Production effects related to mastitis and mastitis economics in dairy cattle herds

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Review article

Production effects related to mastitis and mastitis economics in dairy cattle herds

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Abstract – Mastitis is the most prevalent production disease in dairy herds world-wide and is responsible for several production effects. Milk yield and composition can be affected by a more or less severe short-term depression and, in case of no cure, by a long-acting effect, and, sometimes, an overlapping effect to the next lactation. Summary values in the literature for losses of milk production were proposed at 375 kg for a clinical case (5% at the lactation level) and at 0.5 kg per 2-fold increase of crude SCC of a cow. Due to the withdrawal period after treatment, composition changes in milk can almost be neglected in economic calculations. Lethality rate for clinical mastitis is very low on the average, while anticipated culling occurs more frequently after clinical and subclinical mastitis (relative risk between 1.5 and 5.0). The economics of mastitis needs to be addressed at the farm level and, per se, depends on local and regional epidemiological, managerial and economic conditions. To assess the direct economic impact of mastitis, costs (i.e. extra resource use) and losses (i.e. reduced revenues) have to be aggregated. To support decision making for udder health control, it is necessary to use a marginal approach, based on the comparison of the losses avoided and the additional costs of modified plans, compared to the existing ones.

dairy cattle / mastitis / milk somatic cell count / milk yield / longevity / economics

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1. INTRODUCTION

Mastitis is considered to be the most frequent and most costly production disease in dairy herds of developed countries (e.g. [24, 44, 50]). The assessment of the economic worthiness of a control programme for mastitis has to be supported by a reliable evaluation of the economic losses caused by the disease and the knowledge of the costs of the implementation of that programme. The losses are revenues not earned, while the control costs are real expenditures [42]. Sometimes economic calculations use unrealistic estimates of production losses associated for mastitis and an over-estimated impact. This can then lead to a wrong decision, seen from an economic point of view.

The decrease in milk production per cow due to the clinical and subclinical prevalence of mastitis is usually recognised as the main pathway in causing the economic losses due to the disease [16, 53]. Although, this primordial place may sometimes be challenged under a strict quota limitation, as in France [57]. Previously published papers about the effect of mastitis on milk yield and composition are quite abundant and several discrepancies between the estimated yield losses have been reported [31, 32]. Other production effects (*sensu largo*) of mastitis, i.e. mainly reduced longevity and short term lethality, also have to be taken into account to assess the economic losses, but they have not been documented so much. The effects on body weight and feed intake have only been scarcely studied, and are therefore never quite integrated into the economic calculations. Other components of economic losses of mastitis generally included in the calculations are penalties or loss of premiums related to the somatic cell count of the bulk tank milk and

the milk withdrawn during and after antibiotic treatment. To obtain the total economic impact (sometimes so-called total cost) of mastitis, all these losses (revenues not earned) are added to the control costs (actual expenditures related to treatments and preventive measures, and extra labour to apply them) [42].

The aim of this paper was to provide a summary view of the recent papers published on the production effects and economics of mastitis, considering only on-farm consequences and not addressing public health or dairy processing concerns. Due to an intrinsic non-comparability of the results from economic studies under different production systems and prices contexts [55], our review will be limited to a methodological discussion and examples of that aspect.

2. EFFECTS ON MILK PRODUCTION

2.1. Effects on milk yield

2.1.1. Clinical mastitis

The effects of clinical mastitis on milk production were last reviewed by Hortet and Seegers [31] and only two papers were published thereafter [46, 51].

All the studies relied on observational designs. Large samples (in terms of the number of herds included) were generally samples where only a monthly measurement of individual milk yield was done, whereas samples consisting of a small number of herds had a more frequent milk yield measurement. Almost all clinical cases considered in the reviewed studies were treated cases. The assessment of loss

in milk yield (not including the withdrawal of milk due to treatment, which depends on local regulations) was performed using several time-frames of study: lactation, shorter periods, or long-term effects by including inter-lactation carry-over effects, and using several types of comparison or modelling approaches [31].

The main recent results (literature published since 1990) for lactational-level yield losses are displayed in Table I. Of course, these estimates depend on the average time of occurrence within the lactation, which is probably not comparable between the different studies. Estimates of average lactational loss due to a clinical case ranged from non significant or very low values in some studies to values higher than 700 kg of milk in others.

The results for short term estimates of yield loss (Tab. II) ranged from 0 to 100 kg of milk, when reassessed on a monthly basis around or following occurrence. Higher estimates (40 to 160 kg per month) for monthly loss were found for calculation periods longer than two months (Tab. II). Recently, this observation was confirmed by the results of Østergaard and Gröhn [46]. Figure 1 shows first, that in this study, multiparous cows experiencing clinical mastitis were higher producing cows and that, therefore, losses were underestimated (a shifted curve has been added in the figure to illustrate this). Figure 1 also shows that the magnitude of loss re-increased from the second week post-occurrence. This profile is probably related to a beneficial effect of treatment during a few days after occurrence, followed, in some cows, by a resumption of infection and a chronic expression.

