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Original article

Effect of mastitis and related-germ on milk yield and composition during naturally-occurring udder infections in dairy cows

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Abstract — The effect of mastitis and related-germs on milk chemical composition (protein and lactose contents) and milk somatic cell count (SCC) was investigated in 501 milk quarter samples during two consecutive years in cows from three experimental herds. Each infected quarter was matched by a healthy one in the same udder, as a control. Milk protein and mineral assays were performed in a subsample of 128 milks. Staphylococci were the most frequently isolated germs (*Staphylococcus aureus*: 27%, coagulase-negative Staphylococci: 26%, Streptococci: 21%). Major milk pathogens (*Staphylococcus aureus, Streptococcus uberis* or *Escherichia coli*) associated with clinical signs of mastitis were accompanied by higher SCC (+1.6 log·mL^{-1}, $P < 0.01$), lower lactose concentration (–7.6 g·kg^{-1}, $P < 0.01$), higher protein concentration (+3.3 g·kg^{-1}, $P < 0.01$) and higher soluble protein concentrations (IgG and BSA), hence a sharp decrease in the casein/protein ratio (–10 percentage points, $P < 0.01$). Changes were more marked when *Escherichia coli* was present. *Corynebacterium bovis* did not alter milk chemical composition whereas coagulase-negative Staphylococci slightly reduced lactose concentration (–1.8 g·kg^{-1}) and increased SCC (+0.37 log·mL^{-1}). Calcium and phosphorus milk contents were hardly modified by the presence of microorganisms. The decrease in milk yield during clinical mastitis varied from 1.6 kg·d^{-1} in the presence of *Staphylococcus aureus* to 15 kg·d^{-1} in the presence of *Escherichia coli.*

mastitis / milk composition / microorganism

Résumé — **Effet du type de mammite et du germe sur la production et la composition du lait lors d'infections mammaires naturelles chez la vache laitière.** L'effet de la nature des germes pathogènes présents dans le lait sur la composition chimique (taux protéique, taux de lactose) et la numération cellulaire du lait a été étudié à partir d'un échantillon de 501 laits de quartiers prélevés au

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cours de 2 années consécutives sur les vaches de 3 troupeaux expérimentaux. Pour chaque quartier infecté, un quartier sain de la même mamelle a servi de témoin. Sur 128 de ces prélèvements, des analyses de la composition minérale et protéique des laits ont été réalisées. Les germes les plus fréquemment observés ont été les staphylocoques (27 % de *Staphylococcus aureus* et 26 % de staphylocoques à coagulase négative) et les streptocoques (21 %). Lorsqu'elle a été associée à des signes de mammites cliniques, la présence d'un germe majeur (*Staphylococcus aureus, Streptococcus uberis* ou *Escherichia coli*) s'est accompagnée d'une augmentation de la numération cellulaire (+1,6 log·mL^{-1}, $P < 0,01$), d'une diminution de la teneur en lactose (–7,6 g·kg^{-1}, $P < 0,01$), d'une augmentation du taux protéique (+3,3 g·kg^{-1}, $P < 0,01$) et du taux de protéines solubles (IgG et BSA), de sorte que le rapport caséines/protéines a fortement diminué (–10 points de pourcentage, $P < 0,01$). Ces modifications ont été les plus importantes en présence d'*Escherichia coli*. La présence de *Corynebacterium bovis* n'a pas modifié la composition chimique du lait, alors que celle de staphylocoques à coagulase négative a légèrement mais significativement entraîné une diminution de la teneur en lactose (–1,8 g·kg^{-1}) et une augmentation de la numération cellulaire (+0,37 log·mL^{-1}). Les teneurs en calcium et en phosphore n'ont été que peu modifiées par la présence de germes pathogènes. La chute de production au moment de la mammite clinique a varié de 1,6 kg·j^{-1} en présence de *Staphylococcus aureus* à 15,5 kg·j^{-1} en présence d'*Escherichia coli*.

