A Stochastic Model Simulating Pathogen-Specific Mastitis Control in a Dairy Herd

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ABSTRACT

The objective of this study was to develop a model simulating mastitis control in dairy herds and to investigate how sensitive the model is when varying the effect parameters according to the uncertainty. The model simulates 9 pathogen-specific mastitis types, each of which can be subclinical or clinical. The clinical cases can be 1 of 4 severities defined according to the effect of the mastitis case: mild, moderate, severe, and permanent effect. The risk factors include lactation stage, parity, yield level, previous diseases, season, and contagious spread of the infection from herd mates. Occurrence of mastitis is modeled to have direct effects on feed intake, body weight, milk yield, somatic cell count in the milk, subsequent mastitis cases within the cow and in herd mates, voluntary and involuntary culling, mortality, and milk withdrawal. Thirty-five scenarios were simulated to study model behavior and model sensitivity. The consequences per cow/yr of mastitis in the default simulated herd included 0.42 clinical mastitis occurrences, 0.56 subclinical mastitis occurrences, loss of 385-kg milk yield, a 1.3% reduced feed intake, 61-kg milk withdrawal and €146 in reduced economic net return. Based on scenarios demonstrating model behavior and sensitivity analysis, the model appears to produce valid consequences of mastitis control strategies. Representation of the effect of subclinical mastitis and of variation in mastitis severity was concluded in this study to be important when modeling mastitis economics in a dairy herd. The model offers the opportunity to study the long-term herd specific effects of a wide range of control strategies against mastitis.

(Key words: simulation, mastitis control, dairy herd)

Abbreviation key: BTSCC = bulk tank somatic cell count, ECM = energy-corrected milk, OR = odds ratio, SFU = Scandinavian feed units.

INTRODUCTION

Mastitis is the most prevalent production disease in dairy herds worldwide and is responsible for several production effects (Seegers et al., 2003). The available control strategies against mastitis are numerous and the application of these varies from herd to herd (Barnouin et al., 2004). As the biological knowledge about mastitis is continuously enhanced and new control measures are provided, the evaluation of alternative control strategies has become a repeatedly important decision problem for herd management. Furthermore, there is a trend toward larger herds implying less labor time per cow and increased risk of spread of infections due to more cows per milking unit.

The consequences of implementing a control strategy against mastitis are expected to vary considerably from herd to herd when evaluating the herd level outcome such as drug use, number of clinical cases, culling rate, bulk tank SCC (BTSCC), and economics. This is due to differences between herds regarding already applied strategies against mastitis, reproduction and culling strategy, yield level, actual prices, and costs. Furthermore, farmers may differ in their preferences, which means that a certain set of technical and economic effects of a mastitis control strategy will be considered appealing in one herd but not in another herd. The methodological issues invoked in evaluating the consequences at herd level of such alternative control strategies in different herds has been addressed within the discipline of animal health economics (McInerney et al., 1992; Seegers et al., 2003). The method of stochastic simulation modeling has been suggested as a relevant way to assess the economic worthiness of control strategies (Allore and Erb, 1999; Seegers et al., 2003). One example of the dynamics within a herd, which needs to be accounted for, is that a farmer can decide to cull an extra cow to decrease the BTSCC and hence, not sell a heifer (Seegers et al., 2003). Such relationships cannot be extrapolated easily from mastitis effect at the cow level. This emphasizes the relevance of herd simulation for mastitis economics.

A number of stochastic models simulating the consequences of mastitis at herd level have been published.

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These include discrete-event stochastic simulation models from the United States (SIMMAST; Allore et al., 1998), France (ECOMAN/ECOMAST; Seegers et al., 2000, 2003), and Denmark (Østergaard et al., 2003), and dynamic programming models from The Netherlands (Houben et al., 1994), United Kingdom (Yalcin and Stott, 2000), and United States (Gröhn et al., 2003). Schepers and Dijkhuizen (1991) reviewed earlier studies on mastitis economics. The stochastic models differ significantly in the factors that are included. The models of Houben et al. (1994), Gröhn et al. (2003), and Yalcin and Stott (2000) focused on optimal culling strategies. Two models simulate pathogen-specific mastitis and the aspects of SCC in the milk (Allore et al., 1998; Seegers et al., 2000, 2003). These aspects are considered important for a model to simulate a variety of control strategies against mastitis because the payments for milk are increasingly related to BTSCC. Recent studies have emphasized the importance of the difference in the effects on SCC and milk yield from different mastitis pathogens (De Haas et al., 2002, 2004; Gröhn et al., 2004).

The SIMMAST and the ECOMAST models differ in their representation of reproduction and culling management. The ECOMAST model is mechanistic in the sense that it accounts for the current state of the herd when triggering the culling decisions. This is similar to the SimHerd model (Østergaard et al., 2003), which focuses more on the interactions with reproduction and culling strategies. However, the fact that the ECOMAST model simulates with daily time steps and the choice of programming software have restricted the possibility to study the long term effects of the interaction with reproduction and culling strategy in the herd. The objectives of this study were to develop a model simulating mastitis control in dairy herds that allows long term effects to be studied in conjunction with the effects of different pathogen types, and to investigate how sensitive the model is when varying the effect parameters according to the uncertainty.

MATERIALS AND METHODS

Model Structure

The model developed in this study, SimHerd IV, is an extended and modified version of the mechanistic, dynamic, and stochastic dairy herd model, SimHerd III (Østergaard et al., 2003), including modeling of SCC and mastitis. The model is programmed for a 32-bit Pascal compiler.

The SCC of the individual cow are modeled by the natural logarithm of cells/mL of milk as a function of parity, lactation stage, mastitis occurrence, and with a Gaussian random effect. The model simulates patho-

Table 1. Nine mastitis types based on the mastitis agents included in the model.

Mastitis type	Mastitis pathogen
1	<i>Staphylococcus aureus</i>
2	<i>Streptococcus dysgalactiae</i>
3	<i>Streptococcus uberis</i>
4	<i>Escherichia coli</i>
5	<i>Klebsiella</i> spp.
6	<i>Arcanobacterium pyogenes</i>
7	Minor pathogens
8	No pathogen isolated
9	<i>Streptococcus agalactiae</i>

gen-specific mastitis types. This is because differences have been found in effect of mastitis depending on the pathogen involved (De Haas et al., 2002; Gröhn et al., 2004). Nine mastitis types based on the mastitis agents are represented (Table 1). Subsequently, the numbers in Table 1 are used as reference for these mastitis types. The model structure is the same for all mastitis types. However, each mastitis type is modeled as a separate disease independent of the other mastitis types. Each mastitis type therefore has its own set of variables for the risk factors and the effects of the disease.