The internal validity of the provided estimates depends on the relevance of the models used and, especially, on how the fact that the production level is also a risk factor for clinical mastitis is handled [34]. Moreover, the main weakness of the published results is the unavoidable bias of selection due to the fact that the most severe cases cannot be truly studied when the short term fatality risk is not null and when a very strong depression of milk yield can lead to very early culling. Only Lescourret and Coulon [40] tried to account for these cases by simply considering that the production loss was total after death or culling.

Regarding the external validity, in addition to the previously underlined differences in statistical modelling approaches, some likely differences between study samples probably generate normal discrepancies, for instance: differences in the prevalence of the pathogens involved, in clinical case definition, in the efficacy of treatments, etc.

Remaining questions and areas for further research are numerous:

(1) How to deal relevantly with the production level as a risk factor for the occurrence and factor of underestimation? Several attempts have been made to compare a modelled/expected yield to the observed yield. However, using previous lactation performance or previous test-day results to predict the expected yield does not avoid the drawback (in small or mid-sized samples) that unrecorded effects on yield can interfere. Moreover, this approach cannot be done for primiparous cows or early lactation.

(2) The effects of parity and lactation stage at the occurrence still remain unclear: the results are controversial, especially regarding parity, when comparing those of Houben et al. [34] and Rajala-Schultz and Gröhn [50], for instance.

(3) The same lack of research exists for carry-over effects from a lactation to the following one: only Houben et al. [34] recently studied this aspect. This question probably has to be combined with that of the assessment of differences due to the involved pathogens. The literature reports only non significant differences between pathogens. However, the samples were small, leading to a possible lack of power (in Bartlett et al. [6] and Wilson and Sears [63]).

Table I. Summary of study samples and the results in studies dealing with milk loss due to clinical mastitis at lactation level and published since 1990 (partly taken from Hortet and Seegers [31]).

Reference	Animals			Loss		Comment
	Parity	Milk (kg/lact)	Breed[a]	Mean (kg)	%	
Houben et al. [34]	1	6 433	H	31 to 128	0.5 to 2.0	Effect of 1 to ≥ 3 quarter-cases within current lactation
Myllys and Rautala [45]	1	5 564	A & F	32.8	0.6	Mastitis only from 7 d. before to 7 d. after calving
Pedraza [48]	1	4 639	H	749	–[b]	
Rajala-Schultz and Gröhn [50]	1		A	294, 348, 110	–[b]	Effect of occurrence before peak, between peak and 120 d. and later, respectively
Houben et al. [34]	2	7 632	H	155 to 448	2.0 to 5.8	Effect of 1 to ≥ 3 quarter-cases within current lactation
Wolf and Jahnke [64]	2	4 572	H	205	4.4	
Rajala-Schultz and Gröhn [50]	2		A	284, 300, 220	–[b]	Effect of occurrence before peak, between peak and 120 d. and later, respectively
Firat [23]	≥ 2	6 027	H	231	3.8	
Pedraza [48]	≥ 2	5 256	H	734	–[b]	
Houben et al. [34]	3	8 286	H	NS[c]	NS	Effect of 1 to ≥ 3 quarter-cases within current lactation
Rajala-Schultz and Gröhn [50]	3		A	509, 352, 387	–[b]	Effect of occurrence before peak, between peak and 120 d. and later, respectively
Rajala-Schultz and Gröhn [50]	> 3		A	552, 329, 357	–[b]	Effect of occurrence before peak, between peak and 120 d. and later, respectively
Hoblet et al. [30]	all	8 430	H & J	75–206	0.9–2.4	
Lescourret and Coulon [40]	all	5 032	H & M	313 ± 207	6.2	Summarised by reviewers

[a] H: Holstein-Friesian or Friesian; A: Ayrshire; J: Jersey; M: Montbéliarde.
[b] –: Calculation not applicable due to no data on incidence rate or lactational yield presented in the paper.
[c] NS: not significant.

Table II. Short-term milk-yield loss due to clinical mastitis (partly taken from Hortet and Seegers, [31]).

