

Original article

## Evolution of milk somatic cell count of cows grazing an alpine pasture according to the infection of udder by pathogens

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**Abstract** — The infection of udder by a pathogen and milk somatic cell count (SCC) of 220 quarters from 55 Abondance and Tarentaise cows coming from three Northern Alps herds were analysed three times: in June when cows use a valley pasture before turning to highland pasture and in July and September, respectively at the beginning and the end of highland pasture grazing period. During the three periods, 31% of the experimental quarters were free of infection, 61% were infected by a minor pathogen and 8% were infected by a major pathogen. In quarters infected by a major pathogen, SCC was constantly high (> 1 600 000 cells/ml in the three periods). SCC of uninfected quarters remained below 60 000 cells/ml in the three periods whereas SCC of quarters infected by a minor pathogen averaged 89 000 cells/ml in June and 512 000 cells/ml in September. For the latter, SCC was all the higher as the infection was older. Results are discussed according to the highland grazing conditions that may have had an impact on SCC.

**milk / somatic cell count / udder infection / highland pasture**

**Résumé** — **Évolution au cours de l'alpage de la concentration en cellules somatiques du lait de vache en relation avec l'état infectieux des mamelles.** Le statut infectieux et la concentration en cellules somatiques (CCS) du lait de 220 quartiers issus de 55 vaches de race Abondance et Tarentaise ont été analysés à 3 reprises – avant la montée en alpage lorsque les animaux utilisent des pâturages de vallée (juin), en début de saison d'alpage (juillet) et en fin de saison d'alpage (septembre) – dans 3 troupeaux des Alpes du Nord. Les animaux ont été choisis sur la base de leur concentration en cellules somatiques hivernale (toujours inférieure à 100 000 et 500 000 cellules/ml pour respectivement 75 % et 25 % des animaux). La CCS des laits des quartiers a été mise en relation avec le statut infectieux des quartiers, la conformation des mamelles, le stade de lactation des animaux, le troupeau

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et la période. Au cours des 3 périodes, 31 % des quartiers analysés ont été trouvés non infectés, 61 % étaient infectés par un pathogène mineur et 8 % étaient infectés par un pathogène majeur. La CCS des laits de quartiers a varié en premier lieu en fonction de leur état infectieux : celle des quartiers non infectés a été de 46 000 cellules/ml alors que celle des quartiers infectés par un pathogène mineur et majeur a été respectivement de 152 000 et 1 622 000 cellules/ml. En comparaison, l'influence des autres facteurs testés a été moindre bien que significative. Une forte interaction entre le statut infectieux et la période a également été observée. La CCS des quartiers infectés par un pathogène majeur a été constamment élevée (supérieure à 1 600 000 cellules/ml aux 3 périodes). La CCS des quartiers non infectés est restée inférieure à 60 000 cellules/ml aux 3 périodes alors que celle des quartiers infectés par un pathogène mineur était en moyenne de 89 000 avant la montée en alpage et de 512 000 cellules/ml en fin d'alpage. L'augmentation a été d'autant plus importante que l'infection était plus ancienne. La CCS des laits de quartiers infectés par un pathogène mineur a diminué après la descente d'alpage (-128 000 cellules/ml). Les résultats sont discutés en fonction des différentes conditions de l'alpage pouvant expliquer son effet sur la CCS. Ces résultats montrent que dans les régions où l'utilisation d'alpages est fréquente, il est important de prévenir les infections, y compris celles dues à des pathogènes mineurs qui, dans ces conditions, peuvent contribuer de manière plus importante à la CCS des laits de tank et donc pénaliser l'éleveur.