Mastitis occurrence was defined as the event where a mastitis pathogen begins (week of occurrence) to cause subclinical or clinical mastitis symptoms in the affected cow. State variables of the individual cow include, for each mastitis type: infectiousness, severity, parity, and days after occurrence of a previous mastitis case. The severity is represented by subclinical and 4 clinical severities: mild, moderate, severe, and permanent effect. The severity is defined according to the effect of the mastitis case on production and treatment costs. The severity of a new case is triggered by a random drawing from a discrete distribution where user-definable values define the relative probability of each severity. It is triggered in 2 steps. First, it is triggered if it is a subclinical or a clinical case and, if it becomes a clinical case, then secondly the clinical severity is triggered. The distribution among subclinical and clinical cases can be defined for the lactating and the dry period separately. Because we found no evidence in the literature about the severity of recurrent mastitis cases, we assumed that a new case of the same mastitis type within the same lactation had the same severity as the previous occurrence of the same mastitis type, if the triggered severity of the new case otherwise would have caused a smaller reduction in milk yield. A new case of another mastitis types is allowed any severity.

The risk factors for each mastitis type includes lactation stage (baseline), parity (1, 2, and 3+), yield level

(continuous), presence of previous cases of the same mastitis type, presence of previous milk fever case, season (2 seasons), and contagious spread of the infection from herd mates.

Mastitis is modeled to have direct effects on feed intake, BW, milk yield, SCC, subsequent mastitis cases within cows and in herd mates (described under risk factors), voluntary and involuntary culling, mortality, and milk withdrawal. The effects modify the “without-disease” response of the cow. If the cow has a history of more mastitis types or other diseases, then the effects are combined by successively considering the disease-modified responses of the cow as the “without-disease” level.

The effects of moderate clinical mastitis cases on daily feed intake, BW gain, and milk yield are specified by proportions of the without-disease level according to the time since occurrence of the specific mastitis type. These effects on milk yield, weight loss, and feed intake are interrelated in the model. The effect of moderate clinical mastitis on SCC is modeled by adding the without-disease level with a profile for number of SCC per milliliter of milk according to the time since the occurrence of the specific mastitis type. The SCC can be specified to have a direct effect on the milk yield of the cow according the parity and lactation stage of the cow (Hortet et al., 1999). No direct effect of SCC on milk yield can be specified to represent conditional independent milk yield and SCC effects given the state of mastitis. Milk withdrawal due to mastitis treatments is modeled for a specifiable number of days after occurrence of a clinical mastitis type.

The effects of the severities subclinical, mild clinical, and severe clinical cases on the feed intake, BW, milk yield, and SCC are modeled as proportional corrections of the effect of the moderate clinical case. The effect of clinical cases with permanent effect is modeled by a proportional permanent reduction in the yield capacity of the affected cow.

Default Parameterization of the Model

The general production and management strategy. The default parameter values were intended to represent a typical loose housing system and management strategy for a 200-cow dairy herd with additional young stock. This included cows of the Holstein breed with an average yield capacity of 38 kg of energy-corrected milk (ECM), which is defined with reference to the first 24 wk of third lactation. The standard deviation in yield capacity between animals was 3 kg and the standard deviation in yield capacity between successive lactations within cows was 3 kg. The total risk of stillbirth and death among calves was set to be

0.12 and 0.08 for calves born from primiparous and multiparous cows, respectively. It was assumed that the lactating cows were fed ad libitum with 1 of 3 mixtures depending on parity, lactation stage, and yield level. The energy density of the mixtures differed, and were equivalent to net energy intakes of 20.0, 16.1, and 13.5 Scandinavian feed units (SFU), respectively, for a third-parity cow 3 mo after calving and with an average feed intake capacity. One SFU equals 7.89 MJ of net energy for lactation.

Involuntary culling was defined as a constant risk of 0.0033/wk for all cows. Voluntary culling was based on whether the cow became pregnant within the AI period. Cows with a milk yield higher or lower than the parity-specific herd-average were specified to have an AI period of 259 or 217 d, respectively. The AI periods were initiated 35 d after calving and cows were dried off 7 wk before calving. A cow not pregnant after the AI period was replaced when a heifer entered the herd and the particular cows are the lowest yielding candidates for voluntary culling. Nonpregnant cows producing less than 10 kg of milk per d were culled immediately. The high reproductive efficiency was assumed by a conception rate to d 14 after conception of 0.62 (second or later cycles) and an estrus detection rate of 0.50, respectively (fourth or later cycles).

SCC in uninfected cows. The default parameterization of mean of the natural logarithm of SCC per milliliter of milk in cows not affected by the modeled mastitis cases was based on De Haas et al. (2002). The profile through the lactation was modeled by a 3-phase linear spline function. Random variation was added by multiplying the mean value with a standardized normal distribution $N(0, 0.875)$. Figure 1 illustrates the profiles using the default values.

Baseline risk as function of lactation stage. The shape of the default baseline risk functions for all mastitis types for both subclinical and clinical cases is based on the curve illustrated by Seegers et al. (2000). The results of Gröhn et al. (2004) indicate similar mean and median DIM of first clinical occurrence of different mastitis pathogens. As the level of the curves are scaled relative to the variables of the risk in the week of calving, this variable's value from each mastitis type has been used to calibrate to an incidence of clinical mastitis cases per cow/yr of 0.42, which corresponds to typical Danish figures for mastitis treatment during the lactation period and at drying off. The distribution among the different mastitis types was based on Danish national statistics (Mejeriforeningen, 1999), where 96% of the reported pathogens that correspond to the mastitis types in the model had a distribution of: 0.13, 0.13, 0.27, 0.16, 0.01, 0.04, 0.08, 0.17,

Figure 1. Default parameterized SCC through lactation in primiparous (●) and multiparous (▲) cows not affected by the modeled mastitis. The 95% confidence intervals for primiparous cows are shown.

and 0.01 for mastitis type 1 through 9, respectively. A comparable distribution among pathogen-specific mastitis types has been found by De Haas et al. (2003) and Sargeant et al. (1998). The probabilities of a new case being subclinical were assumed to be: 0.83, 0.42, 0.34, 0.05, 0.05, 0.09, 0.77, 0.50, and 0.50 for mastitis types 1 through 9, respectively. These probabilities were based on Mejeriforeningen (1999). The prevalence of mastitis pathogens during the dry period has been found to be as high as 40% (Green et al., 2005). However, mastitis infections occurring during the dry period are usually not seen as clinical cases before calving (Bradley and Green, 2004). Accordingly, we assumed the probability of a new case occurring during dry period to be clinical to be reduced to one-third of the probability assumed during the lactating period. The default baseline risk function is illustrated in Figure 2.

