# STUDYING POPULATION DYNAMICS OF UDDER INFECTIONS IN DAIRY HERDS

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Les dynamiques des populations de germes lors d'infections de la mamelle par des germes pathogènes mineurs ont été étudiées dans sept troupeaux laitiers pour discuter des études de ces infections dans des conditions de terrain. La contagiosité des infections intramammaires a été définie comme le ratio de propagation de base,  $R_o$ , calculé à partir du ratio du taux de transmission b et du taux de guérisons a des infections.  $R_o$  peut être interprété comme étant le nombre de nouvelles infections par quartier infecté, dans un troupeau sain. Des différences notables ont été observées entre troupeaux. Le ratio  $R_o$  de Corynebacterium bovis a été >1 à partir des quartiers non désinfectés dans tous les troupeaux, cependant Ro a varié autour de 1 dans les quartiers désinfectés et pour les germes coagulase-néqatifs de la famille des Micrococcaceae.

La variabilité de  $R_o$  calculé indique que les résultats calculés à partir d'enquêtes de terrain ne sont pas seulement influencés par les propriétés des germes pathogènes étudiés, ce qui joue sur la répétabilité des mesures. L'estimation de  $R_o$  qui est un paramètre relié de façon basale au pathogène est, dans des études de terrain, apparemment influencé par plusieurs facteurs liés à la vache et à l'élevage. Par conséquent, la discussion porte sur le fait de savoir s'il vaut mieux exprimer les résultats à partir de  $R_t$  qui ne nécessite pas d'avoir une population complète de germes potentiels au début de l'infection. Les facteurs influençant les dynamiques des infections intramammaires et leurs effets sur l'estimation de  $R_t$  sont discutés.

## INTRODUCTION

Mastitis pathogens can spread according to different patterns. Environmental bacteria, such as coliform bacteria, are present in high numbers in the environment in which the cows live. These bacteria infect mammary glands and cause mastitis, but generally do not spread from one cow to another (Lam et al., 1996a). Although contagious pathogens, such as *Staphylococcus aureus*, have also been isolated from the environment, their main reservoir is in infected quarters (Roberson et al., 1994). Intramammary *S. aureus* infections can spread from cow to cow following a Reed-Frost transmission model (Lam et al., 1996b).

The ability of an agent to spread in a population is summarized in the basic reproduction ratio ( $R_0$ ). The  $R_0$  is defined as the average number of secondary cases caused by the first (typical) infectious individual during its entire infectious period in a fully susceptible population. Thus,  $R_0$  of a mastitis pathogen can be interpreted as the number of new infections per infected quarter in an uninfected herd. In this paper it will be discussed how field data on the spread of mastitis pathogens should be handled and interpreted, what the value of these data is to describe the population dynamics of contagious udder infections, and whether or not it is possible to calculate  $R_0$  from this type of data.

# MATERIALS AND METHODS

An intensive longitudinal study on the dynamics of udder infections was done in seven dairy herds with a low bulk milk somatic cell count (Lam et al., 1996a). The main objective of the study was to evaluate the effect of discontinuation of postmilking teat disinfection (PMTD) on the incidence of clinical mastitis in this type of herds. To this end PMTD was discontinued on half of the quarters of all cows in the seven herds. Udder infections with contagious minor pathogens were studied in these herds during a 20 month period. The average number of cows per herd was 56.2 (SD 12.7). Throughout the study, foremilk samples were collected from the quarters of all lactating cows at 5-week intervals. The farmers collected samples from cows that calved during the trial, within seven days of calving, and from cows at drying-off. Additionally all quarters showing signs of clinical mastitis were sampled by the farmers.

Data were analyzed using a discrete regression model, which has earlier been described in detail for the transmission of *S. aureus* infections (Lam et al., 1996b). In this type of model, denoted as SIR model, units (quarters) are defined as either susceptible (S), infectious (I), or resistant (R) (de Jong et al., 1994). In the SIR model, the incidence of new infections is dependant on the prevalence of susceptible quarters, the prevalence of existing infections, and the transmission of infections. The transmission of infections is quantified with a transmission rate  $\beta$ , and a cure rate of infections  $\alpha$ . The transmission rate  $\beta$  can be interpreted as the probability per unit of time that one infectious quarter will infect another quarter, and can be estimated from the SIR model. The cure rate  $\alpha$  is based on the estimated end-points of infections, due to either spontaneous elimination, antibiotic treatment, culling, or the end of the study. The cure rate was estimated is the inverse of the mean duration of infection. Subsequently  $R_0$  can be calculated from the transmission rate  $\beta$  and the cure rate  $\alpha$  of primary infections, as:

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$$\mathsf{R}_{\theta} = \frac{\beta}{\alpha}$$

In this paper the data on the minor pathogens *Corynebacterium bovis*, and coagulase-negative Micrococcaceae (CNM) will be discussed.