mammites / composition chimique du lait / germes

1. INTRODUCTION

The economic impact of udder infections can be highly detrimental [3, 6], since losses resulting from the treatment-related ban on milk commercialisation and the very costs of treatment add to direct production losses. Studies investigating the impact of mastitis on milk production have produced widely variable results according to cow physiological conditions, seasonal factors or cow milk potential [11, 16]. Also, mastitis is accompanied by significant modifications of milk chemical composition [15, 23] with both a reduced synthesis and altered cell permeability [1]. Such modifications affect protein and mineral fractions, in particular, carry major consequences for milk technological properties [23] and appear linked with the mastitis germ. If a number of studies investigating possible links between germ and milk composition have been conducted in experimentally induced mastitis [9, 20, 29, 32, 40], few have been carried out on naturally-occurring mastitis [21].

The study, as a part of a wider programme on mastitis risk of recurrence [8], was aimed at assessing the relationship between a set of milk chemical parameters (including protein and mineral concentrations), the mastitis germ involved and the type of mastitis (clinical or subclinical) and was based on observations and measurements performed on three experimental dairy herds. Milk specimens were sampled from each quarter of cows with mastitis to assess the individual animal effect within the statistical analysis of the results.

2. MATERIALS AND METHODS

This study was conducted in three experimental INRA herds (Theix, Marcenat, Orcival) made up of Holstein (75%), Montbéliarde (15%) and Tarentaise (10%) cows (3000 to 7500 kg per lactation). During two consecutive years (September 1997 – March 1999), the clinical mastitis cows were systematically subjected to sterile milk quarter sampling just before mastitis

treatment and, when possible, 30 days later. Out of 412 available lactations, 155 clinical mastitis were visually diagnosed through the udder and milk, according to Gasqui and Barnouin's rating table [7]. Mastitis was treated with antibiotics administered topically to the udder or by systemic administration in severely affected cows. At drying off, the cows systematically received an udder antibiotherapy. No anti-mastitis vaccine was administered.

Most calvings took place between September and January (85%). In the winter, the cows were kept either in tethered stables (Theix and Marcenat) or in cubicles (Orcival). The diets were composed of forage representing 70–80% of the total dry matter (grass silage and/or hay in Marcenat and Orcival, grass silage, hay, and maize silage in Theix) and were supplemented with concentrates based on cereal and soya-meal. The cows were turned to the pasture between May 1st and November 1st. Milkings were performed on-site (Theix) or in milking parlours (Orcival and Marcenat).

2.1. Milk sampling and analysis

Two types of milk specimens were sampled from each quarter, sterile or not. Sterile specimens were sampled according to the method described by Mialot [19]. Standard procedures according to the International Dairy Federation guidelines [25] were applied for milk bacteriological analysis. Microorganism identification used API kits. Milk bacteriology was performed at the INRA Animal Epidemiology Research Unit (Saint-Genès-Champanelle, France).

Non-sterile specimens, collected after the sterile ones, were divided into two samples. A preservative (bionopol) was added to one of those samples that was used to determine protein and lactose contents (infra-red method, Combifoss 5400, Foss Electric, Denmark) and somatic cell count (fluoro-opto-electronic method, Fossomatic, Foss Electric, Denmark). These analyses were performed at the Laboratoire Interprofessionnel Laitier (Saint-Genès-Champanelle, France).

Another sample, which was immediately frozen, was used to determine casein [30], calcium (atom absorption spectrometry, [13]), phosphorus (spectrometry, [12]), immunoglobulin G (IgG) and serum albumin (BSA) [18]. These analyses were performed at the Laboratoire Interprofessionnel Laitier (Saint-Genès-Champanelle, France) and at the INRA Herbivore Research Unit (Saint-Genès-Champanelle, France).