Risk factor for mastitis occurrence. The default values for the variables for the effect of parity were based on Danish national figures and estimates of Gröhn et al. (2004). These resulted in odds ratios (OR) of 0.60 and 0.75 for parity 1 and 2, respectively, with parity 3+ as reference.

Ingvarstsen et al. (2003) concluded from a literature review that mastitis was the only disease where there was a clear relationship between milk yield and risk of occurrence. Houben et al. (1993) estimated OR in the order of 2.6 and 1.7 for cows producing above average and average milk yield relative to cows producing

below average, respectively. The default values of this effect were based on the value used by Østergaard et al. (2003), which was an OR of 1.04 per kg of milk potential above herd average. Milk potential was defined according to average daily yield during wk 1 to 24 after third calving in the absence of disease (Østergaard et al., 2003). As the default average yield potential was 38 kg, a cow with a 4-kg higher (~10%) yield potential will have an increased mastitis OR = $(1.04)^4 = 1.17$. No evidence was found for specific mastitis types, hence these default values were used for all mastitis types.

The default values of the variables for the recurrence of a mastitis type that previously occurred more recently than last dry off were all assumed to have an OR of 1.20 based on the results of Rajala and Gröhn (1998) and De Haas et al. (2003). Estimates of Houben et al. (1993) indicate larger effects (OR between 1.9 and 2.5). The default duration of this effect was assumed permanent until next dry off. No evidence was found for specific mastitis types, hence this default value was used for all mastitis types.

The default values of the variables for the recurrence of a mastitis type that previously occurred before last dry off were assumed to be an OR of 2.5 based on a number of studies with OR of 3.8 (Calavas et al., 1996), 1.49 (Peeler et al., 1994), 2.0 to 3.0 (Houben et al., 1993), and 2.05 (Bigras-Poulin et al., 1990). The default duration of this effect was assumed to persist until the cow had a new occurrence of the same masti-

Figure 2. Example of the baseline risk function in mastitis occurrence (weekly risk).

tis type. No evidence was found for specific mastitis types, hence these default values were used for all mastitis types.

Evidence of an increased risk of mastitis caused by the previous occurrence of other diseases has mainly been found for milk fever and the default values in this study of the variable for the effect of milk fever on the risk of mastitis was assumed to be an OR of 1.10 based on the estimate applied by Østergaard et al. (2003). No evidence was found on either the duration of this effect or for the effect of specific mastitis types, hence we assumed the duration of 2 mo and the default values were used for all mastitis types.

The default values of the variables for the effect of season on the risk of mastitis were specified to represent no seasonal effect. The part of the model for contagiousness from herd mates with the same mastitis type (clinical or subclinical) was not used in this study. Consequently, in the default parameters, we modeled all mastitis types through the baseline risk function and the other risk factors. This was chosen in this study because we wanted to study the long-term effects of an assumed constant spread of the infectious mastitis types rather than studying the progress of an infection over time in a herd.

Effects of mastitis occurrence. The default values of the variables for the distribution among the mild, moderate, severe, and permanent clinical cases were specified, respectively, as 0.40, 0.50, 0.10, and 0.00 for the mastitis types 3 (*Streptococcus uberis*), 4 (*Escherichia coli*), 5 (*Klebsiella* spp.), 7 (minor pathogens),

and 8 (no pathogens isolated). This distribution among the mild, moderate, and severe clinical cases was suggested by Seegers et al. (2003) based on a literature review on the effect of mastitis on milk yield. For mastitis types 1 (*Staphylococcus aureus*) and 2 (*Streptococcus dysgalactiae*), the default distribution among mild, moderate, severe, and permanent clinical cases were 0.40, 0.50, 0.05, and 0.05, based on an assumption of 5% of severe clinical cases resulting in reduction of the yield capacity of the cow such as blinding off one quarter. For mastitis type 9 (*Strep. agalactiae*), we assumed that all of the severe clinical cases caused this permanent effect, specified by the distribution: 0.40, 0.50, 0.0, and 0.10. For mastitis type 6 (*Arcanobacterium pyogenes*), we assumed that 50% of all clinical cases resulted in the permanent effect specified by the distribution: 0.20, 0.25, 0.05, and 0.50.

The default values for the effects of the mild and severe clinical cases compared with the effects of moderate clinical cases on feed intake, BW, milk yield, and SCC, were specified to be 0.10 and 2.5, respectively. These values are also based on suggestions from Seegers et al. (2003). The default values for the effects of the subclinical cases, compared with the effects of moderate clinical cases on these responses, were specified to be 0.50. The default value for the permanent effect of permanent clinical cases on yield capacity was specified to be a 15% reduction. The value is supported by the results of Gröhn et al. (2004), where the effect of *A. pyogenes* caused a 15% reduction in milk yield 80 d after mastitis occurrence. The effect on feed in-

Table 2. Default values for the 2-phased linear spline function for direct milk reduction caused by moderate clinical mastitis types.

