An Epidemiologic Study of Disease in 32 Registered Holstein Dairy Herds in British Columbia

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ABSTRACT

Data recorded in a herd health management system were obtained from 32 registered Holstein dairy herds from British Columbia. Frequencies of disease were described, and the effect of herd, age, year, season, and the interrelationships between diseases within a lactation on the occurrence of disease were evaluated. Lactational incidence rates were computed for diseases with a short period of risk (ie, udder edema, milk fever, retained placenta, metritis, displaced abomasum, and ketosis), whereas for diseases with a longer period of risk (ie, cystic ovaries, mastitis and stable footrot), incidence densities were calculated. Overall, the disease incidence was low and showed an increase in frequency by year, which we attributed to more observing and complete recording by the owner, rather than an actual increase in disease incidence. Most diseases occurred early in lactation and their frequency increased with lactation number; the exception was udder edema, which occurred mainly during the first 2 lactations.

An informal path model of disease interrelationships was made conditional on herd. Based on the results we inferred 2 independent pathways: one started by udder edema, and the other by milk fever. Udder edema was directly associated with mastitis occurrence from 0 to 30 d in lactation, metritis, and cystic ovaries. Mastitis from 0–30 d in lactation increased the risk of both mastitis from 31–150 d in lactation and cystic ovaries. Both of these increased the risk of late lactation mastitis. Milk fever was directly related with displaced abomasum, which increased the risk of footrot. In general, diseases that occurred in early lactation tended to increase the risk of other diseases later in lactation.

RÉSUMÉ

Les données accumulées dans un régime de régie de santé de troupeau de 32 troupeaux laitiers Holstein enregistrés de la Colombie-Britannique ont été obtenus et la fréquence des maladies fut décrite. Une évaluation fut faite afin de vérifier les associations entre maladies et les effets des variables troupeau, âge, année, et saison sur la présence des maladies. Les risques d'incidences lactationnelles furent calculés pour des conditions avec une courte période de risque (ie, œdème mammaire, parésie de parturition, rétention placentaire, métrite, déplacement de caillette et acétonémie) alors que pour des problèmes avec une période de risque allongée (ie, ovaires kystiques, mammite et piétin d'étable) des densités d'indices furent calculées. De façon générale, l'incidence de maladie était faible et montrait une augmentation relative plutôt que réelle de fréquence par année, attribuable probablement au fait que les propriétaires observaient plus fréquemment et prenaient des notes plus complètes. La plupart des maladies se produisaient plus fréquemment en début de lactation et leur fréquence augmentait avec le nombre de lactation; la seule exception fut l'œdème mammaire qui fut observé principalement durant les deux premières lactation. Un modèle causal a été utilisé en présupposant l'appartenance au troupeau. Deux modèles ont été déduits à partir de nos résultats; un débutant avec l'œdème mammaire, et un autre avec la parésie de parturition. L'œdème de la glande mammaire était directement associé à la présence de mammite entre les jours 0 et 30 de lactation, de métrite et d'ovaires kystiques. La présence de mammite aux jours 0 à 30 de lactation augmentait le risque de mammite aux jours 31 à 150 de lactation de même que les ovaires kystiques. La présence de ces deux problèmes augmentait le risque de mammite en période avancée de lactation. La parésie de parturition était reliée directement au déplacement de caillette, qui augmentait le risque de piétin. De manière générale, les maladies se produisant tôt en début de lactation avaient tendance à augmenter le risque d'apparition de d'autres maladies plus tard au cours de la lactation.

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INTRODUCTION

Periparturient health disorders of dairy cows affect herd performance and can have a considerable negative impact on the revenue of the producer (1), as well as on the welfare of the animal (2). Although periparturient diseases often result in a decrease in milk production or an extended period from calving to conception, an increase in milk production was observed in cows that developed cystic ovarian disease (3).

The authors of a recent review (4) presented incidences and interrelationships among diseases in the same lactation. Calavas (5) investigated associations between diseases in subsequent lactations and noted that certain diseases were at increased risk of recurring or were predisposing to other health disorders in subsequent lactations.

