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

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

III. METABOLIC DISEASES

By
Yrjö Gröhn, Hannu Saloniemi and *Jouko Syväjärvi*

GRÖHN, YRJÖ, HANNU SALONIEMI and JOUKO SYVÄJÄRVI: *An epidemiological and genetic study on registered diseases in Finnish Ayrshire cattle. III. Metabolic diseases.* Acta vet. scand. 1986, 27, 209—222. — The epidemiology of clinical ketosis, hypocalcaemia and hypomagnesaemia was examined. In addition, the genetic variability of ketosis and parturient paresis was investigated. The data set consisted of the lactation records of 70,775 Finnish Ayrshire dairy cows. Each cow was under observation for 2 days before and for 305 days after calving. Lactation incidence rates (%) were: ketosis 6.0, parturient paresis 3.8, non-parturient paresis 0.6, hypomagnesaemic tetany, outdoor 0.6, and indoor 0.2. These diseases formed 22 % of all first treatments by veterinarians during farm visits. 92 % of the cases of ketosis occurred with 8 weeks of parturition, with the highest occurrence 3—5 weeks after calving. Four % of cases of parturient paresis occurred before, and 45 % within 24 h after calving. When cases were categorized by month of calving the risk of ketosis was higher during indoor feeding (October-April) than during outdoor feeding (May-September). The risk of parturient paresis did not significantly vary with month of calving. The occurrence of ketosis increased with parity up to the 4th and decreased thereafter. The occurrence of parturient paresis increased with parity. Both the increase in herd milk yield and the increase in individual milk yields were positively associated with the occurrence of ketosis and parturient paresis. The cows with a history of the reproductive tract infection had a higher risk of contracting ketosis. Heritability estimates for ketosis in various parity groups were from 1.6 % to 4.1 % on the binomial scale (corresponding to 7.3 %—14.4 % on the normal scale), and for parturient paresis from 3.5 to 10.5% (corresponding from 18.3 % to 27.4 %). The genetic correlation between ketosis and parturient paresis, and these and current milk production for all material were insignificant.

disease documentation; heritability; ketosis; parturient paresis; non-parturient paresis; hypomagnesaemic tetany; dairy cows.

Past successes in preventive veterinary medicine have made possible intensive animal production systems (*Schwabe* 1982). In the current dairy industry there are three main disease complexes that are less dramatic but require understanding of multifactorial diseases: reproductive disorders, metabolic diseases and mastitis. All of these diseases have been and still are the subject of intensive research. The clinical picture, aetiology and pathogenesis of the most common metabolic diseases have been reviewed thoroughly (see e.g. *Littledike et al.* 1981). In recent years the research interest in epidemiology of metabolic diseases has increased (*Øverby et al.* 1974, *Saloniemi & Roine* 1981, *Solbu* 1983, *Dohoo et al.* 1983, *Curtis et al.* 1984). The investigators have also been interested in the heritability of susceptibility to metabolic diseases (*O'Bleness et al.* 1960, *Van Vleck* 1964, *Norman & Van Vleck* 1972, *Dyrenthal et al.* 1972). To date the main reason for limited research regarding the genetic determination of metabolic diseases has been the lack of sufficient health data on the scale necessary for a study of this kind. With the institution of more sophisticated disease registry systems, the number of genetic studies of these diseases has increased (*Solbu* 1984, *Gröhn et al.* 1984, *Dohoo & Martin* 1984).

In our previous papers, the data set, the occurrence of the most common dairy cow diseases, the effects of possible risk factors on culling (*Gröhn et al.* 1986), and the epidemiology and genetic variability of reproductive disorders (*Saloniemi et al.* 1986) were investigated. The purpose of the current work was to examine the epidemiology and genetic variability of ketosis and parturient paresis in Finnish Ayrshire cattle. In addition, the objective was to investigate the occurrence of non-parturient paresis and outdoor and indoor hypomagnesaemic tetany.

