

The influence of different factors on resistance of dairy cows to mastitis

**A.E. Bolgov, E.P. Karmanova, L.N. Muravja, V.E. Makarova,
S.G. Shterkel and N.V. Grishina**

*Department of Animal Breeding, Petrozavodsk State University
Lenin 33, 185640 Petrozavodsk, Russia*

(Received 30 April 2001; revised version 19 February 2002; accepted 9 May 2002)

ABSTRACT

The frequency of mastitis in more than 28 thousand cows was investigated. We report on how age and level of productivity, season of the year, "milkmaid" factor, udder properties, and hereditary factors influence mastitis frequency. The influence of the mothers and fathers on resistance or predisposition of the daughters to mastitis is shown. Among daughters from sick mothers the disease is two and more times higher than in the offspring of healthy cows; the frequency of mastitis ranges among families from 0 up to 57%. High variability of the sire's breeding value on mastitis stability (from -4.2 up to +24.0%) was found established. The factors of mastitis inheritance are low, on average 0.1.

It is possible to estimate the bull's effect on resistance to mastitis in first-calving cows if a significantly large group is available. Among the bulls assessed in mastitis, sires were found to be plus proven (29.6%), neutral (44.4%) and poor proven (26.0%) in relation to the resistance parameter. The ranks of the bulls on mastitis frequency in the daughters and on sire's breeding value are characterized by high reproducibility (0.7 and more). The hypothesis about the polygenic type of inheritance of mastitis was confirmed on the basis of histogram analysis of the distribution of the half-sibs and female families on mastitis frequency.

The annual realized genetic trend in mastitis varied from 2 up to 9% depending on the cow's age. It is possible and necessary to select dairy cattle to increase resistance to mastitis simultaneously with selection for milk productivity.

KEY WORDS: mastitis, heredity, genetic resistance, selection, cattle

INTRODUCTION

Mastitis (udder inflammation) is one of the most widespread and expensive illnesses of cows. The frequency of the disease varies within the range of 12-40%, and in separate herds - up to 50-80% (Miller and Dorn, 1990; Boettcher and Van Doornmaal, 1999; Juga et al., 1999; Kopytin and Novicov, 1999; Pryce et al., 1999; Van der Beek, 1999; Kalmykova, 2000). The decrease in milk productivity from a sick cow ranges from 3.2 up to 25% of annual yield (Bolgov and Karmanova, 1989). The losses from one case of mastitis are equivalent to the cost of 240 kg of milk in Denmark (Eriksson, 1991) and 400-470 kg in the USA (Kaneene and Hurd, 1990). The occurrence and distribution of this disease depends on set of the factors, first of all on paratypic factors (Arney et al., 1996; Fourichon et al., 1996; Kaustell et al., 1996), immune system status (Schukken et al., 1994), age of animals (Sandholm, 1995; Pösö and Mäntysaari, 1996), and level of milk production in herds (Heuven et al., 1988; Banos and Shook, 1990; Boettcher et al., 1992; Collean and le Bihan-Duval, 1995).

In recent years the role of genetic factors in distribution of mastitis is being actively investigated (Andersen, 1978; Karmanova and Bolgov, 1979; Egan, 1984; Garkavi and Briede, 1984; Emanuelsson et al., 1988; Shook and Schutz, 1994; Zhang et al., 1994; Boichard and Rupp, 2000). Nonetheless, practical achievements in counteracting mastitis and increasing resistance to this disease remain insignificant. The purpose of our work was to study the influence of various factors on the resistance of dairy cows to mastitis.

MATERIAL AND METHODS

Animals

On the basis of regular and long-term (1976-1999) monitoring, information about mastitis in more than 28 thousand cows in seven large herds in the Republic of Karelia (Russia) was collected and processed. Herds (800-1000 cows) of Ayrshire, Kholmogor and Brown Latvian breeds were observed. In all of the farms, the cows were kept in typical cattle yards of 200-400 heads, in summer for 110-120 days the animals were on pasture.

The feeding level of the cows in the majority of the farms was similar and ranged from 3700 to 4800 feeding units per cow per year with a milk yield of 3300-4500 kg. In the Sortavalsky breeding farm the cost of forage and level of efficiency were higher: 5000-5300 feeding units and 4600-5100 kg of milk, respectively. Milking was done three times a day in the first half of lactation and twice in the second half. One operator of a milking machine handled 30-35 cows.

Diagnosis of mastitis

Dimastine test. The dimastine test was used for diagnosis of subclinical mastitis (Mutovin, 1974). This test is akin to the California Mastitis Test (CMT). Dimastine is a solution of surfactants. The dimastine test indirectly specifies the amount of leukocytes in milk. After ending of milking 1 ml of milk (alveolar milk) from every nipple of the udder was placed in the appropriate opening of a milk-control plate and mixed with 1 ml of 5% dimastine solution for 20-30 sec with a glass rod. The character of the clot was evaluated by its density and colour. Cows were considered to have mastitis if the dimastine reaction was assessed as +++ or ++++ accompanied by a positive sedimentation test.

The sedimentation test (Mutovin, 1974) was applied for confirmation of the diagnosis in case of positive and doubtful results of the dimastine test. After completing milking, 10-15 ml of alveolar milk from each quarter of the udder was placed in separate test tubes and maintained at a temperature of 4-8°C for 16-18 h. The estimation of milk colour, presence and volume of the sediment, height and character of the cream layer was then done. The absence of sediment was considered a negative result (absence of mastitis), weak sediment, doubtful result, appreciable and strong deposit of height 0.1-0.15 cm and more, presence of mastitis. Clinical mastitis was determined from the character of milk secretion and condition of the udder.

Collection and processing of information

Milk from each cow was tested for subclinical mastitis from 4 up to 7 times during lactation, and indices of clinical mastitis were also taken in to account. Cows were considered healthy if they did not have a single episode of mastitis during lactation. If cows had any form of mastitis one time during lactation, they were considered sick. The incidence was judged by the contribution of sick animals expressed in percent to the total number of investigated animals over a specific time. The influence of the year of inspection, season of the year, qualifications of the personnel, age of animals, level of dairy production, properties of the udder, and hereditary factors on the disease was investigated. Separate milking and accounting of milk from each quarter of the udder were carried out for estimation of uniformity of udder development.

