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A Long Term Study on the Health Status and Performance of Sows on Different Feed Allowances during Late Pregnancy

III. *Escherichia coli* and other bacteria, total cell content, polymorphonuclear leucocytes and pH in colostrum and milk during the first 3 weeks of lactation

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Persson, A., A. Pedersen Mörner and W. Kuhl: A long term study on the health status and performance of sows on different feed allowances during late pregnancy. III. Escheria coli and other bacteria, total cell content, polymorphonuclear leucocytes and pH in colostrum and milk during the first 3 weeks of lactation. Acta vet. scand. 1996, 37, 293-313. – The objectives of this study were to (1) estimate the clinical status of the mammary glands and (2) compare it with the bacteriological findings, the total cell content (TCC) and its percentage of polymorphonuclear leucocytes (PMNLs) and pH in colostrum and milk secretion of sows on 2 different feeding regimes, high versus low, during late pregnancy. The milk samples were collected from both agalactia post partum (APP) sows and clinically healthy sows. Sows with a rectal temperature exceeding 39.5°C within 48 h after parturition were considered to be diseased in APP and treated medically. The sows were sampled on days 1, 3, 8 and 22 of lactation during 6 consecutive lactations.

Irrespective of feeding regimes, 49 out of 77 lactations among the APP sows and 15 out of 96 lactations among the clinically healthy sows revealed *E. coli* in pure cultures with a concomitant TCC exceeding 10×10^6 cell/ml already on the first day of lactation. The healthy sows with *E. coli* infection were denominated as being subclinically infected sows. The intensity in growth of *E. coli* successively declined, and the bacteria were finally eliminated between days 3 and 8 of lactation. The TCC were 82×10^6 cells/ml and 157×10^6 cells/ml in the clinically and subclinically *E. coli* infected glands, respectively, on the first day of sampling. The TCC declined gradually in both groups of sows, but was still higher than in bacteriologically negative milk on day 22 of lactation. The percentages of PMNLs were 66% and 79% in clinically and subclinically infected glands, respectively, on day 1 of lactation, thereafter decreasing to approximately 50% on day 22 of lactation in both groups of sows.

In APP sows, swelling, reddening and/or soreness were registered in 38 out of 87 mammary glands with *E. coli* mastitis on the first sampling occasion.

The TCC in bacteriologically negative colostrum and milk collected from APP sows on day 1 of lactation was significantly higher, 2.27×10^6 cells/ml, when compared with the TCC in bacteriologically negative milk secretion from the clinically healthy or subclinically infected sows, 1.38×10^6 cells/ml versus 1.51×10^6 cells/ml, respectively. The PMNLs were higher on day 1 in clinically healthy sows, 59.6%, than in subclinically infected and APP sows (43.5% and 48.3% respectively).

The pH in secretion from clinically or subclinically *E. coli* infected glands (6.57 versus 6.46) were higher than in bacteriologically negative colostrum samples (6.29) from clinically diseased sows on the first day of sampling. On day 22 of lactation, pH-values had

stabilized on a level of approximately 7.00 in all milk samples from earlier bacteriologically positive or negative mammary glands.
The 2 feeding regimes, low versus high, were not found to influence TCC, PMNLs or pH except for TCC in bacteriologically negative samples of APP sows (2.69 versus 3.62).
The lactation number influenced the PMNLs in both groups of sows with *E. coli* infected mammary glands, and both the TCC and PMNLs in bacteriologically negative colostrum and milk.

agalactia; post partum; feeding; mastitis.

Introduction

Mastitis in sows caused by coliform bacteria and established during the first postparturient days is today considered to be strongly implicated in the lactation failure often denominated agalactia post partum (APP). Sows with mastitis during the immediate postparturient period show elevated rectal temperature together with clinical signs such as anorexia, lower or ceased feed and water consumption. The concomitant firmed, swelled, reddened and sore mammary glands might easily result in reluctance to nurse their piglets *(Hermansson et al.* 1978, *Persson et al.* 1989). Although complete agalactia is the final and most severe consequence of this lactation failure, early detection and adequate treatment of the disease often allows colostrum and milk production to be maintained on a level sufficient to eliminate the risk of subsequent starvation and high susceptibility to infections in the offspring *(Jorsal* 1983).

Following palpation of mammary glands with physiological oedema in newly farrowed sows, a tentative diagnosis of mastitis can be proposed. Necropsy of diseased and healthy sows with complementary histological and microbiological examinations has substantiated the diagnosis of mastitis in a majority of the cases *(Ringarp* 1960, *Martin et al.* 1967, *Armstrong et al.* 1968, *Thurman et al.* 1970, *Bertschinger et al.* 1977, *Middleton-Williams et al.* 1977, *Ross et al.* 1981). *Cross et al.* (1958) and *Swarbrick* (1968) used biopsy technique to obtain single or repeated samples from mammary glands, which made it possible to study histological changes during the lactation period immediately after farrowing.

Post mortem examinations of mammary glands from clinically healthy and diseased sows euthanasied shortly postfarrowing have contributed sufficient microbiological and histological results on the etiology and pathogenesis of the APP to allow us to exploit the colostrum and milk secretions as a source for combined bacteriological, cytological, and chemical analysis to set the diagnosis. *Ringarp* (1960) examined 167 milk samples from sows with agalactia toxaemica and obtained *Escherichia coli (E. coli)* in pure cultures in 78 samples. These acute cases of mastitis of galactogenic origin were claimed to be independent entities, and he considered agal. toxaemica to be caused by gastro-intestinal infections and intoxications.

From sows that farrowed within the previous 48 h, *Armstrong et al.* (1968) and *Ross et al.* (1975) presented bacteriological results obtained from milk secretion sampled after thorough cleansing of the teat ends. *E. coli* and *Klebsiella* spp. were the major findings in their studies. Further, *Ross et al.* (1981) collected milk samples 1 to 2 days post partum from anesthetized sows, which immediately afterwards were exsanguinated. *E. coli* were isolated in 29 out of 37 mastitic glands from agalactic sows. Other bacteriological findings were *Klebsiella* spp, *Staphylococcus* spp. and β–hemolytic

streptococci. Cytological analysis revealed, on average, a total somatic cell count of 34.8 ± 38.3×10^6/ml milk from these mastitic mammary glands. According to *Wegmann* (1985) the criteria for an *E. coli* mastitis in the sow is a total cell count exceeding 5.0×10^6 cells/ml and a percentage of polymorphonuclear leucocytes, (PMNLs), ≥70%. *Bertschinger et al.* (1990) used the same methods and criteria to determine the reduction of the incidence of puerperal mastitis by protection of the mammary glands against faecal contamination *(E. coli).*

The aims of the present investigation were to count total cell content (TCC) and the PMNLs and measure the pH in either bacteriologically negative or *E. coli* positive colostrum and milk samples from sows clinically diseased in APP and from sows clinically healthy with or without mammary glands subclinically infected with *E. coli.*

Materials and methods

Animals and feeding

A total of 39 pairs of full sibs (Swedish Landrace×Swedish Yorkshire) were used in the experiment out of which 24 entered the trial as gilts. Each sow was then, when possible, kept for 6 parities.

One sow in each pair was randomly allocated to the control group and the other to the experimental group. Three weeks before expected farrowing, the sows were transferred to the farrowing pens. All sows were kept loose during farrowing and lactation.