Reference	Lactation stage at occurrence	Losses	
		Mean (kg)	Duration or estimation period
Bartlett et al. [6]	≤ 150 DIM	113	Cumulated for 60 days PMO [a]
	> 150 DIM	72	Cumulated for 60 days PMO
Deluyker et al. [19]	1–21 DIM	127.6	0–49 days
	50–119 DIM	250.1	0–79 days
Houben et al. [34]	1 WBC [b] to 5 WIM [c]	14 to 235 [d]	Cumulated for 5 WIM
	1 WBC to 14 WIM	0 to 444 [d]	Cumulated for 14 WIM
	1 WBC to 23 WIM	47 to 511 [d]	Cumulated for 23 WIM
	All stages	mainly NS[e]	≤ 1 month significant decrease in only some stages of lactation in parities 1 and 3
Luquet et al. [41]	< peak	NS	≤ 4 wk on average
	> 5 and ≤ 10 WIM	1.24/d.	< 5 wk on average
	> 10 WIM	0.83/d.	< 5 wk on average
Wilson and Sears [63]	NA	4.3	Estimated from 7 d. before to 14 d. after case occurrence
Østergaard and Gröhn [46]	All stages	65 (primiparous) 117 (multiparous)	Estimated on 9 weeks (including 3 weeks before case occurrence) in energy corrected milk Main loss was after occurrence: see also Figure 1

[a] PMO: post mastitis occurrence.
[b] WBC: week before calving.
[c] WIM: week in milk.
[d] 1 to ≥ 3 quarter-cases (1st, 2nd or 3rd lactation) without case in previous lactation.
[e] NS: not significant.

(4) What is the most-relevant way to express the losses: kg or %? Or, in other terms, does the production level affect the magnitude of losses? Until now, this has never been correctly answered, despite the availability of new sophisticated statistical packages (repeated measures, multilevel modelling, …) that could possibly help.

(5) What would have been the losses in the absence of treatment, or what is the true impact of treatment on yield losses? No real work on this question has been performed.

To summarise, a reasonable (and probably underestimated) average cumulated loss of 375 kg (about 5%) can be proposed for a so-called average clinical case, occurring in the second month of lactation in a Holstein cow. However the losses are very variable. To take this variability into account, it can be proposed that out of 10 cases, 4 lead to a quite negligible loss, 5 to an average loss, and 1 case to a very high loss (about 1000 kg) [31].

2.1.2. Elevated somatic cell counts

The effects of an elevated somatic cell count (SCC) on milk production were last reviewed by Hortet and Seegers [32] and
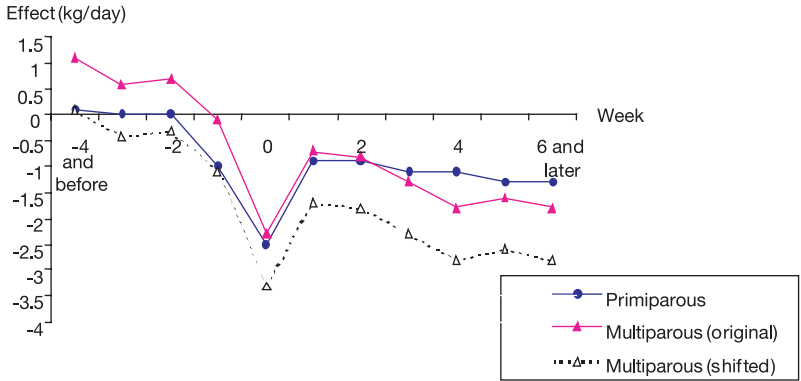
Figure 1. Profiles for daily energy corrected yield in cows with a clinical mastitis case around the week of occurrence (week 0) in deviation from the yield of non-mastitic cows [46]. The shifted curve (broken line) for multiparous cows (effect - 1 kg) was drawn by review authors.

only two papers have been published since [33, 37].

All the studies used retrospective data-analysis protocols. The assessment of the loss in milk yield was almost done ignoring the clinical cases (except in Hortet et al. [33] and also in Koldeweij et al. [37] who selected lactation sequences without clinical cases). The calculations relied on regression models at the test-day level, or, less frequently, at the lactation level. As for clinical mastitis, two main types of comparisons were performed: yield in test-days with vs. without elevated SCC or the expected yield vs. the observed one. As described in detail in the review of Hortet and Seegers [32], the variables used to describe SCC in the regression models differed. SCC values were mainly log-transformed or categorised, to be used as continuous or categorical independent variables. Except for the stage of lactation, the other independent regression variables included in the models differed widely.

To express a loss, the definition of a so-called "non infected" level of SCC is needed, allowing to compare milk yield in this category to that in higher SCC level categories. This "non infected" level can relevantly be set to ≤ 50 000 cells/mL,

based on Laevens et al. [39] and Schepers et al. [54], in contemporary Holstein cows and herds with a low prevalence. However, most of the results are only expressed for one unit of increase in Log(SCC) or for a two-fold increase of crude SCC. This helps to overcome the consequences of the use of different thresholds for the non infected status.

