**Salmonella Dublin infection in young dairy calves: Transmission parameters estimated from field data and an SIR-model**

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**Abstract**

In this study we used field data collected from October 2001 to January 2002 to estimate number of days of faecal excretion of *Salmonella* Dublin bacteria and time to seroconversion in infected calves below the age of 180 days. Based on these estimates all calves in four endemically infected dairy herds were grouped into the following infection states: susceptible (S), infectious (I) and resistant/recovered (R). Resistant calves had either acquired maternal antibodies through colostrum or they have recovered from previous infection and had a high level of antibodies directed against *Salmonella* Dublin possibly protecting them from becoming infected again until the level of antibodies had decreased to sufficiently low levels. Using the antibody measurements and faecal excretion periods, it was possible to assign the most likely infection state to each calf per week of the study period.

Estimates of transmission parameter, \(\beta\), were obtained from a generalised linear model relating the number of new infections to the proportion of susceptible and infectious calves per week. From \(\beta\), the reproduction ratio \(R\) at steady state and the basic reproduction ratio \(R_0\) were estimated for each herd and across herds. The \(R_0\) denotes the average number of new infections caused by one infectious individual that is introduced to a fully susceptible population. The point estimates for \(R_0\) ranged from 1.1 to 2.7 in the study herds. However, the confidence intervals were wide. Data were too limited to show possible significant differences in the parameters between the study herds. However, the tendency in the data suggested that there may be important differences. Across herds the \(R_0\) was close to two suggesting that on average one infectious calf will produce two new infectious calves when introduced into a fully susceptible population under typical Danish dairy production systems. Further,
the analyses indicated that environmental contamination from infectious calves plays an important role in transmitting *Salmonella* Dublin between calves.

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**Keywords:** *Salmonella* Dublin; Transmission; SIR; Calves

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### 1. Introduction

*Salmonella* Dublin is a cause of concern in the cattle industry, because it is a zoonosis causing severe invasive infections in humans and because it causes economic and welfare losses in infected herds (Peters, 1985; Helms et al., 2003). The infection has a tendency to become endemic in many cattle herds in Denmark. When attempting to control *Salmonella* Dublin infections in such dairy herds it is critical to intervene in the calf barn where the infection spreads readily. However, not much is known about the infection dynamics of *Salmonella* Dublin in calf barns of endemically infected herds, because most information comes from outbreak situations and clinical cases (Richardson and Watson, 1971; Wray et al., 1989). Knowledge about the basic reproduction number, $R_0$, is useful for modelling the infection and the effect of potential intervention strategies. The net reproduction number, $R$, at steady state is one, meaning that on average every individual that becomes infected succeeds in transmitting the infection to one other individual during its infectious period (Anderson and May, 1991, p. 17). However, $R_0$ must be above one for any endemically stable disease, meaning that when one infectious animal is introduced into a fully susceptible population on average more than one animal will become infected and thus outbreaks may also occur. In endemically infected herds, the proportion of susceptible animals varies over time. Thus, the infection may die out, or a new outbreak may occur. The size of the outbreak is mainly related to the number of susceptible individuals in the herd (Anderson and May, 1991, pp. 68–69). This is supported by varying clinical signs over time and fluctuating seroprevalence of *Salmonella* Dublin in infected herds that makes it reasonable to assume that even in endemically infected herds, smaller outbreaks are occurring intermittently over time.

The aims of the study were to (1) estimate length of the infectious periods and serological response to infection in calves below 180 days of age from field data, (2) illustrate fluctuations in size of the infection states $S$ (susceptible), $I$ (infectious) and $R$ (recovered/resistant) over time and (3) to estimate the transmission parameters, $\beta$, $R$ and $R_0$ for *Salmonella* Dublin among young calves (<180 days old) in four Danish dairy herds with long-term infection on the premises.