The animals in the control group (C) were fed 2.4 kg/day for the first 100 days of pregnancy and then 3.4 kg/day from day 100 of gestation until farrowing. The experimental group (E) was fed 1.0 kg/day during the last 15 days of gestation. The experimental group was offered extra feed between days 30 and 100 of gestation. The total amount of feed given during the gestation period was equalized for both groups. The composition of the feed was described by *Persson et al.* (1989). During lactation the daily feed allowance was successively increased for all sows to a maximum of 4.0 kg + 0.2 per piglet within 14 days after farrowing.

Clinical examination and collection of colostrum and milk samples

The rectal temperature was monitored every morning and evening, starting 2 days before expected farrowing and continuing until 2 days after parturition. Sows with a temperature exceeding 39.5°C within 48 h after parturition were considered to be diseased in agalactia post partum (APP) and were clinically examined and treated parenterally with antibiotics and oxytocin (Partoxin vet, Pherrovet, Malmö, Sweden). Severely affected animals were also treated with corticosteroids.

Colostrum samples were collected on the day of parturition, day 1, from all healthy sows farrowing during working hours and from diseased sows regardless of time of farrowing. The sows not showing symptoms of disease were milk sampled as healthy controls, but no clinical examination was performed. Piglets were removed from the sow 30 min before milking. The udder was thoroughly washed with soap solution and warm water followed by careful disinfection with iodine and 70% alcohol. Oxytocin was given i.m. (20 IU) to promote milk let-down. The 2 first streams of secretion were discarded from both canals of the teat. Approximately 0.5 ml secretion was collected for bacteriological examination in sterile plastic tubes followed by an additional 2 ml for cytological analysis. Milk from both canals of each teat was pooled. Four to 8 mammary glands of each sow were milked. The colostrum and milk samples from APP sows were collected from mammary glands with clinical signs of mastitis such as hardening, swelling, and erythema and simultaneously from the mammary gland opposite the

Table 1A. Classification of colostrum and milk samples in sows according to clinical status, bacteriology and cytology.

Health status	Definition of samples
I. Agalactia post partum sows	
A1 " "	→ *E. coli* infected glands, TCC>10×10^6 cells/ml
A2 " "	→ noninfected glands
II. Clinically healthy sows	
A1 With subclinical mastitis	→ *E. coli* infected glands, TCC>10×10^6 cells/ml
A2 " " "	→ noninfected glands
B Without subclinical mastitis	→ noninfected glands

Table 1B. Number of colostrum and milk samples obtained for bacteriological and cytological analysis during 6 consecutive lactations. The sows are divided into the following groups: agalactia post partum (IA1, IA2) [77 lactations from 44 sows], healthy sows with subclinical mastitis (IIA1, IIA2) [31 lactations from 28 sows] and healthy sows without mastitis (IIB) [65 lactations from 39 sows].

Clinical status of the sows	Lactation number						
	1	2	3	4	5	6	*All*
IA1+IA2. Agalactia post partum							
Number of lactations	5	12	13	21	16	10	*77*
Number of samples	33	66	122	184	215	101	*721*
II. Healthy sows							
A1+A2. With subclinical mastitis							
Number of lactations	3	5	10	2	3	8	*31*
Number of samples	20	44	115	21	38	60	*298*
B. Without subclinical mastitis							
Number of lactations	13	8	10	9	15	10	*65*
Number of samples	97	107	110	111	184	117	*726*

inflamed one. The sampling procedure was repeated 2, 7 and 21 days after the first sampling. The milk samples collected during working hours were immediately sent to the laboratory and cultured bacteriologically. Preparation of smears for total and differential cell counts and pH-measurements were performed. Milk samples collected outside working hours were stored cold or at –20°C until analysed. Within the 3 categories of sows, defined later in this text as clinically diseased (APP) and clinically healthy with or without subclinical *E. coli* mastitis in one or more mammary glands, 32%, 13% and 17%, respectively, of the milk samples were stored cold until analysed. The number of colostrum and milk samples collected are presented in Tables 1B and 1C. Due to undesired bacteriological contamination connected with the sampling procedure or by other bacteriological growth, some of the cell counts were excluded. Colostrum and milk samples were collected during altogether 77 lactations of APP sows and 96 lactations of clinically healthy sows.

Table 1C. Number of colostrum and milk samples obtained for bacteriological and cytological analysis on four sampling occasions during lactation. The number of samples within brackets are samples with growth of *E. coli* on day 1 plus samples that are bacteriologically negative. The sows are either clinically diseased in agalactia post partum (IA1, IA2) or clinically healthy with (IIA1, IIA2) or without (IIB) subclinical mastitis.

Clinical status the sows	Day of sampling				*All*
	1	3	8	22	
IA1+IA2. Agalactia post partum					
Number of samples	229 (87+142)	175 (72+103)	167 (66+101)	150 (62+88)	*721*
II. Healthy sows					
A1+A2. With subclinical mastitis					
Number of samples	119 (17+102)	71 (13+58)	61 (14+47)	47 (15+32)	*298*
B. Without subclinical mastitis					
Number of samples	249	210	149	118	*726*

Cell count

The total cell count was performed according to a method originally described by *Prescott & Breed* (1910) and modified based on the norms issued by the Sub-committee on Screening Tests, United States National Mastitis Council (1968).

Two smears were prepared from each sample on carefully cleaned slides. 0.01 ml of milk was placed on the slide by means of a microsyringe. Each drop was spread over an area of 0.5×2 cm (1 sq.cm) and allowed to dry over-night. Methanol was then used as fixative and the smears were again allowed to dry until the next day. On day 3, the fixed smears were immersed in Newman's stain and allowed to dry overnight. On day 4 stained smears were carefully washed in water and again allowed to dry until day 5 when total cell count and differential counts of polymorphonuclear leucocytes were carried out at a magnification of ×1000 under oil in a light microscope.

Bacteriological examination

With a calibrated plastic inoculating loop (A/S Nunc, Roskilde, Denmark), approximately 10 µl of the colostrum and milk was spread onto blood agar containing 5% horse blood and onto agar containing 1% lactose. The agar plates were examined after incubation for 24 h at 37°C. In addition, duplicate blood agar plates were incubated anaerobically for 24 h at 37°C in BBL Gas Pak® System Oxoid Gas Generating Kit (Oxoid Ltd., Hampshire, England). Bacterial colonies were identified macroscopically, microscopically, and by API 20E biochemical profiles following the manufacturer's instruction (API 20E System S.A., La Balmes Grottes, France). Identified bacteria were also analysed serologically regarding O-antigen and fimbrial structures. Sensitivity tests of *Escherichia coli* to different antibiotics were also performed. Regarding the bacteriology analyses, more comprehensive information is accessible in a manuscript by *Pedersen et al.* (1996).

When the agar plates were examined, >100 CFU / inoculum, (colony forming units), revealed a profuse growth of *E. coli,* 10–100 CFU/inoculum gave a moderate growth of *E. coli* and <10 CFU/inoculum a sparse growth of *E. coli.*

Determination of pH

The pH of fresh colostrum and milk delivered immediately to the laboratory was determined with a digital pH-meter (Orion Research model 701 A).