The objectives of this study were to describe the frequency of clinical diseases in registered Holstein dairy farms in one area of British Columbia, and to evaluate the effect of herd, age (ie, lactation number), year, season, and interrelationships between clinical diseases on the occurrence of disease within a lactation.

MATERIALS AND METHODS

STUDY POPULATION

The data were collected from 32 registered Holstein farms in the Fraser Valley, British Columbia, from January 1993 to April 1996. The herds averaged 110 cows with freestall housing. Most farms fed a total mixed ration (TMR) and most used the provincial DHI service. Almost all herds were serviced by one veterinary clinic where the senior author worked as part of her graduate training. Veterinary service was predominantly provided by up to 4 veterinarians and herd health checks were usually performed by the same veterinarian. Diagnoses were made and recorded by both the producer and the veterinarian.

HERD INCLUSION/EXCLUSION CRITERIA

Only herds recording reproductive diseases were included. As not all herd owners kept complete health records, lactation records in herds with at least one clinical disease occurrence recorded within a category of disease (ie, mastitis, lameness, metabolic, nutritional, and other) during the first 365 d in lactation were assigned 0/1 values to denote the recording of disease occurrences in specific cows. Otherwise, missing values were assigned to that disease category for all lactations in the herd.

DATA COLLECTION AND HANDLING

The original data on disease and fertility were entered on a voluntary basis in VAMPP (6), an on-farm record-keeping and management program. The data were validated by this system at the moment of entering, based on birth date, calving date and culling date; the former 2 validations were based on biological and temporal plausibility.

After downloading the data for analyses, the data were checked with the aid of the Statistical Analyses System (7), and biologically illogical values were either verified, corrected or coded missing, as appropriate. Data on cows that calved for the first time before an age of 600 d were excluded. Also, data on cows that calved or were culled at an age greater than 3 times the standard deviation above the mean for that lactation were omitted (eg. for lactation one, cows were removed that calved beyond 1034 d of age), resulting in the removal of 676 records. Data on clinical diseases with at least 50 recorded occurrences were maintained. The diseases of interest in order of temporal occurrence and implied causality were analyzed using an informal path model (8,9) in a manner similar to the study of Klerx and Smolders (10). The ordering of the diseases in this study was udder edema, milk fever, retained placenta, metritis, displaced abomasum, ketosis, mastitis, cystic ovaries and footrot. Metritis included cases of metritis, endometritis or pyometra. Mastitis included all causes/cases of udder infection (since most cases were not cultured to determine the microbiological cause of the inflammation).

The period of interest for disease occurrence and reproductive performance was from January 1, 1994 to December 31, 1995. Only the first occurrence of disease within a lactation was used; these were coded as 0 for not having the disease and 1 for having the disease of interest. The first occurrence of mastitis in each of three time periods (0–30, 31–150, and 151–365 d postpartum) was noted. These time periods were chosen to correspond to the immediate puerperium, the peak milk period, and the last half of the lactation period. New occurrences were based on at least 14 d between the previous and current incidence of mastitis.

DISEASE INCIDENCE

Disease incidence was calculated either as a risk rate (lactational incidence rate) for diseases with a short period of risk, or as a true rate (incidence density) (4) for diseases with a long period of risk. The following formulae were used:

Lactational incidence risk, by lactation:

$$\frac{\text{No. of 1st occurrences of a specific disease in a lactation}}{\text{No. of lactations at risk}}$$

Incidence density, by lactation:

$$\frac{\text{No. of 1st occurrences of a specific disease}}{\left(\frac{\text{No. of cows at risk starting lact.}_i + \text{No. at risk at end of lact.}_i}{2}\right)}$$

The lactations at risk were computed as the number of cows initiating a lactation. The number of cows at risk at the start of a lactation was the total number of cows initiating a lactation. The number at risk at the end of the lactation was the total number of cows at the end of a lactation that had not developed the disease of interest.

The standard error (SE) of the means of the lactational incidence rate and the incidence density were estimated with Proc Means (7) across averages of disease frequency by lactations and across averages of disease frequency by herd within year.