Time relative to mastitis occurrence	Reduction in percentage of without-disease level of each mastitis type ¹						
	1	2, 3, and 9	4	5	6	7	8
Week of occurrence	14	16	32	24	32	3	16
Three weeks after occurrence ²	0.90	1.00	0.45	0.58	1.05	1.00	0.77
Daily slope afterwards ²	-0.0020	-0.0075	-0.0027	-0.0035	-0.0095	-0.0010	-0.0060

¹Mastitis types: 1 = *Staphylococcus aureus*, 2 = *Streptococcus dysgalactiae*, 3 = *Streptococcus uberis*, 4 = *Escherichia coli*, 5 = *Klebsiella* spp., 6 = *Arcanobacterium pyogenes*, 7 = minor pathogens, 8 = no pathogen isolated, and 9 = *Streptococcus agalactiae*.

²Relative to the reduction in the week of occurrence.

take, BW, milk yield, and SCC was not assumed to stop at dry off for any mastitis severity. However, except for the permanent clinical case, all these effects were assumed to decline over time.

The default values of the variables for the effect of clinical mastitis on voluntary culling are specified as 0-d reduced insemination period. This means that the effect of clinical mastitis on voluntary culling is represented exclusively by the indirect effect of reduced milk yield on insemination period and thereby on involuntary culling. This default value was specified for all mastitis types.

The default values of the variables for the effect of clinical mastitis on mortality are specified as a 0.3 and 2.0% risk of death at the time of occurrence of mastitis, where the high risk is applied for the gram-negative pathogens (*E. coli* and *Klebsiella* spp.; Wilesmith et al., 1986; Gardner et al., 1990; Seegers et al., 2003). The default values of the variables for the effect of clinical mastitis on involuntary culling were specified as 0% for all mastitis types. Furthermore, this default value was chosen to represent the fact that a cow was not culled for slaughtering directly caused by mastitis in the week where mastitis occurred. The effect of mastitis on voluntary culling is represented through the indirect effect of milk yield.

The default values of the variables for the effect of moderate clinical mastitis on daily feed intake were specified as a 7% reduction in the first week of mastitis occurrence and linear decline in the size of the reduction of -0.33%/d thereafter. This means a 4.7 and 2.4% reduction in the second and third week, respectively. These values were based the estimates of Bareille et al. (2003) and supplemental analyses of the study of Østergaard and Gröhn (2000). The latter study showed 3.7, 3.5, 2.9, and 1.3% reductions in kilograms of DM intake in the week before diagnosis, the week of diagnosis, and the subsequent 2 wk, respectively, for multiparous cows with acute clinical mastitis. No evidence was found for specific mastitis types, hence these default values were used for all mastitis types except for

mastitis type 7 (minor pathogens), where we assumed no reduction in feed intake. The reduction in feed intake caused indirectly 3.1, 2.1, and 1.1% (1.2, 0.8, and 0.4 kg) reductions in daily milk yield for an average multiparous cow during the corresponding 3 wk. Correspondingly, BW was indirectly reduced by 0.37% (2 kg) during the 3 wk.

In the default parameterization of the model, we modeled conditional independent milk yield and SCC effects given the state of mastitis. This means that no direct effect of SCC on milk yield was specified. This approach was because the true mastitis state of the cow is known in the model. The default values for the variables for the effect of moderate clinical mastitis on milk yield were based on the estimates for multiparous cows of (Gröhn et al., 2004). Gröhn et al. (2004) did not analyze for mastitis types 2 (*Strep. dysgalactiae*), 3 (*Strep. uberis*), and 9 (*Strep. agalactiae*) separately. We used their estimates for *Streptococcus* spp. for all these mastitis types. Through the parameterization of the 3-phase linear spline function, we only specified a 2-phase function with a certain reduction in the week of occurrence, a certain reduction 3 week after occurrence, and slope describing reduction thereafter. The first 2 parameters were parameterized to fit the relative yield reduction from the estimates of Gröhn et al. (2004) in the corresponding time point after mastitis occurrence and the slope parameter was parameterized by fitting the relative slope between wk 4 and 10 from the same results. Table 2 summarizes the default values for the 2-phase linear spline function for the direct milk reduction caused by moderate clinical mastitis types. Because the modeled feed intake reduction caused 3.1, 2.1, and 1.1% reductions in milk yield during the first 3 wk, respectively, we only specified the additional effect from the estimates of Gröhn et al. (2004).

The default values of the variables for the effect of moderate clinical mastitis on daily weight gain were specified so that BW was unaffected due to the mastitis occurrence (Østergaard and Gröhn, 1999; Seegers et

Figure 3. Daily feed intake in Scandinavian feed units (SFU) and daily milk yield in kilograms of energy-corrected milk (ECM) of a third-parity cow with average yield capacity simulated with (●) and without (△) moderate clinical mastitis 42 d after calving for mastitis type 1 (*Staph. aureus*).

al., 2003). Accordingly, the indirect effects of the reduced feed intake and milk yield on BW were counter-balanced. Specifically, the linear slope of the effect depending on time after mastitis occurrence was parameterized to fit unaffected BW 4 and 18 wk after mastitis occurrence. Figure 3 illustrates daily feed intake and daily milk yield of a third-parity cow with average yield capacity simulated with and without moderate clinical mastitis 42 d after calving for mastitis type 1 (*Staph. aureus*).

The default values for the variables for the effect of clinical mastitis on milk withdrawal were based on 7-d withdrawal from the day of mastitis occurrence.

The default values for the variables for the effect of moderate clinical mastitis on SCC were based on results of De Haas et al. (2002). The 3-phase linear spline functions were parameterized based on the reported estimates and curves for SCC depending on time after mastitis occurrence and by defining spline points at 1 and 3 wk after mastitis occurrence. The SCC values were corrected toward the SCC level prior to 21 d before mastitis occurrence. No estimates were reported for mastitis type 5 (*Klebsiella* spp.) and 6 (*A. pyogenes*). We established the type 5 default values by using the values for mastitis type 4 (*E. coli*). The default values for mastitis type 6 (*A. pyogenes*) were assumed the same as for mastitis type 1 (*Staph. aureus*). Table 3 summarizes the default values for the 3-phased linear

spline function for the direct SCC increase caused by moderate clinical mastitis types. Figure 4 illustrates SCC of a third-parity cow simulated with and without moderate mastitis (type 1, *Staph. aureus*) 42 d after calving, respectively.

