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Bioeconomic modeling of intervention against clinical mastitis caused by contagious pathogens

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ABSTRACT

The objective of this study was to assess the epidemiologic and economic consequences of intervention against contagious clinical mastitis during lactation. A bioeconomic model of intramammary infections (IMI) was used to simulate contagious spread of *Staphylococcus aureus*, *Streptococcus uberis*, and *Streptococcus dysgalactiae*, and an environmental spread of *Escherichia coli* IMI in a 100-cow dairy herd during 1 quota year. The costs of clinical IMI, subclinical IMI, and intervention were calculated into the total annual net costs of IMI during lactation per scenario and compared with a default scenario. Input parameter values were based on the scientific literature. The scenarios were 3-d intramammary lactational treatment (default), 5-d intramammary treatment, 5-d intramammary treatment and 3-d systemic treatment, 3-d intramammary treatment and culling bacteriologically unrecovered clinical IMI cows, and 5-d intramammary treatment and culling bacteriologically unrecovered clinical IMI cows. Sensitivity analysis was conducted on parameter input values. The results showed that interventions including antibiotic treatment combined with culling unrecovered clinical IMI cows resulted in the lowest transmission, number of IMI cases, and persistent subclinical IMI cases. Nonetheless, the high associated costs of culling bacteriologically unrecovered clinical IMI cows made the other scenarios with a long and intensive antibiotic treatment, but without culling, the most cost effective. The model was sensitive to changes to the cure rate of clinical IMI following treatment, but the ranking of the intervention scenarios did not change. The model was most sensitive to the changes to the transmission rate of *Staph. aureus*. The ranking of the intervention scenarios changed at low transmission rate of this pathogen, in which the default scenario became the most cost-effective scenario. In case of high transmission of contagious IMI pathogens, long and intensive treatment of clinical IMI should be preceded by strategies that lower the transmission.

Key words: clinical mastitis, treatment, economic model, transmission

INTRODUCTION

Clinical IMI is a frequent and costly disease in dairy cattle herds (Halasa et al. 2007). It is also a major welfare problem for dairy cows (Broom, 1991). Clinical IMI is usually treated with 3-d intramammary treatment, but could be extended depending on the severity of the case and the treatment regimen (Barkema et al., 2006). The success of treatment is highly dependent on the causative agent (e.g., McDougall et al., 2007; Bradley and Green, 2009). Several treatment regimens have been suggested, including culling of the infected cows (Steeneveld et al., 2011). Treated, but unrecovered cases could persist as chronic subclinical IMI, amplifying the risk of infection to other herd mates (Halasa et al., 2009). Until recently, several attempts have been made to assess the cost-effectiveness of interventions against clinical IMI, including antimicrobial treatment and culling of IMI cows. For instance, Bar et al. (2008) developed a dynamic programming model to support farmers in decisions related to treatment of clinical IMI. Steeneveld et al. (2011) investigated the effectiveness of cow-specific treatment of clinical IMI cases in short- and long-term antibiotic treatment regimens, supported with systemic antibiotic treatment. Pinzón-Sánchez et al. (2011) estimated the economic consequence of treatment strategies for mild and moderate clinical mastitis cases using decision tree analysis. Heikkilä et al. (2012) used the dynamic programming approach to estimate the costs of clinical IMI taking into account the indirect costs of premature culling of the clinical IMI cases.

Nonetheless, previous assessments of the economic consequence of interventions against clinical IMI have ignored modeling the dynamics of transmission of contagious IMI pathogens between cows at the herd level. When modeling the spread of a contagious infection between individuals, usually a Reed-Frost model (Becker, 1989) is used, in which the probability of infection is