The central tendency reported in the review of Hortet and Seegers [32] was, for the daily study level, a decrease of 0.5 kg per two-fold increase of crude SCC (0.4 in primiparous and 0.6 in multiparous cows). A further assessment by Hortet et al. [33], based on French data, found the same magnitude in average losses as that in the cited review paper. Moreover, this latter study also showed that the magnitude of the loss was influenced both by the lactation number and by lactation stage (at least in parities higher than the first one). Quite surprisingly, when considering that milk yield of a cow is higher in early lactation, the losses were found to be higher, at the same SCC value, in late rather than in early lactation. This is illustrated in Figure 2. In another recent paper [37], based on a large Swedish dataset, similar magnitude of losses as the cited review was found in multiparous cows: the loss was 2.04 kg per unit
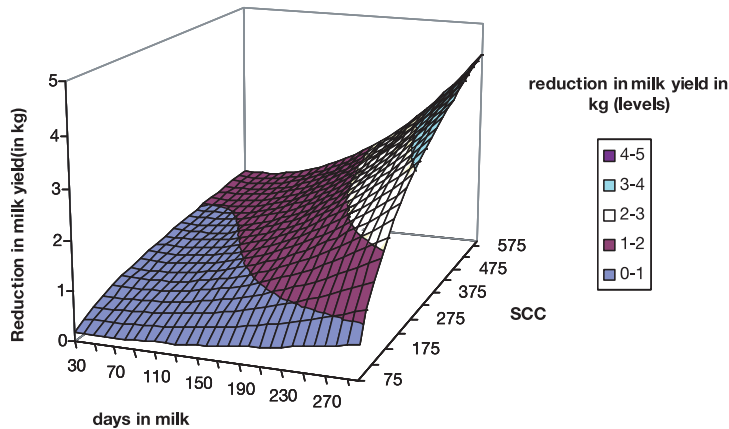
Figure 2. Reduction in milk yield according to SCC and days in milk for cows in 2nd lactation (reproduced from [33] with permission).

of increase in $Log_{10}(SCC.10^{-3})$, which corresponded to about 0.6 kg per two-fold increase of crude SCC. The authors found a slightly lower loss in primiparous cows (half of that calculated for multiparous cows). To assess the milk loss, these authors used pre-corrected yields with fixed adjustment effects calculated from only the non infected test-days. Logically this should have resulted in higher estimates for losses, when compared to the previously used method of analysis, but this was not the case. No obvious explanation can be hypothesised to explain this observation.

Differences in SCC lactation curves have been described according to the pathogen involved in the infection process (e.g., De Haas et al. [17]). An unsolved question is then: are the losses dependent on the nature of the pathogen or are they only dependent on the inflammation magnitude measured by SCC?

The effects of parity and lactation number described in Hortet et al. [33] have to be confirmed. Until now, as for the consequences of clinical cases, no real reliable assessment of an influence of the production level on the magnitude of the loss has been done: the production level was only

statistically accounted for in some studies, like in Koldeweij et al. [37]. Statistical analysis models used to answer this difficult point should cover two aspects: production as a risk factor, as already mentioned, and the diluting effect of higher milk yield on SCC [20].

On the contrary to what is commonly accepted, an elevated SCC does not only represent the subacute or chronic inflammation of the udder but also the acute phases. Especially when the incidence of clinical phases is high, their impact is partially accounted for, due to the fact that the cows are usually sampled when the test-day falls just a few days after a clinical phase. In that time, the level of SCC has not decreased to normal and thus still reflects the consequence of the acute inflammation. This was probably the case in most of the studies. This leads then to an overestimation of the global milk loss, when adding the elevated SSC effect and the clinical cases effect. Further studies should pay more attention to this problem.

To summarise, the effect of SCC on individual cow yield can conservatively be accounted for by a decrease of 0.5 kg per two-fold increase of SCC starting over

50 000 cells/mL. When needed, more precise predictions according to lactation stage and lactation number can be made.

2.2. Effects on milk composition and quality

Fat and protein yields, as well as fat and protein contents of milk, are modified by intramammary infection, but quantification of these modifications on large samples remains scarce (for review: [31, 32]). As known for a long time, fat yields are depressed (for example: Philpot [49]). The main mechanism involved is the reduction of milk volume. Nevertheless it has still not yet been demonstrated whether every mastitis case gives a decrease in the fat content of the 4-quarter milk, although most studies conclude in favour of this trend [32]. This is due to the 4-quarter observation level: a possible effect can best be seen in one-quarter milk when there is a severe clinical inflammation of that quarter. Regarding proteins, less knowledge exists. The total protein yield is also depressed [34, 37]), but most of the papers and arguments favour an apparent increased protein content of milk in association with an infection. This is due to the fact that protein content is estimated from the nitrogenic content of milk. The actual true casein content is depressed and inflammatory non coagulable proteins are increased [4, 5]. In addition to this, the proteolytic activity of the milk is increased, which makes the problem greater for the cheese manufacturer.

To summarise, when looking at the lactational level and only to what is the farmer's main concern (impact on milk price), fat and protein contents are only very little modified, according to Houben et al. [34] and Myllis and Rautala [45]. Moreover, considering the withdrawal period after clinical case treatment, the loss in fat and protein yields can be considered quite proportional to the loss in milk quantity. Therefore, economic calculations can neglect the effects on milk composition, except where

a special opportunity for high valorisation by specific milk pricing would exist.