MATERIALS AND METHODS

Data

The data used in this study have been presented previously (*Gröhn et al.* 1986). The lactation records of 70,775 Finnish Ayrshire dairy cows were complete. The cows that calved during the period between January 1, 1983 and December 31, 1983 were under observation for 2 days before and for 305 days after calving. The cows belonged to the milk registry and health and pedigree data were available. The data included only those cases

treated by veterinarians during farm visits. The metabolic diseases studied were: ketosis, parturient and non-parturient paresis, and hypomagnesemic tetany outdoor and indoor. The diagnoses were made according to ordinary clinical methods in field conditions. Only the first diagnoses were considered, and repeated treatments or treatments by telephone prescription were excluded.

Statistical analysis

All statistical analyses were carried out using the Statistical Analyses System (*Ray* 1982). The occurrence of a metabolic disease was expressed using the term lactation incidence rate (*Erb & Martin* 1980). The possible effects of certain factors on contracting ketosis or parturient paresis were determined using a logistic regression model (*Cox* 1970, *Feinberg* 1980).

For genetic analysis, sire and error components of variance and covariances were recomputed by using the following least square model: $Y_{ijklm} = \mu + a_i + c_j + m_k + s_l + e_{ijklm}$,
where Y = the disease in question recorded as 1 or 0 depending whether the cow has been treated or not by a veterinarian. For estimates of covariances the summed trait of diseases and 305-day milk yield were also analysed as dependent variables. The milk yield was expressed as the deviation from the mean of the herd milk production level;
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—841; > 841 days, in the 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-April, May-August and September-December);
m_k = the effect of kth herd milk production class (the mean of 305-day milk production of the cows in the herd grouped as < 4,870; 4,870—6,150 and > 6,150 kg);
s_l = the effect of the cow's sire;
e_{ijklm} = a residual component.

All factors were considered fixed except sire and error term, which were considered random. The analyses were also done by absorbing the herd effects. Because the absorption had only a

slight effect on the estimates of heritabilities, the results are given without the absorption in these analyses. The analysis was carried out separately for each parity group and for all parities together. Only those sires with more than 25 daughters within each parity group, and the sires with more than 50 daughters for the total material were included in genetic analyses. Error and sire covariances were estimated as half the difference between the respective variances of the summed traits minus each of respective variances of the traits. The genetic correlations, uncorrected and corrected heritabilities and standard errors were computed using the methods described previously (*Gröhn et al.* 1986).

RESULTS

Lactation incidence rates for some metabolic diseases in relation to parity are given in Table 1. All of these diseases increased with parity, except ketosis which decreased after the 4th parity and hypomagnesaemic tetany after the 6th parity. These diseases made up 21.6 % of all first treatments by veterinarians. Ketosis and parturient paresis were the most common metabolic diagnoses.

Table 1. Lactation incidence rate (%) of some metabolic diseases in relation to parity in 70,775 Finnish Ayrshire cows.

Disease	Parity					All	% of all first treatment
	1	2	3—4	5—6	> 6		
Ketosis	4.37	5.14	7.87	7.38	5.04	6.0	12.0
Parturient paresis	0.05	0.40	3.94	10.79	12.23	3.8	7.6
Non-parturient paresis	0.10	0.30	0.64	1.05	1.54	0.6	1.2
Hypomagnesaemic tetany, outdoor	0.08	0.15	0.41	0.56	0.45	0.3	0.6
Hypomagnesaemic tetany, indoor	0.07	0.09	0.19	0.22	0.11	0.1	0.2

In general, calvings were equally distributed throughout the year, except for an increase in March, April and May and a slight decrease in June and July (Table 2). The monthly distribution of treated cases of ketosis and parturient paresis partly reflected calving distribution (Table 2). When cases were categorized by

T a b l e 2. Monthly distribution of calvings, treated cases of ketosis and parturient paresis, and the risk of ketosis and parturient paresis by month of calving for 70,775 Finnish Ayrshire cows.