## lait / concentration en cellules somatiques / état infectieux / alpage

### 1. INTRODUCTION

Somatic cell count (SCC) is a significant factor of milk quality valuation. It is a well known indicator of udder inflammation secondary to infection [17, 23] whose basic risks are linked to the animals [16] or husbandry conditions (milking, housing) [21, 23]. Udder infections are responsible for a decrease in milk yield [6, 14] and alteration of the milk characteristics [2, 12] which may induce sanitary or technological consequences [4, 23]. In the absence of mastitis, the lactation stage or parity [2, 20, 22, 24], the age [13, 20] or the breed [8] have an impact on milk SCC. It has recently been demonstrated, under experimental conditions, that prolonged and forced walking by cows could induce an increase in SCC [7] all the more marked as the udder was previously infected [9]. Milk SCC in Northern Alps herds sometimes remains high in certain situations, in summer in particular and especially in breeders who use alpine pastures [1], without any clear explanation for that finding.

The aim of this study was to analyse the respective effects of udder infection by

pathogens and husbandry conditions of herds grazing an alpine pasture on SCC evolution during the summer period.

### 2. MATERIALS AND METHODS

#### 2.1. Animal and herd characteristics (Tab. I)

The study was conducted in 3 herds using alpine pastures in the area where Beaufort cheese is produced (Savoie, France). Herds were composed of cows coming from a single farm on one case and from a grouping of cows from several farms on the other two. Stocking rate, steepness, displacement of the milking machine and milking methods were different from one herd to the other (Tab. I). In particular, teat cleaning was limited to a hand wiping in herd 1 whereas the udders were scrubbed with a single-use piece of dry paper in herd 2 or washed in water and wiped with a single-use paper in herd 3. At the end of milking, grease was occasionally (herds 1 and 3) or systematically (herd 2) used. In addition, a disinfectant was systematically sprayed on the teats of herd 3 cows.

**Table I.** Main characteristics of farms and herds.

	Herd 1	Herd 2	Herd 3
Number of cow during winter	43	35	113
Number of cows in alpine pasture	43	120	200
Winter (1997) SCC (cells/ml) *	40 000	80 000	150 000
SCC in alpine pasture season (1996) (cells/ml) *	350 000	500 000	600 000
Minimum elevation of alpine pasture (m)	1300	1200	1300
Maximum elevation of alpine pasture (m)	1900	2000	2300
<b>Period 1</b>			
Milk yield (kg/cow/day)	16.2	16.9	19.8
Fat (g/kg)	39.0	37.3	35.7
Protein (g/kg)	29.4	32.5	33.4
Lactation stage (days)	107	143	142
Non infected quarters (%)	53	21	35
Displacements of cows**	++	+	+
Steepness of the grazing areas**	0	++	++
<b>Period 2</b>			
Milk yield (kg/cow/day)	19.2	15.0	17.1
Fat (g/kg)	44.4	38.6	31.3
Protein (g/kg)	30.7	32.1	32.5
Non infected quarters (%)	33	36	25
Displacements of cows**	+	+	+
Steepness of the grazing areas**	+	++	++
<b>Period 3</b>			
Milk yield (kg/cow/day)	9.0	10.0	14.6
Fat (g/kg)	53.4	36.5	33.4
Protein (g/kg)	31.2	34.9	34.4
Non infected quarters (%)	28	24	25
Displacements of cows**	++	+	+++
Steepness of the grazing areas**	++	+++	+++
Decrease of milk yield between periods 1 et 3 (%)	45	41	26

\* Herd geometric mean (data from Contrôle Laitier Savoie (73)).

\*\* 0; nil. +; small. ++; medium. +++; important.

Fifteen to 20 Abondance and Tarentaise cows were selected from each herd according to their lactation stage (under 5 months at the beginning of the trial) and to their SCC in January and April preceding the trial (data provided by the Contrôle Laitier de la Savoie, 1997). To include a high number of non-infected quarters before going up to alpine pasture, 75% of the animals picked in each herd had a SCC constantly below 100 000 from January to April. Other cows had SCC always below 500 000 cells/ml.