STATISTICAL ANALYSES

The effect of herd, age, year, season, and the interrelationships between diseases on the occurrence of disease within a lactation were evaluated. There were 4 seasons of calving, January to March, April to June, July to September and October to December; 2 y of calving, 1993–1994 and 1995 (year was not of primary importance,

but as recording had changed with year, confounding of associations was possible), and for the 4 lactation groupings, lactation 1, 2, 3–4, and lactation 5 and greater.

Because of the inherent intra-herd correlation between binary outcomes, statistical modeling of the data using the ordinary logistic regression is inappropriate. Several approaches have been proposed to analyze binary data gathered in clusters (11–13).

Our analyses were performed with a mixed random and fixed effects logistic model (GLIMMIX) (14) with herd as a random effect and lactation, year and season of calving included as fixed effects. The year-season interaction was also included when significantly ($P < 0.05$) related to the outcome. The covariates entered were diseases that occurred, on average, prior to the disease of interest. Covariates were removed, when the level of significance (ie, 5%) was exceeded. The GLIMMIX model used to obtain estimated effects was:

$$\text{logit}[E(y)] = X\beta + Zu$$

On the logit scale, expectations on the observation y were additive. In this equation, y is the observed occurrence of disease (0/1); X is the design matrix for the fixed effects; β is the vector of regression parameters to describe the dependence of the disease risk on the independent variables; Z is the design matrix for the random effects, and u is the vector of random herd effects.

Estimates of random effects were expressed as a deviation from the population average. The estimates of herd effects were cluster specific. In general, if clustering is minimal, the random effect could be removed; however, herd was maintained in all models to prevent confounding.

RESULTS

The data set included data on 32 herds with 12 471 lactations from 7542 cows. The descriptive data on cow numbers, lactations, age and culling are presented in Table I. The time of first disease occurrence within a lactation on 12 471 lactations is shown in Table II; the number of occurrences per interval is given for the periods 0 to 30, 31 to 150 and 151 to 365 d in lactation. Most of the diseases occurred during the first 30 d of lactation. Cases of cystic ovaries peaked in the interval from 31 to 150 d, while metritis and mastitis remained at a high level during that period. Footrot occurred with nearly equal frequency throughout the lactation.

TABLE I. Total number of cows, culled cows, age in days at calving and at culling in Holstein cows in Fraser Valley, British Columbia, 1994–1995

	Number		Mean age (d)		Standard deviation	
Lactation	Cows	Culled	Calving	Culling	Calving	Culling
1	4422	893	802	965	75	178
2	3248	736	1228	1436	131	206
3	2084	537	1638	1836	159	236
4	1240	362	2053	2247	227	292
5	697	232	2485	2686	303	410
6	345	124	2891	3079	339	316
7	154	67	3265	3447	389	496
8	63	25	3662	3733	326	226
9	34	15	3981	4202	208	252
10	17	8	4451	4471	327	215
11	5	3	5006	5225	536	683
12	1	0	4980	—	—	—

TABLE II. Days post partum to first occurrence of clinical disease in Holstein cows in Fraser Valley, British Columbia, 1994–1995

Category	Days from calving to first disease occurrence			Total number
and Disease	0–30	31–150	151–365	of cases
Other diseases				
Udder edema	108	1	4[b]	113
Metabolic disorders				
Milk fever	127	2	4[b]	133
Ketosis	51	6	1[b]	58
Nutritional disorders				
Displaced abomasum	55	16	4[b]	75
Reproductive findings				
Retained placenta[a]	242			242
Metritis	637	553	40	1230
Cystic ovary	21	439	128	588
Mastitis	238	245	170	653
Lameness				
Footrot	71	69	52	192

[a]Retained placenta could be recorded between 0–10 d post partum
[b]Likely prepartum cases for next lactation

The frequency of disease(s) by lactation number is shown in Table III. Most diseases, with the exception of udder edema and footrot, showed an increased risk of occurrence with lactation number, especially between the 1st and 5th lactations for retained placenta, mastitis and milk fever. Udder edema displayed significant decreases in frequency between lactations 2 and 5, ketosis increased between lactations 1 and 3–4, and cystic ovaries increased between lactations 1 and 3. The risk of metritis did not exhibit a monotonic rise in occurrence by lactation number.