	Month												Year
	J	F	M	A	M	J	J	A	S	O	N	D	
Calvings per month (% of yearly total)	6.9	6.7	10.6	14.2	9.9	6.5	5.7	7.4	7.8	8.9	8.7	7.5	100.0
Ketosis cases per month (% of yearly total)	9.1	10.4	17.1	18.5	4.0	1.3	1.1	2.4	5.2	8.7	9.9	12.3	100.0
Risk of ketosis by month of calving (%)	7.9	9.4	9.7	7.9	2.4	1.2	1.1	2.0	4.0	5.9	7.7	9.8	6.0
Parturient paresis cases per month (% of yearly total)	6.9	6.4	12.3	17.2	9.4	7.6	4.5	4.9	7.1	8.1	8.1	7.5	100.0
Risk of parturient paresis by month of calving (%)	3.8	3.7	4.4	4.6	3.6	4.4	3.0	2.5	3.4	3.5	3.9	3.8	3.8

month of calving the risk of ketosis was higher during indoor feeding (October-April) than during outdoor feeding (May-September) (Table 2). In the logit regression model calving season was a significant factor in explaining the probability of contracting ketosis (Table 4). The risk of parturient paresis did not significantly vary by month of calving (Table 2). 4 % of parturient paresis cases occurred before, and 45 % within 24 h after calving (Table 3). 92 % of ketosis cases occurred within 8 weeks after parturition, with the highest occurrence 3—5 weeks after calving (Table 3).

T a b l e 3. Weeks post partum of occurrence of clinical ketosis and days pre and post partum of occurrence of parturient paresis for 70,775 Finnish Ayrshire cows.

Cases of ketosis	Weeks post partum									
	1	2	3	4	5	6	7	8	9	10—12
Percentage of total	4.1	10.0	16.0	21.6	17.6	11.3	6.7	4.2	2.3	3.3
Cumulative percentage	4.1	14.1	30.1	51.7	69.3	80.6	87.3	91.5	93.8	97.1
Cases of parturient paresis	**Days pre and post partum**									
	—2	—1	0	1	2	3	4	5	6	7
Percentage of total	0.8	3.5	44.9	33.3	9.4	2.5	1.5	1,1	2.0	1.0
Cumulative percentage	0.8	4.3	49.2	82.5	91.9	94.4	95.9	97.0	99.0	100.0

The logistic regression model selected to explain the probability of contracting ketosis included 5 main effects: parity, calving season, herd class average for milk, cow's milk yield as the deviation from the mean of herd's milk production level and the history of the reproductive tract infection. The G^2 statistic for this model was 347.6 with 304 degrees of freedom ($P = 0.0432$); the P-value indicates that there are other factors than those in the model, which may improve the fit. However, for large sets of data the G^2-statistic tends to reject virtually any model, thus being of limited value for practical purposes. Here the ratio of G^2 to the degree of freedom is near to one (expectation under a good model) and the easy interpretation of the model motivates its use. The model selected to explain contracting parturient paresis included the same main effects, except calving season and the history of reproductive tract infection. The G^2 statistic for this model was insignificant ($P = 0.2801$), implying a reasonable fit to the data.

All main effects in both models were significant and further inclusion of main effects did not substantially improve the fit.

Table 4 lists the estimated values for the main effect parameters in the logistic regression model for the probability of contracting ketosis. For instance, the cows with parity 3—4, spring calving season, highest herd milk yield and highest cow's milk yield and with the history of infection of the reproductive tract post partum had the highest expected odds of contracting ketosis of $e^{(-2.567 + 0.243 + 0.638 + 0.192 + 0.158 + 0.452)} = e^{-0.884} = 0.413$ (equivalent to a 29.2 % chance of contracting ketosis). The lowest odds were for the cows with parity > 6, summer calving, lowest herd milk yield, lowest cow's milk yield and without the history of reproductive tract infection of $e^{(-2.567 - 0.265 - 0.990 - 0.186 - 0.163 - 0.452)} = e^{-4.623} = 0.0098$ (equivalent to a 1.0 % chance of contracting ketosis). The odds ratio comparing the odds is $0.413 / 0.0098 = 42.2$. This means that cows with a combination of the highest risk variables are 42.2 times as likely to contract ketosis as those with the combination of lowest risk variables. Similar comparisons can be made by forming such ratios between any two combination groups and using the denominator as the comparison level. Table 4 lists the estimated odds (probabilities in parenthesis) of contracting ketosis, as well. The estimated odds and probabilities are conditional on the given factor level and adjusted for the other factors. The odds ratios computed from Table 4 are stable over all levels of other factors.