### 2. Materials and methods

#### 2.1. Study herds and sampling

The estimates were obtained by the use of field data collected in Denmark in 2001–2002 and a generalised linear model relating the number of new infections to the proportion of
susceptible and infectious calves per week. The data was collected as part of a large project known as “the Kongeåuproject” through which previous knowledge of the four study herds was gathered (Andersen et al., 2000). These four herds were included in the study because they had several Salmonella Dublin positive cultures over a period of at least 1 year. They were therefore considered endemically infected with the bacteria. Clinical signs of salmonellosis were not obvious in these herds before the study period began. The sample collection was organised so that all calves that were born in the study period (a total of 88 calves) were sampled every 3–4 days for the first 4 weeks after birth and then once per week. All neighbouring calves in the same barn areas were sampled once per week. In total 181 calves were sampled in the study period. The number of calves varied between 16 and 69 per herd. Calves were sampled between 1 and 24 times each, on average 9.4 (S.D. = 7.2) times. Every sample event involved collection of an un-stabilised blood sample from the jugular vein and a rectally collected faecal sample. It was attempted to collect a minimum of 25 g of faecal matter at each sampling. However, this often proved difficult in the very young calves. Blood samples were transported to the Veterinary Department of Steins Laboratory in Ladelund for detection of antibodies directed against Salmonella Dublin lipopolysaccharide (LPS) as described below.

2.2. Bacteriology

Faecal samples were cultured in the above-mentioned laboratory for presence of salmonella bacteria by a conventional method described and evaluated elsewhere (Nielsen et al., 2004). The sensitivity of the faecal culture method has been estimated to be between 6% and 32% depending on the age of the animal when pooling of samples was used before individual follow-up on positive pools. In the present study, all faecal samples were cultured individually and the calves were very young, so the sensitivity was close to the highest obtainable, probably around 25–50% (Richardson and Fawcett, 1973). The specificity was assumed to be 100%, as typing of all salmonella-positive isolates was performed at the Institute for Food and Veterinary Research in Copenhagen.

2.3. ELISA

Blood samples were analysed for presence of antibodies directed against Salmonella Dublin O-antigen based LPS using an enzyme-linked immunosorbent assay (ELISA) that has been described in detail and evaluated elsewhere (Nielsen and Erbsøll, 2004; Nielsen et al., 2004). An ODC%-value, which is a background corrected ratio of the test sample optical density (OD) to a known positive reference sample, was calculated for each sample as follows:

\[
\text{ODC\%} = \frac{(\text{OD}_{\text{sample}} - \text{OD}_{\text{neg ref}})}{(\text{OD}_{\text{pos ref}} - \text{OD}_{\text{neg ref}})} \times 100
\]

where \(\text{OD}_{\text{sample}}\) is the mean value of two test wells, \(\text{OD}_{\text{neg ref}}\) and \(\text{OD}_{\text{pos ref}}\) are the mean values of four reference wells in the ELISA plates, respectively. The sensitivity of the serum ELISA at the cut-off value used in the present study (25 ODC%) was approximately 40–46% and the specificity 89–98% for animals between 0 and 99 days of age (Nielsen and
For calves from 100 days and older, the sensitivity was estimated to be 82–85% and the specificity 88–97%. The reason for the low sensitivity in calves younger than 11–12 weeks of age is most likely due to a poor capability to produce antibodies by this age group of calves. This was documented in another study and had to be taken into account when determining the infection states of the calves in our study (Da Roden et al., 1992). The non-optimal specificity may be due to maternal antibodies in this age group. Seroconversion was defined as at least a doubling of the ODC% to above 30 between two sample events. These criteria were based on a mix of results from previous studies and practical experience with the ELISA (Robertsson et al., 1982; Nielsen, 2003).

2.4. Infection status of the calves

To analyse data for transmission parameters, the infection status susceptible (S), infectious (I) and recovered/resistant (R) of all calves was determined for every week of the sampling period by both faecal shedding and by serology. In the absence of reasonable sensitivity of the bacteriological culture method, serology offers another way to determine the infection status (Veling et al., 2000).

Calves were given status S when there was no bacterial growth in the faecal samples and the ODC% was below 25. Status I was assigned from the day that calves had a positive bacteriological culture and 17 days onwards. This average period was estimated from the data from culture positive calves (see Section 3). Additionally, calves were assigned status I based on seroconversion. The infectious period was set to start 36 days prior to the recorded date of seroconversion and 17 days onwards from that date, if the calf was below the age of 100 days at time of seroconversion.

