Incidences and Effects of Diseases on the Performance of Swedish Dairy Herds Stratified by Production

U. EMANUELSON* and P. A. OLTENACU†
*Swedish Association for Livestock Breeding and Production, S-631 84 Eskilstuna, Sweden
†Department of Animal Science, Cornell University, Ithaca, NY 14853

ABSTRACT

Incidences of diseases and their effects on reproductive performance and risk of culling in herds stratified by production and estrus detection efficiency were studied. Data were from the Swedish milk and disease recording systems and consisted of records for 33,748 first parity Swedish Friesian cows. A standardized mixed threshold model was used for statistical analyses of categorical outcome variables, and an ordinary linear mixed model was used for continuous outcome variables.

An increase in production was associated with increased frequencies of treatments of most diseases, shorter intervals from calving to first artificial insemination, fewer days open, and lower culling rates. Cows treated for metritis, silent estrus, and cystic ovaries had an increased number of days to first artificial insemination and more days open. However, the negative consequences of these diseases on reproductive performance decreased as herd production increased. The risk of culling was higher for cows treated for dystocia, cystic ovaries, and mastitis, but the increase in the risk of culling was lower for higher producing herds. Similar trends were observed when herds were stratified by estrus detection efficiency. The results support the hypothesis that herd management, as characterized by milk production or estrus detection efficiency, is important in the incidences and consequences of diseases. Herd management, measured directly or indirectly, should be considered when the health status or cost of disease for a given herd is evaluated.

(**Key words**: dairy cows, risk factors, herd production, estrus detection efficiency)

Abbreviation key: **CM** = clinical mastitis, **CO** = cystic ovaries, **EDE** = estrus detection efficiency, **RP** = retained placenta, **SES** = silent estrus, **VWP** = voluntary waiting period.

INTRODUCTION

The increase in milk production over the last four decades has been accompanied by an increase in health problems and, subsequently, health costs. Solbu (13) showed that, in Norway, between 1950 and 1978, along with a doubling in milk production per cow, the number of veterinary treatments per 100 cows increased from 40 to 110.

Milk production, reproduction, and health are the principal factors that affect the profitability of a dairy herd. In recent years, the dairy industry agenda in many countries has been dominated by health-related problems. The increased interest in the control of these problems focuses around three main points. First, a continuing increase in milk production has made health problems more prevalent and increasingly important. Second, because a surplus of milk exists in many countries and a quota system is in place in Canada and several European countries as well as low milk prices in other countries, emphasis has shifted from an increase in output to a reduction in the cost of production as a means of increasing or maintaining profitability. Finally, as incomes rise and social values change, more attention is given to the health aspects of dairy production and the welfare of farm animals.

One prerequisite for effective health management is accurate knowledge of the factors that affect the health status of a cow, such as parity, season, or health history, and of the association between health problems and other economically important traits, such as milk production, reproduction, and length of productive life. The parameters required for health management are specific to population and time and need to be estimated periodically.

Field data are generally used to estimate parameters for the population of interest. However, health management varies among herds, and it can

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be hypothesized that this variation may influence the incidences and consequences of diseases. Better herd health management is reflected in more effective strategies for the identification and treatment of sick cows. Health problems may be identified earlier and with better follow-up, implying that the detrimental effects of diseases on the performance of cows may vary according to herd management. Estimates based on populations may exaggerate the losses associated with diseases in some herds and underestimate the losses in others. Therefore, there is a need for estimates that are specific for herds with different health management practices.

However, the large data sets needed for epidemiological studies often come from general databases on production and diseases, and information regarding herd management is most often lacking in these databases. Therefore, other options for the evaluation of herd management have to be investigated, and one alternative is to use indicator variables, such as milk production or measures of reproductive performance, as proxies.

The objective of this study was to evaluate the hypothesis that incidences and effects of diseases on reproductive performance and culling vary with herd management. Levels of milk production and estrus detection efficiencies (**EDE**) were used to categorize herd management.

