J. Anim. Breed. Genet. 117 (2000), 97–103
© 2000 Blackwell Wissenschafts-Verlag, Berlin
ISSN 0931–2668

Ms. received: 5.1.1999

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Estimation of heritability for hip dysplasia in German Shepherd Dogs in Finland

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Summary

The heritability of hip dysplasia in the German Shepherd Dog was estimated by applying the animal model and the Restricted Maximum Likelihood (REML) method to a data-set which consisted of the hip scores of 10 335 dogs. Fixed effects of the model were the month and the year of birth, screening age, the panelist responsible for screening and the origin of the animal's sire. The litter and the breeder had only minor effects on hip joints. Heritability estimates were moderate (0.31–0.35). The moderate heritability, which was found in this study, enables a much better genetic gain in the breeding programme, if proper evaluation methods, such as BLUP animal model, and effective selection is used instead of phenotypic selection.

Zusammenfassung

Schätzung der Heritabilität der Hüftgelenksdysplasie beim Deutschen Schäferhund in Finnland.

Die Heritabilität der Hüftgelenksdysplasie beim Deutschen Schäferhund wurde mit Hilfe des Tiermodells und der Restricted Maximum Likelihood (REML) Methode anhand von Hüftgelenksgutachten von 10 335 Hunden geschätzt. Als fixe Effekte wurden im Modell 'Geburtsmonat' und '-jahr', 'Röntgenalter', Einfluß des 'Gutachters' und 'Herkunft des Vaters' berücksichtigt. Die Effekte 'Wurf' und 'Züchter' hatten nur einen geringen Einfluß auf die Hüftgelenke. Die Heritabilitätsschätzungen betrugen 0.31 bis 0.35. Die in dieser Studie geschätzten Heritabilitäten ermöglichen es, zusammen mit geeigneten Methoden, wie beispielsweise dem BLUP-Tiermodell und einer effektiven Selektion, einen schnelleren Zuchtfortschritt zu erreichen, als nur phänotypisch zu selektieren.

Introduction

The hip dysplasia control programme for German Shepherds in Finland was started as early as 1963 and since then there has been some type of minimum criteria for breeding stock. At present, a five-point scoring system is used in grading the hip status (Appendix A), and since 1986 only nonaffected or mildly affected (grades A to C) dogs have been allowed to be used in breeding. Thus, the selection has been based on phenotypic grading of hip dysplasia status by radiographic examination. However, this phenotypic selection has not been successful, as in a previous study (LEPPÄNEN and SALONIEMI 1999) no phenotypic progress could be shown, and the disease prevalence had instead increased during the time of restrictions. The reason for the poor success of phenotypic selection is that no effective genotypic selection has taken place during the time period and so the realized selection differential of parent animals calculated from estimated breeding values was practically zero (LEPPÄNEN et al. 2000).

In other studies, heritability estimates for hip dysplasia in German Shepherd Dogs have varied from 0.1 to 0.6 (HENRICSON et al. 1966; LEIGHTON et al. 1977; HEDHAMMAR et al. 1979; LINGAAS and HEIM 1987; SCHWARZ 1989; DISTL et al. 1991; LEIGHTON 1997; SWENSON et al. 1997). These estimates are affected by the population in question and the

Table 1. **Number of hip dysplasia-scored dogs bred by different breeders, and the number of dams and litters from which the dogs originated**

	Breeders		Dams	Litters
Dogs	No.	%	No.	No.
<11	933	80.71	1050	1465
11–30	147	12.72	462	1051
31–50	39	3.37	302	793
51–100	25	2.16	284	1051
101–150	7	0.60	144	449
151–200	2	0.17	48	306
200–300	3	0.26	256	573
Total	1156		2546	5688

statistical method used for calculations. Most previous studies have used various least square models or regression analysis (LEIGHTON et al. 1977; HEDHAMMAR et al. 1979; LINGAAS and HEIM 1987; DISTL et al. 1991; LEIGHTON 1997; SWENSON et al. 1997). Estimates also differ in respect of whether the genetic component used is the maternal, paternal or half sib component. As breeder effect can be confounded with the maternal effect, heritability estimates with paternal half sibs are probably the most reliable of these three components (DISTL et al. 1991). It might be expected that even mass selection based on phenotypic evaluation – if carried out effectively – should make reasonable progress in decreasing the prevalence of the defect with such moderate to high heritability estimates.

The purpose of this study was to estimate heritability of hip dysplasia in German Shepherd Dogs in Finland.

Materials and methods

Materials

The data consisted of registered German Shepherd Dogs and their hip dysplasia screening results. The dogs were officially screened by the Finnish Kennel Club between 1 January 1988 and 31 December 1996. During this period 10706 results were registered. As some dogs were screened more than once, only the latest and thus the valid result of each dog was included in the material, so that the final number of hip dysplasia results was reduced to 10335. The dogs originated from 5688 litters of 1156 breeders (Table 1). The preliminary data set included the identification number of the dog and its parents, date of birth and date of hip dysplasia screening, sex of the dog and the screening results. At screening the dogs were scored in five categories from A to E, where A and B are classified nonaffected; C mildly, D moderately and E severely affected (Table 2). During the study period five panelists have screened all officially registered radiographs. The data was supplemented with additional information about the breeder, the litter identification number, litter size, whether either of the parents was imported, the identification of the hip dysplasia panelist responsible for each screening and hip dysplasia scores of the parents when available. The same basic data was used for composing the pedigree information. The total number of animals in the pedigree was 13892.