Classification of colostrum and milk samples according to clinical status, bacteriology and cytology

Before the statistical analysis was performed, the colostrum and milk samples were divided into 2 categories according to the health status of the sows (Table 1A). The sows were clinically classified as clinically diseased (agalactia post partum) or clinically healthy. In clinically diseased sows the mammary glands were considered to be mastitic, if the bacteriological and cytotological analyses revealed *E. coli* in pure culture with a concomitant total cell content, TCC, exceeding 10×10^6 cells/ml on the first sampling occasion. The threshold value of 10×10^6 cells/ml was established from bacteriologically negative colostrum and milk samples in clinically healthy sows by calculating the mean ± 2 SD, and was found to be close to 10×10^6 cells/ml. When bacteriological culturing was performed from colostrum and milk samples collected from clinically healthy sows, a subgroup revealed growth of *E. coli* in pure cultures and with a TCC exceeding 10×10^6 cells/ml on the first or second sampling occasion from one or more mammary glands. This group was denoted subclinical *E. coli* mastitic sows.

Statistical analyses were performed on TCC, PMNLs and pH from mammary glands clinically or subclinically infected with *E. coli,* but also from bacteriologically negative colostrum and milk samples obtained from all sows (Table 1A).

Statistical methods

Statistical evaluation was performed using analysis of variance (GLM procedures) available from the Statistical Analysis System (SAS 1985).

The material was statistically analysed in 2 groups: a) samples from *E. coli* infected glands and b) samples from non-infected glands. The response variables: TCC, PMNLs and the pH-value were analysed according to different statistical models.

Model 1: The effect of feeding and the effect of health status of the sows and the interaction between these 2 effects. Feeding was represented by 2 classes, restricted (E) versus high feeding level (C). Health status in material a was represented by 2 classes; sows clinically infected with *E. coli* and sows healthy but subclinically infected with *E. coli,* together with a TCC exceeding 10×10^6 cells/ml in at least one mammary gland on the first sampling occasion. Health status in material b was represented by 3 classes; sows clinically diseased (agal. post partum), sows clinically healthy but subclinically infected with *E. coli,* and sows clinically healthy without subclinical matitis.

Model 2: The effect of day of sampling (4 classes; days 1, 3, 8 and 22 after farrowing), the effect of health status of the sows (2 or 3 classes; see the health status as designated above) and the interaction between these 2 effects.

Model 3: The effect of lactation number (6 classes; lactations 1 to 6), the effect of health status of the sows (2 or 3 classes; see above) and the interaction between these 2 effects.

Using the analysis of variance requires that the observations are normally distributed. Since the variable TCC is far from normally distributed, these values were transformed by using logarithmic transformation before statistical evaluations were performed. The statistical tests are presented directly from obtained results, while the numerical values of the TCC are presented as antilogarithmics of least-square means for the log-values (later called geometric mean)

Table 2. Bacteriological growth on the first sampling occasion and TCC in colostrum and milk from agalactic and clinically healthy sows. A comparison between sows in groups C and E with mastitis (TCC>10×10^6 cells/ml), or with no mastitis. Numbers within brackets indicate that the TCC did not exceed 10×10^6 cells/ml until the second sampling occasion. Figures presented in *italics* denote lactations from the group of sows with bacteriological growth and selected to the subclinical group, although *4* sows (*1+3*) did not reach the TCC-threshold.

	Agalactia post partum			Clinically healthy				*All*
	C	E	All	C		E		
Number of lactations	51	26	77	Mastitis 10	No mastitis 27	Mastitis 17	No mastitis 42	*96*
Bacteriology								
E. coli								
–TCC>10	33(+4)	16(+4[1])	49(+8)	9		6(+8)		15(+8)
–TCC≤10	4	2	6		1+1		3+2	4+3
Other species								
–TCC>10	2	2	4	1		(+3)		1(+3)
–TCC≤10	2		2		1		9	10
No growth	6	2	8		24		28	52

1. Two of these sows did not fulfil any of the two definitions of *E. coli* with a concomitant TCC>10×10^6 cells/ml until the second sampling.

with a 95% confidence interval within parenthesis.

Results

Bacteriology

Bacteriology with special reference to Escherichia coli. The bacteriological findings in the colostrum and milk in sows with agalactia post partum and in clinically healthy sows are presented in Table 2. As is evident from this table, *E. coli* infection was diagnosed in 41 out of 51 (= 80%) lactations in the control sows and in 22 out of 26 (= 84%) in the experimental sows. Information is missing from one sow belonging to the control group. In a few cases (compare Table 2) other bacterial infections were diagnosed, such as α- and β-hemolytic streptococci, *Staphylococcus* spp., anaerobic grampositive cocci, and *Enterobacter agglomerans*. In the majority of the *E. coli* positive samples the TCC exceeded 10×10^6 cells/ml already on the first sampling occasion (Table 2). *E. coli* positive samples from 4 lactations in the control group and 2 lactations in the experimental group revealed no elevation in TCC on either the first or the second sampling occasion. In 6 lactations in the control group and 2 lactations in the experimental group, no bacterial infection was diagnosed. Two of these sows in the control group showed elevated TCC (>10×10^6 cells/ml) on day one of sampling and another 2 sows were clinically affected with metritis.

Among the clinically healthy sows, *E. coli* infection was diagnosed in samples from one or more mammary glands on the first sampling occasion in 11 out of 37 (= 30%) lactations in the control group and in 19 out of 59 (= 32%) lactations in the experimental group. In 15 out of 23 lactations with *E. coli* infection, the TCC exceeded 10×10^6 cells/ml already on the first sampling occasion.

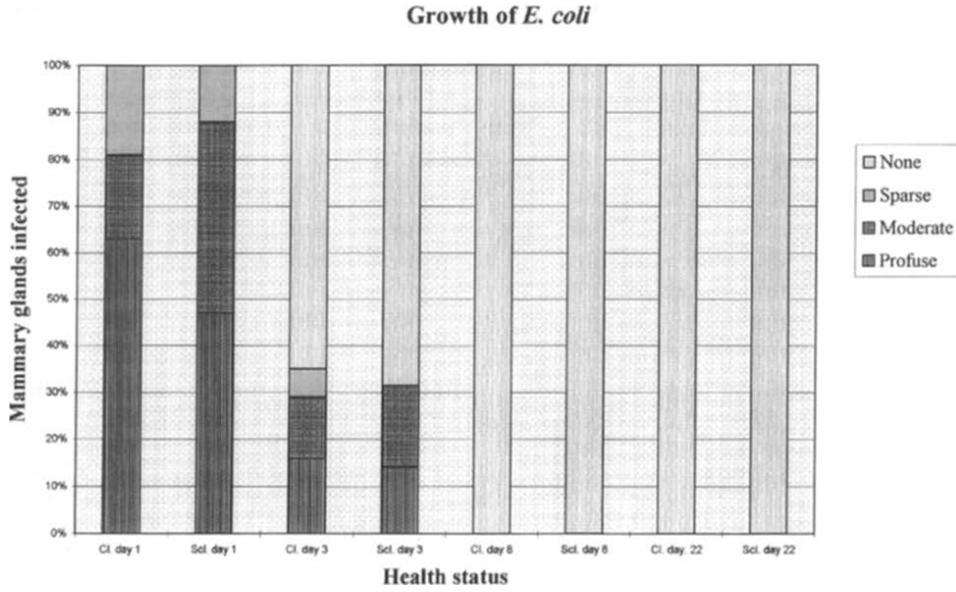

Figure 1. Colostrum and milk samples from agalactia post partum sows (116 mammary glands) and clinically healthy sows (29 mammary glands), which revealed growth of *E. coli* already on the first sampling occasion. Each bar is, on day 1 of lactation, divided into 3 separate parts, which depict a profuse growth of *E. coli,* comparable with >100 CFU/ inoculum, a moderate growth comparable with 10-100 CFU/inoculum and a sparse growth comparable with <10 CFU/inoculum. On subsequent sampling days (3, 8 and 22) open bars indicate bacteriologically negative samples.