MATERIALS AND METHODS

Data

Data for this study were from first parity cows of the Swedish Friesian breed in the Skara region of Sweden. The Skara region was chosen because it closely mirrors the general Swedish population of dairy herds with respect to herd size, breed distribution, and housing conditions and because reliable and complete records on health, production, and reproduction were available for this region. All cows that calved between January 1, 1983 (i.e., the starting date for the official disease recording scheme) and December 31, 1987 were included. The latter date was chosen to give all cows ample time to obtain complete records at the time of data collection. Only herds with >14 recorded calvings were eligible for inclusion in the study. The total data set consisted of 33,748 cows in 1090 herds.

Information available for each individual cow record included complete identification (e.g., herd, breed, and cow identification), all dates (i.e., birth, calving, breedings prior to and after first calving, and culling or next calving), milk production, and all diagnosed diseases as recorded in the national disease recording system (2).

The disease traits considered as outcome variables were stillbirth, dystocia, retained placenta (**RP**), metritis, ketosis, silent estrus (**SES**), cystic ovaries (**CO**), and clinical mastitis (**CM**). Only the first diagnosis of each disease was considered. Stillbirth included late abortions, delivery of a dead calf, or death within 12 h after parturition. Dystocia included all cases requiring veterinary assistance. Retained placenta was diagnosed when the placenta was retained for >24 h after parturition. Metritis included diagnoses of acute puerperal metritis, endometritis, and pyometra. Ketosis was recorded only after veterinary diagnosis of clinical ketosis within the first 50 d after parturition. Cystic ovaries described the presence of ovarian structures determined by rectal palpation, and SES represented either organic or functional anestrus. Essentially, all diagnoses of CO and SES resulted from infertility examinations during the breeding period and were associated with problematic cows. With respect to CM, we hypothesized that the epidemiology was different depending on when CM was first diagnosed (11). Episodes of CM that occurred during the following periods were regarded as separate episodes: prior to or at calving, between d 1 and 50 of lactation, between d 51 and 250 of lactation, and between d 251 and the end of lactation. However, only the first diagnosis was considered (i.e., a cow that had been diagnosed with CM prior to calving was not at risk for CM during the second period). Some diagnosed disorders were also regarded as risk factors or predictor variables (Table 1), and each record was checked, therefore, so that only diseases as a risk factor occurring before diseases as outcomes remained in the analysis.

The consequences of diseases on days from calving to first AI, days open, and culling were studied. When the effects of diseases on fertility traits were studied, we restricted the data to cows with a verified second calving because pregnancy could only be verified in those records and they would all have had at least one AI. We were aware that this procedure could have introduced some bias, but the alternative, to assume pregnancy for all cows from the last AI, could have also introduced some bias. Culling included all reasons for removal from the herd (i.e., also sold for dairy purposes).

Herds were classified according to mean milk production. Mean milk production was based on kilograms of 4% fat-corrected cumulative milk production for the first 200 d for all first parity cows in the herd. Cut-off points were chosen to yield four classes; approximately the same number of herds was included

TABLE 1. Statistical models.

Outcome variable	Independent variable
Stillbirth (SB)	Base[1]
Dystocia	Base
Retained placenta (RP)	Base + SB + dystocia + CM0[2]
Metritis	Base + SB + dystocia + CM0 + RP
Silent estrus (SES)	Base + SB + dystocia + (CM0 + CM1) + RP + metritis + ketosis
Cystic ovaries (CO)	Base + SB + dystocia + (CM0 + CM1) + RP + metritis + ketosis
CM	
CM0	Base
CM1	Base + SB + dystocia + RP
CM2	Base + SB + dystocia + RP + metritis + ketosis
CM3	Base + SB + dystocia + RP + metritis + ketosis + SES + CO
Ketosis	Base + SB + dystocia + CM0 + RP
Days to first AI	Base + SB + dystocia + (CM0 + CM1) + RP + metritis + ketosis + SES + CO
Days open	Base + SB + dystocia + (CM0 + CM1) + RP + metritis + ketosis + SES + CO
Culling	Base + SB + dystocia + (CM0 + CM1 + CM2 + CM3) + RP + metritis + ketosis + SES + CO

[1]Effects of herd, year and season of calving, and age at calving.