Seroconversion in calves older than 100 days lead the infectious period to be estimated to begin 14 days prior to seroconversion and the infectious period would be set to be shorter (12 days). Status I was followed by status R for 14 days unless new infection occurred within those 14 days. In that case, the calf was defined to be continuously infectious. Status R was also assigned to calves that had an ODC% above 25 and were not culture positive. This could for example be newborn calves with maternally derived antibodies or calves that continued to have high antibody levels beyond the designated 14 days recovered period following an infectious period.

Because calves older than 1 month were sampled on a weekly basis, the time step for the analyses was a week. Therefore, calves that were sampled twice weekly were assigned the same status for the whole week. When calves became infectious (changed from S to I) and when they recovered (went from I to R), the whole week was assigned I. This aggregation of data into weekly steps changed the minimum infectious period from 12 or 17 days to 3 weeks in the model. When calves were losing their maternal immunity (went from R to S), the whole week was defined S. New infections were defined each time a calf became infectious after a susceptible period. Examples of infection groups for two calves are shown in Fig. 1A and B.

2.5. Statistical analysis

To estimate the transmission parameter, $\beta$, we used the framework of a simple SIR-model for transmission of Salmonella Dublin between calves. The model is illustrated in
Homogeneous mixing of the calves was assumed. Calves were considered born into either the S or the R compartment depending on whether they received Salmonella-specific antibodies through colostrum. After an infectious period, calves were considered resistant for at least 14 days or until their antibody levels fell below the cut-off value of 25 ODC%.

New infections were assumed to occur at the rate $\beta((S/I)/N + E)$, where $\beta$ is the infection rate, $S$ the number of susceptible individuals, $I$ the number of infectious individuals, $E$ an
external environmental infectious component and $N$ is the total number of animals present in the given time period (Geenen et al., 2005). According to this model, the number of new infections, $C$, in each time interval, $\Delta t$, was assumed to be Poisson distributed and had the following expected value ($e(C)$):

$$e(C) = \frac{\beta}{N} \left( \frac{SI}{N} + E \right) \Delta t$$

$log(\beta)$ was estimated with a generalised linear model (GLM) using the Genmod procedure in SAS$^{\text{1}}$, Version 9.1 (SAS Institute Inc., 2002) with the response variable $C$, log($SI/N + E$) as offset (with $\Delta t$ being 1 week) and a log link function. The external component ($E$) was added to correct for potential infection from the environment of the calves when no infectious calves were present. Because $E$ was unknown, several levels of $E$ from very low (0.001) to high (0.2) was tested in the model to check model fit to the data and to evaluate the effect of the size of $E$ on the parameter estimates. To estimate the 95% confidence interval for $log(\beta)$, the standard error (S.E.) was calculated as the two-sided confidence coefficient assuming a normal distribution and multiplied by the standard error from the model: $log(\beta) \pm 1.96 \times$ S.E. The overdispersion parameter was estimated from the scaled deviance statistics (McCullagh and Nelder, 1989). The overdispersion parameter allows for possible dependence between grouped animals. Also, from a more practical point of view, it ensures that any lack-of-fit that remains after careful inspection and possible modification of the model, is reflected by larger standard errors and more conservative inference. An overdispersion parameter close to one indicates that the data follow a Poisson distribution.

The basic reproduction ratio ($R_0$) is the average number of secondary cases per week produced by one infected individual during the entire infectious period (Diekmann et al., 1990). $R_0$ was estimated by the following formula:

$$R_0 = \frac{\beta}{\gamma}$$

where $\gamma$ is the recovery rate and $1/\gamma$ is the estimated average infectious period in weeks which was estimated from the field data.
Another approximate method used to estimate the $R_0$ for *Salmonella* Dublin in the study herds was used to check the influence of the external component ($E$) on the above estimates. This method assumes that at equilibrium an approximation of the $R_0$ is related to the proportion of susceptible individuals in the population: $R_0 = 1/(S/N)$. The proportion of susceptible individuals was calculated as the average proportion of susceptible individuals over the entire study period. The net reproduction number $R$ was calculated as $R_0$ multiplied with the proportion of susceptible calves (Anderson and May, 1991, p. 17). This method would only apply if the $E$-component was low.

