Acta vet. scand. 1986, 27, 223—234.

From the Finnish Animal Breeding Association, Vantaa, and the Department of Animal Hygiene, College of Veterinary Medicine, Helsinki, Finland.

AN EPIDEMIOLOGICAL AND GENETIC STUDY ON REGISTERED DISEASES IN FINNISH AYRSHIRE CATTLE

IV. CLINICAL MASTITIS

By

Jouko Syväjärvi, Hannu Saloniemi and *Yrjö Gröhn*

SYVÄJÄRVI, JOUKO, HANNU SALONIEMI and YRJÖ GRÖHN: *An epidemiological and genetic study on registered diseases in Finnish Ayrshire cattle. IV. Clinical mastitis.* Acta vet. scand. 1986, 27, 223—234. — The epidemiology and genetic variability of clinical mastitis were examined. The data consisted of 70,775 Finnish Ayrshire cows. All cows were from milk recorded herds and calved during 1983. Each cow was under observation from the date of calving for 305 days. Only clinical mastitis diagnosed and treated by a veterinarian during the farm visits were taken into account. The lactation incidence rate of clinical mastitis (LIR) was 5.4 %. The cows calving in April-May had the highest LIR, but the seasonal variation was relatively small. The LIR increased with parity from the first to sixth parity. The cows treated for parturient paresis, infertility or ketosis had a higher risk of clinical mastitis than cows not treated for these diseases. The LIR was higher in herds with a high milk production level. The highest odds ratio estimated from parameters of the logit model was 14.8. The heritability estimates for clinical mastitis on the binomial scale were 3.2 % in parity 1, 1.6 % in parity 2, 0.6 % in parity 3 and 4, and 0.8 % in all parities (corresponding to 19.7 %, 8.3 %, 2.6 % and 3.8 % on the normal scale). These estimates indicate sufficient assurance for progeny testing of bulls and some possibilities of genetic selection against clinical mastitis. Genetic correlations between clinical mastitis and 305-days milk yield were 0.39 in parity 1, 0.51 in parity 2, 0.18 in parity 3—4 and 0.58 in all parities. This means that the best sires for milk yield had daughters with a higher LIR for clinical mastitis than the other sires.

disease documentation; heritability; dairy cows.

Intensive and systematic work against mastitis has started in many countries. In 1983, the International Dairy Federation sent a questionnaire concerning mastitis control to

member countries of the organisation. The 23 countries replying to this questionnaire had more than 54 million milking cows. All countries recommended mastitis control measures (*IDF* 1985). According to *Miller* (1984), the mastitis control centers on sanitation, antibiotics and, as a last resort, culling. In spite of control, intensified treatments with antibiotics, and extensive research, mastitis is the most important disease problem in the dairy industry (*Dodd* 1985). To some extent, modern dairy management with liberal use of antibiotics and an aim to save labour, conflicts with mastitis control (*Miller* 1984). More exact knowledge of the epidemiology and genetics of mastitis is needed for creating an appropriate preventive and control program.

The state level health registries in the Nordic countries are valuable data banks for investigating the epidemiological and genetic background of mastitis. In this work the data of the Finnish health registry were analysed. The first objective of this study was to analyze certain epidemiological characteristics of clinical mastitis in Finnish Ayrshire cattle. The second aim was to estimate the heritability of clinical mastitis and the genetic correlation between that and milk yield.

MATERIAL AND METHODS

The health and milk registry data have been described in detail in our previous report (*Gröhn et al.* 1986). The data comprised 70,775 Finnish Ayrshire cows, which calved in 1983. From selected communities all milk recorded herds and all Ayrshire cows were included in the data. Each cow was under observation for 305 days after calving. A cow which was diagnosed and treated by a veterinarian during the farm visits as a clinical case of mastitis was considered as an incident of mastitis. In Finland, approximately 70 % of all mastitis cases are treated by prescription (*Saloniemi* 1980). This means that the cases treated by a veterinarian during the farm visits are usually more severe and require systematic therapy. The lactation incidence rate (LIR), a term used by *Erb & Martin* (1980), was used to describe the treatment frequency of clinical mastitis. All statistical analyses were carried out by the Statistical Analysis System (*Ray* 1982). In analysing the effect of epidemiological factors on the occurrence of clinical mastitis a logit model — the FUNCAT procedure of the SAS-package — was used and maximum likelihood estimates were computed (*Cox* 1970, *Feinberg* 1980).