Two aspects of quality are not detailed here. Mastitis also affects the bacterial count of the milk, but the elevation generated is almost negligible after the withdrawal period. Moreover, when errors occur, antibiotic treatments (for clinical cases or at drying-off) put the farm at a higher risk to experience positive results for growth inhibitor detection tests applied on the bulk tank milk.

3. EFFECTS ON LONGEVITY AND OTHER PRODUCTION EFFECTS

3.1. Survival and longevity

3.1.1. Short term effect: lethality

Only a few papers have dealt with the topic. Based on the annual mortality rates given in the original papers, we calculated a mastitis-attributable annual mortality rate of 0.22% in Holstein herds in western France [22] and 0.19% in dairy herds in Northern Ireland [43]. Gram negative pathogens lead to a higher fatality rate. This was shown by Bradley and Green [14] who reported a three-times higher specific mortality rate (0.6% of the lactating cows) and a high fatality rate (2.2%) in 6 herds, due to a high incidence of Gram-negative-bacteria induced clinical cases. Upon studying necropsy records, Hazlett et al. [28] already reported that, out of 145 cases that had bacteriological results, *Escherichia coli* was involved in 74% of them, *Klebsiella* sp. and *Staphylococcus aureus* in about 8% of them each.

3.1.2. Mid term effect: longevity

The effects of clinical mastitis and elevated somatic cell counts on longevity were recently included in the review of Beaudeau et al. [10]. Only one paper dealing with risk factors for the specific culling

Table III. Effect of clinical mastitis and elevated SCC on relative risk for culling.

Type of mastitis	Reference	Risk of culling	Additional information
Clinical mastitis	Beaudeau et al. [8]	1.5	Diagnosis < 90 d on late culling (all lactation ranks)
	Beaudeau et al. [9]	4.0	Diagnosis during the dry period in 1st lactation
		1.3	Diagnosis < 45 d in lactation < 3
	Gröhn et al. [27]	1.9	Diagnosis < 30 d; on culling < 30 d
		3.0	60 < diagnosis < 150 d; on 120 < culling < 180 d
	Rajala-Schultz et al. [51]	1.4 to 2.6	
Elevated SCC	Beaudeau et al. [9]	1.2	SCC = 300 000–800 000 c/mL; throughout lactation 1
		1.7	SCC ≥ 800 000 c/mL; throughout lactation 1

reason 'mastitis' has been published since [52].

The impact of clinical mastitis and elevated SCC in the culling process can simply be described by culling reasons. Among health-related culling reasons, those related to udder disorders were the second most frequent: mastitis-related reasons counted for 5 to 17% of all culling circumstances [7, 21, 56], and reached 28.5% when high SCC and teat injury were added [60]. However, declared culling reasons are more or less subjective, and therefore they rather provide information on the farmer's reactions than constitute an objective evaluation of the impact of health disorders on longevity.

Another approach consists of quantifying the effect of mastitis on the risk of culling, mainly on a lactational basis and ignoring the culling reason. Logistic regression or survival analysis were used to provide risk estimates associated with mastitis and/or elevated SCC, which were most of the time adjusted for other putative risk factors (other health disorders, reproductive performance, milk yield, parity, etc.). The recent results are displayed in Table III. The study of Roxström and Strandberg [52] considered cows culled for mastitis as the declared culling reason. An increased risk of culling in cows that have experienced clinical mastitis or elevated SCC is reported

in all available studies, regardless of the differences between breeds, study periods and designs. The risk of being more rapidly culled after clinical mastitis occurrence exists for all stages of lactation. However, cases occurring in early lactation [8, 27] and during the dry period [9] are associated with the highest risk. Culling subsequent to mastitis occurs either very early in lactation, probably in relation to milking disability, or is delayed to the end of lactation [52, 56]. A substantially increased risk of culling consecutive to teat injuries has also been reported by Beaudeau et al. [7, 8], probably because these disorders can disable a quarter or increase the risk for mastitis.

The differential impact of clinical mastitis, depending on its stage of lactation of occurrence shows that farmers mainly account for subsequent and future milk yield to make the culling decision. The effect of mastitis on the subsequent short-term milk yield (cf. supra) may partly explain why mastitis occurring before the peak of lactation has a large impact on culling. Gröhn et al. [27] showed that the impact of mastitis occurring within 30 d postpartum on culling was mainly indirect, i.e. mediated through lower milk yield. Indeed, its effect was no longer significant in models containing a descriptor of subsequent milk yield. Furthermore, despite a

low incidence rate, mastitis during the dry period is probably associated with a decrease of the expected yield in the next lactation, or, in some countries, a fear of the consequences of an *Actinomyces pyogenes* infection.

To summarise, the risk for a cow of being culled following the occurrence of clinical mastitis or elevated SCC is increased by a factor of 1.5 to 5, mainly depending on the severity of the milk drop and the farmer's anticipation concerning the future yield of the cow.