3. Results

3.1. Time of infectiousness and seroconversion

Based on the laboratory results of 19 calves that shed *Salmonella* Dublin in the study period, the average time of infectiousness (shedding of bacteria) was estimated to be 17 days (range 3–68 days) and the average time from onset of shedding to seroconversion in calves in this age group was estimated to be 36 days (range 11–67 days) (Table 1). For the model this resulted in a minimum infectious period of 3 weeks due to aggregation of data into weekly time steps. New cases appeared to arise in seven out of 16 (44%) weeks with no infectious animals in the previous week as opposed to 11 out of 48 (23%) weeks with infectious animals in the previous week.

3.2. Transmission parameters

Table 2 contains the results of the log-linear regression for the four herds as fixed effects with $E$ set to 0.1 which produced reasonable model fit, and Table 3 contains the estimate across all four herds with a correction for repeated observations within herd for different levels of $E$. The log-linear model with the four herds as fixed effects was overdispersed (deviance/d.f. = 2.4). The average infectious period in weeks that was used to estimate $R_0$ was 3 weeks.

Though the estimates appeared to vary between the herds, the confidence limits were wide and therefore significant difference between herds was not demonstrated. Fig. 3a–d illustrates the fluctuations in the size of the different infection states $S, I, R$ and total number of calves ($N$) per week of the study period according to the data and the definitions. Herd

Table 1
Descriptive statistics for 19 calves ($N$) that were faecal culture positive for *Salmonella* Dublin in four endemically infected Danish dairy herds

<table>
<thead>
<tr>
<th>Variables</th>
<th>$N$</th>
<th>Mean</th>
<th>S.D.</th>
<th>Median</th>
<th>Min–max</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at start of infectious period (days)</td>
<td>19</td>
<td>40</td>
<td>23</td>
<td>43</td>
<td>3–70</td>
</tr>
<tr>
<td>Infectious period (days)</td>
<td>19</td>
<td>17</td>
<td>19</td>
<td>10</td>
<td>3–68</td>
</tr>
<tr>
<td>Age at seroconversion (days)</td>
<td>10</td>
<td>75</td>
<td>15</td>
<td>76</td>
<td>52–100</td>
</tr>
<tr>
<td>Time from start of shedding to seroconversion</td>
<td>10</td>
<td>36</td>
<td>17</td>
<td>28</td>
<td>11–67</td>
</tr>
</tbody>
</table>

Nine animals that excreted bacteria did not show seroconversion in the study period.
two had a peak in infections, which can be considered a small outbreak among the calves in weeks 5–8. This was reflected in the $R_0$ estimate for this herd. Herd four appeared to have experienced a similar outbreak in weeks 10–12, but the herd was very small and thus there were only few observations available for the model estimations resulting in a very wide confidence interval. Across herds, the $R_0$ estimate of *Salmonella* Dublin was significantly higher than one for all tested values of $E$, indicating that upon introduction to a fully susceptible calf population an infectious calf would on average infect approximately two other calves and therefore be likely to cause an outbreak.

The estimates from the approximate method of $R_0$ calculation were similar ($R_0 = 1.8–2.7$) (Table 4) to the $R_0$ estimates from the model ($R_0 = 1.1–2.7$) (Tables 2 and 3).

### 4. Discussion

The data set was unique in that all young calves in four herds were sampled at least once a week for 12 weeks. The data were used to estimate the transmission rate of *Salmonella* Dublin based on the new cases in each time period in young calves. However, the data only covered calves up to 180 days of age. It would be preferable to be able to include several age groups or the entire herd. Such data collection is extremely time-consuming and expensive, in particular if bacteriological culture needs to be performed on all samples. Because ELISA measurements do not give a very good indication of whether an animal is infectious, recovering from infection or a latent carrier, bacteriological culture is needed for this type of study (House et al., 1993; Hoorfar et al., 1996; Veling et al., 2000). On the

### Table 2

Estimated transmission parameter ($\beta$), standard error (S.E.) and basic reproduction number ($R_0$) for *Salmonella* Dublin in young calves in four Danish dairy herds based on an average infectious period of 3 weeks and the risk posed by environment contamination fixed at 0.1