2.2. Data processing

Statistical analysis was performed on the samples (n = 154) for which an infected quarter and a control quarter were available, so that the animal effect could be taken into account. In 105 pairs of healthy quarters from the same udder, we had previously checked that the quarter position (forward or rear) had no influence on milk composition, whether the other quarters were infected or not by a pathogen (Tab. I). We also verified that milk composition of the healthy quarters was not affected by the infection of other quarter(s) of the same udder, regardless of the existence or not of clinical mastitis in that quarter (Tab. II). Somatic cell count, however, was slightly, but significantly increased in a healthy quarter when one or several other quarters of the same udder were infected by a pathogen (Tab. II).

Out of the 154 specimens, 81 matched clinical mastitis and 73 were controls collected 30 days after clinical sign occurrence. Only 501 of the 616 milk specimens from corresponding quarters were of any use, the remaining 115 being either contaminated by a mixture of germs (n = 77) or very unfrequent germs (n = 25) or samples that could not be analysed (n = 13). Protein and lactose assays and somatic cell count were performed on those 501 milk specimens. For casein, mineral, IgG and BSA

Table I. Composition of non-infected quarter milk according to the position of the quarter (forward or rear).

	Quarter		RSD[1]	Significance[2]
	Forward	Rear		
n	105	105		
Lactation stage (d)	115	115		
Protein ($g \cdot kg^{-1}$)	32.3	32.5	0.9	NS
Lactose ($g \cdot kg^{-1}$)	47.5	47.2	2.8	NS
Cells ($log \cdot mL^{-1}$)	4.67	4.64	0.48	NS

[1]Residual standard deviation.
[2]NS: $P > 0.05$.

Table II. Milk composition of non-infected quarters according to clinical and infectious status of the other quarters of the same udder.

	Infectious status			RSD[3]	Significance[4]
	Negative[1]	Positive[2]	Positive[2]		
Clinical mastitis	no	no	yes		
n	76	70	64		
Lactation stage (d)	102	133	111		
Protein ($g \cdot kg^{-1}$)	32.1	32.9	32.1	3.7	NS
Lactose ($g \cdot kg^{-1}$)	47.1	47.6	47.3	4.3	NS
Cells ($log \cdot mL^{-1}$)	4.45a	4.72b	4.76b	0.61	**

[1] The 3 other quarters were not infected.
[2] At least one of the 3 other quarters was infected.
[3] Residual standard deviation.
[4] NS: $P > 0.05$, *: $P < 0.05$, **: $P < 0.01$.

evaluation, 128 quarter samples were selected, so as to make up a sufficient number of specimens for each of the main germs: *Escherichia coli* and *Streptococcus uberis* in clinical mastitis, coagulase-negative staphylococcus and *Corynebacterium bovis* when there was no clinical sign, and *Staphylococcus aureus* in the presence or absence of clinical signs.

The data were analysed using analysis of variance [31]. The factors introduced in the model were the germ, ranked according to the presence or absence of clinical signs, and the cow. Since our statistical model adjusted for the cow effect, it took into account lactation number, lactation stage and other individual and herd effects. To assess the effect of the germ on the milk yield, the cases that occurred after the milk yield peak and in which a single germ was involved and for which the daily milk yield was available, were selected (n = 36).

3. RESULTS

3.1. Milk bacteriology

Out of the 501 milk specimens analysed, 270 were free of any germs. The bacteriological results of the other 231 specimens are displayed in Table III. Staphylococci were the most present, either *Staphylococcus aureus* as major pathogens or coagulase-negative staphylococci for minor ones.

Table III. Bacteriological results observed on the 501 quarter milk samples.

Type of germ	n	Clinical mastitis	
		No	Yes
No germ	270	262	8
Major pathogens			
. *Staphylococcus aureus*	62	48	14
. *Streptococcus uberis*	20	6	14
. *Escherichia coli*	9	1	8
. Other streptococci	29	15	14
. Other germs[1]	19	15	4
Minor pathogens			
. Coagulase-negative staphylococci	60	57	3
. *Corynebacterium bovis*	32	29	3

[1] Mainly *Corynebacterium* spp.