## 2.2. Sampling and analyses

In total, 220 quarters from 55 cows were followed-up. Milk samples were taken during three periods: before turning to alpine pasture when cows were grazing on valley pasture and when milking was performed outside with the same milking machine and persons as in alpine pasture (26 May–7 June), at the beginning of alpine pasture season (7–16 July) and at the end of the alpine pasture season (29 August–4 September). In

the course of each period two milk samples were collected from each experimental quarter: one for SCC and the other one for bacteriological characterisation. During periods 1 and 2, these two samplings were performed with one-day interval and during the third period, the specimen for bacteriological analyse was sampled 6 days before and 6 and 9 days after that for SCC in herds 1, 2 and 3, respectively. Of 18 cows of herds 2 and 3 (46 quarters), an additional sampling was performed after returning from alpine pasture (late October), when cows were grazing in the valley. Samples for bacteriology and SCC were collected from the same milking.

Samples were collected manually during the evening milking after the first ejection. Samples for SCC (30 ml) were added bronopol and stored at +4 °C. Somatic cells were counted by epifluorescence microscopy (Fossomatic 360, Foss Electric, Hillerød, Denmark). Quarter milks for bacteriological analyses were sampled in an aseptic manner. The samples were immediately chilled to +4 °C and frozen (−18 °C) within

3 hours following collection. In the laboratory, 0.025 ml of each sample of quarter milk were spread on sheep blood gelose with esculin. After aerobic incubation for 24 to 48 h at 37 °C, the germs were identified according to international NMC guidelines [15].

Pathogens identified were divided in two groups [17] (Tab. II): major pathogens (*Staphylococcus aureus*, *Streptococcus dysgalactiae*, *Streptococcus agalactiae*, *Streptococcus uberis* and other *Streptococcus* sp.) and minor pathogens (negative coagulase *Staphylococcus*: SC- and *Corynebacterium bovis*: CB). Quarters were considered as not infected when microbiological test was negative. Quarters were considered as infected by a minor pathogen when at least one was present in the absence of a major pathogen. Lastly, quarters were considered infected by a major pathogen when at least one was present with or without a minor pathogen.

Individual milk yield, fat and protein content and the lactation stage results were given by the Contrôle Laitier de la Savoie.

**Table II.** Inventory of the udder pathogens.

Infectious status	Number of samples	%
No pathogen	201	31
Minor pathogens	387	61
Negative coagulase <i>Staphylococcus</i>	46	
<i>Corynebacterium bovis</i>	269	
2 minors pathogens	72	
Major pathogens	52	8
<i>Staphylococcus aureus</i>	16	
<i>Streptococcus dysgalactiae</i>	2	
<i>Streptococcus agalactiae</i>	23	
<i>Streptococcus uberis</i> *		
<i>Streptococcus species</i>	1	
2 majors pathogens	4	
1 major + 1 minor pathogen	6	
Subtotal	640	100
Non usable samples	20	
Total	660	

\* Always with other minor pathogen.

### 2.3 Observations on the cows and on the pastures

The cows' udder conformation was assessed once in the course of the trial, from the height of the tip of rear teats in relation to the hock: above, at the same height or below the horizontal plane passing by the top of the hock. During the third period, when the herds were using areas near the top of the alpine pastures, cows displacement was assessed by plotting them individually on a 1/3000th chart every 30 minutes between the morning milking and sunset. During the first two periods, the herds were placed on small plots and these observations were not made. Because of the proximity of the milking machine and the restricted area of the night plot, the mean daily displacement of the cows was below 3 km.

The quarters that were treated for mastitis, the treatment products used and the administration methods were noted during each period.

### 2.4. Statistical analyses

The quarters that underwent medical treatment between two samplings of the same period ( $n = 12$ ) or for which the type of pathogen had changed from one period to another without the quarter being treated ( $n = 21$ ) were excluded from data analysis. Statistical analyses for SCC were performed based on data expressed as decimal logarithms. The results presented in the tables, the text and figures have been converted into geometric means.