3.2. Other effects

Feed intake

Only peripartum clinical mastitis was studied. It was associated to a significant decrease of dry matter intake and estimated net energy supply before and after calving [67]. However in this study, 2/3 of the mastitis cases were preceded or concomitant with other disorders. Therefore, the observed depression of intake cannot be attributed to mastitis only.

Loss in body weight

On the contrary to metabolic disorders, acute clinical mastitis does not quite depress the body weight. Only a non significant effect was described by Østergaard and Gröhn [46] in multiparous cows during the week of occurrence.

4. ECONOMIC IMPACT OF MASTITIS AND ECONOMICS OF MASTITIS CONTROL

The latest review of papers dealing with mastitis economics was done by Schepers and Dijkhuizen in 1991 [53]. Several new studies have been published since. Most deal with the economics of control, and sometimes only with partial aspects like culling of mastitic cows [35, 61], rather than

with the question of the measurement of the whole economic impact of the disease.

4.1. Methodological issues

To assess the direct economic impact of mastitis, costs (i.e. extra-resources used) and losses (i.e. reduced revenues) have to be quantified and aggregated. To allow a comparison of decisions for health management, it is necessary to use a marginal approach, based on the comparison of the output/input ratio [42]. The choice of an alternative control plan instead of the current one, is grounded when the losses avoided by this new control plan are larger than the additional costs of this plan (Fig. 3).

Control costs include expenditures which can be measured directly from invoices or calculated according to standard treatment and prevention costs, and from labour time for treatment and prevention. Losses correspond to not earned monetary incomes. They are first calculated from the direct modifications of milk price, especially those due to penalties for high SCC. In addition to that, an economic translation
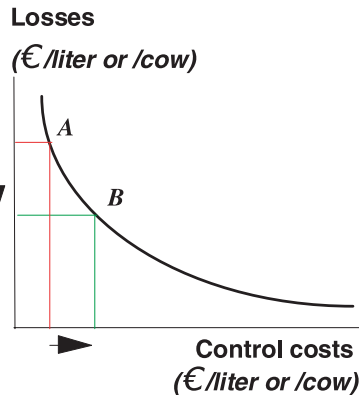
Figure 3. Conceptual approach for cost-effectiveness assessment in a modified control plan for mastitis (avoided losses have to be larger than additional costs when moving from A to B).
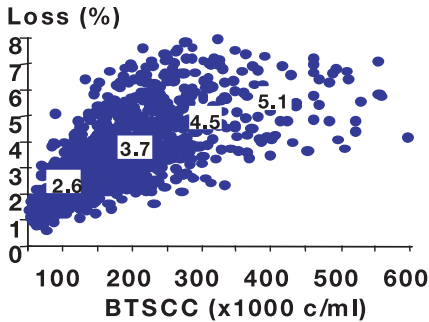
Figure 4. Calculated herd-level loss at a test-day according to BTSCC (somatic cell count of bulk tank milk) for dairy herds in western France [57].

of two types of effects is needed: (i) the decrease of the herd productivity through the several so-called production effects; and (ii) the non-ability of the milk produced for sale (withdrawal after treatments or rejection from collection).

The economics of mastitis needs to be addressed at the farm level. The relationship between the bulk tank milk somatic cell count (BTSCC) and the herd-level loss in milk yield is not directly the same as at an individual test-day level. Compared to the individual level, herd-level losses are higher for low values in BTSCC and lower for high values (see Fig. 4 to illustrate this point). The herd level also implies integrating the decisions made by farmers, often with incomplete information, in addition to the basic biological facts. Therefore some herd-level effects are not strictly limited to the aggregation of basic individual effects. Moreover, at the herd level, some compensation or buffer mechanisms can act [58]. For example, a farmer decides to cull cows with high SCC, more based on the BTSCC and the milk pricing system, than on the absolute values of the individual SCC results of the cows. For another example, a farmer can decide to cull an extra-cow to decrease the BTSCC and give up to sell a heifer. This should be accounted for and is rather complex to model.

Two main groups of approaches have been used in mastitis economics:

(1) The first one is only observational and tries to assess the effect associated from an observed variation in economic results within a sample of farms, comparing high vs. low prevalence or with vs. without the implementation of a control action. The economic variables considered in that case are often simple and can be limited to milk price or revenue from delivered milk, more or less diminished for some control action costs, like in Ott and Novak [47]. More elaborated economic results, like gross margin or net profit, are influenced by much heavier factors and confusion may occur. This is complicated by the fact that farmers often modify their herd management. This group of approaches provides thus incomplete or possibly confounded results.

(2) The second group of approaches relies on modelling and simulation. The most frequent strategy is a combination of results from an observational study and further modelling-simulation steps. Observed data address the variation in occurrence and/or the variation in technical effects in a surveyed sample of farms after/before or with/without the implementation of control plans. Modelling-simulation steps are then applied to these data to calculate the production effects and/or the economic consequences [1, 25, 26, 44]. Sometimes variation in the occurrence of mastitis, technical production effects and economic effects are all simulated [13, 68]. Complete validation of the simulation tools can sometimes become problematic, due to the absence of the needed data to conduct a goodness-of-fit procedure.

