Effect of agent-specific clinical mastitis on herdlife in 2 New York dairies

YT Gröhn*[1], RN González[2], DJ Wilson[2], H Schulte[2], G Bennett[2], JA Hertl[1], YH Schukken[2]
[1]Section of Epidemiology and [2]Quality Milk Production Services (QMPS), Dept. of Population Medicine and Diagnostic Sciences, Cornell University, Ithaca, NY 14853

SUMMARY

We used a Cox proportional hazards (PH) model with time-dependent covariates (TDCs) to estimate the effects of clinical mastitis (CM) without agent identification (generic CM (GCM)) and CM with specific agent identification (SCM) (*Streptococcus* spp., *Staphylococcus aureus, Staph.* spp., *Escherichia coli, Klebsiella* spp., *Arcanobacterium pyogenes* and 'no agent isolated') on herdlife in 2697 cows in 2 New York farms. Other variables included farm, calving season, parity,weekly milk yield (MY) and other diseases. Culling often occurred soon after GCM diagnosis. Including MY reduced GCM's effect on culling, due to MY's intervening effect. Most SCM, particularly *Klebsiella* spp., greatly reduced herdlife. Treating CM as time-dependent let us see its varying effects on herdlife.

INTRODUCTION

Dairies cull cows for 5 main reasons: age, disease, MY, reproduction and lactation stage. CM is an important dairy disease. Few studies have incorporated TDCs (3) or SCM's effects on culling. Survival analysis (SA), using the Cox PH model (1), is a good way to study factors affecting herdlife. It describes the hazard of an event (e.g. culling) at any time. SA uses all subjects, whether they experienced the event or not. Our aim was to study effects of GCM and SCM on herdlife, using SA with TDCs.

MATERIALS AND METHODS

We collected data from 2 New York State farms from 10/1/99-7/31/01 (Farm A) and 10/1/99-3/31/01 (Farm B). Milk samples were microbiologically diagnosed at QMPS. Besides CM, we modeled 8 other diseases (dystocia, milk fever, retained placenta, metritis, ketosis, displaced abomasum (DA), lameness, ovarian cyst) as possible confounders. We fit Cox PH models in a Fortran program (2); it allows TDCs in SA. We followed cows for one lactation, from calving until next calving, culling or study end. Cows that did not have the event of interest (culling) were censored. Data were stratified by farm. The output, hazard ratios (HRs), measure a factor's effect on herdlife.

For 6 agents *(Strep.* spp., *Staph. aureus, Staph.* spp., *E. coli, Klebsiella* spp. and 'no agent isolated'), CM and culling could occur either before or after 120d. This day was chosen based on the distribution of CM cases. HR's were obtained for 3 intervals: 1) CM and culling both before 120d; 2) CM before 120d and culling after 120d and 3) CM and culling both after 120d. There were too few cases of *A. pyogenes* to divide the lactation, so it was coded 'did/did not occur'. GCM and culling could occur 1-7d, 8-30d, 31-120d, 121-200d and >200d. We also made intervals for other diseases based on their occurrence.

We fit 7 models for GCM's effect on herdlife: 1) CM; 2) Parity, calving season, CM; 3) Parity, calving season, MY, other diseases (see above), CM; 4) Parity, calving season, other diseases (see above), CM; 5) Parity, calving season, MY, CM; 6) Parity, calving season, other significant diseases (DA, lameness, ovarian cyst), CM; 7) Parity, calving season, MY, other significant diseases (DA, ovarian cyst), CM. SCM's effects on herdlife are from Model 6, which was deemed most appropriate.

RESULTS AND DISCUSSION

Farm A had more cases of CM than Farm B (Table 1); the latter tended to record only more severe cases. Cull rates in both farms were relatively low (around 20%) because of herd expansion. The incidence of CM due to *Strep.* spp., *Staph. aureus* and 'no agent isolated' rose with age (Table 2). The incidence of *Staph.* spp. CM was the same for Parity 1 and 2 cows and higher in older cows. *A. pyogenes* was more common in older cows. *E. coli* and *Klebsiella* spp. had no trend with age although they were generally more common in older cows. GCM increased with age.

Cows with GCM were more likely to be culled than non-GCM cows (Table 3). Culling was more likely soon after diagnosis. HRs were lower in models with MY, indicating that MY is an intervening variable; CM affects MY, which in turn affects culling. The increased risk is actually due to CM: including MY underestimates CM's effect on culling.