In a first step, all useable SCC were processed by analysis of variance (GLM procedure [19]) by introducing in the model the effects of the infectious status, the period, the herd, the lactation stage of the cows during the first period (3 classes: < 100 days, between 100 and 150 days and > 150 days), the height of the teat bottom in relation to the hock and the interactions between infection

and period, herd and period and lactation stage and period. The latter interaction was not significant and was not included in the final model.

In a second step, SCC evolutions were explored according to the infectious status of quarters in the three periods. Three hundred and ninety six observations were retained, distributed among five groups: quarters not infected in any period, not infected in the first two periods then infected by a minor pathogen, not infected in the first period then infected by a minor pathogen in the last two periods, infected by a minor pathogen in the three periods, and lastly infected by a major pathogen in the three periods. The statistical model used was identical to that previously described.

Lastly, SCC in quarters infected by minor pathogens in periods 3 and 4 was compared by analysis of variance by introducing the period and the quarter in the model.

## 3. RESULTS

On average, individual daily milk yield varied from 17.7 kg during period 1 and 11.3 kg during period 3 (Tab. I). Clinical mastitis were noted in 38 quarters (9 in period 2 and 29 in period 3). These quarters were medically treated. None of those quarters was used in the second statistical analysis. In contrast, for the first analysis, only those quarters that were treated between the sampling of the bacteriological specimen and that for SCC specimen were excluded (4 in period 2 and 8 in period 3).

### 3.1. Inventory of non infected and infected quarters

The proportion of quarters not infected during the 3 sampling periods averaged at 31% (35% in period 1 and 25% in period 3). Although the proportion of non-infected quarters differed from one herd to the other during period 1 (53, 33 and 28% in herds 1, 2 and 3, respectively), the proportion of

non-infected quarters at the end of alpine pasture season was 25% in all 3 herds alike. Minor pathogens were found in 88% of infected quarters. *Corynebacterium bovis* was largely predominant among minor pathogens, by comparison with negative coagulase *Staphylococci* (Tab. II). Major pathogens were found in 12% of infected quarters. They were most often found in herd 3 where they represented 26% of infected quarters. *Streptococcus agalactiae* and *Staphylococcus aureus* were the most frequent. *Streptococcus agalactiae* was only found in herd 3.

### 3.2. SCC variation factors (Tab. III)

The mean SCC of the milk from animals whose lactation stage was under 100 days during the first period was significantly lower ( $P < 0.001$ ) than that of cows whose lactation stage was above 150 days (129 000 vs. 257 000 cells/ml). The mean SCC was 324 000 cells/ml in cows where the tip of the teats was below the hock and below 200 000 cells/ml in those where the teat tip was at the same level or above the hock ( $P < 0.001$ ). The herd also had a significant effect on SCC ( $P < 0.01$ ); it was lower in

**Table III.** Somatic cell count variation factors.

	nb	SCC (cells/ml)	significance
Quarter infectious status			***
Not infected	197	45 709	a
<i>Corynebacterium bovis</i>	246	151 356	b
Negative coagulase <i>Staphylococcus</i>	35	194 984	b
<i>Corynebacterium bovis</i> and negative coagulase <i>Staphylococcus</i>	68	138 038	b
Major pathogen <sup>1</sup>	41	1 621 810	c
Period			***
26 May-7 June	198	100 000	a
7-16 July	195	199 526	b
29 August-4 September	194	398 107	c
Herds			**
1	191	194 984	ab
2	183	257 040	b
3	213	158 489	a
Lactation stage in period 1			***
< 100 days	166	128 825	a
100-150 days	212	234 423	b
> 150 days	209	257 040	b
Height of the tip of the teats			***
above the hock	221	181 970	b
at the same level of the hock	181	131 826	a
below the hock	185	323 594	c
Infectious status * period			***
herd * period			ns

<sup>1</sup>: *Staphylococcus aureus* or *Streptococcus dysgalactiae* or *Streptococcus agalactiae* or *Streptococcus uberis* or other *Streptococcus* sp.

a,b,c: values with different letters are different at 0.05 level of significance.

ns: not significant; \*\* :  $P < 0.01$ ; \*\*\*:  $P < 0.001$ .