The following parameters of uniformity of udder development were taken into account: an index expressing the relation of milk yield from the front quarters to that from all quarters, %; the maximum deviation of milk yield from one quarter from the parameters of an ideal udder (from 25%), %; the maximum difference in milk from the quarters with the highest and lowest efficiency, %; the relation of the milk yield from the most productive quarter to the milk yield from the least produc-

tive one, Max/min index (M/m); the relations of milk yield from the most productive quarter to the total milk yield from quarters, coefficient of uniformity (CU).

The coefficients of genetic correlation between mastitis and milk yield were calculated on the basis of one-factor dispersion and co-variation analysis in groups of half-sibs of the father. The coefficients of heritability in a female line were established by coefficients of phenotypic correlation between mothers and daughters. Heritability on the fathers' side was revealed in correlations between half-sibs of the father. Repeatability of bulls' estimations on resistance of their daughters to mastitis for different lactations was defined according to the coefficient of Spearman rank correlation (r_s).

For the estimation of distribution of related cows on mastitis (female families and half-sibs of the father) histograms were constructed and the following calculated: average arithmetic means, median, mode, coefficients of asymmetry (A_s) and excess (E_x). The errors of representation for A_s and E_x were defined according to commonly known formulas (Lakin, 1980).

The breeding value (BV) of bulls for resistance to mastitis was defined according to the formulas of Karlikov et al. (1979).

RESULTS AND DISCUSSION

Factors influencing the frequency of mastitis

Year of investigation. The frequency of mastitis in the same herd strongly varied in different years (from 11.1 up to 34.9%) and was characterised by high variability ($C_v = 40.8\%$). This testifies to the strong influence of various factors on the condition of cows' udders and consequently it is necessary to introduce a correlation for the year of study.

Breed. Cows of different breeds differed in their resistance to mastitis. Ayrshire cows had mastitis less often than Kholmogor and Latvian brown cows (12.3% to 17.6 and 33.1%, respectively). Pure-bred Ayrshires in the Sortavalsky cattle breeding farm were especially resistant to this disease, only 7.8% cows were ill. According to the literature, no breed of cattle can be considered free from mastitis. Madsen et al. (1987) and Saloniemi (1995) show that the majority or practically all breeds of cattle are susceptible to mastitis, but resistance of animals to this disease in different breeds varies.

The age of cows had a significant influence on the frequency of mastitis (Table 1). On our farms the first-calving cows were most resistant to mastitis. In seven herds the average frequency grew from 5.5% in the first lactation to 30.5% in the eighth.

TABLE I

Mastitis frequency of different age cows (average of 7 herds)

Lactation, number	Tested cows n	Mastitic cows	
		n	%
1	2596	142	5.5
2	2748	286	10.4
3	2261	354	15.7
4	1602	338	21.1
5	1185	289	24.4
6	760	191	25.1
7	422	121	28.7
8 and older	459	140	30.5

In practice, early identification of animals with an increased predisposition to mastitis is necessary. We found that the cows that suffered from mastitis in the first two lactations experienced another episode in the third lactation on average in 2.8, and in some herds, even 4.6 times more often than cows that were healthy when young (37.8 vs 13.6%). The coefficient of mastitis recurrence between specific groups of cows equalled on average 0.216 ± 0.024 ($P < 0.001$). Obviously, the level of mastitis resistance of cows that had been ill still remains below the threshold of harmful influence of the environment. As the result they experienced recurrence of this udder disease. Decreased resistance to mastitis with age was reported by Borozdin et al. (1993), Sandholm and Pyörälä (1995), and many others.

Season of the year. The season of the year exerted a certain influence on mastitis frequency. Long-term observations in the Zaitsev state farm have shown that the lowest disease rate was in the winter, 16.9%. In the spring the disease rate sharply grew (almost double) and reached 32.7% ($P < 0.001$). In the summer, in the pasture period, it declined for a short period, but remained on a high level (28.9%). In the autumn the number of sick cows decreased significantly (to 21.5%; $P < 0.05$). Similar data have been reported in the works of Schultze et al. (1985) and Rabold et al. (1988). On other farms the seasonal distinctions were expressed less distinctly or not at all. The literature data is conflicting. Probably, the season of the year is not a main factor of mastitis, and non-observance of the principles of feeding and maintenance is more important in predisposing to mastitis.

Milkmaid. Differences in the sick rate were revealed in groups of cows that were served by different milkmaids, from 0 up to 62%. The strength of influence of the „milkmaid” factor on the disease rate was low ($\eta^2_x = 0.06$), but statistically significant. It shows that the share of milkmaid qualifications was 6% of all factors,

which has an impact on mastitis frequency. Similar data are found in the works of Klaassen (1989), Sandholm and Pyörälä (1995), and others.

Milk yield. Many authors have studied the dependence of mastitis frequency on the level of dairy production of cows, however, a consensus has not been reached yet. Some researchers note that animals with high milk productivity are affected by mastitis more often (e.g., Soldatov et al., 1988; Badran, 1989). The dependence between milk production and mastitis frequency is not analysed at all in some studies (Janicki and Balukiewicz, 1980; Shokurov et al., 1984). Our results have shown that a weak dependence between milk yield and mastitis take place in herds with different levels of milk production. The extent of influence of milk yield for lactation on mastitis spread was low and doubtful ($\eta^2 = 0.015 - 0.066$). Phenotypic correlation between milk yield and mastitis was low and positive in most cases ($r_b = 0.062$ with variations from - 0.122 up to 0.445).

The mastitis rate corresponded to the average data in herds with 5500 kg milk yield. The cows with a yield less than 3000 kg of milk had high mastitis rates.

