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Epidemiology of Mammary Gland Disorders in Multiparous Finnish Ayrshire Cows

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ABSTRACT

Logistic regression was used to investigate the effects of host characteristics, production, and 23 veterinary diagnoses on the odds of contracting three mammary gland disorders among 41,989 multiparous Finnish Ayrshire cows who calved during 1983.

Cows which had higher previous yields were at increased risk of acute and chronic mastitis, and teat injury. All of the mammary gland disorders were directly interrelated. Abomasal disorder, indoor hypomagnesemia, prolapsed uterus and vagina, and abortion were not risk factors for any of the three mammary gland disorders; however, retained placenta, udder edema, ketosis, nonparturient paresis, rumen acidosis, traumatic reticuloperitonitis, silent heat, cyst and other infertility each was a direct risk factor for at least two mammary gland disorders.

INTRODUCTION

In spite of control, intensified treatments with antibiotics, and extensive research, mastitis is the most important disease problem in the dairy industry (4). For this reason, more exact knowledge by means of extended epidemiological analyses of mastitis and other udder diseases is needed for creating better control programs. The state-level health registries in Nordic countries are valuable data banks for investigating the epidemio-

logical background of these disorders.

Breed, parity, and season as predisposing factors were analyzed in several studies (1, 6, 7, 8, 13) but the sample size has been large (over 50,000 lactations) in only a few (2, 21). Association of mastitis with other diseases such as teat injury (2, 5, 7, 14, 17) and parturient paresis (2, 3, 21) has been reported. Numerous host characteristics and disease relationships still are understood poorly. Our objectives were 1) to generate hypotheses regarding risk factor-disease relationships not previously studied, and 2) to reexamine previously-identified relationships under conditions of maximum practical statistical control of potential confounding by host characteristics, production and other diseases.

A data set of 41,989 multiparous Finnish Ayrshire lactations begun in 1983 was available. The set contained information on 23 diagnoses (all made by veterinarians), herd milk yield, and cow milk yield. Mammary gland disorders were defined broadly to include acute and chronic clinical mastitis, and teat injury.

MATERIALS AND METHODS

Data

The Finnish dairy cow health data recording system has been described previously (11). The data are from multiparous cows from whom data on 23 veterinary diagnoses were collected from 2 days before calving either to a subsequent calving or to removal from

the herd. All cows were pedigreed, were bred by artificial insemination, and were in herds that recorded milk. The herds were from the 76 (out of 461) communities that were judged to have the best record-keeping. The data included only those diagnoses made according to ordinary clinical methods under field conditions by veterinarians during farm visits. Only the first diagnoses of each disease in each lactation were considered. Because approximately 65 % of all mastitis cases in Finland are treated by telephone prescription (without the veterinarian seeing the cow) (16), the cases analyzed in this study probably were the more severe cases and may have required systematic therapy (because the veterinarian did see the cow).

The mammary gland disorders studied were: acute and chronic clinical mastitis, and teat injury (12). Twenty other veterinary diagnoses were considered as risk factors for mammary disorders: dystocia, prolapsed uterus, retained placenta, early metritis (diagnosed within 42 d postpartum), silent heat (anestrus or subestrus), cyst (ovulatory dysfunction including cystic ovaries), prolapsed vagina, late metritis (diagnosed later than 42 d post-partum), other types of infertility (reproductive disorders which were not included in the former diagnoses), abortion, parturient (within 2 d before and 7 d after calving) and nonparturient paresis, udder edema, disorder of the abomasum (most cases probably were abomasal displacements), indoor (hypomagnesemia treated during the indoor season) and outdoor hypomagnesemia (hypomagnesemia treated during the outdoor season), ketosis, rumen

acidosis, traumatic reticuloperitonitis, and foot or leg injury.

Time-ordering of Variables

We copied the edited data set once for each mammary disease. Within each such data set, that one disease became the dependent variable, we then recoded potential risk factor diseases to code = 0 ("absent") if the day in milk (DIM) of the potential risk factor was greater than the DIM of the dependent variable.