Partial budgeting is a commonly used technique to express the economic differences in the second group of approaches. This technique allows to perform a static comparison between "high" and "low" (or null) prevalence situations, the first one being mostly an observed situation (from a survey, for example, or from a typical herd

Table IV. Components included in the partial budget simulation of Fourichon et al. [24, 26] to calculate the economic impact of mastitis (case of no quota constraint).

Unfavourable consequences	Favourable consequences
Decrease in revenues	Increase in revenues
Lower milk sales (decreased yield and discarded milk) Lower milk price Lower calf sales Lower meat sales (cows euthanasied, lower carcass weight, lower carcass price) Dead cows Opportunity costs: margin on hectares used for raising extra heifers	Higher calf sales due to extra replacement heifers Higher meat sales (extra culls, higher carcass weight)
Increase in costs	Decrease in costs
Costs for extra calves up to 15 d (milk replacer, calf veterinary costs, identification costs) Costs for raising heifers from 15 d to 1st calving to replace extra culled cows (milk replacer, concentrates, veterinary costs, identification, variable costs for land hectares used for extra heifers)	Lower use of concentrates Lower use of milk replacer (discarded milk given to calves) Costs for calves due to reduction in calf number (milk replacer, calf veterinary costs, identification costs)

in a country) and the second a simulated one [1, 11, 15, 29, 38, 42, 44, 66]. Table IV displays, as an example, the components included by Fourichon et al. [24, 26] in their economic calculation model when there is no quota constraint. Since designed for a French context, and given the strict quota regulation (no transfer between farms without purchasing or borrowing land), the calculation model proposed by these authors could also simulate and assess the consequences of delivering the allowed quota with more cows (and heifers) than needed (because of the lower production caused by mastitis). This was the most prevalent case in the survey of Fourichon et al. [25] conducted in western France.

Dynamic modelling is a more sophisticated and more relevant way to assess the economic worthiness of a control plan. This technique allows consideration of transient situations and costs [12, 13, 58]. Linear programming and dynamic programming techniques have been implemented several times in economic models applied to farm

management and sometimes to mastitis control: recently, by Houben et al. [35] and Zepeda et al. [68]. There are limits to these deterministic approaches. They imply infinite divisibility of factors, linearity and additivity of effects, and also the certainty of effects. Thus, these techniques are well adapted to optimisation procedures in cases of absence of uncertainty, while in mastitis economics, a large part of the decision making is supported by imperfect knowledge. Therefore simulation techniques mimicking the farmer's decision process and including stochastic effects to model uncertainty and variability are more relevant than the deterministic programming approaches [1, 36].

As underlined by Seegers et al. [55], any value for the economic burden due to mastitis (as for any other clinical disorder), in a herd or a country is, *per se*, of low external validity. Indeed, variations in the prevalence of mastitis and in the nature of pathogens involved are aggregated with: (i) differences in the farming system, herd management and implementation rules of

Table V. Components included in simulations to assess the economic impact or the economic efficiency of control plans for mastitis in recent literature.

Component	Reference of paper [1]												
	1	2	3	4	5	6	7	8	9	10	11	12	13
Decreased milk yield or higher number of cows	X	X	X	X	X	X	X	X	X	X		X	X
Variation in milk composition								X		X		X	X
Veterinary costs	X	X	X	X	X		X	X	X	X		X	X
Treatment costs	X	X	X	X	X	X	X	X	X	X		X	X
Mortality and extra-culling		X	X	X	X			X	X	X	X		X	X
Extra-manpower	X			X	X		X			X			
Penalties, milk produced and not sold	X				X	X		X				X	X
Decrease in feed intake		X	X				X					X	X

[1] Authors: 1: DeGraves and Fetrow [16]; 2: McInerney et al. [42]; 3: Beck et al. [11]; 4: Hillerton et al, [29]; 5: Miller et al. [44]; 6: Van Eenennaam et al., [62]; 7: Kossaibati and Esslemont [38]; 8: Allore and Erb [1]; 9: Bennett et al. [13]; 10: Zepeda et al. [68]; 11: Yalcin et al. [65; 66]; 12: Fourichon et al. [24, 26]; 13: Seegers et al. [58, 59].

treatments; (ii) differences in the upper limit of milk production (fixed quota, possible leasing of quota or no quota); (iii) differences in milk pricing according to tank SCC; and (iv) other differences in prices of production factors. Last point, but not least, when considering the available literature, it appears that the components included in various simulations differ widely, as illustrated in Table V. Therefore, it is not very relevant to compare the results obtained in different spatio-temporal contexts. Thus only some examples will be presented below.