The results of research on the genetic correlation of the examined attributes are inconsistent too. The majority of the studies have found a positive genetic correlation between milk yield and frequency of mastitis, with the estimation from 0.11 up to 0.70 (Jensen et al., 1985; Monardes and Hays, 1985) This connection assumes an increase in the mastitis rate with increased milk productivity. At the same time, some researchers have established a negative genetic correlation (Al-Rawi et al., 1979). Our calculations (Table 2) have shown a positive genetic correlation between milk yield and frequency of mastitis (0.335; 0.318). The coefficients of genetic correlation have appeared highly negative: -0.841 and -0.792 when more objective estimation of the average data for three lactations was carried out. Therefore it is possible to consider that the bulls with a high genetic potential of dairy productivity have daughters with a lowered frequency of mastitis. Low-productive daughters of the bulls have a large risk of udder inflammation. Coffey et al. (1986) and Lyons et al. (1986) have obtained results similar to ours. A weak positive

TABLE 2
Genetic correlation between milk production and mastitis frequency of Ayrshire cows ($P < 0.001$)

Lactation, number	Number of daughters	Coefficient of correlation	
		between mastitis and milk yield for 305 days of lactation	between mastitis and fat production for 305 days of lactation
1	521	0.335 ± 0.039	0.318 ± 0.039
3	336	-0.318 ± 0.045	0.201 ± 0.048
First three	354	-0.841 ± 0.016	-0.792 ± 0.019

genetic correlation between the amount of somatic cells and milk yield has been established by Monardes et al. (1984) for Ayrshire cows in the first lactation (0.36), high negative in the second and subsequent lactations (-0.97), and also for all lactations (-0.50).

Udder properties. Animals with rather lifted udders are more resistant to mastitis. The disease rate among cows in which the distance from the bottom of the udder to the ground equaled 45-55 cm was 25.6%; 56-66 cm, 13.3%; 67 cm and more, 8.3%. Janicki and Balukiewicz (1980), Rogers et al. (1991) and others arrived at a similar conclusion. Seykora and McDaniel (1985) found a significant negative genetic correlation between the distance of the udder from the ground and the amount of somatic cells in milk. Our data testify that the duration of milking influences the frequency of mastitis. The highest resistance to mastitis (7.7% of sick cows) was in groups of quickly milking cows, with a duration of milking less than 5 min, and the greatest susceptibility in groups of cows of the same age that had a milking time of more than 8 min (23.2%). Lindström and Syväjärvi (1978) and Jahnke et al. (1989) reported a weak connection between frequency of mastitis and udder features or milking intensity in the posterity of bulls. In our work the ranks correlation of bulls on mastitis of daughters with ranks on intensity and time of milking was significant, $r_s = -0.659$ and $r_s = 0.618$ ($P < 0.05$), respectively. These data confirm reports on the increased susceptibility of hard-milking cows to mastitis. The wide spreading of hard milking among the posterity of the particular bulls can be considered an indirect criterion of an undesirable genotype on resistance to mastitis.

In the literature there is no common opinion on the question about distinctions between the predisposition of forward and back quarters of the udder to inflammation. In our study, 4690 sick quarters were taken into account. The back quarters of the udder were affected by subclinical mastitis more often in comparison with the forward ones (54.3 vs 45.7% of the total number of affected quarters). The clinical form of mastitis, in contrast, was more often found in the forward quarters (54.4 vs 45.6%). Overall, the probability of disease of the back quarters is higher than of the forward ones. They are more voluminous and milking takes longer, stress factors can inhibit milk causing incomplete milking and predisposition to mastitis. We have established that cows with a more proportional udder are affected by mastitis less often than animals with an expressed disproportion of udder quarters. The highest ranks correlation was obtained between disease and the M/M parameter, which characterizes the relation of milk amount from the quarters with maximum milk yield to the quarter with minimal milk yield ($r_s = 0.740$; $P < 0.001$). The same ranks correlation was found between frequency of mastitis and coefficient of udder uniformity (CU) - $r_s = 0.740$; $P < 0.001$. The ranks correlation of the bulls by illness of the daughters with ranks of maximum difference in milk yield of udders quarters was also high ($r_s = 0.762$; $P < 0.05$).

The parameters of uniformity of udder development can be a criterion of early evaluation of cows for mastitis resistance. Ayrshire cows can be considered to have a potentially steady resistance to udder inflammation if they have an udder index of more than 45%, maximum difference in milk yield of quarters less than 8%, M/M index less than 1.4, and factor of uniformity less than 0.400.

Influence of maternal genotype on stability and susceptibility of daughters to mastitis

We analyzed pairs of mothers and daughters to study how the genotype of the mothers influences the disease of their daughters. Daughters of sick or previously sick mothers were affected by mastitis more often than daughters of healthy mothers (Table 3). The healthy mothers had only 13.6% sick daughters, sick mothers, 34.5% ($\chi^2 = 22.4$; $P < 0.001$) on average, or 2.5 times more.

Other researchers also pointed to a connection between mammary gland disease of the mothers and daughters (Lindström and Syväjärvi, 1978). Consequently, identification of sick cows and priority of using daughters from resistant mothers can be a good means of increasing the animals' resistance to this disease. The coefficients of phenotypic correlation have confirmed the dependence of daughters' susceptibility to mastitis on the genotype of their mothers. For all samples of 1567 mother-daughter pairs, the coefficient of correlation was equal to 0.227 ($P < 0.001$), and $h^2 = 2r = 0.454$. All these data testify that the daughters inherit the predisposition to the disease from their mothers. Our data also have shown that it is worth while to calculate the coefficients of correlation on the basis of the information received on mothers and daughters for several lactations.

TABLE 3

Mastitis disease of cows in relation to healthy their mothers

Farm	Breed	Number of pair	Healthy mothers				Sick mothers			
			their daughters				their daughters			
			n	healthy, n	sick n	%	n	healthy, n	sick n	%
Breed farm										
"Sortavalsky"	Ayrsh	96	71	66	5	7.0	25	19	6	24.0
Farm										
"Bolshevik"	Ayrsh	62	46	43	3	6.5	16	9	7	43.7
Farm										
"Vedlozersky"	Kholm	138	80	64	16	20.0	58	40	18	31.0
Farm named after Zaitsev	Kholm	258	119	88	31	26.0	139	79	60	43.2
Farm named after Zaitsev	Ayrsh	1029	905	794	111	12.3	124	90	34	27.4
All farms		1583	1221	1055	166	13.6	362	237	125	34.5

Resistance in families. The frequency of mastitis in 83 families was investigated. The obtained data testify to the unequal resistance of cows to mastitis in different families, the sick rate varied widely, from 0 to 57%. In the Vedlozersky farm, every ninth family (11.1%) was free from mastitis, while in the Sortavalsky breeding farm, more than half (57.5%).