Tables IV, V and VI show the results of the GLIMMIX modelling. The data comprised 11 795 records on 7151 cows; 4105 animals had one lactation, 2518 animals had 2, and 528 animals had 3 lactations.

Coefficients of lactations and seasons are presented in Table IV. Lactation had a significant effect ($P < 0.05$) on the risk of all diseases under study. There was a significant effect of season ($P < 0.05$) on the risk of mastitis from 0–30 d in lactation, retained placenta, metritis, cystic ovaries, mastitis from 31–150 d in lactation, ketosis, and footrot. The interaction between year and season was significant for retained placenta, mastitis occurrence from 31 to 150 d post partum (pp), metritis, cystic ovaries and stable footrot (not shown).

The variance parameter amongst herds was statistically significant ($P < 0.05$) for milk fever, retained placenta, metritis, ketosis, mastitis from 31–150 d in lactation, cystic ovaries and footrot (Table V), while for all other diseases it was significant at $P < 0.1$.

Figure 1 shows, conditional on herd, an informal path model. The coefficients of the independent

TABLE III. Clinical disease incidence per lactation in Holstein cows in Fraser Valley, British Columbia, 1994–1995

Disease	Lactation	Cases/Total	ID%[a]	LIR%[b]	Standard error %[c]
Udder edema	1	44/2002		2.2	0.7
	2	45/1379		3.3	
	3 & 4	22/1343		1.6	
	≥ 5	1/507		0.2	
Milk fever	1	1/3314		0.03	1.4
	2	12/2445		0.5	
	3 & 4	64/2489		2.6	
	≥ 5	56/1009		5.6	
Retained placenta	1	58/4422		1.3	0.6
	2	60/3248		1.9	
	3 & 4	73/3324		2.2	
	≥ 5	48/1316		3.7	
Metritis	1	423/4422		9.6	1.1
	2	289/3248		8.9	
	3 & 4	335/3324		10.1	
	≥ 5	169/1316		12.8	
Displaced abomasum	1	15/3166		0.5	0.2
	2	19/2300		0.5	
	3 & 4	35/2307		1.1	
	≥ 5	6/958		0.9	
Ketosis	1	9/3314		0.3	0.2
	2	13/2445		0.5	
	3 & 4	27/2489		1.1	
	≥ 5	9/1009	0.9		
Cystic ovaries	1	136/4422	3.2		0.8
	2	161/3248	5.2		
	3 & 4	203/3324	6.5		
	≥ 5	80/1316	6.7		
Mastitis	1	174/3229	5.6		1.0
	2	185/2306	8.4		
	3 & 4	191/2335	8.7		
	≥ 5	92/958	10.5		
Footrot	1	65/3192	2.1		0.1
	2	54/2317	2.5		
	3 & 4	54/2330	2.5		
	≥ 5	18/953	2.1		

[a] incidence density — see Materials and Methods for details
[b] lactational incidence rate — see Materials and Methods for details
[c] standard error of a disease over lactations

variables that were significantly ($P < 0.05$) related to the risk of the disease of interest are also shown (Table VI). Diseases directly linked with udder edema were (i) mastitis at 0 to 30 d, (ii) metritis, and (iii) cystic ovaries. Milk fever was directly associated with the risk of displaced abomasum. The statistically significant odds ratios were mostly greater than 2, with a range from 1.6 to 8.

DISCUSSION

The data originated from registered Holstein dairy herds, whose owners recorded findings in an on-farm record-keeping program. All study herds recorded reproductive disorders, but there were only 11 herds that had recordings for all clinical disease categories. There was an increase in disease frequency by year, which we attribute to better observation and/or more complete recording by the owners rather than an actual increase in disease frequency. Given our knowledge of the herds and the veterinary clinic, we believe the overall recorded disease occurrences are valid; however, given the low frequency of selected diseases in some herds (eg, metritis), we suspect a moderate level of under-reporting.