<table>
<thead>
<tr>
<th>Herd</th>
<th>$\log(\beta)$</th>
<th>S.E.</th>
<th>$R_0$</th>
<th>95% CI of $R_0$</th>
<th>Proportion $S$ (%)</th>
<th>$R$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>−1.00</td>
<td>0.90</td>
<td>1.1</td>
<td>0.2–6.4</td>
<td>55</td>
<td>0.6</td>
</tr>
<tr>
<td>2</td>
<td>−0.13</td>
<td>0.45</td>
<td>2.6</td>
<td>1.1–6.3</td>
<td>37</td>
<td>0.9</td>
</tr>
<tr>
<td>3</td>
<td>−0.51</td>
<td>0.63</td>
<td>1.8</td>
<td>0.5–6.3</td>
<td>54</td>
<td>0.9</td>
</tr>
<tr>
<td>4</td>
<td>−0.11</td>
<td>0.59</td>
<td>2.7</td>
<td>0.9–8.5</td>
<td>52</td>
<td>1.3</td>
</tr>
</tbody>
</table>

Proportion of susceptible animals ($S$) and the net reproduction number ($R$) over the study period.

### Table 3

Estimated transmission parameter ($\beta$), standard error (S.E.) and basic reproduction number ($R_0$) and model fit evaluation (log likelihood) for *Salmonella* Dublin in young calves across four Danish dairy herds based on an average infectious period of 3 weeks at different levels of risk posed by environmental contamination as opposed to transmission by direct contact ($E$)

<table>
<thead>
<tr>
<th>$E$</th>
<th>$\log(\beta)$</th>
<th>S.E.</th>
<th>$R_0$</th>
<th>95% CI of $R_0$</th>
<th>log likelihood</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.001</td>
<td>−0.21</td>
<td>0.13</td>
<td>2.4</td>
<td>1.9–3.1</td>
<td>−27.4</td>
</tr>
<tr>
<td>0.01</td>
<td>−0.21</td>
<td>0.13</td>
<td>2.4</td>
<td>1.9–3.2</td>
<td>−27.9</td>
</tr>
<tr>
<td>0.1</td>
<td>−0.31</td>
<td>0.14</td>
<td>2.2</td>
<td>1.7–2.9</td>
<td>−28.7</td>
</tr>
<tr>
<td>0.2</td>
<td>−0.44</td>
<td>0.15</td>
<td>1.9</td>
<td>1.4–2.6</td>
<td>−29.1</td>
</tr>
</tbody>
</table>
Fig. 3. The dynamics of the size of infection groups $S$, $I$, $R$ and the total number of calves, $N$, in the population per week of the study period in four dairy herds. The large fluctuation in $N$ is due to bull calves being sold from the herds around 2 weeks of age and movement of calves in groups between barn areas.
Fig. 3. (Continued).
other hand, it is known that conventional bacteriological culture also lacks sensitivity in cattle faecal samples and there is large variation in the duration of excretion of bacteria between individual calves (as illustrated by our data in Table 1). Correct classification into infection states is therefore difficult to obtain for this infection (Richardson and Fawcett, 1973; Nielsen et al., 2004). Thus, we may have underestimated both the number of infectious calves in each time step and the number of new infections, which again could affect the $R_0$ estimates.

For optimal estimation of transmission parameters, the time between each sampling should preferably be as short as the average generation interval, i.e. the time from one animal becomes infectious to the time the second case infected by the first case becomes infectious. For *Salmonella* Dublin the generation interval is probably only between 3 and 7 days, which is why at least weekly samples are required if the estimations are based on field data. Infection rates, duration of infection and recovery rates could possibly be estimated using a Bayesian model though the data used here may be too limited to improve the posterior estimates. For the present model we have mainly used the serological changes over time and faecal culture results rather than single ELISA results. Therefore, adjusting for sensitivity and specificity is not easily done. In a Bayesian model knowledge about test accuracy could be included.

The Poisson model is only an approximation to the real transmission dynamics. In particular, when the number of susceptible animals is small and the infection intensity high, then the expected number given by the model will overestimate the true expected number in the next time step. In this study this is unlikely to affect the estimates, because the expected number of new cases was never high, and the number of susceptible animals rarely low (Fig. 3).