Simulated scenarios and analyses of simulated results. We simulated 35 scenarios to study the model behavior and sensitivity. Definitions of the scenarios are provided in Table 4. The scenarios for studying model behavior were general reduction in mastitis incidence and mastitis effects. These scenarios were studied by comparing each of them with the default (DEF) scenario. The scenarios for studying model sensitivity were selected to represent potential key uncertainties on mastitis effect parameters. To study how sensitive the model is when varying the mastitis effect parameters, we compared the contrast between the default and the no mastitis scenarios (DEF and NOMA) with the contrasts between each of the sensitivity scenarios and the NOMA scenario. Each scenario was simulated over 10 yr and the mean results from the last 5 yr of simulation were used to study the long-term effects of the scenarios. In this way, the influence of the state of the initial herd was diminished. Each scenario was replicated in 1000 simulation runs, which ensured highly stable estimates. Based on analyses of variance, least significant differences ($P < 0.05$) were estimated for the comparison of the scenarios simu-

Table 3. Default values for the 3-phased linear spline function for direct SCC×1000 increase caused by moderate clinical mastitis types.

Time relative to mastitis occurrence	Increase in SCC×1000 over the without-disease level for each mastitis type ¹						
	1 and 6	2	3	4 and 5	7	8	9
Week of occurrence	1644	1774	1788	1666	1660	1440	1773
One week after occurrence	344	374	588	416	474	493	373
Three week after occurrence	118	154	257	158	266	233	154
Daily slope afterwards	-559	-1117	-1397	-559	-999	-1317	-1000

¹Mastitis types: 1 = *Staphylococcus aureus*, 2 = *Streptococcus dysgalactiae*, 3 = *Streptococcus uberis*, 4 = *Escherichia coli*, 5 = *Klebsiella* spp., 6 = *Arcanobacterium pyogenes*, 7 = minor pathogens, 8 = no pathogen isolated, and 9 = *Streptococcus agalactiae*.

lated for studying model behavior. Further statistical comparisons between contrasting scenarios were not applied because any failure to attain statistical significance could be the result of too few stochastic simulation runs.

The economic consequences of the simulated scenarios were studied by applying a set of assumed 2004–2005 Danish prices and costs for the corresponding technical results (Table 5). Economic net return was calculated as sales income less variable costs (feed, AI, veterinary assistance, medicine, and other costs) for cows and heifers. Labor and management costs were not included as variable expenses.

RESULTS

Effect of Eliminating Mastitis and Assuming Only Mild Clinical Cases

Technical and economic effects of the scenarios DEF, NOMA, NOIN, NOEV and MILD are presented in Table 6. In the scenario DEF, the incidence per cow/yr of mastitis types 1 through 9 were 0.053, 0.055, 0.108, 0.065, 0.004, 0.018, 0.043, 0.071, and 0.003, respectively.

The culling percentage was reduced by 0.83 percentage units in the NOMA scenario and by 0.37 and 0.35

Figure 4. Somatic cell count (per mL) of a third-parity cow simulated with (●) and without (△) moderate clinical mastitis (type 1, *Staph. aureus*) 42 d after calving.

Table 4. List of scenarios to study model behavior and model sensitivity.

Scenario	Definition
Model behavior scenarios	
DEF	Default
NOMV	Elimination of mastitis type ¹ <i>N</i> , where <i>N</i> = {1, 2, 3, 4+5, 6, 7, 8, 9}
NOMA	Elimination of all mastitis types ¹
NOIN	Elimination of infectious mastitis types ¹ (type 1, 2, 7, and 9)
NOEN	Elimination of environmental mastitis type ¹ (type 3, 4, 5, 6, and 8)
MILDV	Clinical cases of mastitis type ¹ <i>N</i> , where <i>N</i> = {1, 2, 3, 4+5, 6, 7, 8, 9} being mild clinical cases
MILD	All clinical cases being mild clinical cases
Model sensitivity scenarios	
SUB1	Half of subclinical cases assumed to be clinical cases
SUB2	Subclinical cases assumed to have effect on feed intake, milk yield, BW, and SCC like moderate clinical cases
PER	Moderate and severe clinical cases assumed to be permanent cases
NOPER	Permanent cases assumed to be nonpermanent clinical cases
MOD	Nonpermanent clinical cases assumed to be moderate clinical cases
DRY	Effects of nonpermanent mastitis cases assumed to stop at dry off regarding feed intake, BW, milk yield and SCC
CUL1	Clinical cases assumed to reduce the insemination period by 28 d
CUL2	Clinical cases assumed to reduce the insemination period by 56 d
MOR1	Effect of clinical mastitis on cow mortality assumed to be zero
MOR2	Effect of clinical mastitis on cow mortality assumed to be doubled
WD1	4-d milk withdrawal after mastitis treatment assumed
WD2	10-d milk withdrawal after mastitis treatment assumed

¹Mastitis types: 1 = *Staphylococcus aureus*, 2 = *Streptococcus dysgalactiae*, 3 = *Streptococcus uberis*, 4 = *Escherichia coli*, 5 = *Klebsiella* spp., 6 = *Arcanobacterium pyogenes*, 7 = minor pathogens, 8 = no pathogen isolated, and 9 = *Streptococcus agalactiae*.

for NOIN and NOEV scenarios, respectively. In all the remaining scenarios, the effect was less than 0.20 percentage units. The number of cow/yr (cow feeding days divided by 364) and prevalence of primiparous cows was also stable except for a minor effect in the NOMA scenario. This indicates only minor effects of mastitis on herd demography.

Milk yield per cow/yr was increased in the range of 133 kg (NOM1) to 3 kg (NOM9) when eliminating a single mastitis type. Generally, a higher effect on milk yield was found for mastitis types with higher inci-

dence in the default herd. By dividing by the number of mastitis cases per cow/yr in the DEF scenario, the effect of eliminating all mastitis types can be expressed as $[385/(0.420+0.563)]$, i.e., 392 kg of ECM per case of mastitis (subclinical and clinical). The elimination of infectious mastitis (NOIN) had a greater effect on milk yield than elimination of environmental mastitis types (NOEV). The scenario for all clinical cases being mild for each mastitis type increased the milk yield by up to 33 kg of ECM per cow/yr. The scenario for all clinical cases being mild for all mastitis types increased the

Table 5. Prices and costs (in €).