3.2. Milk yield evolution according to the type of germ

Whichever the germ involved, milk yield decreased only from the date when clinical signs of mastitis were observed. The decrease (difference between the mean milk yield of days 3 and 4 before mastitis detection and the mean milk yield of days 0 and 1 after mastitis detection) varied greatly according to the type of germ (Fig. 1): with *Escherichia coli*, milk yield decreased by an average of 15.5 kg per day over two days ($P < 0.01$) and never resumed its baseline level, even after three weeks. With *Streptoccocus uberis* and *Staphylococcus aureus* the decrease was low (2.9 kg·d^{-1} and 1.6 kg·d^{-1}, respectively) and not significant. With the other major germs, the decrease was intermediate (5.4 kg·d^{-1}, $P < 0.01$) and did not persist beyond 3 to 4 days. The sharp differences in baseline production levels were due both to the time of occurrence and to the different milk yield potentials according to the type of the germ: in our sample, *Escherichia coli* and *Staphylococcus aureus* mastitis occurred in early lactation (7 out of 8 cases and 7 out of 10 during the first 100 days, respectively) but the former involved cows with much higher milk yield potentials than the latter (31.3 and 24.6 kg per day on average during the 5th week of lactation, respectively). Mastitis ascribable to *Streptoccocus uberis* and other major germs affected high producing cows (29.2 and 29.9 kg per day on average during the 5th week of lactation, respectively) but they occurred at a much later stage of lactation (7 out of 13 cases and 1 out of 5 during the first 100 days, respectively).

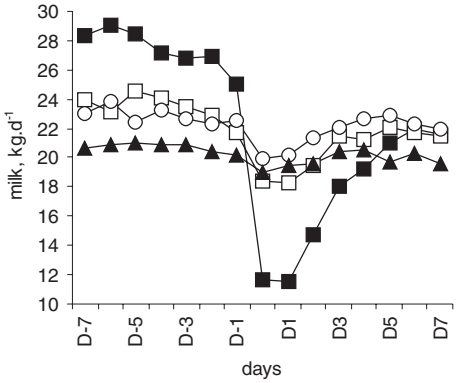

Figure 1. Milk yied pattern around the day of detection of clinical mastitis according to the germ: ■ *Escherichia coli*, n = 8; ○ *Streptococus uberis*, n=5; ▲ *Staphylococcus aureus*, n = 10; □ other major germs, n = 13.

3.3. Effect of the germ on milk chemical composition (Tabs. IV and V)

In 91% of the cases, the presence of a minor pathogen was not accompanied by any clinical sign of mastitis. Clinical signs accompanied the presence of a major pathogen in 38% of the cases, with a wide variability according to the germ (from 22% with *Staphylococcus aureus* to 89% with *Escherichia coli*). No germ was detected at all in three percent of the clinical cases. The presence of a minor germ was accompanied by an increase in somatic cell count (+0.29 log·mL^{-1}, $P < 0.01$), although it did not change milk chemical composition (Tab. IV). However, when considering the two types of minor germs found in this study separately (coagulase-negative staphylococci and *Corynebacterium bovis*), it was noted that no milk characteristic was degraded in the presence of *Corynebacterium bovis*, whereas lactose concentration was lower (–1.8 g·kg^{-1}, $P < 0.05$) and somatic cell count was higher (+0.37 log·mL^{-1}, $P < 0.01$) in the presence of coagulase-negative staphylococci than in healthy quarters. The presence of a major germ was accompanied by very significant changes in milk composition when it was associated with clinical signs of mastitis. In particular, milk proteins increased by 3.3 g·kg^{-1} ($P < 0.01$), exclusively because of soluble proteins (IgG, BSA), so that the casein/protein ratio decreased by almost 10 percentage points in the presence of clinical signs. Also, lactose decreased by 7.6 g·kg^{-1} and somatic cell count increased by 1.6 log·mL^{-1}. These modifications differed according to the germ involved (Tab. V): they were indeed more marked with *Escherichia coli*, whose presence in milk was linked to a decrease in lactose concentration and casein/protein ratio, which reached 13.6 g·kg^{-1} and 19 percentage points, respectively. Conversely, modifications were less significant with *Streptococcus uberis*, IgG and BSA concentrations not being significantly different with this germ from those observed in