In the control records (lactations in which the dependent variable was absent), a dummy "cut-off" DIM was assigned for counting or not counting the risk factor diseases. The assignment was at random but in proportion to the DIMs observed for the dependent variable (10). Thus, there was equal "opportunity" for risk factor diseases to be counted as present in both cases and controls.

Statistical Analysis

All statistical analyses were carried out using the Statistical Analysis System (20). A 3-stage process was used to model each outcome disease. Only risk factor diseases associated at $P \leq .10$ with the outcome in unconditional, 2 x 2 chi-square tests (the first stage) were advanced to the second stage.

The second modelling stage began with a multiple logistic regression model that included all the risk factor diseases that passed the first stage, plus parity (2, 3-4, 5-6, >6), season of calving, and cow and herd milk yield classes from the previous

lactation. Season had 3 classes (spring = January - April, summer = May - August, fall = September - December). Wald's test ($Z = \beta/SE(\beta)$) was used to test the significance of the terms in the model. Any variables not significant at $P \leq .10$ were removed and the logistic regression was rerun with the remaining variables. Wald's tests were done again and the process continued until a model was obtained in which all variables were significant at $P \leq .10$.

In the third stage of the modelling, community was forced into the models and the level of significance was lowered to 5%. Any predictor variable (except community) with $P > .05$ by Wald's test was removed and the reduced model was rerun until final models were obtained in which the only variables were community plus those with coefficients significant at 5%.

RESULTS AND DISCUSSION

Occurrence

The lactation incidence rates (Table 1) of mastitis were near those reported by Dohoo et al. (6; 3,5 %) and Curtis et al. (3; 5.4 %). The total incidence of clinical mastitis in Finland and in other Scandinavian countries has been higher (10-30 %; 21) than in our data. One has to remember that this material included only veterinary-treated cases (likely the more serious cases of clinical mastitis). The median times in milk at diagnoses of mastitis requiring systemic (22 days) or local (54 days) therapy

or of teat injury (94 days) studied by Dohoo et al. (6) were shorter than those found by us.

Demographic Characteristics

The increasing risk of clinical mastitis with parity (Tables 2 and 4) is well known (e.g. 3, 6), however the OR's for older cows were not as strong as reported elsewhere (6). One explanation may be that mastitic cows are culled more intensively in Finland than in North America and the cows with higher resistance to mastitis will stay in the herd (11). The risk of teat injury was not related to parity (Table 3), as noted also by Dohoo and Martin (6).

The cows that calved in January-August were slightly more likely to contract teat injury than those who calved in September-December (Table 3). Calving season was not a risk factor for acute or chronic mastitis (Tables 2 and 4).

Higher individual-cow's milk yield in the previous lactation increased the risks of teat injury and of acute and chronic clinical mastitis (Tables 2-4). Dohoo and Martin (5) did not find any association between (deviation from herd average) previous milk production and clinical mastitis or teat injury in their Canadian material, but in a large Finnish study they did find a large genetic correlation between milk yield and clinical mastitis (r_g = 0.57; 21). In contrast, herd milk yield was not related to teat injury, acute or chronic mastitis.

Disease Risk Factors

The G^2 statistics for lack of fit for all models were non-significant (all P's > .42), implying reasonable fits to the data. Relationships among mammary gland disorders are combined in Figure 1 from the four (i.e., udder edema, acute and chronic mastitis, and teat injury) logistic regression models. We have also included udder edema in this figure, although it was reported earlier as a metabolic disorder (10). Demographic variables were excluded from the figure. Because the arrows come from four different time-ordered sub-sets, it is possible to see e.g., that the cows with teat injury have greater odds of acute mastitis and the cows with acute mastitis have greater odds of teat injury.