Table 4. Estimates of the parameters included in the logit model used in the analysis of contracting ketosis for 51,449 Finnish Ayrshire cows (cows in first lactation excluded).

Parameter	Estimated value	Estimated odds [a]	
Intercept	—2.567	0.077	(7.2)
Parity			
—			
2	—0.102	0.069	(6.5)
3—4	0.243	0.098	(8.9)
5—6	0.124	0.087	(8.0)
> 6	—0.265	0.059	(5.6)
Calving season			
January-April	0.638	0.145	(12.7)
May-August	—0.990	0.029	(2.8)
September-December	0.352	0.109	(9.8)
Herd milk yield in previous lactation (kg)			
< 4870	—0.186	0.064	(6.0)
< 6150	—0.006	0.076	(7.1)
≥ 6150	0.192	0.093	(8.5)
Cow's milk yield in previous lactation (the deviation from the mean, kg)			
<—1000	—0.163	0.065	(6.1)
<— 500	—0.158	0.066	(6.2)
< 500	0.032	0.079	(7.3)
< 1000	0.131	0.088	(8.1)
≥ 1000	0.158	0.090	(8.3)
Infection of the reproductive tract post partum < 42 days			
yes	0.452	0.121	(10.8)
no	—0.452	0.049	(4.7)

[a] The estimated odds (probabilities in parenthesis) are for contracting ketosis conditional on the given factor level and adjusted for the other factors.

In Table 5 the estimated values, odds and probabilities of contracting parturient paresis are given. The cows with parity > 6, highest herd milk yield and highest cow milk yield had the highest odds of contracting parturient paresis. The lowest odds are for the cows with parity 2, lowest herd milk yield and lowest cow milk yield.

The estimates of heritabilities for ketosis and parturient paresis and genetic correlations between those and milk produc-

Table 5. Estimates of the parameters included in the logit model used in the analysis of contracting parturient paresis for 51,449 Finnish Ayrshire cows (cows in first lactation excluded).

Parameter	Estimated value	Estimated odds [a]	
Intercept	—3.251	0.039	(3.8)
Parity			
—			
2	—2.235	0.004	(0.4)
3—4	—0.011	0.038	(3.7)
5—6	1.041	0.110	(9.9)
> 6	1.205	0.129	(11.4)
Herd milk yield in previous lactation (kg)			
< 4870	—0.417	0.026	(2.5)
< 6150	0.058	0.041	(3.9)
≥6150	0.359	0.056	(5.3)
Cow's milk yield in previous lactation (the deviation from the mean, kg)			
<—1000	—0.146	0.034	(3.3)
<— 500	—0.085	0.036	(3.5)
< 500	—0.002	0.039	(3.8)
< 1000	0.067	0.041	(3.9)
> 1000	0.166	0.046	(4.4)

[a] The estimated odds (probabilities in parenthesis) are for contracting parturient paresis conditional on the given factor level and adjusted for the other factors.

tion in the current lactation are given in Table 6. Heritability estimates for ketosis and parturient paresis differed clearly from zero. The estimate for parturient paresis increased with parity. Genetic correlations between ketosis and parturient paresis, and these and current milk production were inconsistent in various parity groups and the correlations for the total material were insignificant.