Genotype of bulls as a factor of resistance of cows to mastitis

The genotype of the sire significantly influenced the frequency of mastitis in populations. In 1989-1994 the incidence of mastitis in the daughters of different bulls varied from 9.0 to 34.7%, on average 21.9% for the first three lactations.

The best bulls Sumrak 259, Atlas 348 and Into 101/205 were classified on the basis of data for the first lactation. The sick rate of their daughters did not exceed 3.5%. At the same time the sick rate of the first-calving daughters of bulls Unga 207/2 and Dictor 1170 was 18.5 and 20.7%, respectively. The distinctions between the best and worse bulls deviate from a zero hypothesis. The best bulls have kept their status in subsequent lactations. In other herds the best and worse bulls also were picked out based on mastitis resistance of their daughters. The distinctions in resistance to mastitis in the daughters of different bulls were shown by Merkurieva et al. (1980), Wolf and Schönstedt (1982) and others.

Distribution of groups of related animals. Histograms of distribution of the half-sisters of the father (Figure 1) and female families on mastitis (Figure 2) were constructed for definitions such as inheritance of resistance to mastitis. The calcu-

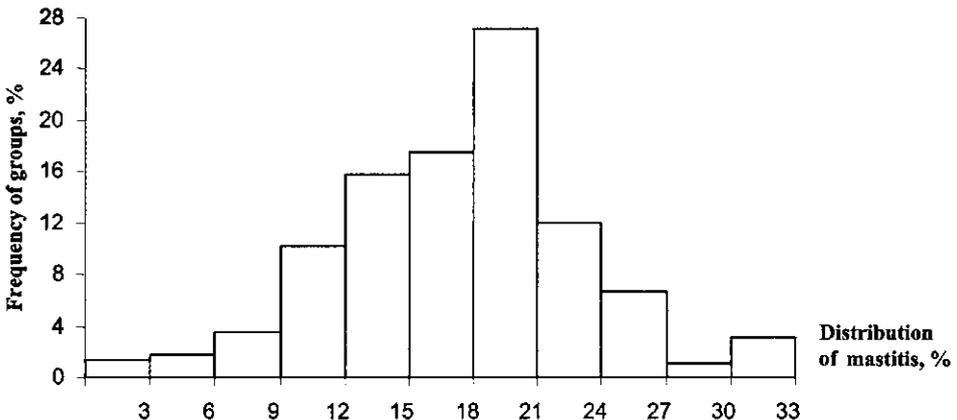


Figure 1. Distribution of infection among daughters of Kholmogor breed sires through all lactations (35 sires, 2876 lactations)

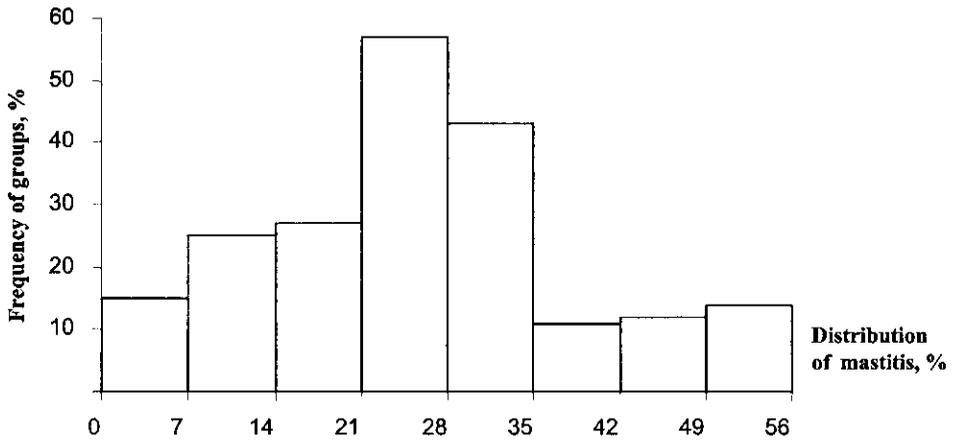


Figure 2. Distribution of Kholmogor breed families according to their susceptibility to mastitis

lations showed that in the father's half-sibs, the arithmetic average (19.2), median (19.5) and mode (20.6) were similar. The coefficient of asymmetry was equal to -0.074 ± 0.045 ($t_{As} = 1.64 < 3$), and coefficient of excess $+0.356 \pm 0.091$ ($t_{Es} = 3.91 > 3$). This means that the distribution of groups of daughters of different bulls in terms of mastitis is actually normal, though with the presence of some positive excess. In female families the arithmetic average (29.0), median (30.2) and mode (29.2) coincide even more, and the criterion of reliability of coefficients of asymmetry and excess was less than 3:

$$A_s = -0.127 \pm 0.162 \quad (t_{As} = 0.78 < 3) \quad \text{and} \quad E_x = -0.463 \pm 0.324 \quad (t_{Ex} = 1.43 < 3).$$

Consequently, the distinction between empirical and Gauss curves corresponds to a zero hypothesis, and the presented distribution of half-sisters on the father's side and families of cows submits to the law of normal distribution. Usually such a law is characteristic of quantitative attributes that are caused by the additive action of genes. This fact testifies to the benefit of the hypothesis about the polygenic type of inheritance of resistance or susceptibility of cows to mastitis.

Reproducibility of bull evaluations on mastitis frequency of their daughters. Reproducibility was judged by the coefficient of ranks (r_s) of mastitis frequency in daughters in different lactations. The ranks of the Ayrshire breed bulls for the first, second and third lactation correlated with the estimation for the first three: $+0.605$ ($P < 0.05$), $+0.573$ ($P < 0.05$) and $+0.773$ ($P < 0.01$). The stability of the evaluation of bulls on mastitis resistance depends on the number of daughters. The reproducibility of bull evaluation for different lactations increases as the number of daughters increases.