Residual Standard Deviation = 0.55 (log (cells/ml)).

herd 3 (158 000 cells/ml), higher in herd 2 (257 000 cells/ml) and medium in herd 1 (195 000 cells/ml).

The mean SCC of quarters not infected or infected by a minor or major pathogen was 46 000, 152 000 and 1 622 000 cells/ml, respectively ( $P < 0.001$ ). No significant difference was noted between the SCC of quarters infected by a minor pathogen according to the infectious agent (CB, SC- or CB/SC-) ( $P > 0.1$ ). The SCC means were higher during the two alpine pasture periods ( $P < 0.001$ ); 100 000 cells/ml in period 1, 200 000 cells/ml in period 2 and 398 000 cells/ml in period 3. There was no interaction between infection and herd ( $P > 0.1$ ). In contrast, a strong interaction was noted between infection and the period ( $P < 0.001$ ).

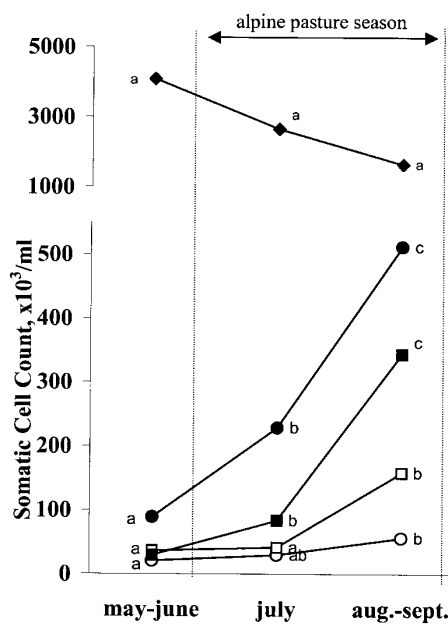
### 3.3. SCC evolution according to quarter infectious status

The mean SCC of quarters not infected in the 3 periods increased significantly ( $P < 0.01$ ) between periods 1 and 3 (Fig. 1) although that increase was not important (+ 36 000 cells/ml). The SCC of quarters infected by a major pathogen in the three periods was always above 1 600 000 cells/ml; the average decrease noted was not significant. In contrast, the mean SCC of quarters infected by a minor pathogen in the three periods increased much more between periods 1 and 3 (+ 423 000 cells/ml,  $P < 0.001$ ). That increase was all the more marked as infection occurred earlier (Fig. 1): the mean SCC of quarters that became infected between periods 1 and 2 and remained infected to the end increased by 315 000 cells/ml ( $P < 0.001$ ) whereas that of quarters that became infected between periods 2 and 3 increased by 122 000 cells/ml ( $P < 0.001$ ). The mean SCC in period 3 was higher in herd 2 (Fig. 2) where SCC was higher than in the other 2 herds in period 1 ( $P < 0.001$ ). Also, between periods 2 and 3, the mean increase in SCC was higher in herd 3 than in herd 1. After returning from alpine pasture,

the mean SCC of quarters infected by a minor pathogen decreased significantly ( $P < 0.05$ ): 300 000 cells/ml in period 3 and 182 000 cells/ml in period 4 (Fig. 3).