[2]Clinical mastitis (CM) was divided into episodes diagnosed prior to or at calving (CM0), between d 1 and 50 after calving (CM1), between d 51 and 250 after calving (CM2), and between d 251 after calving and the end of lactation (CM3).

in each class. The cut-off points used, in addition to the minimum and maximum values, were 3953, 4203, and 4431 kg, respectively.

Herds were also classified according to mean EDE. Information on intervals between calving and AI for individual cows were used to calculate the voluntary waiting period (**VWP**) and prebreeding EDE according to principles described by Fetrow et al. (6). Cows that were not inseminated and cows with >150 d between calving and first AI were excluded. The VWP for a herd was defined as the number of days postcalving when 5% of the cows had received their first AI. From that date, four periods of 20 d (equivalent to one estrus period) were defined (i.e., VWP to VWP + 20 d, VWP + 21 d to VWP + 40 d, VWP + 41 d to VWP + 60 d, and VWP + 61 d to VWP + 80 d, respectively). A fifth period covered VWP + 81 d to 150 DIM. The EDE for periods 1 through 4 was calculated as the number of cows inseminated during the period divided by the number of cows available for AI:

$$EDE_w = n_w / \sum_{w}^{5} n_i$$

where EDE_w = EDE during period w, and n_w = number of cows inseminated during period w (w = 1 to 4). The EDE of the herd was calculated as the mean of EDE_1 and EDE_2. The cut-off points used for stratification of herds were 0.40, 0.50, and 0.60, respectively.

Statistical Analysis

Separate analyses were performed for each herd production and EDE group. An ordinary mixed linear model was used for the analysis of days to first AI and days open. A standardized mixed threshold model, assuming that observed values were expressions of underlying, unobservable, normally distributed liability variables, was used for the disease traits and for culling. The following basic mixed model was used for both linear and threshold analyses:

$$y_{ijklm} = h_i + t_j + s_k + a_l + e_{ijklm}$$

where

y_{ijklm} = record on cow ijklm,
h_i = random effect of herd i,
t_j = fixed effect of year of calving j,
s_k = fixed effect of calving season k,
a_l = fixed effect of age at calving l, and
e_{ijklm} = random residual effect.

Five separate years, 1983 to 1987, and four calving seasons, January to March, April to June, July to September, and October to December, were considered. Age at first calving was ≤24 mo, >24 mo and ≤28 mo, >28 mo and ≤32 mo, or >32 mo. The random herd and residual effects were assumed to have 0 means; to be normally distributed with variances equal to σ_h^2 and σ_e^2, respectively; and to be mutually uncorrelated.

The statistical models used to analyze each outcome variable are described in Table 1. The basic model was used when studying stillbirth, dystocia, and CM prior to or at calving. The base model was expanded to include additional risk factor diseases when other traits were analyzed.

A computer program for analysis of categorical traits, which were based on principles described by Misztal et al. (9), was used in the threshold analyses. The equations were solved iteratively using Fisher's scoring and a modification of the expectation-maximization algorithm for variance component estimation. Variance components were estimated by using a REML algorithm for threshold models. Twenty rounds of Fisher's scoring iterations were made for each analysis, and eight rounds of variance compo-

TABLE 2. Predicted incidences[1] of diseases in herds stratified by production.[2]

	Milk production			
Disease	2869 to 3952 kg	3953 to 4202 kg	4203 to 4430 kg	4431 to 5824 kg
Stillbirth	1.3	1.7	1.4	2.2
Dystocia	0.8	0.3	0.7	1.0
Retained placenta	3.9	4.7	3.3	4.5
Metritis	1.2	1.6	1.8	2.3
Silent estrus	2.4	2.0	3.2	4.0
Cystic ovaries	0.0	0.5	0.4	0.9
Clinical mastitis (CM)[3]				
CM0	0.8	0.7	1.2	1.8
CM1	3.4	4.7	4.3	4.4
CM2	3.1	4.5	3.7	5.0
CM3	1.8	1.7	2.1	1.5
Ketosis	0.7	0.1	0.9	1.0

[1]Estimated using a threshold model.