Intensity in growth of E. coli. The intensity of *E. coli* growth in colostrum and milk samples obtained from mammary glands in sows clinically or subclinically infected with *E. coli* already at the first sampling are presented in Figure 1. In the clinically diseased sows, 87 mammary glands out of 116, and in the subclinical group, 17 out of 29, exceeded the TCC threshold of 10×10^6 cells/ml already on the first day of sampling. Growth of *E. coli* was revealed only on days 1 and 3.

In clinically diseased sows the bar shows a profuse growth of *E. coli* in 63% of the infected mammary glands, a moderate growth in 18% and a sparse growth in 19% on day one of sampling. On day 3 of lactation, 35% of the milk samples of the clinically infected still had growth of *E. coli* with an intensity grading of 6% profuse, 13% moderate and 16% sparse.

In the subclinically infected sows the intensity of *E. coli* growth was 52% profuse, 34% moderate and 14% sparse on day one of sampling. On day 3, there was moderate growth of *E. coli* in 21% and sparse growth in 17% of the mammary glands.

Relationship between laboratory and clinical diagnosis. In the clinically diseased sows, palpation of the mammary glands infected with *E. coli* and a TCC>10×10^6 cells/ml on the first sampling occasion demonstrated that 38 of 87 mammary glands were mostly characterized by swelling, but also reddening and/or soreness. On day 3 of sampling, these changes were only noted in 10 of the 38

Table 3. The TCC in colostrum and milk samples with growth of *E. coli* in pure cultures. A comparison between sows clinically (IA1) or subclinically (IIA1) infected with *E. coli.* The TCC-values ($\times 10^{-6}$/ml) are presented as geometric means with a 95% confidence interval. The statistical analysis was performed on the original TCC-values after logarithmic transformation and the presentation emanates from these calculations. Numbers of glands are given in parentheses ( ), the number of corresponding sows are given in square brackets [ ].

Day of lactation	TCC in colostrum and milk with growth of *E. coli*	
	IA1	IIA1
1	82.27 (61.12-110.72) $^{a\,A}$ (87) [31]	156.65 (80.00-306.73) $^{a\,A}$ (17) [16]
3	58.08 (41.90-80.50) $^{a\,A}$ (72) [29]	21.65 (10.04-46.69) $^{b\,B}$ (13) [12]
8	13.34 (9.49-18.77) $^{b\,A}$ (66) [28]	4.40 (2.10-9.22) $^{c\,B}$ (14) [13]
22	8.24 (5.80-11.72) $^{b\,A}$ (62) [28]	3.28 (1.60-6.70) $^{c\,B}$ (15) [14]

Mean values within a column without a small superscript letter in common differ significantly ($p<0.01$).
Mean values between columns but on the same day, without a large superscript letter in common, differ significantly ($p<0.05$).

mammary glands. Mammary glands from sows in the experimental group seemed to be easier to palpate; 18 of 26 mammary glands were found to be abnormal compared with 20 of 61 mammary glands from the sows in the control group.

The TCC and its percentage share of PMNLs in colostrum and milk samples with growth of E. coli

The Total Cell Content, TCC, counted in colostrum and milk samples with growth of *E. coli* in pure cultures is presented as geometric mean values with a 95% confidence interval in Table 3.

In sows clinically infected with *E. coli,* the TCC was 82.27×10^6 cells/ml on day one. The TCC decreased gradually from day 1 to day 22, the decrease being significant between days 3 and 8 ($p<0.001$).

In sows subclinically infected with *E. coli,* the TCC in colostrum samples was 156.65×10^6 cells/ml on day one. The TCC decreased thereafter gradually to 3.28×10^6 cells/ml on day 22, the decrease being significant from day 1 to day 3 ($p<0.001$) and from day 3 to day 8 ($p<0.01$).

When a comparison was performed between the 2 groups of sows, the TCC values on day 1 were numerically higher ($p = 0.08$) in the subclinical (156.65) than in the clinical (82.27) sows. On the subsequent sampling days (3,8 and 22), the TCC values were higher in the clinically infected sows than in the subclinical sows ($p<0.05$).

In Table 4, PMNLs expressed as percentages of

Table 4. The PMNLs expressed as its percentage share of the TCC, in colostrum and milk samples with growth of *E. coli* in pure cultures and a TCC exceeding 10×10^6 cells/ml on day one of lactation. A comparison between sows clinically (IA1) or subclinically (IIA1) infected with *E. coli.* The percentage values of PMNLs are represented as least-square mean ± S.E.M. Numbers of glands are given in parentheses ( ), and number of corresponding sows are given in square brackets [ ].

Day of lactation	PMNLs, % in colostrum and milk with growth of *E. coli*	
	IA1	IIA1
1	65.8±2.7 $^{a\,A}$ (87) [31]	79.0±6.4 $^{a\,A}$ (16) [15]
3	54.3±3.0 $^{a\,A}$ (72) [29]	52.9±7.1 $^{b\,A}$ (13) [12]
8	52.1±3.1 $^{b\,A}$ (66) [28]	43.4±6.8 $^{b\,A}$ (14) [13]
22	48.4±3.2 $^{b\,A}$ (62) [28]	54.0±6.6 $^{b\,A}$ (15) [14]

Percentage values within a column without a small superscript letter in common differ significantly ($p<0.01$). Percentage values between columns but on the same day, with a large superscript letter in common, do not differ significantly ($p>0.05$).

the TCC are presented. Within both the clinically and subclinically *E. coli* infected sows there were significant decreases ($p<0.01$) from day 1 to day 3 (65.8 versus 54.3 and 79.0 versus 52.9, respectively). The percentage of PMNLs was numerically higher ($p = 0.0581$) in the subclinically infected sows than in the clinically infected sows (79.0% versus 65.8%). On days 8 and 22 the percentages of PMNLs for both groups of sows were stabilized on a level between 54.0 and 43.4.

The TCC and its percentage of PMNLs in colostrum and milk samples from noninfected mammary glands from sows diagnosed as clinically healthy, clinically healthy but subclinically infected with E. coli in some glands, or from clinically diseased sows – agalactia post partum

The results are presented in Table 5. In agalactic sows and in sows subclinically infected with *E. coli,* the geometric mean of the TCC-values increased ($p<0.001$) from day 1 to day 3 and thereafter decreased ($p<0.01$) to day 8. There was no change in TCC from day 8 to day 22.

In healthy sows, the TCC increased significantly ($p<0.001$) from day 1 to day 3 (1.38 versus 3.18). On day 22, the TCC values, 2.10×10^6 cells/ml, were significantly lower ($p<0.05$) than on day 3.