To have the estimates of sire and error (co)variance components, the following least square model — GLM procedure of the SAS-package — was used:

$Y_{ijklm} = \mu + a_i + c_j + h_k + s_l + e_{ijklm}$

where Y = clinical mastitis coded as 1 or 0 depending on whether the cow was treated by a veterinary surgeon or not. For estimates of covariances the summed trait of clinical mastitis and 305-day yield, and the traits separately, were also analysed as dependent variables;

μ = the theoretical mean;

a_i = the effect of the ith age class at calving (age at calving was grouped for the cows in the first parity < 751; 751—840 and > 840, int he second parity < 1,111; 1,111—1,200 and > 1,200 days, and in the other parities the parity grouping 3—4, 5—6, and > 6 was used);

c_j = the effect of the jth calving season (grouped as January-May and June-December);

h_k = the effect of the kth herd;

s_l = the effect of the lth sire;

e_{ijklm} = error term.

The sire and error terms were considered random and the other factors fixed. The effect of herd was removed by absorption. Removal of herd effects partially eliminates variation in the ability of the dairyman to recognize a disease and to call a veterinarian because the ability of the dairyman is confounded with the herd effect. The analysis was done separately for the first 3 parity groups and all parities. Only those sires with more than 25 daughters within each parity group, and the sires with more than 50 daughters in the whole material were included in genetic analyses. Error and sire covariances were estimated as half the differences between the respective variance of the summed traits minus each of respective the variances of the traits. The genetic correlations, uncorrected and corrected heritabilities and standard error of those were computed using the methods described by *Gröhn et al.* (1986).

RESULTS AND DISCUSSION

Disease occurrence

The overall lactation incidence rate (LIR) was 5.38 % (Table 1). In an earlier Finnish study (*Lindström & Syväjärvi* 1978), the

incidence of clinical mastitis observed by the owners was 28 % and in studies from the area of an ambulatory clinic (*Saloniemi* 1980, *Saloniemi & Roine* 1981), the incidences of clinical mastitis treated by a veterinarian during farm visits were 9.6 % and 10.2 %. The total incidence of clinical mastitis (treated during the farm visits and by telephone prescription) was 32 % in the same area (*Saloniemi* 1980). In other Nordic countries the incidence of clinical mastitis seems to vary from 16 % to 32 % (*Elleby et al.* 1969, *Bäckström et al.* 1975, *Bakken & Gudding* 1982, *Solbu* 1983). In a report by *Dohoo et al.* (1984) the lactation incidence rate of mastitis requiring systemic therapy was 3.5 % and in a material by *Curtis et al.* (1985) 5.4 %. One has to consider that the incidence level depends highly on the definition of the diagnoses and the registration system for the data.

Approximately one third of the cows calved during March-May and also the treatment frequency was highest during the same months (Table 1). The variation of LIR by month was much slighter than the variation of calvings and treatments. Two thirds of all treatmetns (Table 2) were given during 2 months after calving. Thus, like many other diseases the appearence of clinical mastitis was inked with the variation of calving, but a real seasonal variation was very small (*Erb & Martin* 1978,

Table 1. Counts and percentages of calvings and lactation incidence rate (LIR) of clinical mastitis by month of calving for 70,775 Finnish Ayrshire cows.

Month of calving	Calvings		Clinical mastitis		
	Number	% of total	Number	% of total	LIR (%)
January	4906	6.93	266	6.98	5.42
February	4767	6.74	279	7.32	5.85
March	7538	10.65	450	11.81	5.97
April	10056	14.21	617	16.20	6.14
May	6953	9.82	452	11.87	6.50
June	4593	6.49	237	6.22	5.16
July	4068	5.75	235	6.17	5.78
August	5308	7.50	252	6.62	4.75
September	5535	7.82	266	6.98	4.81
October	6300	8.90	281	7.38	4.46
November	5491	7.76	249	6.54	4.53
December	5260	7.43	225	5.91	4.28
Total	70755	100.00	3809	100.00	5.38

Table 2. Occurrence of first treatment of clinical mastitis by 30-day intervals up to 305 days post partum for 70,775 Finnish Ayrshire cows.