TABLE IV. Estimates (logistic coefficients) of association between season and lactation with disease in Holsteins in Fraser Valley, British Columbia 1994–1995

		Disease										
		Udder edema	Milk fever	Retained placenta	Mastitis 0–30 d pp[a]	Metritis	Displaced abomasum	Ketosis	Mastitis 31–150 d pp[b]	Cystic ovaries	Mastitis 151–365 d pp[c]	Footrot
Season												
Spring	Estimate	−0.21	0.01	0.68	−0.02	−0.17	0.16	0.64	0.33	−0.45	0.02	0.04
	SE	0.23	0.21	0.23	0.25	0.18	0.31	0.38	0.23	0.24	0.22	0.25
	OR	0.81	1.01	1.97*	0.98	0.85	1.18	1.91	1.39	0.64	1.03	1.04
Summer	Estimate	−0.29	−0.22	0.53	0.53	0.17	−0.24	0.71	−0.51	−0.48	0.03	−0.69
	SE	0.21	0.20	0.22	0.21	0.16	0.32	0.35	0.24	0.22	0.21	0.28
	OR	0.75	0.80	1.70*	1.70*	1.19	0.79	2.04*	0.60*	0.62*	1.03	0.50*
Fall	Estimate	0.01	−0.03	0.10	0.56	−0.39	−0.00	0.63	−1.87	−1.69	−0.06	−1.70
	SE	0.20	0.20	0.25	0.21	0.18	0.31	0.36	0.41	0.33	0.20	0.43
	OR	1.01	0.98	1.10	1.76*	0.68*	1.00	1.88	0.15*	0.19*	0.94	0.18*
Lactation												
2	Estimate	0.79	3.01	0.44	0.12	0.03	0.60	0.96	0.81	0.43	0.27	0.37
	SE	0.17	0.78	0.17	0.19	0.12	0.33	0.38	0.15	0.18	0.19	0.17
	OR	2.21*	20.22*	1.55*	1.13	1.04	1.83	2.62*	2.25*	1.53*	1.31	1.45*
3–4	Estimate	0.16	4.75	0.56	0.40	−0.05	1.24	1.73	0.83	0.78	0.57	0.33
	SE	0.21	0.76	0.16	0.19	0.12	0.30	0.34	0.16	0.18	0.19	0.17
	OR	1.17	115.79*	1.75*	1.49*	0.95	3.46*	5.65*	2.30*	2.17*	1.77*	1.39
≥ 5	Estimate	−1.48	5.66	1.21	0.93	0.49	0.41	1.54	0.84	0.84	0.58	0.21
	SE	0.77	0.76	0.19	0.24	0.17	0.46	0.41	0.20	0.23	0.27	0.25
	OR	0.23	286.69*	3.36*	2.54*	1.63*	3.46	4.65*	2.32*	2.31*	1.78*	1.24

SE — Standard error; OR — Odds ratio; pp — post partum.
[a] Mastitis occurrence between 0 and 30 d pp
[b] Mastitis occurrence between 31 and 150 d pp
[c] Mastitis occurrence between 151 and 365 d pp
* Significant $P < 0.05$