[2]Herd production was measured as the mean cumulative 4% fat-corrected milk production at 200 DIM.

[3]Clinical mastitis was divided into episodes diagnosed prior to or at calving (CM0), between d 1 and 50 after calving (CM1), between d 51 and 250 after calving (CM2), and between d 251 after calving and the end of lactation (CM3).

nent estimation were completed for every second round of Fisher's scoring.

Linear model analyses were performed with a package developed by Meyer (7, 8) that yielded REML estimates of variance components. The iterative process was terminated when the difference between successive estimates was <0.01%.

Solutions from standardized threshold models are on the underlying liability scale. However, they were used to predict incidences on the observed scale. The predicted incidences were calculated as $\Phi(\mathbf{k}'\boldsymbol{\beta})$, where Φ = standard normal distribution function, $\mathbf{k}$ = vector of weights, and $\boldsymbol{\beta}$ = vector of solutions. The $\mathbf{k}$ vector was constructed to yield predicted incidences for each subclass in a given factor, keeping all other factors at an average level. Therefore, the numbers in the $\mathbf{k}$ vector that did not correspond with the factor of interest were chosen to give equal weight to all herds, years, seasons, and calving ages. However, the weights represented the overall incidence in the population for the risk factor diseases when such were included in the model.

RESULTS AND DISCUSSION

Predicted incidences of all diseases for herds with different milk production were rather low for most diseases (Table 2) but were consistent with other results published on first parity Swedish Friesian cows (3, 12). However, an increase in predicted incidences of diseases as herd production increased was discernible for many diseases.

Because for some diseases, such as CM, ketosis, and CO, milk production is a known risk factor (5), it is possible that the increase in incidence rate as herd production increased might have been due to production. However, it is important to realize the difference between the true incidence of a disease and the incidence of treatments for a disease; our data were based on treatment records. Therefore, we would argue that the increase could be a consequence of better management because differences in mean milk production among herds were small and because an increase in the incidence rate as herd production increased was also observed for those diseases for which milk production was not considered to be a risk factor. Further support for this assumption is provided by Ekman (1) who found that the incidences of most treatments were higher in well-managed herds than in poorly managed herds.

Predicted incidences of RP and metritis, as affected by the occurrence of other diseases, in herds stratified by production are presented in Table 3. Dystocia and CM prior to or at calving are major risk factors for RP, and risk increases as herd production increases. This relationship is also true for dystocia and RP, which are major risk factors for metritis. We would argue that this increased risk as herd production increases is also a consequence of better management, reflecting a better follow-up on cows with diseases.

Days to first AI and days open are two standard measures of reproductive performance, and one would expect these measurements to decrease as herd management increased. As shown in Tables 4 and 5,

TABLE 3. Effects of diseases on predicted incidences of retained placenta and metritis in herds stratified by production.[1]

	Milk production			
Disease	2869 to 3952 kg	3953 to 4202 kg	4203 to 4430 kg	4431 to 5824 kg
Retained placenta				
+ No other disease	3.9	4.7	3.3	4.5
+ Stillbirth	3.9	4.3	4.5	5.2
+ Dystocia	9.5	15.3	15.0	10.6
+ Clinical mastitis[2]	4.3	7.8	4.1	5.5
Metritis				
+ No other disease	1.2	1.6	1.8	2.3
+ Stillbirth	0.5	1.4	1.4	1.4
+ Dystocia	4.7	7.1	7.2	13.0
+ Clinical mastitis	1.0	0.8	2.0	0.8
+ Retained placenta	4.4	7.9	8.7	9.5

[1]Herd production was measured as the mean cumulative 4% fat-corrected milk production at 200 DIM.

[2]Diagnosed prior to or at calving.