Days post partum	Percentage of total	Cumulative percentage
0—30	48.5	48.5
31—60	11.9	60.4
61—90	9.2	69.6
91—120	8.3	77.9
121—150	6.1	84.0
151—180	4.5	88.5
181—210	3.6	92.1
211—240	30	95.1
241—270	2.5	97.6
271—305	2.4	100.0

Saloniemi & Roine 1981, *Solbu* 1983, *Dohoo et al.* 1984). However, a trend towards a higher LIR during the first half of the year could be seen. The logit model selected to explain the probability of contracting clinical mastitis included 6 main effects. All the effects were highly significant. The G^2 statistic for the model was 204.87 wit h196 degree of freedom, implying a reasonable fit to the data ($P = 0.317$). Table 3 lists the estimated values for main effects, odds and lactation incidence rates. The highest differencies in estimates were among parity classes. For instance, the odds ratio (OR) between parity 5—6 and parity 1 was 2.3. The increasing risk with age is well known (e.g. *Dohoo et al.* 1984, *Curtis et al.* 1985). In our study the risk of mastitis decreased from parity 5—6 to parity > 6 (Table 3). This is in agreement with another Finnish study, where the prevalence of subclinical mastitis decreased after the 6th parity (*Saloniemi* 1980). One explanation may be that mastitis cows are culled and the cows with higher resistance to mastitis will stay. In our earlier report (*Gröhn et al.* 1986) the risk of culling increased with parity per se, but the relative risk of culling for mastitis was lower in older cows. The cows treated for parturient paresis had twice as high a risk for clinical mastitis as those which were not treated (OR = 2.0). *Curtis et al.* (1985) also observed that cows with a history of parturient paresis had an increased risk of mastitis when mastitis was diagnosed during the first month after calving (OR 5.4). They speculated that the recumbent cows are not milked because of parturient pareses and thus they are

Table 3. Estimates of the parameters included in the logit model used in the analysis of contracting clinical mastitis for 70,775 Finnish Ayrshire cows. [a, b, c]

Parameter	Estimated value	Estimated odds	Estimated LIR
Intercept	—2.316	0.099	8.98
Parity			
1	—0.497	0.060	5.66
2	—0.201	0.081	7.47
3—4	0.028	0.101	9.21
5—6	0.325	0.137	12.02
> 6	—.0345	0.070	6.53
Calving season			
January-May	0.090	0.108	9.74
July-December	—0.090	0.090	8.27
Herd milk yield, kg			
< 4870	—0.216	0.079	7.36
4870—6150	0.056	0.104	9.45
> 6150	0.160	0.116	10.38
Parturient paresis			
No	—0.354	0.069	6.48
Yes	0.354	0.141	12.33
Ketosis			
No	—0.188	0.082	7.56
Yes	0.188	0.119	10.64
Infertility [c]			
No	—0.117	0.088	8.07
Yes	0.117	0.111	9.98

[a] The G^2 statistics for the model was 204.87 with 196 degrees of freedom ($P = 0.317$).

[b] The estimated odds and lactation incidence rates (LIR) are for contracting clinical mastitis on the given factor level and adjusted for the other factors.

[c] Infertility = anostrus, suboestrus, ovulatory dysfunction, uterine infections and abortion.

more exposed to udder pathogens. *Dohoo & Martin* (1984) did not find an association between parturient paresis and mastitis, but a high association between abdominal dislocations and ketosis with mastitis. The association between ketosis and clinical mastitis in our material (OR = 1.5) might, at least partly, be explained by the reduced efficiency of bovine blood white cells to reduce the number of mastitis pathogens growing in milk in the presence of elevated levels of ketone bodies (*White & Rattray*

1968). *Dohoo & Martin* (1984) found that the cows treated for clinical mastitis had a higher risk of late reproductive infections. We found a slight association between clinical mastitis and reproductive diseases at the same lactation (OR = 1.3). The cows with parity 5—6, at spring calving season, the highest herd milk yield and with a history of parturient paresis, ketosis and infertility had the highest risk of contracting clinical mastitis (estimated odds = 0.338 equivalent to a 25 % risk). The lowest risk was for the cows with parity 1, autumn calving season, lowest herd milk yield, and without the history of the above diseases (estimated odds = 0.023 equivalent to a 2.2 % risk).