4.2. Recent examples of questions studied and results

4.2.1. Measurement of the impact of mastitis

Some examples are given here (the comparisons between studies are almost not valid, cf. above):

• Dealing only with somatic cell counts and under the pricing system applied in Ontario, Dekkers et al. [18] showed that the impact of one unit in herd average linear somatic cell score (Log2 transformed), i.e. for a two-fold increase, was 19.6 Can. $ per cow-year.

• The calculated total economic impact of clinical mastitis was found to be 119 £ per cow-case and was mainly due to reduced yield and discarded milk. This was 38% of the total impact of common clinical health problems in a group of 90 dairy farms from the United Kingdom in 1995 [38]. Also for UK conditions, losses were assessed reaching 121 Million £ for a mid-incidence level for mastitis (50%) at the country level, by Bennett et al. [13].

• The average cumulated impact of mastitis (clinical and elevated SCC) was 78 € per cow-year (around 11 € per 1000 L of milk) in a study involving 197 herds in western France. Mastitis accounted for 33% of the total impact calculated for health disorders in the surveyed herds [25, 26]. Average mastitis related losses were

two-times higher than mastitis related control costs, representing respectively 22 and 11% of the total impact of the health disorders. A questionable point, in assessing the total impact of mastitis, is that the calculations of losses need to fix a reference "low" level, which can theoretically be set to zero in the incidence of clinical cases and about 100 000 cells/mL in SCC. As long as a zero value in annual incidence is never quite observed, except in small herds, using such a reference leads to a slight overestimation. Therefore, it is advisable to use a more realistic reference level. Authors set that level at the 10th percentile of the clinical incidence in their survey.

4.2.2. Assessment of worthiness of control actions

Some examples of recent results for economic worthiness of control actions are also available (the comparisons between studies are almost not valid, cf. above):

• Using partial budgeting applied to outcomes of a discrete event stochastic model and under US conditions, Allore and co-authors [1, 2, 3] found that, both prevention (forestripping, predipping, cleaning and drying with a single use paper towel and postdipping) and dry cow therapy were relevant in mastitis control strategies for herds having BTSCC over 500 000 cells/mL. Where environmental pathogens are predominant, strategies including vaccination for *E. coli* mastitis provide higher annual profit per cow than strategies not including that vaccine.

• Using a calculation of marginal returns (i.e. the sum of the value of the saved production and of the avoided BTSCC penalties) provided by several control measures applied to herds facing a high BTSCC problem (> 400 000 cells/mL), Yalcin et al. [66] found a total impact of 100 £ per cow-year in Scotland. The authors concluded that, out of these 100 £, one third could have been saved by using more efficient control measures (dry cow therapy, milk-ing machine test and post-milking teat disinfection). However, the technical results of the study came from a static comparison (logistic regression) of farms implementing and farms not implementing the measures. Therefore, these conclusions do not necessarily apply to changes in control plans applied on the same farms, which would probably have been more relevantly studied by a dynamic simulation approach.

• Profits of a control programme to reduce the prevalence of *Staphylococcus aureus* infections are most dependent on the initial prevalence of mastitis and initial BTSCC penalty or premium, than on milk yield or the cost of drying-off treatments, under the US context [68].

• An important contribution to the benefit of mastitis control might originate in a reduction of persistently infected cows by culling, as shown by Yalcin et al. [66], using dynamic programming.

• By comparing several options for control plans applied to high BTSCC herds by a dynamic simulation model, Seegers et al. [58, 59] concluded that, under French conditions, the control strategies including strict culling rules for cows having persistently high SCC, are not relevant in herds with BTSCC above 300 000 cells/mL and delivering their milk quotum, despite a high mastitis prevalence. Although very effective in decreasing BTSCC rapidly when combined with improved prevention, strict culling rules frequently lead to not reaching the quota. This resulted in a lower gross margin, given the (relatively mild) French penalty system for BTSCC and the (rigid) French quota system. Lactational antibiotic treatment of newly incident infections in young cows could be advisable in such cases. Applying strict culling rules becomes more relevant under 300 000 cells/mL in BTSCC (then, they concern less cows and the quota will be more easily fulfilled). The interest of culling is higher when the contagious aspect of mastitis is higher.

5. CONCLUSION

The economic impact of mastitis results from two origins: the control costs (i.e. extra resource use) and the losses (i.e. reduced revenues). Losses are the economic consequences of the production effects and are quite often difficult to assess. The main detrimental production effect induced by mastitis is a more or less persistent decrease in milk yield. Milk composition changes can almost be neglected in economic calculations. Lethality rate for clinical mastitis is very low, except for specific situations of high prevalence of Gram negative infections. Mastitis exposes the cows to a higher risk to experience anticipated culling. Translating these production effects into economic losses has to be done for a specific farm and a specific economic context. Therefore, the results regarding the economic impact of mastitis or the worthiness of control plans should rather not be directly compared.

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