The TCC values from colostrum samples on day 1, 2.27×10^6 cells/ml, obtained from mammary glands in agalactic sows, were significantly higher ($p<0.05$) than from samples on the same day obtained from the other 2 groups of sows (1.51 and 1.38, respectively). On day 3 in agalactic sows the TCC, 4.86×10^6 cells/ml,

Table 5. The TCC in bacteriologically negative colostrum and milk, collected from mammary glands of sows clinically affected by agalactia post partum (IA2), sows clinically healthy with some mammary glands infected with *E. coli* (IIA2) or sows clinically healthy (IIB). The TCC-values ($\times 10^{-6}$/ml) are presented as geometric means with a 95% confidence interval. Numbers of glands are given in parentheses ( ), the number of corresponding sows are given in square brackets [ ]. The statistical analysis was performed on the TCC-values after logarithmic transformation and the statistical presentation emanates from these calculations

Day of lactation	TCC in bacteriologically negative colostrum and milk		
	IA2	IIA2	IIB
1	2.27 (1.79-2.88)$^{a\,A}$ (142) [38]	1.51 (1.14-2.00)$^{a\,B}$ (102) [28]	1.38 (1.15-1.65)$^{a\,B}$ (249) [37]
3	4.86 (3.68-6.41)$^{b\,A}$ (103) [33]	4.15 (2.87-6.01)$^{b\,AC}$ (58) [22]	3.18 (2.61-3.86)$^{b\,BC}$ (210) [35]
8	2.80 (2.12-3.71)$^{a\,A}$ (101) [32]	1.49 (0.99-2.25)$^{a\,B}$ (47) [22]	2.37 (1.88-2.99)$^{bc\,A}$ (149) [34]
22	2.79 (2.07-3.77)$^{a\,A}$ (88) [31]	1.99 (1.21-3.28)$^{a\,A}$ (32) [16]	2.10 (1.62-2.72)$^{c\,A}$ (118) [31]

Mean values within a column without a small superscript letter in common differ significantly ($p<0.05$).
Mean values between columns but on the same day, without a large superscript letter in common, differ significantly ($p<0.05$).

was significantly higher ($p<0.05$) compared with the TCC, 3.18×10^6 cells/ml, obtained from the healthy sows. Sows subclinically infected with *E. coli* revealed a TCC, 1.49×10^6 cells/ml, on day 8 that was significantly lower ($p<0.05$) compared with the other 2 groups of sows.

The percentage of PMNLs on day 1 in bacteriologically negative colostrum from clinically healthy sows, Table 6 was significantly higher ($p<0.001$) than in colostrum from the other 2 groups of sows (59.6% versus 48.3% and 43.5%, respectively). The percentage of PMNLs within the 3 groups of sows thereafter decreased to values between 36.5 and 50.0, but this decrease was not significant in clinically healthy sows with subclinical mastitis.

pH in colostrum and milk with growth of E. coli or without bacteriological growth

Table 7 shows the pH-values in colostrum and milk samples collected from sows clinically or subclinically infected with *E. coli.* The pH-values gradually increased between day 1 and day 8, the increase being significant ($p<0.01$) between day 3 and day 8, thereafter stabilizing on the day 8 level.

The pH-values in colostrum and milk without bacteriological growth and obtained from the 3 groups of sows are presented in Table 8. The pH-values on days 1 and 3 were significantly higher ($p<0.01$) in samples from agalactic sows than in the other 2 groups of sows. There were significant increases in pH between days 1 and 3 ($p<0.001$) and between days 3 and 8

Table 6. The PMNLs expressed as their percentage share of the TCC in bacteriologically negative colostrum and milk from mammary glands of sows affected by agalactia post partum (IA2), sows clinically healthy with some mammary glands infected with *E. coli* (IIA2) or sows clinically healthy (IIB). The percentage values of PMNLs are represented as least-square mean ± S.E.M. Numbers of glands are given in parentheses ( ), the number of corresponding sows are given in square brackets [ ].

Day of lactation	PMNLs, % in bacteriologically negative colostrum and milk		
	IA2	IIA2	IIB
1	$48.3\pm2.2^{ab\,A}$ (139) [38]	$43.5\pm2.7^{a\,A}$ (98) [27]	$59.6\pm1.7^{a\,B}$ (247) [37]
3	$50.0\pm2.6^{a\,A}$ (101) [33]	$43.3\pm3.5^{a\,A}$ (58) [22]	$49.1\pm1.8^{b\,A}$ (207) [35]
8	$44.8\pm2.7^{ab\,A}$ (99) [32]	$36.5\pm3.9^{a\,A}$ (46) [21]	$43.6\pm2.2^{b\,A}$ (146) [34]
22	$42.2\pm2.8^{b\,A}$ (87) [31]	$38.1\pm5.1^{a\,A}$ (27) [15]	$48.9\pm2.5^{b\,A}$ (111) [31]

Percentage values within a column without a small superscript letter in common differ significantly ($p<0.05$). Percentage values between columns but on the same day, without a large superscript letter in common, differ significantly ($p<0.001$).

($p<0.001$) in all 3 groups of sows. Thereafter, the pH stabilized on approximately 7.0 except in healthy sows without subclinical mastitis, where a small but significant decrease ($p< 0.01$) was seen.

The influences of feeding regime and lactation number on TCC, PMNLs and pH in colostrum and milk collected from mammary glands infected with E. coli or from non-infected glands

Mammary glands infected with E. coli. The 2 different feeding regimes did not influence the TCC, the percentage of PMNLs, or the pH in sows clinically or subclinically infected with *E. coli* (Table 9).

The lactation number did not influence the TCC in the 2 clinical groups of sows (Table 10), whereas it did have a significant influence ($p<0.001$) on the percentage of PMNLs. During the 6 consecutive lactations, the sows with clinically or subclinically infected *E. coli* mammary glands showed a PMNL varying from 38.7 to 80.0%. Within both groups of sows, the percentage of PMNLs at the sixth lactation was statistically lower than in the preceding 5 lactations ($p<0.05$).

Due to few pH-values in each lactation it was not possible to analyse the potential influence of lactation number (Table 10).

Mammary glands bacteriologically negative. The 2 different feeding regimes were not found to significantly influence the TCC-values (Table 9) in colostrum and milk samples without bacteriological growth from clinically healthy sows or sows subclinically infected with *E. coli*. In sows belonging to the

Table 7. pH in colostrum and milk with growth of *E. coli* in pure culture. A comparison between mammary glands in sows clinically (IA1) or subclinically (IIA1) infected with *E. coli.* The pH-values are given as least-square means ± S.E.M and the numbers of glands are given in parentheses ( ), the number of corresponding sows are given in square brackets [ ].

Day of lactation	pH in colostrum and milk with growth of E. coli	
	IA1	IIA1
1	6.57±0.07 a (32) [15]	6.46±0.09 a (10) [10]
3	6.66±0.09 a (23) [10]	6.61±0.10 a (8) [8]
8	7.01±0.09 b (24) [11]	7.12±0.10 b (9) [9]
22	6.99±0.09 b (21) [9]	7.24±0.12 b (6) [6]

pH-values within a column without a small superscript letter in common differ significantly (within the first column $p<0.01$ and within the second $p<0.001$).
No statistical evaluation was performed comparing the pH-values between the two clinical categories of sows.

control group and diseased of agalactia post partum, the TCC were significantly higher ($p<0.05$) in colostrum and milk than in diseased sows belonging to the experimental group.

The percentage levels of PMNLs in colostrum and milk samples without bacteriological growth were not influenced by the 2 different feeding regimes (Table 9).

In all 3 clinical groups of sows, the geometric mean value of the TCC (TCC-values below the threshold of 10×10^6 cells/ml) varied between $0.45–2.10\times10^6$ cells/ml in the 6 lactations (Table 10), and the TCC was found to reach a maximum at the fourth or fifth lactation. Lactation number was found to influence the TCC ($p<0.001$).

