be 6–20 months (Barlow et al., 1997) and in naturally infected Australian range cattle it was calculated to be approximately 7 years (Stoneham and Johnstone, 1986 cited by Barlow et al., 1997). Deer appear to transmit the disease more readily than cattle (Morris et al., 1994). Some animals may be severely affected within 6 months, while others may survive without overt signs for several years (Fedoseev et al., 1982, cited by Clifton-Hadley and Wilesmith, 1991; Williams, 1987...
cited by Clifton-Hadley and Wilesmith, 1991). In the present study, no spread of infection was found during more than 5 years in herds D and G or during 3 years in herd C. In herd F, no spread was detected after 6 years, but since <25% of the animals were necropsied this figure is less reliable (Table 1). The choice of the one-year incubation period to drive the model was considered to be appropriate, since (i) the time period from the infected but non-infectious status to the infectious status is very variable, (ii) a time period of one year was used when modelling Johne’s disease (Collins and Morgan, 1991a) and (iii) a lag period of approximately one year was used when modelling tuberculosis in cattle (Barlow et al., 1997).

In the present study, \( k \) (the number of effective contacts made by an individual during a time period) was evaluated. In contrast to the classical Reed-Frost model (Abbey, 1952), where herd size is constant, the investigated deer herds increased in herd size over time. Similar modifications have been made by others (Collins and Morgan, 1991b). Since \( k \) was assumed to be independent of herd size it remained stable. However, in herds where the increase in population size results in a high population density (as seen in one excluded herd: Herd 1 with 22 deer/ha) it may be expected that \( k \) would increase and under those circumstances this assumption may not be valid.

We concluded that despite the uncertainties in the estimation of the incidence of tuberculosis in the observed herds, the obtained \( k \)’s can be used to predict how tuberculosis would be expected to spread in Swedish extensive deer herds. Based on this prediction, it may be possible to estimate the efficacy of meat inspection, as a tool to prove absence of tuberculosis at the herd level. However, caution should be observed in drawing inferences for longer time periods than \( \approx 5–6 \) years after the introduction of the disease, since such periods were outside the range of observations in the present study.

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References


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