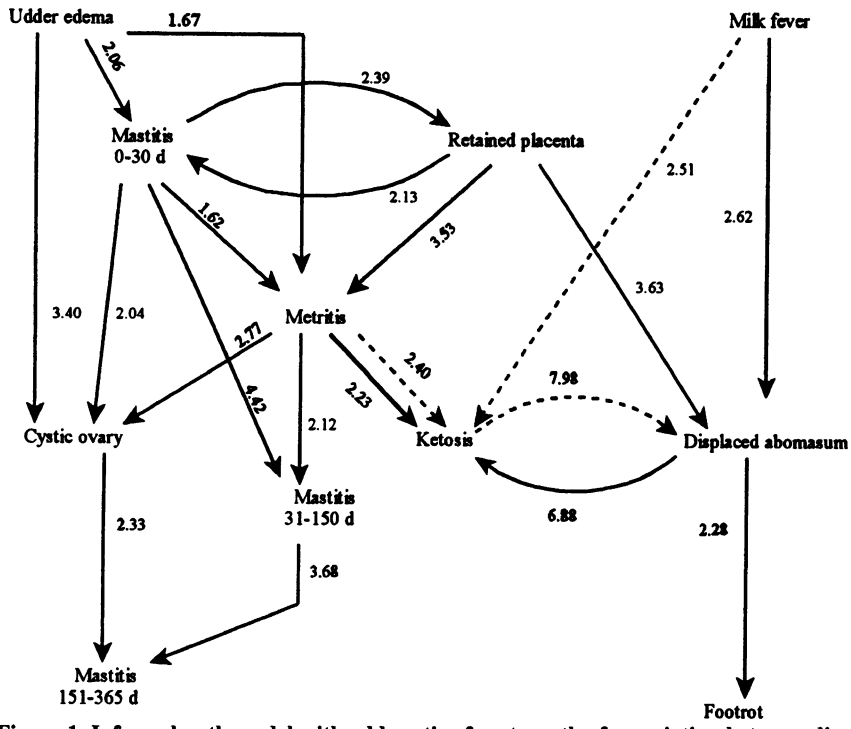

Figure 1. Informal path model with odds ratios for strength of association between diseases. Udder edema and milk fever were the earliest disorders followed by the other diseases shown below them in the graph. Although footrot could occur throughout the lactation we assumed it occurred after displaced abomasum. No connecting arrow indicates no significant association between diseases.

TABLE V. Influence of herd on disease in Holsteins in Fraser Valley, British Columbia

Disease		Herd variance
Udder edema	Estimate	5.23
	SE	3.00
	P	0.08
Milk fever	Estimate	1.74
	SE	0.67
	P	0.01
Retained placenta	Estimate	1.85
	SE	0.77
	P	0.02
Mastitis 0–30 d pp	Estimate	1.50
	SE	0.81
	P	0.07
Metritis	Estimate	0.83
	SE	0.40
	P	0.04
Displaced abomasum	Estimate	0.59
	SE	0.32
	P	0.06
Ketosis	Estimate	1.41
	SE	0.63
	P	0.02
Mastitis 31–150 d pp	Estimate	1.33
	SE	0.54
	P	0.01
Cystic ovaries	Estimate	0.72
	SE	0.37
	P	0.05
Mastitis 151–365 d pp	Estimate	0.76
	SE	0.40
	P	0.06
Footrot	Estimate	2.34
	SE	0.98
	P	0.02

Retained placenta, metritis, mastitis, cystic ovaries and stable footrot were probably regarded, by the owners, as the more important diseases, which could have a substantial impact on economic losses due to involuntary culling, reduced fertility, lower production and increased treatment costs (1,15–17). Udder edema, milk fever, displaced abomasum, and ketosis were probably not regarded as priority diseases, and this might have influenced the completeness of recording of these diseases.

Kelton et al (4) reviewed frequencies of diseases in dairy cows. Disease frequencies found in this study were within the range of the values reviewed, although, most of the disease frequencies were at the lower end of the range (perhaps, in part, for reasons stated previously).

Disregarding our apparently low frequencies of disease, comparing frequencies of disease across studies is difficult because of variation in definitions of disease and in calculations of "rates." Some diseases have been reported as an incidence (18), others as incidence per cow-year at risk or lactational incidence (19), while others reported frequencies as prevalence (20). Other authors also included repeated cases of a disease during the same lactation (21) in their measures.

In general, most diseases occurred early in lactation. This pattern was also detected in earlier studies (22–27). However, footrot occurred evenly spread throughout the lactation, and incidences of cystic ovaries were concentrated from 30 to 150 d pp. The "late" occurrence of the nonreproductive parturition-related diseases is likely due to their occurrence just before the next calving. There is a paucity of reported frequencies on udder edema; however, Dentine and McDaniel (28) found that the frequency of udder edema decreased with lactation number.

A mixed model was used to analyze the interrelationships between diseases with herd included as a random effect. This approach both controls for confounding by herd and allows the intercept parameter to vary from herd to herd. The coefficient of the random effect measures the change in the logit of disease risk in a herd. In general, the herd effect, which estimates the variance attributable to herd for the disease of interest, was statistically significant for most of the diseases under study (Table V). The herd effects were smaller than we expected, but since the study herds were a convenience sample from one veterinary practice, one should not make general inferences about the importance of herd effects in the population based on these data.