Table IV. Effect of the bacteriological and clinical status on milk composition (mean lactation stage: 114 d).

	Bacteriological status				RSD[1]	Significance[2]
	Negative	Minor pathogen	Major pathogen			
Clinical mastitis	No	No	No	Yes		
n	262	86	85	54		
Protein (g·kg^{-1})	32.3a	32.6a	32.6a	35.6b	1.8	**
Lactose (g·kg^{-1})	47.5a	46.6b	46.1b	39.9c	2.9	**
Cells (log·mL^{-1})	4.70a	4.99b	5.58c	6.39d	0.58	**
n	58	21	21	28		
Casein (g·kg^{-1})	26.9	27.2	26.3	26.5	1.5	NS
Casein/proteins (%)	82.7a	82.6a	80.0a	72.3b	4.3	**
IgG (g·kg^{-1})	0.60a	0.56a	0.73a	2.09b	0.90	**
BSA (g·kg^{-1})	0.17a	0.17a	0.22a	1.29b	0.85	**
Calcium (g·kg^{-1})	1.27	1.29	1.27	1.25	0.10	NS
Phosphorus (g·kg^{-1})	0.95a	0.99a	0.93ab	0.91b	0.06	*

[1]Residual standard deviation.
[2] NS: $P > 0.05$, *: $P < 0.05$, **: $P < 0.01$.
IgG: Immunoglobulins G; BSA: Bovine serum albumin.
On the same row, results with different letters are significantly different ($P < 0.05$).

Table V. Effect of the type of germ and the clinical status on milk composition (mean lactation stage: 123 d).

	Germ[1]							RSD[2]	Significance[3]
	No germ	Cbovis	Scoag-	Saureus	Saureus	Suberis	Ecoli		
Clinical mastitis	No				Yes				
n	216	29	57	48	14	14	8		
Protein ($g \cdot kg^{-1}$)	32.5a	32.8a	32.9a	32.9a	34.5b	36.3c	39.3d	1.8	**
Lactose ($g \cdot kg^{-1}$)	47.7a	48.2a	45.9b	45.6b	39.7c	42.0c	34.1d	2.8	**
Cells ($log \cdot mL^{-1}$)	4.69a	4.79ad	5.06d	5.84b	6.58c	5.64b	7.09c	0.51	**
n	58	9	12	21	10	12	6		
Casein ($g \cdot kg^{-1}$)	26.9a	27.6ab	26.9ab	26.3a	25.7ac	28.2b	24.3c	1.4	**
Casein/proteins (%)	82.8a	82.7ab	82.4ab	80.0bd	70.8c	76.7d	64.7c	3.9	**
IgG ($g \cdot kg^{-1}$)	0.60a	0.44a	0.64a	0.73a	2.45b	1.17a	3.33b	0.83	**
BSA ($g \cdot kg^{-1}$)	0.17a	0.15a	0.19a	0.22a	1.11b	0.48a	3.20c	0.72	**
Calcium ($g \cdot kg^{-1}$)	1.27a	1.29abc	1.29ac	1.27abc	1.17b	1.37c	1.13b	0.09	*
Phosphorus ($g \cdot kg^{-1}$)	0.95a	1.03b	0.96ac	0.93ac	0.90cd	0.96abc	0.80d	0.06	**