The coefficients of ranks correlation of Kholmogor bulls in all cases were higher than 0.7 (Table 4) on the basis of information on a large number of daughters in three herds.

With a high number of posterity, the first preliminary judgement about the advantage of the sire can be obtained on the basis of inspection of first-calving cows. Vecht et al. (1985) reported authentic distinctions between bulls on mastitis resistance of their daughters, which were based on the first lactation. The distinctions in resistance of the daughters of different bulls to mastitis appear early in most cases. This gives an opportunity for objective estimation and selection. Our data have also shown that the frequency of mastitis in posterity of particular bulls is not always connected with dairy productivity. Therefore the assessment of sires on dairy production is necessary to supplement the assessment of the resistance of their daughters to mastitis.

TABLE 4
Coefficients of ranks correlation of Kholmogor bulls on mastitis diseases of their daughters, data in the three herds ($P < 0.05$)

Parameters	Comparison of lactation of the daughters					
	first and second	first and third	first and three of first	second and third	second and three of first	third and three of first
Quantity of daughters lactations	1669	1212	2667	1273	2723	2271
rs	0.715*	0.900	1.000	0.900	0.800	0.900

* $P > 0.05$

Heritability of resistance to mastitis from the fathers. According to literature data, the coefficients of heritability (h^2) of mastitis frequency vary strongly, from 0 to 0.5. The high coefficients (more than 0.4) are obtained when methods of calculating h^2 on the correlation of parameter of the mothers and daughters are used. By definition h^2 by half-sibs method on the father on the large experimental material the coefficient was in the limits of 0.1-0.2 (e.g., Karmanova and Bolgov, 1979; Ernst and Karlikov, 1983). In a few works, h^2 is reported as less than 0.1 (Emanuelson et al., 1988; Duda and Pirchner, 1989; Monardes et al., 1990).

In our work the heritability of mastitis frequency was calculated on separately taken lactations and on average data for a few lactations of half-sibs of the father. This evens out the influence of most environment factors. In the Kholmogor breed the heritability factors of mastitis frequency were low, but statistically significant (Table 5).

TABLE 5

Heritability of mastitis' resistance on fathers (on the data of three herds, Kholmogor breed)

Lactation	Parameters				
	number of sires	number of daughters	average number of daughters on one sires	h ²	P
First	6	804	134	0.171	<0.001
Second	6	865	144	0.088	<0.01
Third	5	408	82	0.098	<0.05
Three of first lactation	5	1863	373	0.135	<0.001

The number of daughters per bull in our dispersion complex reached 134-144. The highest h² was for the first lactation of the daughters, 0.171, for the second and third lactation, 0.085 and 0.098, respectively in our case. Laben (1979) reported a very high h² (0.48) by results of the first lactation, with an average number of 81 daughters for each of 197 bulls.

In the Ayrshire breed we also obtained the highest coefficient of mastitis heritability for first-calving cows (0.200; P<0.01), for subsequent lactations it was reduced to 0.035-0.091. For all lactations in our material h² was equal to 0.088 (P<0.05).

The results of dispersion analyses in our study have shown that the genetic variability (or additive variability) also is included in phenotypic variety on frequency of mastitis. According to the results of the estimation for the first lactation the genetic dispersion σ_s^2 is equal to 0.055, casual σ_E^2 - 0.034, criterion of Fisher, 1.62 (P<0.01); for the second lactation, respectively 0.115, 0.093, 1.24 (P>0.05); for the third, 0.147, 0.113, 1.30 (P>0.05). The influence of the father's genotype on mastitis resistance of posterity was showed especially precisely by analysis of results for all lactations: $\sigma_s^2 = 0.231$, $\sigma_E^2 = 0.107$, F= 2.16 (P<0.001).

Therefore, analysis of the coefficients of heritability testifies to the presence of significant and real genetic variability of mastitis resistance in herds and the opportunity to select for resistance to the disease. Generalizing the obtained results, it is possible to conclude that in Kholmogor and Ayrshire herds, h² of mastitis frequency is equal to about 0.1 on average with variations for separate lactations from 0.04 to 0.24. The values of h² that were established in our work give an opportunity to already confidently assess the genotype of the bull on mastitis resistance on the first lactation in the presence of a sufficiently large number of daughters. The scale for definition of the minimal number of daughters necessary for 70% accuracy of bull assessment depending on the meaning of the coefficient of heritability, was designed in our work.

Bull value (BV) on resistance to mastitis strongly varied. The BV of Kholmogor bulls varied from - 3.7 to 21.9% (Table 6). Zhikler 634 was the best bull, his BV equalled -3.7%. The minus sign „minus” means that this bull improves resistance to mastitis and reduces disease frequency in relation to the average frequency of mastitis in herds. Animals with a high and low breeding value that showed a wide variability from - 4.2 to 24.0%, were also found among the Ayrshire bulls. Consequently, the bulls transfer to their daughters unequal concentrations of additive genes of resistance to mastitis. The calculations showed that the ranks of sires on breeding value have rather high reproducibility according to their estimation on cows of different age $-r_s=0.772-1.00$. Zhang et al. (1994) reported a significant correlation (0.62) of breeding value of Canadian Holstein bulls on somatic cells for the first and next lactations of daughters. After the assessment of bulls on breeding value for mastitis resistance we divided all animals into three breeding categories: plus proven sires, 29.6%, neutral, 44.4%, poor proven, 26.0% of resistance parameter.