## 4. DISCUSSION

In our trial, the mean SCC of milk in May-June was clearly lower than noted by Agabriel et al. [1] in the same period in 50 Northern Alps herds. This can be explained by our decision to include in our sample a large proportion of quarters that were not infected before turning to alpine pasture. That proportion was slightly lower than observed by Faye et al. [10] in cows in early lactation in Brittany farms. The number of



**Figure 1.** Evolution of milk somatic cell count (geometric means) of quarters not infected in the 3 periods (○; 26 quarters), of quarters infected by a minor pathogen in period 3 (□; 8 quarters), in periods 2 and 3 (■; 24 quarters), in the 3 periods (●; 68 quarters) and of quarters infected by a major pathogen in the 3 periods (◆; 6 quarters). Within each evolution, values corresponding to the points with different letters are different at 0.05 level of significance.

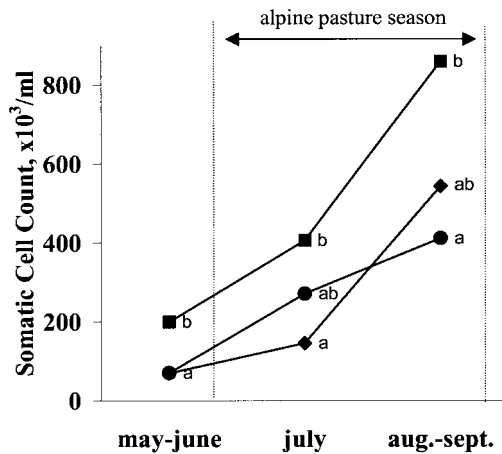
quarters that became infected between the beginning and the end of the study (3 months) was low in herds 2 and 3 and clearly greater in herd 1, where none of the infection-limiting measures were taken [23]. As already observed by a number of authors [5, 10, 23], the pathogens qualified as minor were the most frequent. In contrast, the predominance of *Streptococcus agalactiae* among major pathogens was a surprise because this potentially human pathogen is classically less frequent [10, 21].

Due to the difficulty to sample aseptic specimens during milking in alpine pasture, it was not possible to collect specimens for bacteriology and SCC determination during the same milking. But it is unlikely that this may influence the results because the probability of a quarter becoming infected between the 2 samplings (knowing that 10% of quarters became infected in 100 days) was only 1‰ during the first 2 period and 7‰ during the third period.

This study provided a confirmation and ranking of the known effects of infection of udder by pathogens, lactation stage [8] and udder conformation [11, 16] on SCC. Although they were significant, the lactation stage and udder conformation effects were marginal compared to that of quarter infection (approximately 10 times lower). The latter effect was very close to that

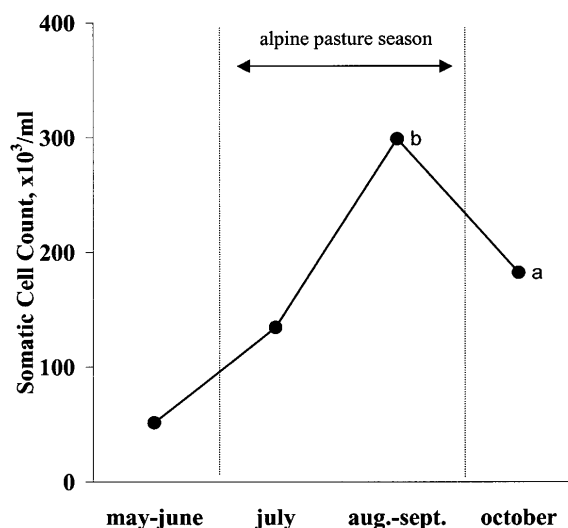
observed by other authors [3, 22]. Our results also showed that type of minor pathogens had no specific effect on SCC, as already noticed in goats [18]. A herd effect was also evidenced regardless of quarter infection, lactation stage and udder conformation, although no factor was found to explain it. It is possible, however, that the herd effect overshadows a cow effect because when taken into account in the statistical model, the herd effect loses significance.