DISCUSSION

A common feature for ketosis, hypocalcaemia and hypomagnesaemia is the imbalance between the supply of nutrients and the requirements of the animal (*Littledike et al.* 1981). Intensive selection for milk yield can be assumed to narrow the nutritional and managerial spectrum over which the animals can maintain metabolite homeostasis. Therefore, it is important to register all

Table 6. Estimates of heritabilities (h^2) for ketosis and parturient paresis and genetic correlations (rg) between those and milk production in current lactation for Finnish Ayrshire cattle.[a, b, c]

	Parity					
	1	2	3—4	5—6	> 6	All
Number of sires	190	150	178	45	22	259
Mean number of daughters per sire	70	62	61	60	47	127
Ketosis						
h^2 uncorrected	0.016	0.016	0.026	0.041	0.020	0.021
standard error	0.008	0.009	0.010	0.023	0.034	0.005
h^2 corrected	0.078	0.073	0.088	0.144	0.089	0.084
Parturient paresis						
h^2 uncorrected	N.E.	I.E.	0.035	0.087	0.105	0.038
standard error			0.018	0.033	0.058	0.006
h^2 corrected			0.183	0.244	0.274	0.204
Genetic correlations						
Ketosis × parturient paresis	N.E.	I.E.	0.062	—0.216	—0.783	—0.068
standard error			0.240	0.314	0.253	0.132
Ketosis × milk yield	0.297	0.185	0.196	—0.292	I.E.	0.100
standard error	0.147	0.206	0.160	0.263		0.103
Parturient paresis × milk yield	N.E.	I.E.	—0.052	—0.432	I.E.	—0.088
standard error			0.148	0.189		0.088

[a] The correction was made by the multiplication factor $P(1—P)/z^2$, where P was the incidence of the disease and z the height of the ordinate of the normaldistribution at that incidence.

[b] N.E. = not estimated due to the extremely low lactation incidence rate.

[c] I.E = illogical estimate.

changes in the occurrence of these diseases, to investigate effects of possible risk factors and the genetic variation. From a breeding point of view the most important question is to know the genetic correlation between disease susceptibility and production ability.

In this study the incidence rates for ketosis (6.0 %) and non-parturient paresis (0.6 %) are approximately the same as, that for hypomagnesaemic tetany (outdoor 0.6 % and indoor 0.2 %) is higher than, and that for parturient paresis (3.8 %) is lower

than in a previous Finnish paper (*Saloniemi & Roine* 1981). The rates for ketosis and parturient paresis are higher in Norway (*Solbu* 1983) and lower or approximately the same in Sweden (*Bäckström et al.* 1975) and Denmark (*Elleby et al.* 1969).

For ketosis the period of highest risk was 3—5 weeks after calving as reported earlier (*Øverby et al.* 1974, *Gröhn et al.* 1983). *Dohoo et al.* (1983) found an earlier occurrence than we did. The occurrence of parturient paresis in relation to calving agrees with a finding by *Dyrendahl et al.* (1972).

A real seasonal pattern in the risk of ketosis by month of calving (Table 2) was verified by a logistic regression model (Table 4). The risk of ketosis was higher during indoor feeding (October-April) than during outdoor feeding (May-September). Variation in the risk of parturient paresis by month of calving was not apperent as reported by *Ekesbo* (1966) and *Saloniemi & Roine* (1981). The effects of parity were investigated by determining both lactation incidence rates in the various parity groups, (Table 1) and by estimating odds in the logit model (Tables 4 and 5). The incidence rate of clinical ketosis has been reported to peak in the age ranges 4—7 years (*Erb & Martin* 1978), 5—8 years (*Øverby et al.* 1974) and at 9 years (*Dohoo & Martin* 1984). The peak in this study was at parity 3—4 (approximately 5—6 years of age). The increase of the risk of parturient paresis with parity supports findings by *Dyrendahl et al.* (1972) and *Dohoo & Martin* (1984).

Both the increase in herd milk yield and the increase in individual cow's milk yield increased the risk of ketosis and parturient paresis (Tables 4 and 5). Although there are other reports (*Pehrsson* 1966, *Øverby et al.* 1974, *Solbu* 1983) supporting this finding, it is difficult to interpret the significance of the connection between milk yield and ketosis or parturient paresis. This difficulty has been discussed in a previous paper (*Gröhn et al.* 1984). Assuming that the association between milk yield and ketosis and parturient paresis is real and does not just reflect better care for the higher yielders, other explanations may be discussed. A genetic analysis may also clarify this question.