Abomasal disorder, indoor hypomagnesemia, prolapsed uterus and vagina, and abortion were not associated directly with any of the mammary gland disorders (Tables 2 - 4).

Teat injury directly increased the odds of udder edema, acute and chronic mastitis - and was itself contributed to by several reproductive disorders. The association between some infertility disorders and teat injury may suggest the changed behavior of the cows which may induce teat injuries. An association between traumatic injuries of the teat and mastitis has been documented in many studies (2, 5, 7, 15, 17, 19). Injuries to udder are considered one of the most important factors in the etiology of acute mastitis (7); this is supported also by the high ORs (6.9).

Udder edema was a risk factor for acute and chronic mastitis and was contributed to by retained placenta, teat injury, and

acute and chronic mastitis. Retained placenta was also a risk factor for mastitis. The association between preceding inflammatory diseases and mastitis may indicate either the real risk of these diseases or the decreased resistance of the cow against all kinds of inflammatory diseases. Schukken et al. (18) suggested that the relationship between retained placenta and mastitis is mediated by the peripartum peripheral leucocyte activity. There were several other common risk factors for acute and chronic mastitis (Figure 1).

General Discussion

In contrast to most other epidemiologic studies on this topic, our cows all were Finnish Ayrshires; differences in results between our study and others may reflect merely breed or geographic differences. Our relatively large sample size allowed us to explore relationships involving rarer disorders, but does not guarantee that observed associations are of economic importance. Finally, we investigated so many relationships that some of those "discovered" (and especially those not previously observed in other studies) may in fact represent Type I errors.

A strength of our study is that <u>all</u> prior diagnoses were allowed as potential risk factors (rather than excluding all diagnoses of a disease based on average date of diagnosis in the full data set). However, we also allowed concurrent diagnoses to be considered as risk factors, and it is possible that a cow would be observed more closely for additional diseases once one disease

was noticed. This increased "index of suspicion" would bias towards observing (a few) additional significant relationships.

ACKNOWLEDGEMENTS

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TABLE 1. Rates of cases treated by veterinarians for 41,989 multiparous Finnish Ayrshire lactations (calvings in 1983 in 5,661 herds).

Diagnosis	Lactational incidence rate %	Lactational incidence rate Number	Median day postpartum of diagnosis
Parturient paresis	5.3	2229	1
Udder edema	.3	141	14
Disorder of the abomasum	.6	243	21
Indoor hypomagnesemia	.2	66	22
Ketosis	6.6	2755	28
Nonparturient paresis	1.2	488	43
Outdoor hypomagnesemia	.4	188	44
Dystocia	1.0	427	0
Prolapsed uterus	.2	68	0
Retained placenta	5.3	2229	2
Early metritis	2.2	912	16
Silent heat	4.5	1886	89
Cyst	7.5	3166	89
Prolapsed vagina	.1	48	94
Late metritis	1.1	448	104
Other infertility	2.0	855	129
Abortion	.4	165	-
Rumen acidosis	.3	131	56
Traumatic reticuloperitonitis	.5	218	113
Acute mastitis	7.0	2946	44
Teat injury	3.1	1301	95
Chronic mastitis	2.0	860	103
Foot or leg injury	1.8	768	65

TABLE 2. The final logistic regression model used for acute mastitis (41,989 multiparous Finnish Ayrshire cows).