This study mainly revealed a strong interaction between the infection of udder and the period. This variation in quarter behaviour according to their infectious status cannot be ascribed to the lactation stage, parity, age or breed [2, 8, 13, 20, 24] because these characteristics were very close in both groups of cows. The decrease of SCC in quarters infected by a minor pathogen, as observed after returning from alpine pasture, clearly demonstrates the effect of alpine pasture conditions. The level reached remained higher than that before turning to alpine pasture but the difference then was most likely due to the lactation stage (285 days on average at the time of the 4th sampling). Our results were very close to those of Coulon et al. [9] who noted a higher increase in SCC in infected quarters than in non infected quarters after a 10 km/day forced walk. In this study, when we made an estimate, the



**Figure 2.** Evolution of milk somatic cell count (geometric means) of quarters infected by a minor pathogen in the 3 periods within the 3 herds (●, herd 1 (27 quarters); ■, herd 2 (18 quarters); ◆, herd 3 (21 quarters)). Within each period, values corresponding to the points with different letters are different at 0.05 level of significance.





**Figure 3.** Evolution of milk somatic cell count (geometric means) of 30 quarters infected by minor pathogens in the 4 periods. Values corresponding to the points with different letters are different at 0.05 level of significance.

distances covered by the cows were always below 3 km/day. The effort produced by the cows must nonetheless have been significant considering the elevation range and the steepness of the plots. It was indeed in herd 3, managed under the most adverse conditions in the third period, that the SCC increase (between periods 2 and 3) was the highest. Also, as evidenced in a case of stress due to high temperature [25], other factors related to the cows' exposure to weather conditions (wide temperature ranges, rain) or dietary factors (composition of pasture grass) may also have played a role without the possibility for us to verify it as a part of this study.

As for walking [9], it is probable that the different increases in SCC were due to higher cell permeability secondary to a combined effects of non-infectious inflammation of udders due to alpine pasture conditions and microbial infection.

## 5. CONCLUSIONS

This study provides at least partial explanation for the high increases in SCC observed in alpine pasture grazing. The results indeed show that even in the absence

of contamination by major pathogens, herd milks can reach SCC near 500 000 cell/ml at the end of alpine pasture period, considering that the frequency of non-infected quarters at the end of alpine pasture season was only 25% in a group of cows whose SCC before turning to alpine pasture was below that classically observed. This study thus showed that it is of particular importance that udders be exempt of any pathogen before turning to alpine pasture. In practice, these increases are also due to infections occurring during alpine pasture season. In areas where alpine pasture is often used, it is all the more important to prevent infection, including those induced by minor pathogens, which under such conditions can contribute to a greater extent in the SCC of milk and consequently penalise breeders.