In accordance with previous studies, and bearing in mind that herd and lactation effects were controlled to prevent confounding, season of calving was not associated with udder edema (28), milk fever (22,29), and displaced abomasum (22). Because we had data for only 2 y, we cannot make strong inferences about reasons for seasonality or seasonal differences across years (interaction).

Significant lactation effects were present for all diseases. Note that the associations with lactation in Table IV are more refined than those in Table II, because a number of potential confounding variables are controlled in the GLIMMIX model.

Udder edema occurred mostly in first and second lactation, which was also found by Dentine and McDaniel (28). The incidence of milk fever significantly increased with lactation,

TABLE VI. Logistic regression coefficient estimates, standard errors, and odds ratios for associations between diseases based on a path model structure[a]

Dependent variable[a]		Independent variables								
		Udder edema	Milk fever	Retained placenta	Mastitis 0–30 d pp	Metritis	Displaced abomasum	Ketosis	Mastitis 31–150 d pp	Cystic ovaries
Udder edema		NA	NS							
Milk fever			NA							
Retained placenta	Estimate				0.87					
	SE				0.24					
	OR				2.39					
Mastitis 0–30 d pp	Estimate	0.72		0.76						
	SE	0.32		0.28						
	OR	2.06		2.13						
Metritis	Estimate	0.51		1.26		0.48				
	SE	0.23		0.18		0.19				
	OR	1.67		3.53		1.62				
Displaced abomasum[b]	Estimate		0.96	1.29/1.33*[d]				2.08*		
	SE		0.44	0.34/0.34*				0.45*		
	OR		2.61	3.63/3.79*				7.98*		
Ketosis[c]	Estimate		0.92*			0.80/0.87*	1.93			
	SE		0.39*			0.28/0.28*	0.41			
	OR		2.51*			2.23/2.40*	6.88			
Mastitis 31–150 d pp	Estimate				1.52	0.67				
	SE				0.17	0.14				
	OR				4.56	1.95				
Cystic ovaries	Estimate	1.22			0.71	1.02				
	SE	0.33			0.26	0.16				
	OR	3.40			2.04	2.77				
Mastitis 151–365 d pp	Estimate								1.30	0.85
	SE								0.20	0.25
	OR								3.68	2.33
Footrot	Estimate						0.82			
	SE						0.38			
	OR						2.28			

SE — standard error; OR — odds ratio; pp — post partum

[a] Ordering of health disorders reflects their putative causal ordering, beginning with udder edema and milk fever

[b] Displaced abomasum could occur via 2 different paths, ie, by milk fever and retained placenta or by retained placenta and ketosis

[c] Ketosis could occur via 2 different paths, ie, milk fever and metritis or by metritis and displaced abomasum

[d] Displaced abomasum in one model is associated with milk fever and retained placenta, whereas in the other model displaced abomasum is associated with retained placenta and ketosis (shown by *)

which is in agreement with other studies (22,25,26).

The risk of retained placenta increased significantly with lactation, which was also found by other researchers (3,23,25,27), but not by Dohoo et al (22) who found irregular patterns for age. These irregular patterns for age (22) may have been caused by the small number of observations or by the statistical model they used.

Metritis was associated with a significant increase in odds for lactation 5 and higher. This was in agreement with Erb and Martin (27), but differed from Dohoo et al (22) and Grohn et al (23), who found no differences in odds between lactations, and Markusfeld (30) who found the highest risk of metritis for first lactation cows.

Our results for abomasal displacement and ketosis are in contrast to those of Dohoo et al (22) and Markusfeld (30), who did not find an association between parity and displaced abomasum. The increase of ketosis by lactation agreed with previous research (22,26,28,31). Ketosis has been reported to be associated with immune suppression (32), and hence possibly related to the occurrence of mastitis and footrot.

The risk of cystic ovaries significantly increased for lactations 3 and 4 animals compared to lactation 1. This was in agreement with other authors (22,27), who found an increase in odds of cystic ovarian disease with age.

In the present study we found an increase in mastitis occurrence with lactation, which was previously found by Dohoo et al (22). Footrot was the only disorder that was not significantly associated with lactation in our study; however, Dohoo et al (22) found an increase in odds of lameness with age. This difference was probably caused by pooling different types of lameness into one category. Dohoo et al (22) included all problems of feet and legs in one general category, and we used only specific cases of footrot.