TABLE 4. Effects of the occurrence of disease on days to first AI (DFAI) in herds stratified by production.[1]

	Milk production			
	2869 to 3952 kg	3953 to 4202 kg	4203 to 4430 kg	4431 to 5824 kg
Overall mean DFAI	83.3	81.8	80.7	77.9
Disease				
Retained placenta	+3.3	+4.1	+3.0	+2.0
Metritis	+6.7	+7.9	+3.6	+5.2
Silent estrus	+37.0	+32.0	+30.9	+29.9
Cystic ovaries	+11.0	+29.3	+35.2	+22.0

[1]Herd production was measured as the mean cumulative 4% fat-corrected milk production at 200 DIM.

herd means decreased for both measurements from low producing herds to high producing herds when herds were stratified according to production.

Diseases known to affect reproductive performance are RP, metritis, CO, and SES (12). The effects of these diseases on days to first AI and days open are shown in Tables 4 and 5, respectively. For low producing herds, the increase in days open associated with these four diseases was 8.7, 20.7, 44.8, and 46.6 d, respectively, and these negative effects decreased as herd production increased. This decreasing negative effect further supports the argument that there are differences attributable to herd health management among herds stratified according to production.

Possible explanations for a smaller detrimental effect on the performance of cows in herds under good management are that health problems are identified earlier and have a better prognosis and that better follow-up is pursued after treatment. Median days from calving to treatment for SES and CO were 10 to 20 d shorter in herds under good management than in herds under poor management.

Effects of diseases on risk of culling in herds stratified by production are shown in Table 6. Among the diseases considered, dystocia, CO, CM prior to or at calving, and CM between 1 and 50 DIM are major risk factors for culling, and, except for dystocia, the risk of culling decreases as herd production increases. The lack of change in the risk of culling that is associated with dystocia could be explained by the fact that these are cases that required the assistance of a veterinarian, and the risk of culling for such severe cases is expected to be similar across herds stratified by herd management. Conversely, the effect of dystocia as a risk factor for RP and metritis, which, if increased risk for sequelae represent better follow-up as we are suggesting, should be influenced by herd management, was shown to increase as herd production increased (Table 3).

The results presented can be used to show that the cost associated with a disease varies among herds. A case of CO, for instance, increased days open between 39 and 60 d for herds with different levels of milk

TABLE 5. Effects of the occurrence of disease on days open (DO) in herds stratified by production.[1]

	Milk production			
	2869 to 3952 kg	3953 to 4202 kg	4203 to 4430 kg	4431 to 5824 kg
Overall mean DO	106.7	105.0	103.3	99.0
Disease				
Retained placenta	+8.7	+2.5	+5.9	+1.7
Metritis	+20.7	+18.1	+14.8	+14.9
Silent estrus	+44.8	+38.0	+34.6	+33.4
Cystic ovaries	+46.6	+59.7	+44.4	+39.3

[1]Herd production was measured as the mean cumulative 4% fat-corrected milk production at 200 DIM.

TABLE 6. Effects of the occurrence of disease on risk of culling in herds stratified by production.[1]

	Milk production			
Disease	2869 to 3952 kg	3953 to 4202 kg	4203 to 4430 kg	4431 to 5824 kg
Stillbirth				
No	24.1	23.4	21.6	21.4
Yes	29.2	29.4	26.7	26.2
Dystocia				
No	24.1	23.4	21.6	21.4
Yes	36.0	41.1	43.5	41.1
Retained placenta				
No	23.9	23.4	21.9	21.4
Yes	26.7	33.4	25.7	22.4
Metritis				
No	24.0	23.6	21.9	21.5
Yes	24.5	31.6	25.7	20.4
Silent estrus				
No	24.2	23.8	22.0	21.5
Yes	19.5	25.0	22.2	20.9
Cystic ovaries				
No	23.8	23.7	21.9	21.3
Yes	55.8	44.2	38.5	38.2
Clinical mastitis (CM)[2]				
No	23.5	23.2	21.4	20.8
CM0	41.4	46.0	38.6	38.1
CM1	41.5	39.7	40.8	34.5
CM2	24.4	23.5	21.3	24.5
CM3	5.7	8.0	6.3	5.9
Ketosis				
No	24.0	23.8	22.1	21.4
Yes	21.2	23.4	16.6	23.9

[1]Herd production was measured as the mean cumulative 4% fat-corrected milk production at 200 DIM. Overall incidences of culling for the four strata were 28.4, 30.3, 26.6, and 25.1%, respectively.