TABLE 6

Breeding value of the Kholmogor bulls according to resistance to mastitis (data on three herds, the first lactation of daughters)

Bulls	Number of daughters	Diseases of daughters, %	BV, %
Zhikler 634	326	2.5	-3.7
Udachnik 5	138	2.9	0.3
Zaslon 725	80	7.5	9.0
Zodchij 2284	101	13.0	19.2
Zavar 2270	60	16.0	21.9

Possible genetic progress

Effect of selection (E_M) was found as the product of coefficient of regression ($R_{D/M}$) on selection differential (S_M): $E_M = R_{D/M} \times S_M$. The predicted effect of selection was low and equal to 0.089 on all surveyed herds (Table 7). This means that with the removal of cows-mothers that were sick or had suffered from mastitis, the number of healthy daughters in the following generation will increase by 8.9%. The annual effect of selection on mastitis resistance will make 1.8% if the interval between generations of cows equals 5 years. Skripnichenko (1991) obtained similar parameters on possible effects of selection. With the registration of different quantities of lactation in the Zaitsev state farm (Ayrshire breed) a smaller effect of selection is predicted. The number of healthy daughters will rise by 4.53%, the annual effect of selection will reach 0.9% with selection of mothers for the first three lactations. Consequently, the breeding of all healthy mothers without exception can supply the same genetic progress, as well as selection for milk yield. How-

TABLE 7

Parameters of possible selection effect on mastitis resistance caused by selection of mothers

Farm	Breed	$R_{d/m}$	S_m	$E_m, \%$
"Bolshevik"	Ayrshire	0.372	0.258	9.5
"Sortavalsky"	Ayrshire	0.169	0.260	4.3
Named after Zaitsev	Kholmogor	0.171	0.538	9.1
"Vedlozersky"	Kholmogor	0.110	0.420	4.6
All herds		0.208	0.429	8.9

ever, owing to low productivity part of the healthy cows are removed, and some highly productive animals with a predisposition to mastitis are used in the herd. Therefore, actual effect of selection on decreasing mastitis will be less for mass selection. However, this does not mean that such selection should not be carried out.

Realized genetic trend

We estimated the realized genetic trend of resistance of cows to mastitis by the method of Smith (1962) updated by Kuznetsov (1983). The annual effect of selection taking into account the daughters of the first lactation is equal to +6.10 and +9.38%, respectively in two variants of calculation. This means that the share of first-calving cows, that are free from mastitis, is increased by 6-9% in a herd annually due to the influence of hereditary factors. The realized genetic progress was lower, from 2 to 5% for estimation of some lactations. Consequently, and with absence of direct selection on resistance to mastitis in herds, positive genetic shifts in frequency of mastitis are nevertheless observed.

CONCLUSIONS

Many factors influence the frequency of mastitis in cows. This makes it necessary to optimise paratypic factors, improve feeding conditions, maintenance and handling of cows, increase udder quality by selection, perfection of milking technology and qualifications of personnel. In our work the important role of genetic factors in resistance of cows to mastitis was shown on different selection levels. The presented results prove the necessity and possibility of using genetic methods to increase the resistance of dairy cattle to mastitis. Therefore, within the framework of complex programs of addressing the issue of mastitis it is necessary to undertake efforts to accumulate or increase the concentration of resistance genes in populations of cattle to the disease. Bulls can exert an essential influence on increasing

genetic progress in resistance of cows to mastitis. Assessment of the impact of bulls on the resistance of their daughters to mastitis and use of prepotent sires are necessary together with the assessment of effect on milk production. It is possible to expect a genetically fixed reduction of mastitis frequency if the parameter of mastitis resistance is included among selection attributes and with the realization of the appropriate selection.

REFERENCES

- Al-Rawi A.A., Laben R.C., Pollak E.J., 1979. Genetic analysis of California Mastitis Test records. 2. Score for resistance to elevated tests. *J. Dairy Sci.* 62, 1125-1131
- Andersen E., 1978. On the possibility of breeding for genetic resistance to disease in cattle. 29th Annual Meeting EAAP, Stockholm, p. 52
- Arney D., Phillips C.J.C., Chiy P.C., 1996. Alkali metal effect on dairy cow production and somatic cell count. In: J.A.M. van Arendonk (Editor). *Book of Abstracts of the 47th Annual Meeting of the EAAP, Lillehammer (Norway)*, p. 155
- Badran A.E., 1989. Genetic analysis of mastitis score in Friesian cows and its relationship to environmental factors. *Indian J. Anim. Sci.* 59, 703-706
- Banos G., Shook G.E., 1990. Genotype by environment interaction and genetic correlation among parities for somatic cell count and milk yield. *J. Dairy Sci.* 73, 2563-2573
- Boettcher P.J., Van Doornmaal B.J., 1999. Tools for selection for functional traits in Canada. In: H. Aumann, V. Ducrocq, N. Gengler, A. Groen, E. Strandberg, H. Sölkner (Editors). *Proceedings of International Workshop on EU Concerted Action on Genetic Improvement of Functional Traits in Cattle (GIFT); Breeding Goals and Selection Schemes*. Wageningen Press, Wageningen (The Netherlands), pp. 29-39
- Boettcher P.S., Hansen L.B., Van Raden P.M., Ernst C.A., 1992. Genetic evaluation of Holstein bulls for somatic cell in milk of daughters. *J. Dairy Sci.* 75, 1127-1137
- Boichard D., Rupp R., 2001. Phenotypic and genetic relationships between somatic cell counts and clinical mastitis in French dairy Holstein cows. *Proceeding of the Interbull Technical Workshop, Verden (Germany)*, pp. 66-72
- Bolgov A.E., Karmanova E.P., 1989. *The Using of Ayrshire Cattle for the Improvement of Milk Breeds (in Russian)*. Moscow
- Borozdin E.K., Kleberg K.V., Zimin G.J., 1993. *Resistance of cattle to mastitis (in Russian)*. ASRB Institute, Moscow, pp. 207
- Coffey E.M., Vinson W.E., Pearson R.E., 1986. Potential of somatic cell concentration in milk as a sire selection criterion to reduce mastitis in dairy cattle parities. *J. Dairy Sci.* 69, 2163-2172
- Collean J.S., le Bihan-Duval E., 1995. A simulation study of selection methods to improve mastitis resistance of dairy cows. *J. Dairy Sci.* 78, 659-671
- Egan J., 1984. Mastitis - a review. *Irish Vet. News* 3, 5-18
- Emanuelson U., Dannel B., Philipsson J., 1988. Genetic parameters for clinical mastitis, somatic cell counts and milk production estimated by multiple-trait restricted maximum likelihood. *J. Dairy Sci.* 2, 467-476
- Eriksson J.Å., 1991. *Mastitis in Cattle Breeding for Diseases Resistance in Farm Animals*. ED School of Agricultural and Forest Sciences, University of Wales, Bangor, pp. 394-410