The $R_0$ estimates around two indicated that *Salmonella* Dublin would not spread very rapidly through susceptible populations under management systems similar to the ones in these herds. This makes sense because *Salmonella* Dublin is an infection that primarily spreads via the faecal-to-oral route and under typical Danish dairy herd conditions young calves with individual housing up to about 6–8 weeks of age, do not necessarily have a lot of direct contact between many neighbouring calves. On the other hand, direct contact between calves may not be the only factor leading to transmission of infection. High contamination of the environment by infectious calves and to some extend adult cows may also lead to transmission. In a simulation model it would be possible to allow the contamination level of the environment to depend on the number of infectious animals in the barn area in the previous time steps.

The number of calves per herd was too small to determine the differences between herds but there was an indication that in some herds *Salmonella* Dublin may spread faster than in

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**Table 4**

Approximation of the basic reproduction number ($R_0$) for *Salmonella* Dublin in young calves based on the average proportion of susceptible ($S/N$) individuals in a 12–16 study period in four Danish dairy herds

<table>
<thead>
<tr>
<th>Variables</th>
<th>$S/N$</th>
<th>S.D.</th>
<th>$N$</th>
<th>$R_0$</th>
<th>95% CI of $R_0$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Herd 1</td>
<td>0.55</td>
<td>0.18</td>
<td>14</td>
<td>1.8</td>
<td>1.6–2.2</td>
</tr>
<tr>
<td>Herd 2</td>
<td>0.37</td>
<td>0.09</td>
<td>16</td>
<td>2.7</td>
<td>2.4–3.1</td>
</tr>
<tr>
<td>Herd 3</td>
<td>0.54</td>
<td>0.11</td>
<td>16</td>
<td>1.9</td>
<td>1.7–2.1</td>
</tr>
<tr>
<td>Herd 4</td>
<td>0.52</td>
<td>0.24</td>
<td>11</td>
<td>1.9</td>
<td>1.5–2.6</td>
</tr>
<tr>
<td>Across herds</td>
<td>0.49</td>
<td>0.17</td>
<td>57</td>
<td>2.0</td>
<td>1.9–2.2</td>
</tr>
</tbody>
</table>
and that small outbreaks occurred during some time periods. This could be due to hygienic conditions in the herds, housing and management of the calves. Earlier studies on risk factors for the spread of *Salmonella* Dublin confirm that herd management, but also co-infections with other diseases such as BVD aggravate an outbreak (Wray and Roeder, 1987; Veling et al., 2002).

The point estimate for the reproduction ratio at equilibrium (*R*) was between 0.6 and 1.3 which was expected, because the herds were infected with *Salmonella* Dublin for several years and thus were in an endemic situation. However, under endemic situations there may be fluctuations in the proportion of susceptible animals leading the net reproduction ratio to increase, meaning that the transmission of bacteria between animals has increased periodically, whereas during other periods the herd immunity level would be sufficiently high that no or very little transmission of bacteria would occur (Anderson and May, 1991).

The model fit to the data was not optimal. The model was overdispersed, which indicated that there was more variation in the number of new infections than expected and the standard errors of the transmission rate had to be inflated to correct for this effect. The poor fit was probably a result of the fact that at times there were no infectious calves in the herd but new cases did occur (Fig. 3). Therefore, we included an external component (*E*) in the model, and the model fit did in fact improve with increased levels of environmental contamination indicating that this is an essential source of new infections. This suggests that *E* needs to be included in the model, however it is quite likely that the environmental contamination came from calves that where infectious not long before the weeks with no infectious animals present and that the bacteria survived in the environment. Thus, it is likely to be highly dependent on the number of infectious calves in the previous time steps. It is advisable to explore the effect of such an environmental component by a simulation model in which this component is allowed to vary stochastically or dependent on the number of infectious animals in the previous time steps.

Another source of biased *R*₀ results is that we may have misclassified some calves as non-infectious though they were in fact shedding bacteria. Few studies were available to aid in defining the infectious periods and recovery rates, and the main study available was based on clinical experiments, but confirmed the time of infectiousness in our study (Robertsson, 1984). Also, it must be expected that the individual variation of infectiousness is large. Such individual variation in length of infectious periods and time of onset of infectiousness was not fully included in the analyses.

The next steps will be to include the parameters in a stochastic simulation model, in which the heterogeneity among calves and the infectiousness of other age groups in the herd can be included. The external component could be an environmental compartment related to the number of infectious animals in the lactating cows and survival of the bacteria in the environment (Wray et al., 1989). However, this poses even higher demands on the data sources available.

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