Except for 'no agent isolated', SCM increased culling (Table 4). *Klebsiella* spp. was most detrimental before 120d (HR=3.3). After 120d, cows with *Staph. aureus*, *Staph.* spp., *E. coli* or *Klebsiella* spp. were at least 3 times more likely to be culled than non-SCM cows. Culling tended to occur soon after diagnosis; HRs were usually lowest for CM diagnosed before 120d and culling occurring after 120d.

CONCLUSIONS

GCM significantly increased culling, especially soon after diagnosis. Including MY in the model reduced its effect, implying that MY is an intervening variable. SCM, especially due to *Staph. aureus*, *Staph. spp*. and *Klebsiella* spp., also had large effects. Treating CM as a TDC gave us better insight into its effect on herdlife. The findings increase our understanding of culling of mastitic cows, both in terms of when in lactation they contract CM, and when they are culled.

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Table 1. Description of 2 New York State dairies: Lactational incidence rates (%) of 1st occurrence of each CM agent and culling rate.

	Dairy A (1107 cows)	Dairy B (1590 cows)
Strep. spp.	8.8%	2.3%
Staph. aureus	4.4%	1.1%
Staph. spp.	2.4%	1.4%
E. coli	8.0%	3.8%
Klebsiella spp.	3.3%	2.1%
A. pyogenes	1.4%	0.6%
No agent isolated	7.7%	2.5%
All agents (GCM)	26.9%	12.1%
Culling rate (%)	21.3%	19.1%

Table 2. Lactational incidence rate of CM pathogens (1st occurrence) by parity in 2697 cows in 2 farms

Disease	Par 1 (n=1261)	Par 2 (n=585)	Par 3 (n=464)	Par 4+ (n=387)
Mastitis caused by:				
Strep. spp.	3.6%	4.1%	5.6%	9.8%
Staph. aureus	1.3%	2.6%	3.4%	4.7%
Staph. spp.	1.5%	1.5%	2.2%	2.8%
E. coli	3.6%	7.4%	6.5%	8.0%
Klebsiella spp.	0.4%	3.2%	5.6%	5.2%
A. pyogenes	0.2%	0.5%	2.2%	2.3%
No agent isolated	2.7%	3.8%	6.7%	9.6%
All agents (GCM)	12.0%	19.0%	23.7%	30.7%

Table 3. Effects of GCM on culling. The numbers are HRs (*: significant at p=0.05). See text for description of the 7 models. The reference categories were cows without CM.

Stage of mastitis, stage of culling	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
M1, C1	1.5	1.4	0.8	1.3	0.8	1.3	0.8
M1, C2	4.3*	3.8*	1.6	3.3*	1.4	3.2*	1.6
M1, C3	2.9*	2.6*	2.6*	2.5*	2.6*	2.5*	2.8*
M1, C4	2.5*	2.3*	4.0*	2.2*	3.6*	2.3*	3.8*
M1, C5	1.9	1.7	1.2	1.7	1.2	1.8	1.2
M2, C2	14.8*	13.3*	3.8*	12.7*	3.3*	12.7*	3.8*
M2, C3	3.1*	2.6*	1.1	2.5*	1.0	2.5*	1.1
M2, C4	2.2	1.8	3.2	1.7	2.3	1.7	3.6
M2, C5	2.5	1.9	2.0	1.9	2.0	1.8	2.1
M3, C3	6.1*	4.8*	1.5	4.4*	1.4	4.5*	1.5
M3, C4	4.6*	3.5*	1.7	3.2*	2.0*	3.2*	1.8*
M3, C5	2.9*	2.2*	2.0*	2.2*	2.0*	2.2*	2.0*
M4, C4	24.1*	20.4*	5.1*	21.2*	5.1*	21.2*	5.1*
M4, C5	2.2*	2.0*	2.1	2.2*	2.1	2.2*	2.1*
M5, C5	4.2*	4.0*	2.5*	4.3*	2.5*	4.2*	2.6*

Table 4. Effects of SCM on culling. The numbers are HRs (*: significant at p=0.05), from Model 6 (all agents in same model). Reference cows were those without the agent-specific CM.

Interval[1]	*Strep.* spp.	*Staph. aureus*	*Staph.* spp.	*E. coli*	*Klebsiella* spp.	No agent isolated	*A. pyogenes*[2]
M1, C1	2.8*	2.8*	1.5	2.5*	3.3*	1.7	2.0*
M1, C2	1.1	2.5*	1.8	1.3	1.6	1.5	--
M2, C2	1.9*	3.0*	3.5*	3.2*	4.1*	1.4	--

[1]M1, C1: Mastitis in 1st 120d, culled in 1st 120d; M1, C2: Mastitis in 1st 120d, culled after 120d
M2, C2: Mastitis after 120d, culled after 120d
[2]As it occurs