#### RESULTS

For C. bovis an overall  $R_0$  was estimated at 1.28 in disinfected quarters, and at 3.81 in non-disinfected quarters. Contagiousness of CNM was found to be lower than of C. bovis, which is reflected in a lower  $R_0$ : 1.01 in disinfected, and 1.76 in non-disinfected quarters.

Notable differences in  $R_0$  between herds were observed for both, CNM and *C. bovis* (Table I). The R<sub>0</sub> of *C. bovis* in non-disinfected quarters was bigger than the critical value 1 in all herds, indicating that these infections will spread in all herds studied if they would not practice PMTD. In all other situations  $R_0$  varied from below to above 1. Variability was higher in non-disinfected than in disinfected quarters, and higher for *C. bovis* than for CNM.

Herd	C. bovis		CNM	
	R <sub>0 d</sub> <sup>1</sup>	R <sub>0 nd</sub> <sup>2</sup>	Rod	R <sub>0 nd</sub>
1	3.27	6.50	1.02	1.41
2	1.33	4.32	0.82	2.31
3	1.24	1.25	0.96	1.97
4	0.43	5.14	1.04	1.67
5	1.23	4.63	1.34	2.16
6	1.25	1.69	0.93	0.55
7	0.56	3.27	0.86	1.48

 Table I

 Estimates for the different herds, of the basic reproduction ratio R<sub>0</sub>, and the efficacy of postmilking teat disinfection against infections with Corynebacterium bovis and coagulase-negative Micrococcaceae.

<sup>1</sup> Basic reproduction ratio in disinfected quarters.

<sup>2</sup> Basic reproduction ratio in non-disinfected quarters.

#### DISCUSSION

Differences between herds in speed and extent of the spread of contagious intramammary infections will be influenced by different factors. Since contagious mastitis pathogens spread from cow to cow following a Reed-Frost transmission model (Lam et al., 1996b), a first factor influencing the spread of infections is the initial number of infectious and susceptible quarters. These factors can be corrected for, as has been done in this study, by calculating  $R_0$ . Differences between herds in  $R_0$  calculated for minor pathogens, however, are obvious. This variability of the calculated  $R_0$  indicates that the results from field studies on dynamics of infections are not only influenced by the properties of the pathogen studied.

An important factor influencing the results is the gradation of 'susceptibility'. In the SIR model a unit is either susceptible, infectious or resistant. In intramammary infections, however, susceptibility is not a binary variable. Genetic factors influence the susceptibility of cows, as may do other factors, such as previous infections with the same pathogen or current or previous infections with other pathogens, or other diseases.

As described for *S. aureus*, R<sub>0</sub> calculated in different time periods in the same herd may also vary considerably (Lam et al., 1996b). This may be due to different reasons, one of them being the 'most susceptible' cows to be infected during an outbreak of disease, selecting 'less susceptible' cows to be non-infected during later phases.

Thirdly, there are management factors that influence the spread of disease. In this study the effect of PMTD was quantified. Other management factors such as hygiene and functioning of the milker and the milking machine probably are at least as important.

Finally,  $R_0$  calculations from field studies assume infected units to be 'typical' infectious units. Generally, passage of infectious agents over animals may affect pathogenicity and thus the capacity to spread. In udder infections, one infected quarter may lead be to a higher infectious pressure than another, and different shedding patterns of bacteria in milk have been described (Sears et al., 1990). When these patterns are a function of time,  $R_0$  will also change in time.

The estimation of R<sub>0</sub>, which may be interpreted as a pathogen related parameter, from field studies, apparently is influenced by many herd and cow factors. Therefore care should be taken when interpreting these data. As

explained above, cow and herd factors will influence the estimation of  $R_0$ . Preferably, the effect of these riskfactors on  $R_0$  should be estimated. Clearly, transmission in a not fully susceptible herd is better described by  $R_t$ , where t is a time component. Apart from this direct effect of the initial infectious status of a population on the spread of disease, that status may also influence the effect of riskfactors on transmission of infections (Koopman et al., 1991). Thus, the effect of herd as well as cow factors on  $R_t$  should be calculated by studying the dynamics of udder infections in many different situations and calculate  $R_t$  in the same way in these different studies, allowing measurement of riskfactors in relation to  $R_t$ . We do realize that it is not always possible to do detailed studies as described in this paper. Reproduction ratio's however, can also be estimated from, for instance, prevalence data (Becker, 1989).

The factors discussed will have consequences for the repeatability of measurements. Field data, however, give information on the 'real life' behaviour of infections. Theoretically in vitro studies would give more reliable information on the properties of the pathogen itself. In vitro experiments are, however, not very good imaginable to study the spread of intramammary infections. Therefore, repeated measurements from the field will lead to the most informative description of the dynamics of infections, and of factors influencing these dynamics.

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