Risk factor	β	SE(β)	Odds ratio	95% C.I. (OR)
Parity				
2	-.19	.04	1.0	-
3-4	-.15	.03	1.0	.9 - 1.2
5-6	.11	.04	1.4	1.2 - 1.5
>6	.23	.04	1.5	1.3 - 1.8
Cow milk yield in previous lactation (305 FCM, kg)				
< 4740	-.25	.05	1.0	-
4740 - 5899	-.10	.03	1.2	1.0 - 1.3
5900 - 7059	.08	.03	1.4	1.2 - 1.6
≥ 7060	.28	.04	1.7	1.5 - 2.0
Parturient paresis	.63	.07	1.9	1.6 - 2.1
Ketosis	.63	.08	1.9	1.6 - 2.2
Nonparturient paresis	1.10	.18	3.0	2.1 - 4.2
Outdoor hypomagnesemia	.78	.28	2.2	1.3 - 3.8
Retained placenta	.74	.07	2.1	1.8 - 2.4
Early metritis	.48	.12	1.6	1.3 - 2.1
Silent heat	.40	.12	1.5	1.2 - 1.9
Late metritis	.75	.23	2.1	1.4 - 3.3
Other infertility	.86	.17	2.4	1.7 - 3.3
Rumen acidosis	.80	.33	2.2	1.2 - 4.2
Traumatic reticuloperitonitis	.69	.31	2.0	1.1 - 3.7
Udder edema	1.24	.28	3.5	2.0 - 6.0
Teat injury	1.93	.09	6.9	5.8 - 8.2
Foot or leg injury	.71	.16	2.0	1.5 - 2.8
(76 communities)				

TABLE 3. The final logistic regression model used for teat injury (41,989 multiparous Finnish Ayrshire cows).

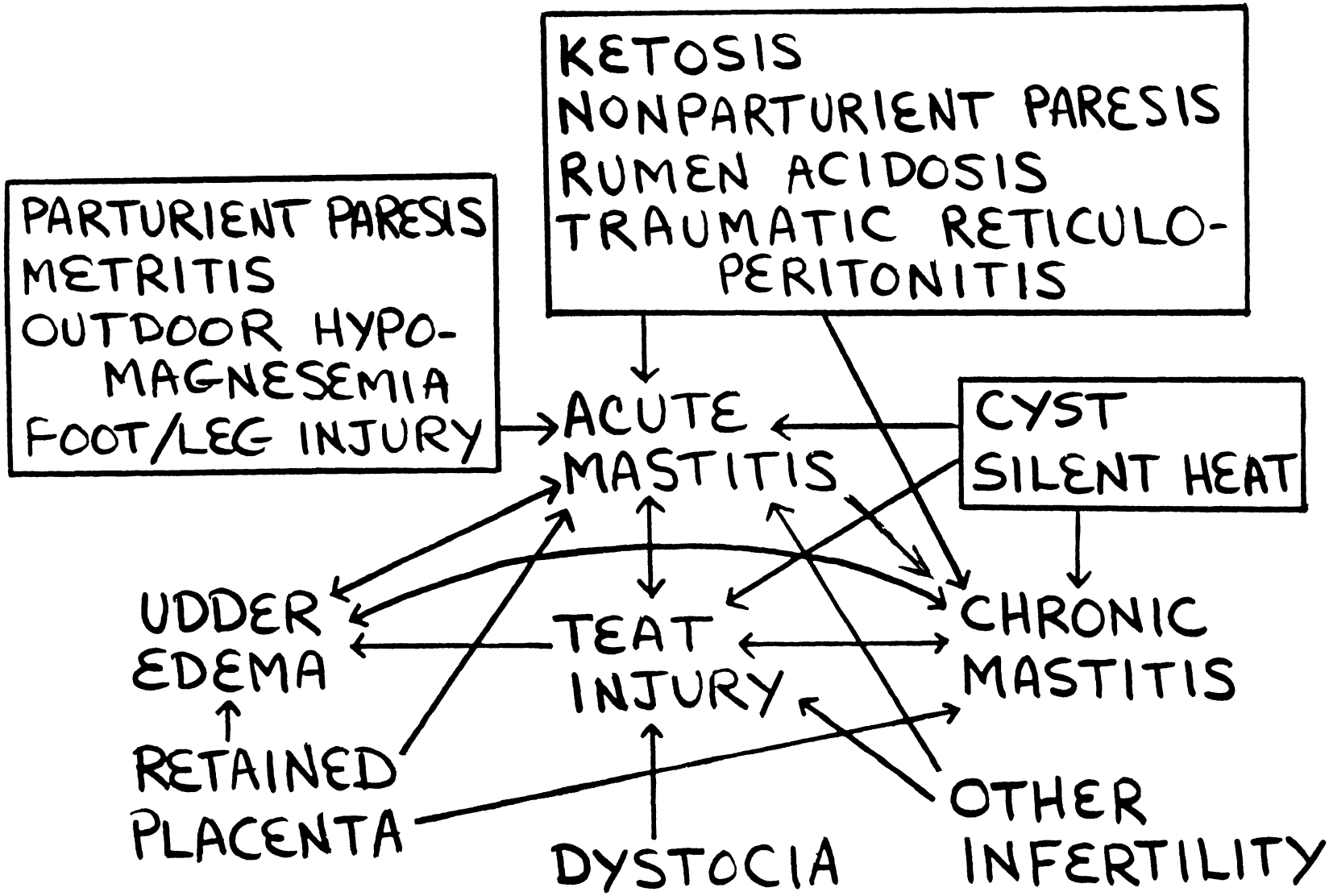
Risk factor	β	SE(β)	Odds ratio	95% C.I. (OR)
Calving season				
January - April	.05	.04	1.2	1.0 - 1.4
May - August	.08	.04	1.2	1.0 - 1.5
September - December	-.13	.04	1.0	-
Cow milk yield in previous lactation (305 FCM, kg)				
< 4740	-.18	.06	1.0	-
4740 - 5899	-.13	.05	1.1	.9 - 1.3
5900 - 7059	.08	.05	1.2	1.0 - 1.4
≥ 7060	.22	.06	1.5	1.2 - 1.8
Dystocia	.47	.23	1.6	1.0 - 2.5
Silent heat	.49	.15	1.6	1.2 - 2.0
Cystic ovary	.45	.12	1.6	1.2 - 2.0
Other infertility	.50	.23	1.7	1.1 - 2.6
Acute mastitis	1.44	.08	4.2	3.6 - 4.9
Chronic mastitis	1.23	.15	3.4	2.5 - 4.6
(76 communities)				