Factor	Price
Milk	€0.282/kg of ECM ¹ ; Correction for bulk tank SCC (BTSCC) per mL: +1.8, +0.9, -3.6, and -9.0% for BTSCC of ≤200,000; 200,000–300,000; 400,000–500,000; and >500,000, respectively. Milk withdrawal used for calves: €0.225/kg of ECM
Livestock	Slaughter cows: €1.43/kg of BW; Slaughter heifers: €403/animal; Bull calves: €108/animal; Pregnant heifers: €874/animal; Dead cows: €-93/animal
Feed cows	Mixed rations for cows: €0.17, €0.16, and €0.15/SFU ² for mix 1, 2, and 3, respectively
Feed heifer	Milk replacer for calves: €0.225/kg milk; Concentrates: €0.16/SFU; Roughage: €0.13/SFU; Grazing first and second year: €0.13 and €0.27/d
Veterinary costs per disease occurrence	Milk fever ³ : €90; Dystocia ³ : €109; Downer (destruction) ³ : €49; Retained placenta ³ : €55; Metritis ³ : €80; Left displaced abomasum ³ : €164; Ketosis ³ : €71; Mastitis (all types): €90
Other costs	Cows ⁴ : €40 per cow/yr; Heifers ⁴ : €13 per heifer/yr; AI: €16/AI
Interest rate of the herd value	6%/yr

¹ECM = Energy-corrected milk.

²SFU = Scandinavian feed unit; one SFU is equivalent to 7.89 MJ net energy for lactation.

³Covering also retreatment costs within current lactation.

⁴Covering average costs for bedding, milk recording, pregnancy test, additional veterinary assistance, and drugs.

Table 6. Technical and economic effects of the default (DEF), no mastitis (NOMA), no infectious mastitis (NOIN), no environmental mastitis (NOEV), and only mild clinical mastitis (MILD) scenarios.

Output variable	Scenario					LSD _{0.95} ¹
	DEF	Mean effect relative to scenario DEF				
	Mean (SD)	NOMA	NOIN	NOEV	MILD	
Replacement, %	33.7 (1.61)	−0.83	−0.37	−0.35	−0.19	0.14
Mean slaughter cow, kg	559 (4.2)	5.7	0.9	4.9	3.1	0.4
Dead cows ²	0.026 (0.005)	−0.002	0	−0.002	0	0.0004
Sale of pregnant heifers ²	0.16 (0.02)	0.005	0.003	0.003	0.001	0.002
Feed intake, SFU ^{2,3}	5906 (21)	77	42	34	44	2
Kilograms of ECM ²	9168 (47)	385	218	159	190	4
Kilograms withdrawal ²	61 (4)	−61	−22	−37	8	0.3
Average SCC, ×1000 cells/mL	169 (2)	−29	−17	−10	−23	0.2
Incidence of clinical mastitis ²	0.42 (0.02)	−0.42	−0.15	−0.27	−0.01	0.002
Incidence of subclinical mastitis ²	0.56 (0.03)	−0.56	−0.41	−0.15	0.02	0.002
Vet treatment cost, ² €	53.4 (2.5)	−37.5	−13.4	−23.8	−0.8	0.2
Net return, ² €	1517 (13)	146	75	73	52	1
Net return/kg of ECM, €	0.165 (0.001)	0.009	0.004	0.005	0.002	0.0001

¹Least significant difference ($P < 0.05$).²Per cow/yr.³SFU = Scandinavian feed unit.

milk yield by 190 kg of ECM per cow/yr, which is almost half of the increase found with the NOMA scenario.

The feed consumption for the cow herd increased by 25 SFU per cow/yr in the NOM1 scenario, which was the largest effect of eliminating a single mastitis type, although other mastitis types had higher incidences of clinical cases. The MILD scenario increased the feed consumption by 44 SFU per cow/yr.

In the NOMA scenario, the sale of slaughter cows increased by 0.006 per cow/yr and the average weight of slaughter cows increased by 5.7 kg. In the other scenarios, the effect was smaller and the total sale of kilograms of slaughter cow per cow/yr was not significantly affected.

The number of dead cows was reduced by 0.0013 and 0.0020 per cow/yr in the NOM4+5 and NOMA scenarios, respectively. No significant effect was found in the other scenarios. The incidences per cow/yr of diseases other than mastitis did not differ significantly between the simulated scenarios.

The average SCC of all milk produced in the herd (including milk withdrawal) was reduced by 29,000, 17,000, and 10,000 cells/mL in the NOMA, NOIN, and NOEV scenarios, respectively. Eliminating single mastitis types reduced average SCC by less than 10,000 cells/mL. The MILD scenario reduced average SCC by 23,000 cells/mL, whereas assuming only mild clinical cases of single mastitis types reduced average SCC by less than 7000 cells/mL.

The economic net return was increased. The increase ranged from €42 (NOM1) to €0.5 (NOM9) due to the elimination of a single mastitis type. The major items

of the account resulting in an increased economic net return of €146 per cow/yr from the NOMA scenario were €119 from milk, €4 from sale of pregnant heifers, €-16 from feed consumption, and €38 from less veterinary cost. The milk sale item is mainly increased milk yield, but also a €0.001 increased milk price per kg of ECM, which is a result of lower BTSCC. By dividing by the corresponding number of mastitis cases per cow/yr in the scenario DEF, the effect of scenario NOMA can be expressed as €148 per case of mastitis (subclinical and clinical) and €347 per case of clinical mastitis. The latter figure assumes that subclinical cases are inherent parts of clinical cases.

The scenarios of eliminating only the infectious (NOIN) and environmental (NOEV) mastitis types increased the net return similarly, but by less than half of the increase found with the NOMA scenario. However, the reduction in incidence of clinical mastitis occurrences was significantly larger from the NOEV scenario than from the NOIN scenario. The increase in net return from the MILD scenario was smaller, which may be explained by no significant effect on milk withdrawal and number of clinical mastitis occurrences.

Sensitivity of the Model After Altering Key Assumptions for the Effect of Mastitis

The economic effects of eliminating mastitis in each of the sensitivity scenarios relative to the effect in the DEF scenario (€146 per cow/yr) are shown in Figure 5.

Figure 5. Model sensitivity of economic net return per cow/yr to the sensitivity scenarios. Sensitivity was measured as the effect of eliminating mastitis in each sensitivity scenario relative to the effect of eliminating mastitis in the default scenario. See Table 4 for the definitions of the scenarios.