Genetic parameters

In estimating the heritability, a linear model was adapted to binomial data. This procedure has theoretical drawbacks, since the underlying assumption about normality and linearity is not fulfilled. Prediction and estimation procedures based on normality are approximative and may yield poor results (e.g. *Danell* 1985, *Mejering* 1985). Based on a threshold concept, scaling factors have been derived to relate estimates of heritability on the observed binary scale to those on the underlying scale (*Dempster & Lerner* 1950, *Gianola* 1982). Simulation studies have shown that these factors yield satisfactory approximations over a wide range of conditions and that the scaling factor of genetic correlation is unity (*Van Vleck* 1972, *Olausson & Rönningen* 1975, *Gianola* 1982). Today a new coherent body of methods is available, which, at least in theory, gives more accurate estimations of genetic parameters (*Gianola* 1980 a, b, *Harville & Mee* 1984, *Mejering* 1985). Because experience with application of the new methods is limited, the older, more approximative methods were used.

One may suggest that binomial data may be converted to continuous data by adding more categories. However, *Banks et al.* (1985) have shown that the inclusion of additional classes does not increase the accuracy of heritability estimates, if one class has a high frequency of observations as in our data. The estimates of heritability of clinical mastitis on the binomial scale and genetic correlations between that and milk yield are given in Table 4. As expected, the heritability estimates were relatively low. In the review by *Miller* (1984) the heritability estimates of clinical mastitis ranged from —0.03 to 0.5, with average 0.12.

Table 4. The estimates of heritability (h^2) and standard errors of clinical mastitis and genetic correlations (r_g) between that and milk yield.[a]

	Parity group			
	1	2	3—4	All
Number of sires	190	149	178	259
Number of daughters per sire	70	63	61	127
Clinical mastitis				
h^2 uncorrected	0.032	0.016	0.006	0.008
standard error	0.013	0.017	0.014	0.004
h^2 corrected	0.197	0.083	0.026	0.038
Genetic correlation				
Clinical mastitis ×				
milk yield	0.388	0.506	0.177	0.574
standard error	0.132	0.236	0.445	0.106

[a] The correction of the heritability estimates was made by the multiplication factor $P(1—P)/z^2$, where P the incidence of clinical mastitis and z the height of the ordinate of the normal distribution at that incidence.

Most of these estimates were greater than in our study. In a previous Finnish paper (*Lindström & Syväjärvi* 1978), the estimates varied from 0.001 to 0.046 in parity 1 and from 0.037 to 0.136 in parity 2. *Solbu* (1984) reported the estimates from 0.001 to 0.018 in Norway and *Philipsson et al.* (1980) from 0.008 to 0.038 in Sweden. As ours, these studies were based on relatively large data of clinically observed mastitis cases in field conditions. One may assume that heritability estimates in late lactation should be higher than in first lactation, because the incidence of mastitis increases with age. However, this was not the case in this study. A reason for conflicting results with an earlier finding (*Lindström & Syväjärvi* 1978) may be that only those cows which completed the 305 day milk yield were included in the data and the cows culled during this time period were excluded. In fact, neither *Shook et al.* (1982) found an increasing trend of heritability with parity by analysing somatic cell counts.

The estimates of genetic correlations between clinical mastitis and 305-day milk yield varied from 0.177 to 0.574 (Table 4). These correlations differed significantly from zero. These correlations imply that daughters of better sires in milk yield were

treated more for clinical mastitis than the daughters of other sires. The positive correlation may partly be apparent because farmers may more easily call a veterinarian for daughters of better sires in milk yield than for daughters of other sires. In addition, daughters of poor sires are more likely to be culled than daughters of better sires. *O'Bleness et al.* (1960) and *Miller et al.* (1975) also found a positive correlation between clinical mastitis and milk yield (0.44 and 0.33, respectively). *Norman & Van Vleck* (1972) and *Lindström & Syväjärvi* (1978) reported a slight negative correlation.

For breeding purpose, the greatest interest is in a heritability estimate in the first lactation. In this material and in the cited literature, the heritability estimates for clinical mastitis varied from 0.001 to 0.05 in the first lactation. According to the recommendation of the European Association of Animal Production (EAAP), the accuracy (r) of the published estimation has to be greater than 0.6. Assuming a 2 % heritability, the recommended accuracy is reached when bulls are progeny tested using 113 daughters. The current progeny testing system with health registry data already provides a reliable base for such evaluation. In Finland, a multiple trait selection index is used in sire selection (*Mäntysaari et al.* 1984). The inclusion of progeny testing for mastitis to the index should be the best way to consider clinical mastitis in sire selection.

ACKNOWLEDGEMENTS

The authors thank Pol. Lic. Juni Palmgren for advice on logistic regression, and the Agricultural Data Processing Centre for supplying computer facilities for statistical analysis.