[2]Clinical mastitis was divided into episodes diagnosed prior to or at calving (CM0), between d 1 and 50 after calving (CM1), between d 51 and 250 after calving (CM2), and between d 251 after calving and the end of lactation (CM3).

production. A 1-d increase in days open was estimated to decrease the net return per year with up to 12 Swedish crowns (14). Thus, the net return for a cow with CO could decrease with 470 Swedish crowns in a high producing herd but with 720 Swedish crowns in a low producing herd. The additional costs that result from an increased risk of culling for cows with CO are also larger for low producing herds, and these costs decrease as herd production increases.

Because herd production is not synonymous with herd management and because milk production also has the disadvantage of being a risk factor for some diseases, we decided to evaluate the effect of herd management using EDE to classify herds.

Predicted incidences of diseases for herds stratified by EDE are presented in Table 7. The trend was similar to what was found for milk production; an increase in incidences of diseases was observed as EDE increased. The only exception was for SES, and the incidence of SES decreased as the EDE of the herd increased. This result was to be expected because, in our data, SES was defined as organic or functional anestrus and its incidence should have decreased as the EDE of the herd increased.

Effects of diseases on days to first AI and days open in herds stratified by EDE are shown in Tables 8 and 9, respectively. Again, trends were similar to what was found when herds were stratified by milk production (i.e., the effect of diseases on reproductive performance decreased as the EDE of the herd increased).

TABLE 7. Predicted incidences[1] of diseases in herds stratified by estrus detection efficiency (EDE).

	EDE			
Disease	0.08 to 0.39	0.40 to 0.49	0.50 to 0.59	0.60 to 1.00
Stillbirth	1.6	2.0	1.5	1.9
Dystocia	0.9	0.2	0.8	0.5
Retained placenta	3.6	4.3	4.4	4.0
Metritis	1.5	2.0	1.2	2.1
Silent estrus	3.9	2.4	3.2	2.4
Cystic ovaries	0.0	0.7	0.5	0.5
Clinical mastitis (CM)[2]				
CM0	0.2	1.0	1.4	1.4
CM1	3.7	4.5	4.0	4.2
CM2	3.5	4.0	4.7	4.1
CM3	2.1	1.8	2.2	2.6
Ketosis	0.6	0.9	0.8	1.0

[1]Estimated using a threshold model.

[2]Clinical mastitis was divided into episodes diagnosed prior to or at calving (CM0), between d 1 and 50 after calving (CM1), between d 51 and 250 after calving (CM2), and between d 251 after calving and the end of lactation (CM3).

TABLE 8. Effects of the occurrence of disease on days to first AI (DFAI) in herds stratified by estrus detection efficiency (EDE).

	EDE			
	0.08 to 0.39	0.40 to 0.49	0.50 to 0.59	0.60 to 1.00
Overall mean DFAI	90.4	81.7	77.2	74.7
Disease				
Retained placenta	+6.7	+1.7	+3.7	+2.1
Metritis	+5.5	+5.3	+10.9	+2.8
Silent estrus	+36.2	+31.7	+28.9	+27.1
Cystic ovaries	+18.8	+32.8	+26.6	+25.5

CONCLUSIONS

Our hypothesis was that incidences and effects of diseases on the performance of cows and length of productive life would vary among herds according to herd management. Because the information available to us did not allow a complete assessment of herd management, we used milk production and EDE as proxies. The results were similar between the two strategies and, thus, supported our hypothesis. The estimated effects might have been even larger if herd management was better assessed. Herd management is probably not well defined by any one parameter, but a combination should be used. Better alternatives to assess herd management such as those suggested by Mohammed (10) and Enevoldsen et al. (4) should be investigated.

The consideration of the relationships between herd management and incidences of treated diseases when treatment records are used in herd health programs is important. A high frequency of treatments is not necessarily a disadvantage. Assessments of health status of herds based on treatment records may be valuable for internal comparisons (i.e., comparisons of the development of a herd over time). However, assessments based on treatment records should be used with caution for external comparisons (i.e., comparisons of different herds). Objective measurements

TABLE 9. Effects of the occurrence of disease on days open (DO) in herds stratified by estrus detection efficiency (EDE).