[1] Cbovis: *Corynebacterium bovis*, Scoag-: Coagulase-negative staphylococci, Saureus: *Staphylococcus aureus*, Suberis: *Streptococcus uberis*, Ecoli: *Escherichia coli.*
[2] Residual standard deviation.
[3] NS: $P > 0.05$, *: $P < 0.05$, **: $P < 0.01$.
IgG: Immunoglobulins G; BSA: Bovine serum albumin.
On the same row, results with different letters are significantly different ($P < 0.05$).

healthy quarters and lactose decreasing only by 5.7 $g \cdot kg^{-1}$. With *Staphylococcus aureus*, intermediate modifications were noted. When this germ was not associated with clinical signs, modifications in milk chemical composition were noticeable (– 2.1 $g \cdot kg^{-1}$ lactose, – 2 percentage points in the casein/protein ratio, + 1.15 $log \cdot mL^{-1}$ in somatic cell count), but not as marked as when *Staphylococcus aureus* was associated to clinical mastitis. Calcium and phosphorus concentrations were negatively affected only in the presence of major pathogens (*Escherichia coli* and *Staphylococcus aureus*) associated with clinical signs. In contrast, milk phosphorus content was higher than in the healthy control in the presence of *Corynebacterium bovis*. *Streptococcus uberis* was accompanied by high calcium contents.

Considering the entire set of points available, the reduction of the casein/protein ratio between healthy and affected quarters of the same udder was closely and linearly linked to that of lactose ($R^2 = 0.69$, $P < 0.001$). That ratio and milk lactose concentration decreased in a curvilinear manner ($R^2 = 0.39$ and 0.46, respectively) with the increase in the somatic cell count.

4. DISCUSSION

This study was based on data from naturally occurring mastitis in experimental farms, which provided a large number of specimens and ensured homogeneous assessment of mastitis clinical signs. Also, when working with milk quarter samples which had healthy controls in the same udder, it was possible to optimally take the individual animal effect into account in data analysis (including the animal's genetic, physiological and nutritional characteristics),

with that effect weighting very significantly in the interpretation of the changes in milk chemical composition. To achieve this, it was hypothesised that the chemical composition of healthy quarters was not affected by the presence of infected quarters in the same udder. This hypothesis may nevertheless be questioned: through the observation of the evolution of milk characteristics with time, trials on mastitis induced by injecting endotoxins have shown that such an independence is not systematic, in particular regarding lactose [20, 34] whose precursors can be reduced as a consequence of infection. This observation was not confirmed in this study because comparing the healthy quarter of a totally healthy udder with those which presented at least one infected quarter, with or without clinical signs, elicited no significant difference in either lactose or protein contents, and only a slight increase in somatic cell count was observed.

The germs identified in this study during clinical mastitis were those commonly observed in France [4, 32]. The proportion of mastitis without any germ was low and comparable to those commonly observed [27]. They could be due to either a non-infectious inflammatory phenomena (traumatic in particular) or a detection problem linked to the fact that we collected a single milk sample at the beginning of milking [27].

On the contrary to what has been suggested in large-scale studies [2, 21], the changes in milk production under the effect of mastitis strongly depend on the type of germ. In particular, *Escherichia coli* mastitis induces the greatest and most durable milk yield reductions, when compared with other types of mastitis. The magnitude of this response suggests that at least in the short term, the production of all quarters in the udder was reduced by a systemic effect of *Escherichia coli* infection [33]. With the other germs, the milk loss of one quarter was more or less compensated by an increase in production of the other quarters [32]. Milk yield evolution patterns that we observed were consistent with those previously described on a weekly observation scale [16]. They validated the hypothesis of a marked effect of the type of germ on milk production and appeared to confirm that the production level and lactation stage are promoting factors for certain types of infection [5]. These results also showed that precise estimates of the milk losses induced by clinical mastitis require that measurements be made daily or weekly to take into account the sometimes very fast resumption (4 days) of pre-infection production levels.