The sire components and the heritability estimates for susceptibility to ketosis and parturient paresis were relatively high (Table 6). However, neither a genetic correlation between ketosis and parturient paresis, nor between these and current milk production differed clearly from zero. This may imply that the

observed association between milk yield and ketosis, or between milk yield and parturient paresis are mainly phenotypic. About 25 % of the variation in milk production results from genetic differences. High yielders are phenotypically more susceptible to ketosis and parturient paresis because their nutritional requirements may not be satisfied. In future research accurate feeding and management data should be included in the studies of metabolic diseases.

From a statistical point of view, as discussed earlier (*Gröhn et al.* 1984), one difficulty is to handle threshold characteristics. The logit regression methodology is an appropriate technique for analysing a binary response factor. However, our approach in genetic analyses was to use traditional animal genetics methodology (linear model) on the binomial scale for probability of occurrence of a disease, and then to convert binomial heritability estimates to estimates on the normal scale. The treatment occurrences are typically all-or-none traits and the underlying assumption about normality is not fulfilled in the data although the assumption about normality of genotypes may still be realistic. Thus, prediction and estimation procedures based on normality are approximative and may yield poor results (*Portnoy* 1982, *Mejering* 1985).

The findings in the current study indicate that there is a genetic component in the aetiology of ketosis and parturient paresis. It seems reasonable to consider at least ketosis in the progeny testing of bulls. The low incidence of parturient paresis in younger animals hinders the inclusion of this trait in the current breeding program. Providing that the linear model in the genetic analysis gave reliable results, the association between these diseases and milk yield is mainly phenotypic. Thus, selection for milk yield does not genetically increase the incidence of these diseases. The management and nutritional requirements of higher yielding animals have to be considered appropriately. The epidemiological findings in this study are only a base enabling understanding of metabolic diseases and further research in feeding and management is clearly necessary.

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SAMMANFATTNING

En epidemiologisk och genetisk undersökning av sjukdomsdata från finsk Ayrshire boskap. III. Metaboliska sjukdomar.

Epidemiologin av klinisk ketos, hypokalcemi och hypomagnesemi undersöktes. Den genetiska variationen av ketos och kalvningsförlamning undersöktes också. Dataset innehöll laktationsresultaten från 70775 finska Ayrshire kor. Varje ko observerades från 2 dagar före till 305 dagar efter kalvningen. Lactation incidence rate var: ketos 6,0, paresis puerperalis 3,8, icke-puerperal pares 0,6, hypomagnesemi under betesperiod 0,6 och under stallperiod 0,2. Dessa sjukdomar omfattade 22 % av alla första veterinärbehandlingar på gårdarna. 92 % av ketosfallen inföll inom 8 veckor efter kalvningen. Den högsta frekvensen konstaterades 3—5 veckor efter kalvningen. 4 % av kalvningsförlamningarna inföll förkalvningen och 45 % inom 24 timmar efter kalvningen. Ketosrisken var högre under stallperioden (oktober-april) än under betesperioden (maj-september), när materialet klassificerades efter kalvningsmånaden. Paresrisken varierade inte signifikant efter kalvningsmånaden. Ketosfrekvensen ökade med laktationsperioder ända till fjärde kalvningen och minskade därefter. Paresrisken ökade med åldern. Hög mjölkproduktion hos både besättning och enskilda kor hade ett positivt samband med förekomsten av ketos och pares. Kor med tidigare infektion i reproduktionsorgan hade en

högre risk att insjukna i ketos. Ärftligheten för ketos i olika laktationsperioder var från 1,6 % till 4,1 % i den binomala skalan (motsvarar från 18.3 % till 27,4 %). Genetisk korrelation mellan ketos och pares och mellan dessa och mjölkproduktion varierade oregelbundet i olika laktationsperioder och en korrelation med hela materialet var osignifikant.

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Reprints may be requested from: Yrjö Gröhn, the College of Veterinary Medicine, P. O. Box 6, 00551 Helsinki, Finland.