- Ernst L.K., Karlikov D.V., 1983. Results and perspectives of scientific research on genetic resistance of agricultural animals to diseases. Genetic resistance of agricultural animals to diseases (in Russian). ASRB Institute, Moscow, pp. 3-4
- Fourichon C., Seegers H., Beaudeau F., Bareille N., 1996. Critical control points analysis for udder health management in dairy herds. In: J.A.M. van Arendonk (Editor). Book of Abstracts of the 47th Annual Meeting of the EAAP, Lillehammer (Norway), p. 152
- Garkavi F.L., Briede D.A., 1984. Hereditary resistance and susceptibility of cows to mastitis (in Russian). *J. Agr. Biol.* 8, 24-26
- Heuven H., Bovenhuis H., Politick R., 1988. Inheritance of somatic cell count and its genetic relationship with milk yield in different parities. *Livest. Prod. Sci.* 18, 115-127
- Jahnke B., Funke U., 1989. Breeding improvement of udder. Methodological aspects (in German). *Fortschr. Landw. Nahrungs.* 27, 103-104
- Janicki C., Balukiewicz A., 1980. Genetic conditions of cow's mastitis appearance (in Polish). *Roczn. Akad. Roln. Poznań* 120, 59-66
- Jensen N.E., Madsen P., Larsen B., 1985. Heritability and markers of resistance against mastitis in the Danish RDM breed. *Kiel. Milchwirt. Forschungsber.* 37, 506-510
- Juga J., Mäntysaari E.A., Pösö J., 1999. Economic response to total merit selection in Finnish Ayrshire breeding. In: H. Aumann, V. Ducrocq, N. Gengler, A. Groen, E. Standberg, H. Sölkner (Editors). *Proceeding of International Workshop on EU Concerted Action on Genetic Improvement of Functional Traits in Cattle (GIFT); Breeding Goals and Selection Schemes.* Wageningen Press, Wageningen (The Netherlands), pp. 79-87
- Kalmykova O.A., 2000. Hereditary determination of resistance to mastitis (in Russian). *Zootekhnija* 4, 11-12
- Kaneene J.B., Hurd H.S., 1990. The National animal health monitoring system in Michigan. *Prev. Vet. Med.* 8, 103-114
- Karlikov D.V., Lebedev M.M., Nahmanson V.M., 1979. Selection of the cattle to resistance of leucoses (in Russian). *Zhivotnovodstvo* 5, 31-34
- Karmanova E.P., Bolgov A.E., 1979. Inheritance of resistance to mastitis in dairy cattle (in Russian). *Genetic* 15, 1298-1303
- Kaustell K.V., Mäntysaari E.A., Huhtanen P., 1996. Utilisation of feed consumption date and contemporary group solution in management of milk recorded herds. In: J.A.M. van Arendonk (Editor). *Book of Abstracts of the 47th Annual Meeting of the EAAP, Lillehammer (Norway)*, p.129
- Klaassen M.N., 1989. Diagnostics, prophylaxis and treatment of cow's mastitis in a dairy complex (in Russian). PhD Thesis. Tartu, pp. 17
- Kuznetsov V.M., 1983. The Estimation of Genetic Changes in the Herds and Populations of Agricultural Animals. *Methodical Recommendations* (in Russian). ASRIBGA, Leningrad, pp. 44
- Kopytin V.K., Novikov O.G., 1999. Cow's mastitis (in Russian). *Veterinaria* 2, 12-14
- Laben R., 1979. The inheritance of mastitis resistance. *J. Dairy Cattle Day* 18, 43
- Lakin G.F., 1980. *Biometry* (in Russian). Moscow
- Lindström U.B., Syväjärvi J., 1978. Use of field records in breeding for mastitis resistance in dairy cattle. *Livest. Prod. Sci.* 5, 29-44
- Lyons D.T., Freeman A.E., Berger P.J., 1986. Evaluation of health related traits in Holstein cattle. *J. Dairy Sci.* 69, 124
- Madsen P.S., Nielsen S.M., Rasmussen M.D., Klastrup O., Jensen N.E., Jensen P.T., Larsen B., Hyldgaard-Jensen J., 1987. Research of the genetically conditioned resistance to mastitis (in Danish). National Institute of Animal Science, Copenhagen (Denmark), pp. 227