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calculated dynamically based on the transmission rate of infection per unit of time, the number of susceptible individuals, and the number of infected individuals (Becker, 1989; Dohoo et al., 2003). This means that the probability of infection is dependent, among other factors, on the number of infected cows within the herd. Thus, when a clinical IMI cow is treated and recovered, it would not only result in direct benefits from, for instance, reducing milk production loss and risk of premature culling, but also in indirect effects from reducing the risk of infection to healthy herd mates. This is expected to decrease the costs of clinical and subclinical IMI at the herd level (Steeneveld et al., 2011). Therefore, previous studies could have underestimated the true effect of interventions against contagious clinical IMI cases, because of ignoring modeling the transmission dynamics of contagious IMI pathogens between cows at the herd level. Thus, economic effects of IMI interventions can be properly studied only when the transmission dynamics of contagious IMI pathogens are modeled. The objective of this study was to estimate the epidemiologic and economic consequence of interventions, including antibiotic treatment and culling, against contagious clinical IMI during lactation using a bioeconomic simulation model incorporating the dynamics of IMI.

MATERIALS AND METHODS

Model Description

The bioeconomic model used in this study is a previously published stochastic and dynamic simulation model of IMI with slight modifications to evaluate the economic consequence of intervention against clinical IMI during lactation in 1 quota year. The model simulates the dynamics of pathogen-specific IMI within dairy herds during lactation (Halasa et al., 2009) and the dry period (Halasa et al., 2010). The model was written using Mathematica 6.0 software (Wolfram Research, Champaign, IL). In brief, the dynamics of *Staphylococcus aureus*, *Streptococcus uberis*, *Streptococcus dysgalactiae*, and *Escherichia coli* were simulated at cow level using a discrete-event simulation model in a 100-cow dairy herd in a milk quota system. Each time period in the model was 2 wk. Reed-Frost models were used to model IMI dynamics of contagious pathogens (*Staph. aureus*, *Strep. uberis*, and *Strep. dysgalactiae*) during the lactation, whereas a Greenwood model was used to model IMI dynamics of *E. coli*. During the dry period, Greenwood models for all 4 pathogens were assumed to represent IMI dynamics. Dry cow therapy was applied to every cow during dry-off.

During lactation, the probability of acquiring an IMI was determined at the beginning of each time period based on the number of pathogen-specific IMI cows in the previous time period, the number of susceptible cows, and a pathogen-specific transmission rate. For *E. coli* IMI, a constant probability of infection was used, based on the cumulative incidence of *E. coli* IMI per 14 cow-days at risk. During the dry period the probability of new IMI per 14 cow-days at risk and the cure of existing IMI at dry off following antimicrobial therapy were implemented as explained previously (Halasa et al., 2010).

Cows were culled due to clinical IMI, subclinical IMI, and other reasons and were replaced by heifers based on the need for new animals to fill the milk quota (Halasa et al., 2009). Culling of clinical IMI cows was changed depending on the simulated scenario as explained in the subsequent section. The calculated difference between the actual and the expected herd-level milk production at each time period was used to decide on the introduction of replacement heifers to fulfill the milk quota (Halasa et al., 2009).

Modeling Intervention Scenarios

Four scenarios of clinical IMI intervention were modeled and were compared with a default scenario of clinical IMI intervention. The default scenario consisted of the normal 3-d intramammary treatment of clinical IMI cases (Steeneveld et al., 2011). Two scenarios consisted of long-duration antibiotic treatment, 1 with supportive systemic treatment, and 2 scenarios consisted of antibiotic treatment combined with culling the treated, but bacteriologically unrecovered cases. The scenarios were (A) the default scenario, with 3-d intramammary treatment; (B) scenario 1, a clinical IMI case received for 5-d intramammary treatment; (C) scenario 2, a clinical IMI case received for 5-d intramammary treatment combined with 3-d systemic antibiotic treatment; (D) scenario 3, similar to the default scenario, but if the clinical IMI case did not bacteriologically recover (confirmed by bacteriological culture), it was culled during the subsequent time period; and (E) scenario 4, similar to scenario 1, but if the clinical case did not bacteriologically recover (confirmed by bacteriological culture), it was culled in the subsequent time period.

Probabilities of cure from clinical IMI, following treatment for the different treatment regimens, were obtained from Steeneveld et al. (2011) and are presented in Table 1 together with other input values. A clinical IMI case was considered a new case when at least 14 d had elapsed since the previous clinical IMI case (Halasa et al., 2009). Clinical flare ups, which are subclinical