The path model structure allows us to postulate an ordering of causation based on biological plausibility and temporal considerations. In these models the earliest occurring diseases may affect the occurrence of all later diseases. The analysis helps identify associations of statistical significance and allows the reader to focus on these as the most likely important causal paths. Note that our model controls for herd, year, season, year-season interaction and lactation.

In the present study, at the clinical level the 2 'earliest' occurring diseases, milk fever and udder edema, were not related to each other. Thus, it appears that there might be 2 distinct causal pathways which start at either udder edema or milk fever (Table VI, Figure 1). Occurrence of udder edema, milk fever, or retained placenta indicated that animals developing these diseases early in lactation had an increased risk of other diseases that occurred later in lactation and these effects came about by

both direct and indirect pathways (Figure 1). For example, the overall impact of udder edema on metritis was direct, as well as indirect via mastitis from 0–30 d pp.

No association between milk fever and retained placenta was found in this study, nor in the study by Klerx and Smolders (10). In earlier studies (23,30) other authors found an association between milk fever and retained placenta, or an indirect pathway between milk fever and retained placenta via dystocia (29). The association between mastitis from 0 to 30 d in lactation and retained placenta (odds ratio = 2.39 and 2.13) represents the unknown causal nature of the interrelationship between them (Figure 1). It is most likely that retained placenta preceded mastitis because retained placenta could only be recorded during the first 10 d of lactation and predominantly occurs during the first 24 h of lactation. The association we observed between retained placenta and mastitis was not noted by Correa et al (29), but was found by Oltenacu and Ekesbo (17). The association we observed between retained placenta and metritis, as well as the association between metritis and cystic ovaries, has been previously described (10,22,23,29,30).

Our finding of an association between mastitis 0–30 d in lactation and cystic ovaries disagreed with the findings of Oltenacu and Ekesbo (17). However, Oltenacu and Ekesbo (17) used a risk period from 0–50 d for mastitis in contrast to our 0–30 d period. This may suggest that only early cases of mastitis are associated with the occurrence of cystic ovaries.

The risk of metritis (Figure 1) was directly increased by mastitis during the first 30 d in lactation and retained placenta, and also by udder edema. Metritis was often an intervening variable; for example, ketosis was indirectly (via metritis) associated with udder edema, mastitis from 0–30 d in lactation, and retained placenta. Displaced abomasum may also have acted as an intervening variable for the indirect risk between retained placenta and ketosis. Bendixon et al (31) found a direct positive association between milk fever in combination or a single occurrence of retained placenta with ketosis. The association we noted between retained placenta and displaced abomasum was explored (29), but not observed by Correa et al (29).

The highest disease to disease risk was obtained when a cow experienced displaced abomasum or ketosis. The association of ketosis with displaced abomasum represents the unknown causal nature of the interrelationship between these 2 diseases. Either could have been the cause of the other, or an unknown factor might have caused both. In some instances ketosis occurred prior to displaced abomasum, whereas in other instances, displaced abomasum occurred prior to ketosis. Both are plausible causal pathways. Cows with displaced abomasum had a 6.88 times greater risk of developing ketosis compared to an animal that did not develop displaced abomasum. Cows with ketosis had a 7.98 times greater risk of developing displaced abomasum than non-ketotic cows. Correa et al (29) explored the risk of ketosis on displaced abomasum, but not vice versa. They (29) found that ketosis was positively associated with displaced abomasum. Footrot was directly positive related with displaced abomasum, and indirectly with milk fever, retained placenta, metritis, and ketosis. Unfortunately, Klerx and Smolders (10) did not include displaced abomasum in their study.

In conclusion, the current study provided insight into disease occurrence in dairy herds by using data from an on-farm record-keeping program. Such data can be useful to assess the incidence of disease, and to quantify associations among diseases. Nonetheless, the incidence of disease may have been biased by the general interest, and hence recording completeness of the herd person. However, the results of this study should motivate producers to record health disorders as this may prove useful for disease prevention. This effort may be helped by our informal path analysis, which showed that incidences of disease early in lactation can have a cumulative effect on the occurrence of other diseases later in lactation.

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