The net return per cow/yr associated with eliminating mastitis was 54% greater when all moderate and severe clinical cases were assumed to have effects like the permanent clinical cases (PER). This high sensitivity to severity of mastitis types was also found when subclinical cases were assumed more severe (SUB2 and SUB1). In the MOD scenario, the sensitivity was +11%; in the remaining scenarios, the sensitivities were less than 5%.

The 54% higher loss in net return due to mastitis in scenario PER compared with the default scenario was provided by even higher sensitivity in term of reduced income from milk sale, which was partly compensated by the largest reduction in feed cost due to mastitis. Feed consumption for the cows in the herd was decreased by 230 SFU per cow/yr. The reduced income from milk sale was mainly a larger milk yield reduction (478 kg of ECM per cow/yr) whereas a lower average SCC of 25,000 cells/mL and reduced milk withdrawal of 9 kg of ECM per cow/yr provided an increased milk price of 0.20%. Compared with the DEF scenario, the incidence of subclinical and clinical mastitis was reduced by 0.06 and 0.07 per cow/yr, respectively, which explains most of the reduction in veterinary treatment cost of €6 per cow/yr. The income from total livestock sale was increased by €3 per cow/yr, which was mainly provided by 16-kg heavier slaughter cows in this scenario compared with the DEF scenario.

The scenarios SUB2 and SUB1 had sensitivity with 36 and 31% higher losses in net return due to mastitis compared with the DEF scenario. In scenario SUB2, the sensitivity was higher on the reduction in income from milk sale (172 kg of ECM per cow/yr), which was slightly compensated by saved feed cost (32 SFU per cow/yr consumed less by cows). The milk withdrawal was not affected significantly but the SCC was increased by 21,000 cells/mL, which caused a 0.4% reduction in milk price. In the SUB1 scenario, a 20% reduction in income from milk sale (74 kg of ECM per cow/yr) was supplemented by a 65% increase in veterinary cost (€24 per cow/yr). The reduction in milk sale comprised 74 kg of ECM produced less per cow/yr and an increased milk withdrawal of 38 kg per cow/yr. The SCC was decreased by 8000 cells/mL.

The sensitivity of +11% from the MOD scenario was essentially caused by the effect on milk sales (reduction in milk production of 47 kg of ECM per cow/yr). Milk withdrawal was not affected significantly in this scenario. The sensitivity was low (<5%) in the following scenarios: NOPER, DRY, CUL1, CUL2, MOR1, MOR2, WD1 and WD2.

DISCUSSION

The average loss in milk yield over lactation due to clinical mastitis is between 4 and 6%, as summarized

by Hortet and Seegers (1998). However, this estimate together with a figure for lactational incidence rate is insufficient to provide reliable estimates of the herd-level milk loss due to mastitis in specific cases. Not only do lactations with mastitis tend to be shortened because of culling but a number of other indirect production effects also exist. As the present study shows, these effects are different for different mastitis types. In our study, the effect of a combination of all mastitis types was estimated to be 385 kg of ECM per cow/yr, which could be expressed as $[385/(0.420+0.563)]$, i.e., 392 kg of ECM per case of mastitis (subclinical and clinical). As milk yield in the scenario without mastitis was 9553 kg of ECM per cow/yr, the milk yield loss of 385 kg due to mastitis accounts for 4.0% of the without-mastitis milk yield at herd level. This compares favorably with other published estimates (see review of Hortet and Seegers, 1998). In economic terms, the loss in net return was €146 per cow/yr which accounts for 9.0% of the without-mastitis net return per cow/yr or 3.4% of the without-mastitis net return per kg of ECM produced. These results indicate that mastitis is economically less important when a milk quota restricts the milk production in a herd.

The result of €146 per cow/yr increase in net return from eliminating mastitis does not include any control costs for labor and investments, and so on. If these control costs were zero, the economic potential in mastitis control would be equivalent to the value of the extra 508 kg (5.5%) of ECM per cow/yr. The scenarios eliminating either environmental or infectious mastitis (NOEV and NOIN) both increased the economic net return by one-half of the increase associated with eliminating all mastitis types. This indicates equal economic importance of the 2 categories of mastitis. The scenario in which moderate and severe clinical cases were assumed mild (MILD) resulted in an increased net return of €52 per cow/yr, which is equivalent to 36% of eliminating all mastitis.

Although the comparison of results from different simulation studies obtained in different spatiotemporal contexts has been questioned (Seegers et al., 2003), we provide a few examples to provide context for our results. Bennett et al. (1999) estimated total economic impact of clinical mastitis to be £119 per cow-case. In our study, we did not estimate the specific effect of the clinical occurrences. However, our estimate for the cost per case (clinical and subclinical) was 14% lower than the estimate of Bennett et al. (1999). The average cumulative impact of mastitis (clinical and elevated SCC) was reported as €78 per cow/yr in a French study (Seegers et al., 2003). The estimate in our study was 86% higher.

Seegers et al. (2003) outlined 2 main approaches for calculating the economic impact of mastitis and economics of mastitis control strategies. The first approach was observational studies, which suffer from the problem that farmers do modify their management during the period of observation; consequently, such studies may be incomplete or have possibly confounded results. The second approach, simulation modeling, is considered more appropriate, especially when dynamic stochastic models are used. The present model is a contribution to such models aiming for more precise estimates on consequences at the herd level of alternative control strategies against mastitis.

Among existing simulation models, the extended SimHerd IV model presented herein bears the closest similarity to SIMMAST (Allore et al., 1998) and ECO-MAST (Seegers et al., 2000) models, but with some important differences. The SimHerd IV model differs by simulating more pathogens, simulating with weekly time steps (as opposed to daily time steps), allowing a cow to have more mastitis types simultaneously, and simulating the effects of mastitis on feed intake and BW. Further, the effects of mastitis on milk yield and SCC can be simulated as independent or correlated responses, and long-term interactions between mastitis control and feeding, and reproduction and culling strategy can be simulated.