REFERENCES

Bakken, G. & R. Gudding: The interdependence between clinical and subclinical mastitis. Acta agric. scand. 1982, *32*, 17—22.

Banks, D. B., I. L. Mao & J. P. Walter: Robustnes of restricted maximum likelihood estimator derived under normality as applied to data with skewed distributions. J. Dairy Sci. 1985, *68*, 1785—1792.

Bäckström, L., J. Mattsson, O. Wallin & B. Vilson: Praktikjournalförsöket i Skaraborgs län 1971—1973. (Practice journal experiment in the county of Skaraborg 1971—1973). Svensk. Vet.-Tidn. 1975, *27*, 238—250, 292—300.

Curtis, C. R., H. N. Erb, C. J. Sniffen, R. D. Smith & D. S. Kronefeld: Path analysis of dry period nutrition, postpartum metabolic and reproductive disorders, and mastitis in Holstein cows. J. Dairy Sci. 1985, *68*, 2347—2360.

Cox, D. R.: Analysis of Binary Data. Chapman and Hall, London 1970.

Danell, Ö.: Theoretical aspects in the estimation of breeding values for all-or-none traits. 36th Ann. Meet. Europ. Assoc. Animal Product., Kallithea, Greece 1985.

Dempster, E. R. & I. M. Lerner: Heritability of threshold characters. Genetics 1950, *35*, 212—236.

Dodd, F. H.: Progress in mastitis control. Kieler Milchw. Forschnungsb. 1985, *37*, 216—223.

Dohoo, I. R. & S. W. Martin: Disease, production and culling in Holstein-Friesian cows. III. Disease and production as determinants of disease. Prev. Vet. Med. 1984, *2*, 671—690.

Dohoo, I. R., S. W. Martin, I. McMillan & B. W. Kennedy: Disease, production and culling in Holstein-Friesian cows. II. Age, season and sire effects. Prev. Vet. Med. 1984, *2*, 655—670.

Elleby, F., P. Jochumsen & H. N. Veirup: Forekomst af kvægsygdomme i 6 danske kontrolforeningar i 1967/1968. (Disease incidence within six Danish milk recording societies). Kgl. Vet.- og Landbohøjsk. Inst. Sterilitetsforskn. Aarsberetn. 1969, p. 77—90.

Erb, H. N. & S. W. Martin: Age, breed and seasonal patterns in the occurrence of ten dairy cow diseases: a case control study. Canad. J. comp. Med. 1978, *42*, 1—9.

Erb, H. N. & S. W. Martin: Relationship between production and reproductive diseases in Holstein cows. Data. J. Dairy Sci. 1980, *63*, 1911—1917.

Feinberg, S. E.: The Analysis of Cross-classified Categorical Data. 2nd ed. MIT Press, Cambridge, Massachusets 1980.

Gianola, D.: A method of sire evaluation for dichotomies. J. Anim. Sci. 1980a, *51*, 1266—1271.

Gianola, D.: Genetic evaluation of animals for traits with categorical responses. J. Anim. Sci. 1980b, *51*, 1272—1276.

Gianola, D.: Theory and analysis of threshold characters. J. Anim. Sci. 1982, *54*, 1079—1095.

Gröhn, Y., H. Saloniemi & J. Syväjärvi: An epidemiological and genetic study on registered diseases in Finnish Ayrshire cattle. I. The data, disease occurrence and culling. Acta vet. scand. 1986, *27*, 182—195.

Harville, D. A. & R. W. Mee: A mixed model procedure for analysing ordered categorical data. Biometrics 1984, *40*, 393—408.

International Dairy Federation (IDF): International progress in mastitis control. Bulletin No. 187, 1985.

Lindström, U. B. & J. Syväjärvi: Use of field recording in breeding for mastitis resistance in dairy cattle. Livest. Prod. Sci. 1978, *5*, 29—44.

Meijering, A.: Practical aspects in the estimation of parameters and breeding values for all-or-none traits. 36th Ann. Meet. Europ. Assoc. Animal Product., Kallithea, Greece 1985.

Miller, R. H., J. R. Owen & E. D. More: Heritability of measures of incidence of clinical mastitis in Jersey cattle. J. Dairy Sci. 1975, *58*, 751.

Miller, R. H.: Traits for sire selection related to udder health and management. J. Dairy Sci. 1984, *67*, 459—471.