- Merkurieva E.K., Skripnichenko G.G., Beljaeva N.V., 1980. Characteristic of Ayrshire cows herd on incidence of subclinical mastitis in connection with genetic accessory (in Russian). Collection of Scientific Works, Moscow Veterinary Academy, 115, 3-7
- Miller G.Y., Dorn C.R., 1990. Costs of dairy cattle diseases to producers in Ohio. *Prev. Vet. Med.* 8, 171-182
- Monardes H.G., Hayes J.F., Moxley J.E., 1984. Heritability of lactation cell count measures and their relationships with milk yield and composition in Ayrshire cows. *J. Dairy Sci.* 67, 2429-2435
- Monardes H.G., Hayes J.F., 1985. Genetic and phenotypic relationships between lactation cell counts and milk yield and composition of Holstein cows. *J. Dairy Sci.* 68, 1250
- Mutovin V.I., 1974. Fight with Mastitis of Cows (in Russian). Moscow
- Pösö J., Mäntysaari E.A., 1996. Relationships between clinical mastitis, somatic cell score and production for the first three lactations of Finnish Ayrshire. *J. Dairy Sci.* 79, 1284-1291
- Pryce J., Simm G., Amer O., Coffy M., Stott A., 1999. Returns from genetic improvement on indices that include production, longevity, mastitis and fertility in UK circumstances. In: H. Aumann, V. Ducrocq, N. Gengler, A. Groen, E. Strandberg, H. Sölkner (Editors). Proceedings of International Workshop on EU Concerted Action on Genetic Improvement of Functional Traits in Cattle (GIFT); Breeding Goals and Selection Schemes. Wageningen Press, Wageningen (The Netherlands), pp. 55-61
- Rabold K., Sastry N.S.R., Metz A., Grimm H., 1988. About influence of environment on mastitis incidence (in German). *Wien. Tierärztl. Moschr.* 75, 249-254
- Rogers G.W., Hargrove G.L., Lawlor T.J., Ebersole J.L., 1991. Correlations among linear type traits and somatic cell counts. *J. Dairy Sci.* 74, 1087-1091
- Saloniemi H., 1995. Use of somatic cell count in udder health work. In: M. Sandholm, T. Honkanen-Buzalski, L. Kaartinen, S. Pyörälä (Editors). *The Bovine Udder and Mastitis*. University of Helsinki, Faculty of Veterinary Medicine, Helsinki, pp. 105-114
- Sandholm M., 1995. Detection of inflammatory changes in milk. In: M. Sandholm, T. Honkanen-Buzalski, L. Kaartinen, S. Pyörälä (Editors). *The Bovine Udder and Mastitis*. University of Helsinki, Faculty of Veterinary Medicine, Helsinki, pp. 89-104
- Sandholm M., Pyörälä S., 1995. Clinical examination of a mastitic cow. In: M. Sandholm, T. Honkanen-Buzalski, L. Kaartinen, S. Pyörälä (Editors). *The Bovine Udder and Mastitis*. University of Helsinki, Faculty of Veterinary Medicine, Helsinki, pp. 83-88
- Schultze W.D., Stroud B.H., Brasso U.B., 1985. Dairy herd problem with mastitis caused by a rapidly growing Mycobacterium species. *Amer. J. Vet. Res.* 46, 42-47
- Schukken Y.U., Mallard B.A., Dekkers J.C.M., Leslie K.E., Stear M.J., 1994. Genetic impact on the risk of intramammary infection following *Staphylococcus aureus* challenge. *J. Dairy Sci.* 77, 639-647
- Seykora A.J., McDaniel B.T., 1985. Heritabilities of teat traits and their relationships with milk yield, somatic cell count, and percent-two minute milk. *J. Dairy Sci.* 68, 2670-2683
- Shokurov A.E., Safonova L.D., Ysupova I.F., 1984. Role of genotype and some factors in cow's mastitis disease (in Russian). *Zhivotnovodstvo* 6, 44-45
- Shook G.E., Schutz M.M., 1994. Selection on somatic cell score to improve resistance to mastitis in the United States. *J. Dairy Sci.* 77, 648-658
- Skripnichenko G.G., 1991. Genetic parameters of dairy cattle natural resistance and their application in selection (in Russian). PhD Thesis. Moscow, pp. 32
- Smith C., 1962. Estimation of genetic change in farm livestock using field records. *Anim. Prod.* 4, 239-251
- Soldatov A.P., Holodkov S.A., 1988. Resistance of Alatau cows to mastitis. Selection of agricultural animals on resistance to diseases and rise of resistance under conditions of industrial technology (in Russian). Moscow, pp. 26-28

- Van der Beek S., 1999. Breeding for profit in the Netherlands. In: H. Aumann, V. Ducrocq, N. Gengler, A. Groen, E. Standberg, H. Solkner (Editors). Proceedings of International Workshop on EU Concerted Action on Genetic Improvement of Functional Traits in Cattle (GIFT); Breeding Goals and Selection Schemes. Wageningen Press, Wageningen (The Netherlands), pp. 75-78
- Vecht U., Shook G.E., Politiek R.D., Grootenhuis G., 1985. Effect of bull selection for somatic cell count in first lactation. *J. Dairy Sci.* 68, 2995-3003
- Wolf J., Schönstedt G., 1982. Breeding aspects of dairy cows mastitis frequency (in German). *Arch. Tierzucht* 25, 461-468
- Zhang W.C., Dekkers J.C., Banos G., Burnside E.B., 1994. Adjustment factors and genetic evaluation for somatic cell score and relationships with other traits in Canadian Holsteins. *J. Dairy Sci.* 77, 659-665

STRESZCZENIE

Wpływ różnych czynników na odporność krów na zapalenie gruczołu mlekowego

W badaniach przeprowadzonych na ponad 28 tysiącach krów określono wpływ wieku i produktywności krów, pory roku, udoju, cech wymienia oraz czynników dziedzicznych na częstotliwość występowania zapalenia gruczołu mlekowego. Stwierdzono wpływ matek i ojców na odporność lub/ oraz skłonność córek do mastitis. Zachorowalność córek pochodzących od chorych matek była dwu- i więcej krotnie większa niż od matek zdrowych, a częstotliwość występowania zapalenia gruczołu mlekowego w różnych rodzinach wahała się od 0 do 57%. Stwierdzono dużą zmienność w wartości hodowlanej buhajów (od -4,2 do +24,0%) dotyczącej odporności na mastitis. Współczynniki odziedziczalności zapalenia gruczołu mlekowego są niskie, średnio 0,1.

Przy dostatecznej liczbie potomstwa możliwa jest ocena odporności buhaja przeciw zapaleniu gruczołu mlekowego u krów przy pierwszym wycieleniu. Spośród ocenianych buhajów 26,6% zaliczono do odpornych, 44,4% - obojętnych oraz 26,0% o niskiej odporności przeciw mastitis. Częstotliwość występowania zapalenia gruczołu mlekowego u córek oraz wartość hodowlana buhajów są wysoce powtarzalne (0,7 i więcej). Hipoteza zakładająca poligeniczny typ dziedziczenia mastitis została potwierdzona na podstawie analizy histogramów dotyczących częstotliwości występowania mastitis u półrodzeństwa i w rodzinach żeńskich.

Roczny genetyczny trend występowania mastitis waha się od 6-9% do 2-5% w zależności od wieku krów. Możliwa i konieczna jest selekcja bydła mlecznego w kierunku zwiększenia odporności na występowanie zapalenia gruczołu mlekowego łącznie z selekcją uwzględniającą produkcję mleka.