The presence of major pathogens in milk quarters induced very significant modifications of milk chemical composition, greater than those described by Miller et al. [21] in naturally occurring mastitis, but equal to those observed by Auldist et al. [1] in the same conditions, and by several authors in experimentally induced mastitis [9, 29, 33, 34, 40]. These modifications were greater with *Escherichia coli*, including a 14 $g \cdot kg^{-1}$ decrease in lactose and a 7 $g \cdot kg^{-1}$ protein increase. This increase was not the result of a concentration effect on the proteins synthesised by the udder because casein was sharply reduced, but rather to an increase in blood proteins. Such changes were mainly linked to two phenomena [28]: reduced secretory activity of the mammary cells and increased permeability of the mammary epithelium. The sharp increases in serum albumin and IgG concentrations are proof of that phenomenon, which promotes the passage of blood proteins in the milk and lactose escape to the blood [33]. Also, clinical mastitis is classically accompanied by an increase in milk proteolytic activity, which contributes to reducing the casein content [1, 14]. Lastly, in severe *Escherichia coli* mastitis, these modifications may also be linked to a reduced input of the blood precursors (glucose, amino acids) necessary for intramammary synthesis, under the effect of the cows' reduced appetite [33, 38].

Out of the three major germs studied, *Streptococcus uberis* was the least consequential regarding milk characteristics, although its immediate effects on milk production of the four quarters was greater than with *Staphylococcus aureus.* With *Streptococcus uberis,* the somatic cell count was similar to that observed with *Staphylococcus aureus* in the absence of clinical signs. Also, the BSA and IgG concentrations of the milks where *Streptococcus uberis* was present were no different from those of healthy quarters. The weak effect of *Streptococcus uberis* on the composition of quarter milk is hard to explain. Nevertheless, the response time to infection may vary according to the pathogenic variability of the strains and their resistance to antibiotics, as well as to the animals' own resistance. That variability was especially revealed with *Streptococcus uberis* [22, 26, 37].

The increase in somatic cell count and the change in milk chemical composition in the presence of *Staphylococcus aureus* were much more marked when that germ was associated with clinical signs. This result reflects the notion of severity, as already evidenced with other germs in experimental mastitis [9]. But even in the absence of clinical signs, the effects of the presence of *Staphylococcus aureus* on milk composition were significant, including in particular a near-to-three-percentage-point reduction of the casein/total protein ratio. This result was consistent with Urech et al.'s findings [36] on the effect of subclinical mastitis on milk protein composition and could be otherwise explained by the chronicity and relative incurability of most *Staphylococcus aureus* mastitis.

The presence in milk of minor pathogens is not frequently associated with clinical signs [39]. In this study, *Corynebacterium bovis* was not accompanied by any degradation of milk production or composition, consistently with Le Van et al.'s study on natural infections [17]. In contrast, the lack of influence of *Corynebacterium bovis* on somatic cell count is not in accordance with the results from other studies involving natural infections [10, 17, 24], which revealed a slight increase in cell count in the presence of *Corynebacterium bovis*. But in these studies, the presence of clinical signs of mastitis was not taken into consideration although it could have explained the increase in somatic cell count. Conversely, even in the absence of clinical signs, the presence of coagulase-negative staphylococci in milk decreased lactose concentration and increased cell count, on the contrary to the results from Timms and Schultz's study [35] where no effect on cell count was found. Insofar as these two parameters are the most sensitive to infection, these modifications reflect a reaction, albeit slight, of the udder to the germ.

In conclusion, this study showed that the modifications of the milk chemical composition are linked to the germ, but also that such changes are much more marked in the presence of clinical signs than in subclinical infections. *Escherichia coli*, which associates the strongest responses, both productionwise and compositionwise, appears to be the germ with the most deleterious economic consequences on milk quality.

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