A key aspect of the validation of a model is the extent to which the uncertainty about the parameter values affects the conclusions to be drawn from the model outcome. A comprehensive sensitivity analysis would be based on a sampling from joint distributions of all parameters. However, this was not feasible with the current model. One problem is a lack of information from the literature to establish interrelationships among different parameters in the model. Another problem is the high number of parameters in the model. Therefore, we chose a simpler approach by using worst cases for uncertainty of some potential key parameters. The sensitivity analysis generally showed a modest relative effect of the uncertainty.

The sensitivity in terms of net return was highest when moderate and severe clinical cases were assumed permanent. As this is an extreme assumption, we consider the sensitivity values of 36 and 31% provided by scenarios SUB2 and SUB1 to be more important. These sensitivities emphasize the importance of the representation of the effect of subclinical mastitis when evaluating mastitis control strategies. The sensitivity of scenario MOD was 11% indicating that the severity of clinical cases was relatively less important than whether the mastitis cases were clinical or subclinical. The inclusion of different severities and not just an average effect of a certain mastitis type, can

be seen as a way to include directly in the model the uncertainty on the biological effect of mastitis. Consequently, this sensitivity of 11% could be interpreted as a reduction in the effect of mastitis when uncertainty of the effect parameters is included in the model. As the remaining scenarios had sensitivities below 5%, the corresponding uncertainties of mastitis effect parameters were considered to be of minor importance.

Another category of model sensitivity is that related to the uncertainty of the applied prices and costs. The most important price factor is the treatment cost per case of clinical mastitis and the milk price (comparison of DEF and NOMA). If the veterinary treatments are done by the farmer and the costs thereby halved (€90 to €45), then the difference in net return between the DEF and the NOMA scenarios would decrease by 25% from €146 to €109 per cow/yr. A 20% change in milk price would affect the difference in net return by 16% from €146 to €122.

A number of possible effects of mastitis have been simplified or ignored due to insufficient information. Lack of evidence exists for the effect of a mastitis case in one lactation on the production in the subsequent lactation other than that explained by an increased mastitis recurrence risk. The low sensitivity from the corresponding DRY scenario, where the effects of non-chronic mastitis cases assumed to stop at dry off regarding feed intake, BW, milk yield, and SCC, indicate that this lack of evidence is not important for evaluating control strategies against mastitis. This was also found for the uncertainty of the effect of clinical mastitis on voluntary culling (CUL1 and CUL2), the effect of clinical mastitis on cow mortality (MOR1 and MOR2), and the effect of number of days of milk withdrawal after mastitis treatment (WD1 and WD2). The sensitivity of the WD1 and WD2 scenarios may be underestimated, because we assumed that all the withdrawal milk was used by the calves in the herd. If this is not the case when the number of days of milk withdrawal increases, then the cost of milk withdrawal increases.

Based on this sensitivity analysis, the model has a low sensitivity to uncertainty on most of the addressed parameters. The highest sensitivity appeared from changing the severity of mastitis occurrences. This emphasizes the importance of representing the different severities in the model and the importance of model parameterization to get reliable estimates in the specific herd situation. The differentiation of mastitis cases in different severities that fundamentally affects the magnitude of losses has also been addressed by Hortet and Seegers (1998) and Kossaibati and Esslemont (2000).

The sensitivity analysis did not include different relative effects among feed intake, milk yield, and BW. From the cow-level default estimates on feed intake reduction, it seems that halving or doubling this effect would not have important consequences on the herd effect of mastitis. However, if mastitis were assumed to have an effect on feed intake for duration significantly longer than 3 wk, then this might introduce important herd effects of mastitis. We could find no evidence for a longer duration of mastitis effect on intake although it should be noted that only few large studies have addressed this issue. In the default parameters, we assumed no effect on BW. This is in accordance with the very little evidence there is on the effect of mastitis on BW. However, an important question raised by combining the mastitis effect parameters is the fact that the marginal feed intake reduction only explains a minor part of the milk yield reduction. This deserves attention in future research when more cow-level data becomes available from epidemiological studies.

In this study, we simulated different infectious mastitis types (*Strep. agalactiae*, *Staph. aureus*, and *Strep. uberis*) but did not take advantage of the possibility of allowing the model to simulate infectious spread of the infection in the herd. Using the part of the model with infectious spread is relevant if the scenarios differ in mastitis pathogen susceptibility and if the purpose of the study is to analyze the progress of infection in a herd over time. We simulated a constant spread of the infectious mastitis types that was represented by certain base risks in the herd. We considered this approach appropriate for simulating the long-term consequences of infectious mastitis types that are controlled to a certain level in the herd.

We assumed that cows were not culled involuntarily as a direct and immediate consequence of mastitis. This might be relevant in cases of *Strep. agalactiae*. However, because of the low incidence of *Strep. agalactiae* in this study, and the assumption of a constant infection spread in the herd and a high voluntary culling rate due to reduced milk yield of the cow, the simulated results would only be affected slightly by the assumption of involuntary immediate culling after *Strep. agalactiae*.

Although, as discussed above, there are a number of issues not fully dealt with by the model and full sensitivity testing was not feasible, the results of the simulated scenarios revealed no major flaws in the model. Overall, the model results at the herd level are logical consequences of the assumptions for each scenario in this study. This supports the validity of the model by means of face validation (Sørensen, 1990). The presented model has not been validated using real data. Validation of this type of model with

real data is generally not feasible because of lack of appropriate data (Sørensen, 1990). Therefore, caution should be exercised in interpreting the results from the model.

The implementation of the mastitis model into the existing SimHerd framework allows simulation of the interaction between mastitis control strategies and various management strategies in a herd (e.g., reproduction, culling, other health problems). This is an important potential of SimHerd IV, because the consequences of mastitis control strategies are expected to vary significantly between herds. The fact that the distribution of different mastitis pathogens differs significantly between herds (Green et al., 2005) can also be simulated with SimHerd IV to obtain more precise herd-specific results.

CONCLUSIONS

A model to simulate pathogen specific mastitis control in a dairy herd was successfully developed. Based on scenarios demonstrating model behavior and sensitivity analysis, the model appears to produce valid consequences of mastitis control strategies. Representation of the effect of subclinical mastitis and of variation in mastitis severity was concluded in this study to be important when modeling mastitis economics in a dairy herd. The model offers the opportunity to study the long-term herd specific effects of a wide range of control strategies against mastitis.

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