In the 3 clinical groups of sows the percentage of PMNLs fluctuated between 20.5 and 58.2 during the 6 lactations (Table 10). In sows clinically diseased or healthy but subclinically infected with *E. coli,* the PMNLs tended to decrease when they reached the fifth and sixth lactations, percentage values varying between 44.4 and 20.5, respectively. This tendency was not found in sows clinically healthy, where the PMNLs remained on a higher level (58.2%). The lactation number significantly influenced ($p<0.001$) the percentage of PMNLs.

The pH was not found to be influenced by the 2 different feeding regimes (Table 9). The influence of lactation number on the pH tended to be significant ($p = 0.0578$). The clinically diseased sows showed a significantly higher ($p<0.001$) mean pH of the lactations than in the other 2 clinical groups of sows (Table 10).

Table 8. A comparison between pH-values obtained from bacteriologically negative colostrum and milk. These samples were collected from mammary glands of sows clinically affected by agalactia post partum (IA2), sows clinically healthy with some mammary glands infected with *E. coli* (IIA2), or sows clinically healthy (IIB). The pH-values are given as least-square means ± S.E.M and the numbers of glands within parentheses ( ), the number of corresponding sows are given in square brackets [ ].

Day of lactation	pH in bacteriologically negative colostrum and milk		
	IA2	IIA2	IIB
1	$6.29\pm0.03^{a\,A}$ (51) [15]	$6.18\pm0.03^{a\,B}$ (57) [14]	$6.18\pm0.01^{a\,B}$ (199) [30]
3	$6.72\pm0.03^{b\,A}$ (55) [20]	$6.57\pm0.03^{b\,B}$ (40) [13]	$6.56\pm0.02^{b\,B}$ (100) [22]
8	$6.97\pm0.04^{c\,A}$ (33) [11]	$7.01\pm0.04^{c\,A}$ (27) [12]	$7.03\pm0.03^{c\,A}$ (56) [19]
22	$6.98\pm0.05^{c\,A}$ (19) [10]	$6.98\pm0.05^{c\,A}$ (16) [8]	$6.91\pm0.03^{d\,A}$ (46) [17]

pH-values within a column without a small superscript letter in common differ significantly. In the first two columns ($p<0.001$) and in the third ($p<0.01$).
pH-values between columns but on the same day without a large superscript letter in common, differ significantly ($p<0.01$).

Discussion

The gram negative bacteria, *Escherichia coli* and *Klebsiella pneumoniae,* have been suggested to be the most common organisms invading the mammary glands in sows directly pre- or postpartum. This invasion often leads to mastitis which, in a majority of cases, is implicated in the agalactia post partum syndrome *(Ringarp* 1960, *Armstrong et al.* 1968, *Ross et al.* 1981, *Wegmann* 1985, *Bertschinger et al.* 1990).

In the present study, *E. coli* was the predominant bacteriological finding in milk samples obtained from the mammary glands in sows clinically or subclinically affected by mastitis. In clinically diseased sows, more than 60% of the *E. coli* infected glands exhibited a profuse growth on day 1 of lactation. In the subclinically infected sows, the corresponding percentage of profuse E. coli growth was just above 50. There was a pronounced decline in the intensity of *E. coli* growth already on day 3. On the third sampling occasion, day 8, no growth of *E. coli* was detected. Our results are well in accordance with *Wegmann* (1985), who found that *E. coli* was the most prominent bacteriological finding during the first 2 days post partum and that no further growth was detected after day 5. In the present study, mastitis was clinically diagnosed in 38 of 87 mammary glands infected with *E. coli.* Clinical symptoms of mastitis were more pronounced in the experimental group than in the control group (18 of 26 glands versus 20 of 61 glands). This difference was earlier reported

Table 9. The potential influence of low (E) and high (C) feeding levels during late pregnancy on TCC, PMNL% and pH in colostrum and milk samples from mammary glands either bacteriologically negative or with growth of *E. coli*. Sows clinically affected by agalactia post partum were compared with clinically healthy sows with some mammary glands infected with *E. coli* (subclinical sows) or with the remaining numbers of clinically healthy sows. The geometric mean values for TCC ($\times 10^{-6}$/ml) and the mean values for PMNLs and pH are here given without confidence interval (TCC) and standard errors (PMNL% and pH).

	Feeding levels		Sign. of difference
	E	C	E-C
E. coli			
TCC			
Agal. p.p. sows	27.6	38.0	n.s.*
Subcl. infect. sows	20.3	12.9	n.s.
PMNL%			
Agal. p.p. sows	57.6	51.8	n.s.
Subcl. infect. sows	59.4	56.8	n.s.
pH			
Agal. p.p. sows	6.88	6.73	n.s.
Subcl. infect. sows	6.82	6.89	n.s.
Bacteriolog. neg.			
TCC			
Agal. p.p. sows	2.69	3.62	$p<0.05$
Subcl. infect. sows	2.61	1.76	n.s. ($p = 0.0507$)
Clin. healthy sows	2.21	2.02	n.s.
PMNL%			
Agal. p.p. sows	47.4	45.2	n.s.
Subcl. infect. sows	42.1	41.1	n.s.
Clin. healthy sows	50.5	52.4	n.s.
pH			
Agal. p.p. sows	6.65	6.70	n.s.
Subcl. infect. sows	6.60	6.51	n.s.
Clin. healthy sows	6.44	6.51	n.s. ($p = 0.0503$)

* n.s. = not significant.

by *Persson et al.* (1989) who found that the overall percentage of agalactic lactations with udder changes was slightly higher in the experimental group than in the control group (88.4% versus 73.1%). The higher feeding intensity within the control sows might have initiated the lactation earlier and resulted in swelling and engorgement of the mammary glands and thus made the clinical examination of the mammary glands more difficult. Early initiation of lactation may be a contributing factor in mastitis in sows *(Gonneratne et al.* 1982).

The reason for the decline and final elimination of coliforms has not beeen totally clarified, but most probably polymorphonuclear leucocytes are involved in the inflammatory response against the infection *(Jain et al.* 1968, *Paape et al.* 1991).

The endocrine changes in sows around parturition, with a pronounced rise in oestrogen, might exert an effect on the immune system. *Magnusson & Einarsson* (1990) found that oestradiol-benzoate injected in ovariectomized gilts was followed by a significant decrease in the total

Table 10. The potential influence of lactation number on TCC, PMNL % and pH in colostrum and milk samples from mammary glands either bacteriologically negative or with growth of *E. coli*. Sows clinically affected by agalactia post partum were compared with clinically healthy sows with some mammary glands infected with *E. coli* (subclinical sows) or with the remaining numbers of clinically healthy sows. The geometric mean values for TCC ($\times 10^{-6}$/ml) and the mean values for PMNL% and pH are here given without confidence interval (TCC) and standard errors (PMNL%, pH).