Mäntysaari, E., J. Syväjärvi & T. Hellman: A multiple trait selection index for progeny tested dairy bulls in Finland. Progeny testing methods in dairy cattle. IDF/EAAP symposium. Prague, September 1984. Bulletin Document 183, 204—212.

Norman, H. D. & L. D. Van Vleck: Type appraisal: II. Variation in type traits due to sires, herds, and years. J. Dairy Sci. 1972, *55*, 1717—1725.

O'Blennes, G. V., L. D. Van Vleck & C. R. Henderson: Heritability of some appraisal traits and their genetic and phenotypic correlation with production. J. Dairy Sci. 1960, *43*, 1490—1498.

Olausson, A. & K. Rönningen: Estimation of genetic parameters for threshold characters. Acta agric. scand. 1975, *25*, 201—208.

Philipsson, J., B. Thafvelin & J. Hedebro-Velander: Genetic studies on disease recordings in first lactation cows of Swedish dairy breeds. Acta agric. scand. 1980, *30*, 325—335.

Ray, A. A.: SAS User's Guide: Statistics. SAS Institute Inc., Carry, NC 1982.

Saloniemi, H.: Udder diseases in dairy cows — field observations on incidence, somatic cell and environmental factors and control. J. scient. agric. Soc. Finl. 1980, *52*, 85—184.

Saloniemi, H. & K. Roine: Field observations on the incidence of bovine clinical mastitis and teat diseases. Nord. Vet.-Med. 1981, *33*, 297—305.

Shook, G. E., F. Ruvna & A. K. A. Ali: Genetic parameters for lactation average of somatic cell concentration in milk. 2nd World Congress on Genetics Applied to Livestock Production. Madrid 1982, *8*, 142—147.

Solbu, H.: Disease recording in Norwegian dairy cattle. I. Disease incidence and non-genetic effects on mastitis, ketosis, and milk fever. Z. Tierz. Zuchtungsbiol. 1983, *100*, 139—157.

Solbu, H.: Disease recording in Norwegian dairy cattle. II. Heritability estimates and progeny testing for mastitis, ketosis and "all diseases". Z. Tierz. Zuchtungsbiol. 1984, *101*, 51—58.

Van Vleck, L. D.: Estimation of heritability of threshold characters. J. Dairy Sci. 1972, *55*, 218—225.

White, F. & E. A. S. Rattray: The in vitro effect of ketone bodies on the growth curves of mastitis organisms in milk. J. comp. Path. 1968, *78*, 179—188.

SAMMANFATTNING

En epidemiologisk och genetisk undersökning av sjukdomsdata från finsk Ayrshire boskap. IV. Klinisk mastit.

Epidemiologin och den genetiska variationen av klinisk mastit undersöktes. Det använda datasetet omfattade 70775 finska Ayrshire kor. Alla kor var från kontrollföreningsanslutna besättningar och hade kalvat under år 1983. Varje ko observerades från kalvningen till den

305 dagen efter denna. Bara kliniska mastiter, diagnostiserade och behandlade av en veterinär i samband med sjukbesök, inkluderades i materialet. Lactation incidence rate (LIR) för klinisk mastit var 5.4 %. Kor, som kalvade under april-maj hade den högsta LIR, men säsongvariationen var relativt liten. LIR ökade från den första laktationen till den sjätte. Kor som behandlades för kalvningsförlamning, infertilitet eller ketos hade en högre risk för klinisk mastit än obehandlade kor. LIR var högre i högproducerande besättningar. Den högsta odds ratio enligt logit model var 14,8. Ärftligheten för klinisk mastit i binomiala skalan var 3,2% (1. aktationen), 1,6 % (2.), 0,6 % (3.—4.) och 0,8 % för hela materialet (motsvarande 19,7 %, 8,3 %, 2,6 % och 3,8 % i den normala skalan). Resultaten är tillräckligt noggranna för avkommebedömning av tjurar och ger några möjligheter för genetisk selektion mot klinisk mastit. Genetisk korrelation mellan klinisk mastit och 305-dagars mjölkproduktion var 0,39 (1. laktationen), 0,51 (2.) och 0.18 (3.—4.). Det betyder, att tjurar som har mest producerande döttrar också har döttror med högsta LIR av klinisk mastit.

(Recived February 6, 1986).

Reprints may be requested from: Jouko Syväjärvi, F.A.B.A., P. O. Box 40, 01301 Vantaa, Finland.