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## REFERENCES

- [1] Agabriel C., Coulon J.B., Sibra C., Journal C., Hauwuy A., Facteurs de variation de la numération cellulaire du lait en exploitation, *Ann. Zootech.* 46 (1997) 13–19.
- [2] Auld M.J., Coats S., Rogers G.L., McDowell G.H., Changes in the composition of milk from healthy and mastitic dairy cows during the lactation cycle, *Austr. J. Exp. Agric.* 35 (1995) 427–436.
- [3] Badinand F., Maîtrise du taux cellulaire du lait, *Rec. Méd. Vét.* 170 (1994) 419–427.
- [4] Barbano D.M., Rasmussen R.R., Lynch J.M., Influence of milk cell count and milk age on cheese yield, *J. Dairy Sci.* 74 (1991) 369–388.
- [5] Bartlett P.C., Miller G.Y., Lance S.E., Heider L.E., Clinical mastitis and intramammary infections on Ohio dairy farms, *Prev. Vet. Med.* 12 (1992) 59–71.
- [6] Bartlett P.C., Van Wijk J., Wilson D.J., Green C.D., Miller G.Y., Majewski G.A., Heider L.E., Temporal patterns of lost milk production following clinical mastitis in a large Michigan Holstein herd, *J. Dairy Sci.* 74 (1991) 1561–1572.
- [7] Coulon J.B., Pradel P., Effect of walking on roughage intake and milk yield and composition of Montbéliardes and Tarentaises dairy cows, *Ann. Zootech.* 46 (1997) 139–146.
- [8] Coulon J.B., Dauver F., Garel J.P., Facteurs de variation de la numération cellulaire du lait chez des vaches laitières indemnes de mammmites cliniques, *Inra Prod. Anim.* 9 (1996) 133–139.
- [9] Coulon J.B., Pradel P., Cochard T., Poutrel B., Effect of extreme walking conditions for dairy cows on milk yield, chemical composition, and somatic cell count, *J. Dairy Sci.* 81 (1998) 994–1003.
- [10] Faye B., Dorr N., Lescourret F., Barnouin J., Chassagne M., Les infections intra-mammaires chez la vache laitière dans l'enquête écopathologique Bretagne, *Inra Prod. Anim.* 7 (1994) 55–65.
- [11] Geer D.V.D., Grommers F.J., Van Houten M., Comparison of dairy cows with low or high rate of udder infection, *Vet. Quart.* 1 (1979) 204–211.
- [12] Harmon R.J., Symposium: mastitis and genetic evaluation for somatic cell count, *J. Dairy Sci.* 77 (1994) 2103–2112.
- [13] Kennedy B.W., Sethar M.S., Tong A.K.W., Moxley J.E., Downey B.R., Environmental factors influencing test-day somatic cell counts in Holsteins, *J. Dairy Sci.* 65 (1982) 275–280.
- [14] Lescourret F., Coulon J.B., Modeling the impact of mastitis on milk production by dairy cows, *J. Dairy Sci.* 77 (1994) 2289–2301.
- [15] National Mastitis Council Inc., Microbiological Procedures for the diagnosis of bovine udder infection, Arlington VA, 2201, USA, 1990.
- [16] Poutrel B., La sensibilité aux mammmites : revue des facteurs liés à la vache, *Ann. Rech. Vét.* 14 (1983) 89–104.
- [17] Poutrel B., Généralités sur les mammmites de la vache laitière. Processus infectieux, épidémiologie, diagnostic, méthodes de contrôle, *Réc. Méd. Vét.* 161 (1985) 497–511.
- [18] Poutrel B., Udder infection of goats by coagulase negative staphylococci, *Vet. Microbiol.* 9 (1984) 131–137.
- [19] Statistical Analysis Systems Institute, SAS User's guide: Statistics, SAS Institute, Inc. Cary, north Carolina, USA, 1992.
- [20] Schutz M.M., Hansen L.B., Steuernagel G.R., Kuck A.L., Variation of milk, fat, protein, and somatic cells for dairy cattle, *J. Dairy Sci.* 73 (1990) 484–493.
- [21] Seegers H., Menard J.L., Fourichon C., Mammmites en élevage bovin laitier : importance actuelle, épidémiologie et plans de prévention, *Renc. Rech. Ruminants* 4 (1997) 233–242.
- [22] Serieys F., Concentration cellulaire du lait individuel de vache : influence de l'état d'infection mammaire, du numéro de lactation, du stade de lactation et de la production laitière, *Ann. Rech. Vét.* 16 (1985) 255–261.
- [23] Serieys F., Les mammmites des vaches laitières, Collection « Le point sur », 3<sup>ème</sup> édition, Institut de l'élevage, Paris, 1995.
- [24] Sheldrake R.F., Hoare R.J.T., Mc Gregor G.D., Lactation stage, parity and infection affecting somatic cells, electrical conductivity and serum albumin in milk, *J. Dairy Sci.* 66 (1983) 542–547.
- [25] Wegner T.N., Schuh J.D., Nelson F.E., Stott G.H., Effect of stress on blood leucocyte and milk somatic cell counts in dairy cows, *J. Dairy Sci.* 59 (1976) 949–956.