	Lactation number						Average of lactations	Level of sign. Lactation
	1	2	3	4	5	6	1-6	
E. coli.								
TCC								n.s.
Agal. p.p. sows	31.62	22.51	18.32	30.01	39.59	30.81	27.96	
Subcl. infect. sows	49.51	–	12.53	9.34	7.75	35.06	Non-est.[2]	
PMNL %								p<0.001
Agal. p.p. sows	80.0[1]	59.4	61.2	64.6	55.2	39.5	59.99	
Subcl. infect. sows	71.3[1]	–	61.6	59.2	69.9[1]	38.7	Non-est.[2]	
pH[3]								
Bacteriol. negative								
TCC[4]								p<0.001
Agal. p.p. sows	1.80	1.43	1.22	1.37	2.01	1.45	1.53[a]	
Subcl. infect. sows	1.10	1.24	1.27	0.45	2.10	1.93	1.21[b]	
Clin. healthy sows	1.01	1.45	1.26	1.85	1.78	1.37	1.42[ab]	
PMNL %								p<0.001
Agal. p.p. sows	38.2	40.9	51.5	58.0	36.7	44.4	44.9[a]	
Subcl. infect. sows	41.1	48.8	45.9	53.0	42.7	20.5	42.0[a]	
Clin. healthy sows	44.2	48.1	54.7	54.4	50.0	58.2	51.6[b]	
pH[5]								n.s.
Agal. p.p. sows ↘							6.68[a]	(p = 0.0578)
Subcl. infect. sows →	6.49	6.59	6.64	6.52	6.53	6.55	6.50[b]	
Clin. healthy sows ↗							6.48[b]	

Geometric mean values without a small superscript letter in common differ significantly (p<0.05).

1. Number of observations are few or below 10.
2. No estimation possible to perform.
3. The influence of lactation on pH within the group of sows with growth of *E. coli* could not be determined because of too few pH-values, especially among the subclinically infected sows (compare Table 7).
4. This analysis was performed from TCC-values emanating from the cell class below 10×10^6 cells/ml.
5. ↘→↗The influence of lactation on pH was evaluated after the individual pH-values in each lactation belonging to each of the three different clinical groups of sows had been added together.

number of lymphocytes in the blood and a concurrent increase in the phagocytic capacity of the polymorphonuclear leucocytes (PMNLs). Conflicting results arose when oestrogen was added to blood cells in vitro, where a decrease in the phagocytic function of the PMNLs was revealed.

Other noncytological defence mechanisms, such as transferrin/lactoferrin, act by withdrawal of, for instance, Fe necessary for replication of *E. coli (Reiter et al.* 1975).

In the agalactia post partum sows, the invading *E. coli* caused a very high TCC in the milk (82.27×10^6 cells/ml). The TCC was still high

on day 3 of lactation, with concomitant presence of *E. coli* in 35% of the originally infected mammary glands. Thereafter TCC decreased significantly but was still elevated (8.24×10^6 cells/ml) compared with the normal TCC on day 22 (1.38×10^6 cells/ml; *Persson et al.* 1996). *Wegmann* (1985) defined mammary glands as being mastitic if the TCC was $>5.0\times10^6$ cells/ml and the percentage of PMNLs ≥70. In their study, 18 out of 41 mastitic mammary glands were infected with *E. coli.* In the 41 mastitic glands, the TCC increased to above 10×10^6 cells/ml milk during the first days post partum and stabilized after 10 days in suckled glands on levels between $2.4–3.1\times10^6$ cells/ml. The clinically healthy sows affected by a subclinical *E. coli* mastitis showed an even higher TCC on day 1 of lactation than the clinically diseased sows. The 2 groups of sows showed a similar decline in TCC throughout the sampling period, but on day 22 the TCC in the group of clinically diseased sows was on a significantly higher level.

The percentage of PMNLs was elevated in colostrum samples from both the clinically and subclinically mastitic sows (65.8% and 79.0%, respectively). The number of PMNLs exceeded the number of PMNLs migrating to the colostrum secretion in bacteriologically negative mammary glands within clinically healthy sows (approximately 60%; *Persson et al.* 1996). In mastitic mammary glands, *Wegmann* (1985) obtained PMNL-values exceeding 80% of the TCC on day 1 of lactation. In their study, the percentage of PMNLs fell already after 2 days to levels below 70%, which is in accordance with values obtained in our study. In our study, however, there was a tendency towards an increase in percentage of PMNLs among the subclinically affected sows. This might indicate a more successful migration of the PMNLs to the mammary glands. In cows, a slow diapedesis of neutrophils appears to be associated with the most severe cases of *E. coli* mastitis *(Hill et al.* 1979). The lower percentage of PMNLs in milk from mammary glands belonging to the clinically affected sows might reflect the neutrophilic leucopenia observed in blood after intravenous or intramammary infusion caused by adequate doses of endotoxin *(Nachreiner et al.* 1972). A higher prevalence of endotoxinemia was observed in dysgalactic than in healthy sows *(Morcoc et al.* 1983).

An attempt was also made to elucidate the influence of mastitis on TCC and PMNLs in bacteriologically negative colostrum and milk samples collected from sows clinically or subclinically infected with *E. coli* in other mammary glands. The results were compared with those from mammary glands without detectable growth of microorganisms in sows designated as healthy. TCC obtained from bacteriologically negative colostrum in sows affected by *E. coli* infection in one or more glands and considered to be clinically diseased revealed a TCC that was higher on day 1 than values obtained from noninfected glands in clinically healthy or subclinically infected sows. One possible explanation of this elevation in TCC among clinically affected sows could be a transient agalactic or hypogalactic condition causing an increase in cell content. This difference disappeared and only one observation remained, on day 8, which was a significantly decreased TCC-value in subclinically infected sows.

The drop in percentage of PMNLs in mammary glands, without any demonstrable infection among the clinically or subclinically infected sows compared with clinically healthy sows without subclinical infections (48.3% and 43.5% versus 59.6%), might indicate that the phagocytic cells have been redistributed to those glands where they are required in the elimination of the invading pathogens. It must, however, be emphasized that the total amount of PMNLs available in clinically diseased sows

is still a level exceeding the total number of PMNLs present in mammary glands belonging to the healthy sows.

The pH-values on day 1 in mammary glands that were clinically or subclinically infected with *E. coli* and where the TCC exceeded 10×10^6 cells/ml were considerably higher than in glands not infected and inflamed within the same sows (6.57 and 6.46 versus 6.29 and 6.18). *Ringarp* (1960) reported an even higher pH (7.0–7.2) in milk secretion collected from mammary glands in sows diseased in "agalactia toxemica". In his study, pH measured in milk from mammary glands diagnosed to have mastitis was usually only slightly higher than in secretion from clinically nonmastitic glands. *Martin et al.* (1967) found that pH in milk from affected sows ranged from 6.5 to 8.0 (average 7.2) compared with 6.0 to 6.9 (average 6.6) in milk from normal sows. In our study, a rise in pH from days 1 to 3 (6.29 and 6.72) was also shown in bacteriologically negative colostrum and milk collected from clinically affected sows. *Ross et al.* (1981) on the other hand, reported a lower pH in mastitic milk compared with normal milk. Their explanation of the deviating results was that only one of the 2 gland-segments were mastitic and that the bulk of milk might have come from the less severely affected segment.

The medical treatment in the present study might have contributed to the declination and final elimination of the *E. coli,* although sows clinically healthy with mammary glands infected of *E. coli* and not receiving any medical treatment eliminated the bacteria spontaneously. In cows, recovery from coliform mastitis is usually attributed to treatment, but also without treatment the *E. coli* cannot be isolated at repeated sampling *(Eberhart et al.* 1979). In the present study the elimination of the infectious stimuli *(E. coli),* spontaneously or possibly attributed to the medical treatment, caused a successive decrease in TCC and PMNLs during the first 3 weeks of lactation to levels that only slightly differed from normal cell count levels (compare *Persson et al.* 1996).