	EDE			
	0.08 to 0.39	0.40 to 0.49	0.50 to 0.59	0.60 to 1.00
Overall mean DO	117.0	105.7	98.9	92.6
Disease				
Retained placenta	+10.2	+3.2	+6.8	+2.0
Metritis	+16.0	+12.8	+21.2	+17.0
Silent estrus	+40.5	+36.6	+34.1	+31.1
Cystic ovaries	+58.3	+51.0	+47.4	+38.2

(e.g., SCC and ketone bodies in milk) are preferable for such purposes. Differences that were observed for the effects of diseases on reproductive performance and culling among herds under different herd management styles need to be considered when the costs of these diseases for various types of herds are estimated. The cost of higher incidences of treatments in well-managed herds may very well be outweighed by the decreased costs caused by the reduction in the detrimental effects of diseases on performance and risk of culling.

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REFERENCES

1 Ekman, T. 1998. A study of dairy herds with constantly low or constantly high bulk milk somatic cell count—with special emphasis on management. Acta Univ. Agric. Sueciae Veterinaria 32. Swed. Univ. Agric. Sci., Uppsala, Sweden.
2 Emanuelson, U. 1988. The national Swedish animal disease recording system. Acta Vet. Scand. Suppl. 84:262–264.
3 Emanuelson, U., P. A. Oltenacu, and Y. T. Gröhn. 1993. Nonlinear mixed model analyses of five production disorders of dairy cattle. J. Dairy Sci. 76:2765–2772.
4 Enevoldsen, C., J. Hindhede, and T. Kristensen. 1996. Dairy herd management types assessed from indicators of health, reproduction, replacement, and milk production. J. Dairy Sci. 79:1221–1236.
5 Erb, H. N. 1987. Interrelationships among production and clinical disease in dairy cattle: a review. Can. Vet. J. 28:326–329.
6 Fetrow, J., D. McClary, R. Harman, K. Butcher, L. Weaver, E. Studer, J. Ehrich, W. Etherington, W. Guterbock, D. Klingborg, J. Reneau, and N. Williamson. 1990. Calculating selected reproductive indices: recommendations of the American Association of Bovine Practitioners. J. Dairy Sci. 73:78–90.
7 Meyer, K. 1986. Restricted maximum likelihood to estimate genetic parameters—in practice. Proc. 3rd World Congr. Genet. Appl. Livest. Prod., Lincoln, NE XIII:454–459.
8 Meyer, K. 1987. Restricted maximum likelihood programs for the analysis of animal breeding data. Program notes prepared for workshop, Mar. 2–6, Orange, New South Wales, Australia. Inst. Anim. Genet., Edinburgh Univ., Edinburgh, Scotland.
9 Misztal, I., D. Gianola, and J. L. Foulley. 1989. Computing aspects of a nonlinear method of sire evaluation for categorical data. J. Dairy Sci. 72:1557–1568.
10 Mohammed, H. O. 1990. A multivariate indexing system for hygiene in relation to the risk of *Mycoplasma gallisepticum* infection in chickens. Prev. Vet. Med. 9:75–83.
11 Oltenacu, P. A., and I. Ekesbo. 1994. Epidemiological study of clinical mastitis in dairy cattle. Vet. Res. 25:208–212.
12 Oltenacu, P. A., A. Frick, and B. Lindhé. 1990. Epidemiological studies of several diseases, reproductive performance, and culling in primiparous Swedish cattle. Prev. Vet. Med. 9:59–74.
13 Solbu, H. 1983. Disease recording in Norwegian dairy cattle. I. Disease incidence and non-genetic effects on mastitis, ketosis and milk fever. Z. Tierz. Zuechtsbiol. 100:139–157.
14 Strandberg, E., and P. A. Oltenacu. 1989. Economic consequences of different calving intervals. Acta Agric. Scand. 39: 407–420.