TABLE 4. The final logistic regression model used for chronic mastitis (41,989 multiparous Finnish Ayrshire cows).

Risk factor	β	SE(β)	Odds ratio	95% C.I. (OR)
Parity				
2	-.33	.07	1.0	-
3-4	-.05	.05	1.3	1.1 - 1.6
5-6	.10	.06	1.5	1.3 - 1.9
>6	.27	.09	1.8	1.4 - 2.4
Cow milk yield in previous lactation (305 FCM, kg)				
< 4740	-.16	.08	1.0	-
4740 - 5899	-.17	.06	1.0	.8 - 1.3
5900 - 7059	-.08	.06	1.1	.9 - 1.4
≥ 7060	.41	.07	1.8	1.4 - 2.3
Ketosis	.65	.12	1.9	1.5 - 2.4
Nonparturient paresis	.82	.27	2.3	1.3 - 3.8
Retained placenta	.48	.13	1.6	1.3 - 2.1
Silent heat	.69	.16	2.0	1.4 - 2.7
Cystic ovary	.39	.14	1.5	1.1 - 1.9
Rumen acidosis	1.35	.39	3.8	1.8 - 8.3
Traumatic reticuloperitonitis	1.07	.36	2.9	1.4 - 5.9
Udder edema	1.31	.29	3.7	2.1 - 6.6
Acute mastitis	.76	.10	2.1	1.8 - 2.6
Teat injury	1.42	.14	4.1	3.1 - 5.4
(76 communities)				

LEGEND FOR FIGURE

Figure 1. Relationships among mammary gland disorders from the four logistic regression models. Demographic variables were excluded from the figure. To simplify the figure there is an arrow between cyst and acute mastitis, although cyst is not a risk factor for acute mastitis.



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