The 2 different feeding regimes during late gestation as a rule did not influence the TCC, the PMNLs, or the pH in milk from bacteriologically negative mammary glands or from glands with *E. coli* mastitis. Agalactia post partum was diagnosed in altogether 52 out of 195 lactations in the control group and 26 out of 181 lactations in the experimental group, respectively *(Persson et al.* 1989). A tendency in the same direction is also the significantly higher TCC in bacteriologically negative milk secretion in the control group of sows compared with the experimental group (3.62 versus 2.69).

The lactation number had an overall significant influence on PMNLs but not on TCC in mammary glands with clinical or subclinical *E. coli* mastitis. In these 2 groups of sows, the sixth lactation revealed a significantly lower percentage of PMNLs, just below 40, compared with the preceding 5 lactations.

The lactation number had a significant influence on the TCC and the PMNLs, but not on the pH in bacteriologically negative colostrum and milk collected from all the clinical groups of sows. The TCC was found to reach a maximum of 1.85 to 2.10×10^6 cells/ml at the fourth or fifth lactation within these 3 groups of sows. The PMNLs reached a maximum at lactations 3 and 4, 53%–58%, in milk secretion from clinically diseased sows or from healthy but subclinically *E. coli* infected sows, thereafter decreasing to levels between 20.5% and 44.4% at the sixth lactation. In contrast to the latter 2 groups, the group of clinically healthy sows reached its maximum in PMNLs, 58.2%, at the sixth lactation.

It should be emphazised that there was a decrease in percentage of PMNLs at the sixth lactation in both bacteriologically negative and *E.*

coli positive milk samples collected from sows clinically diseased or subclinically infected. However, we do not know anything of the percentage level of PMNLs in lactations later than number 6. There seems to be a decrease in defence mechanisms that ought to be further studied.

Conclusions

The objective of this study was to evaluate the potential influence of high (C) versus low (E) feed allowances in sows during late pregancy in 6 consecutive lactations on the following parameters: clinical status of the mammary glands, bacteriology, TCC, PMNLs and pH of the milk secretion.

E. coli infection was diagnosed in 80% of the sows suffering from agalactia post partum and in 30% of the clinically healthy sows irrespective of feeding regime. *E. coli* bacteria were finally eliminated between days 3 and 8 of lactation. The infected glands were considered to be mastitic if the TCC exceeded 10×10^6 cells/ml secretion. Forty-nine out of 63 lactations in clinically diseased and 15 out of 27 lactations in subclinically infected sows revealed *E. coli* infections in one or more mammary glands with a concomitant TCC exceeding the threshold value already on day 1 of lactation. The corresponding PMNLs varied from 65% to 80% on the first day of lactation. The TCC and PMNLs declined gradually to day 22 of lactation, but were still higher compared with values emanating from bacteriologically negative milk sampled on the same day. The pH was higher in colostrum and milk from infected mammary glands than in bacteriologically negative colostrum and milk.

The sows diseased in APP were imposed to medical treatment which might have exerted an effect on the declination and elimination of the *E. coli* and consequently also on the decrease of TCC and PMNLs during the first 3 weeks of lactation. Sows clinically healthy but with subclinical *E. coli* mastitis spontaneously eliminated the bacteria and the TCC and PMNLs decreased in a similar way that has been described from sows affected of APP.

The 2 different feeding regimes did not as a rule influence the TCC, the PMNLs or the pH in mammary glands with *E. coli* mastitis or in bacteriologically negative glands.

The lactation number had a significant influence on the percentage of PMNLs, but not on TCC in mammary glands with *E. coli* mastitis. Further, the lactation number had a significant influence on the TCC and the PMNLs but not on the pH in bacteriologically negative colostrum and milk in all clinical groups of sows.

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Sammanfattning

En långtidsstudie rörande hälsotillstånd och produktionsresultat hos suggor på olika fodergivor under sen dräktighet. III. Escherichia coli och andra bakterier, totalt cellinnehåll, polymorfkärniga leukocyter och pH i kolostrum och mjölk under de första 3 veckorna av laktationen

Syftet med föreliggande studie var att (1) fastställa juvrets kliniska status och (2) jämföra detta med bakteriologiska fynd, det totala cellinnehållet (TCC) och andelen polymorfkärniga leukocyter (PMNL) därav och pH i kolostrum och mjölk från suggor som stått på två olika utfodringsnivåer, låg respektive hög, under sen dräktighet. Mjölkkprov uttogs från såväl sjuka, (agalakti post partum, APP), som från kliniskt friska suggor. Suggor med en rektaltemperatur överstigande 39.5°C inom 48 timmar efter förlossningen betraktades som sjuka och behandlades. Proverna togs på dag 1, 3, 8 och 22 i laktationen under sex på varandra följande laktationer.

Oavsett utfodringsnivå upptäcktes under 49 av 77 laktationer bland APP-suggorna och 15 av 96 laktationer bland de kliniskt friska suggorna *E. coli* i renkultur åtföljt av ett TCC överstigande 10×10^6 celler/ml redan under första dagen av laktationen. Friska suggor med *E. coli* kom att benämnas subkliniskt infekterade. Intensiteten i *E. coli* växten avklingade successivt och bakterierna eliminerades slutligen mellan dag 3 och 8 av laktationen. Första provtagningsdagen uppgick TCC till 82×10^6 celler/ml respektive 157×10^6 celler/ml i de kliniskt och subkliniskt *E. coli* infekterade juverdelarna. TCC sjönk gradvis i de båda suggrupperna men var ändå högre än i bakteriologiskt negativ mjölk på dag 22 i laktationen.

På dag 1 uppgick andelen PMNL till 66% respektive 79% i kliniskt och subkliniskt infekterade juverdelar, varefter nivån sjönk till cirka 50% fram till dag 22 i laktationen inom båda grupperna. Bland APP-suggorna registrerades svullnad, rodnad och/eller ömhet hos 38 av 87 juverdelar med *E. coli* mastit vid första provtagningstillfället.

TCC i bakteriologiskt negativ kolostrum uttaget från APP-suggor under första dagen av laktationen var signifikant högre, $2,27\times10^6$ celler/ml, jämfört med TCC i bakteriologiskt negativ kolostrum från kliniskt friska eller subkliniskt infekterade suggor, $1,38\times10^6$ celler/ml respektive $1,51\times10^6$ celler/ml. PMNL var högre dag 1 bland kliniskt friska suggor, 59,6%, än bland subkliniskt infekterade suggor och APP-suggor (43,5% respektive 48,3%).

Under första provtagningsdagen var pH i kolostrum från kliniskt eller subkliniskt *E. coli* infekterade juverdelar (6,57 respektive 6,46) högre än i bakteriologiskt negativa kolostrumprov (6,29) från kliniskt sjuka suggor. På dag 22 hade pH-värdet stabiliserats på en nivå av cirka 7,00 i alla mjölkprov oavsett om juverdelarna tidigare varit bakteriologiskt positiva eller negativa.

De två utfodringsnivåerna, låg eller hög, påverkade inte TCC, PMNL eller pH med undantag för TCC i bakteriologiskt negativa prover bland APP-suggor (2,69 kontra 3,62).

Laktationsnumret påverkade PMNL bland båda grupperna av *E. coli* infekterade juverdelar, och både TCC och PMNL i bakteriologiskt negativ kolostrum och mjölk.

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