Table 2. **Frequencies of dogs and parent animals in different hip score classes**

Class	dogs (%) (n = 10 335)	sires (%) (n = 788)	dams (%) (n = 1803)
A	28.8	81.3	65.4
B	27.0	13.8	17.5
C	23.4	4.6	16.0
D	15.6	0.3	0.8
E	5.2	0.0	0.3

n = number of dogs, sires or dams whose hip dysplasia score was available

Methods

Statistical methods

In a preliminary analysis various combinations of variables, dysplasia score of the mother and the father, litter size, sex, panelist, year of birth, month of birth, year of screening, age at screening, parental status of imported versus nonimported animals were tested. As fixed effects for the model variables that were tested significant ($p < 0.05$) in preliminary data analysis were chosen.

Variance components for hip dysplasia were estimated by applying the Restricted Maximum Likelihood (REML) method (PATTERSON and THOMPSON 1971). Statistical analysis was carried out using the program package PEST (GROENEVELD 1990) including a variance component estimation programme REML VCE4 (GROENEVELD 1997).

The following linear individual animal model (Model 1a) was assumed when estimating variance components and solutions for fixed effects:

$$y_{ijklmno} = \mu + year_i + month_j + age_k + panelist_l + import_m + a_n + \varepsilon_{ijklmno}$$

where $y_{ijklmno}$ is the hip dysplasia score for each dog; $year_i$ is the fixed effect of the ith year of birth ($i = 1, \ldots, 20$); $month_j$ is the fixed effect of the jth birth month ($j = 1, \ldots, 12$); age_k is the fixed effect of the kth screening age class ($k = 1, \ldots, 9$); $panelist_l$ is the fixed effect of the lth panelist ($l = 1, \ldots, 5$); $import_m$ is the fixed effect of the genetic group of the mth imported or nonimported sire ($m = 1,2$); a_n is the random additive genetic effect of the nth animal; and $\varepsilon_{ijklmno}$ is the random residual effect.

The distributions of a_n and $\varepsilon_{ijklmno}$ were assumed to be with zero means and with $\mathrm{Var}(a) = \mathbf{A}\sigma_a^2$ and $\mathrm{Var}(\varepsilon) = \mathbf{I}\sigma_\varepsilon^2$, where $\mathbf{A}$ is a numerator relationship matrix. The covariances between a and ε were assumed to be zero. Models 1b and 1c were similar to Model 1a but they also included the litter or the breeder as a random effect, respectively. In these models the random effects were assumed to be normally distributed with zero means.

Classification of fixed effects

The hip dysplasia scores were coded as numbers 1–5 with respect to the original scoring letters A–E, so that codes 1 and 2 represent the dogs classified as nonaffected (scores A and B, respectively). The dogs were born during a 20-year period. (1976–1995). Birth months were coded as 1–12, from January to December, respectively; and the panelists were coded with personal numbers 1, 2, 3, 4 or 5. For the screening age the dogs were divided into nine categories (12–16; 17–20, 21–24, 25–28, 29–32, 33–36, 37–42, 43–48 and dogs screened at age of 49 months or older). The dogs were coded into two genetic groups according to their

sires (imported versus nonimported sire). The distributions of dogs in different categories of fixed effects are presented in Table 3.

Results and discussion

More than half the total number of dogs (55.8%) and the majority of the parent animals (95.1% of sires and 92.9% of dams) in the data were graded nonaffected (A or B) and only 5.2% of the dogs were graded E, as severely affected. Of the parent animals less than 1% was graded moderately (D) or severely (E) affected, but 21% were mildly affected (Table 2). Thus the intensity of selection, based on the phenotypes of parent animals has been very low, which in part explains the poor success of the breeding programme.

The mean value of the subjectively recorded hip dysplasia score was 2.42 with a standard deviation of 1.20. The coefficient of the variation was 49.9%. The heritability estimates for hip dysplasia were moderate varying from 0.306 to 0.354. The litter or the breeder had only small effects as random effects. When the litter or the breeder were included in the model, a reduction of additive genetic variance was noted; and in Model 1b residual variance increased (Table 4). In comparison the heritability estimates from parent–offspring regression were 0.09 ± 0.02 (sire–offspring); 0.13 ± 0.02 (dam–of spring) and 0.22 ± 0.02 (mid-parent–offspring), respectively, which were all lower than REML estimates.

In this study we used the REML method that treated hip dysplasia as a continuous trait with an assumed normal distribution. This was justified by the results from many studies where the use of nonlinear methods for categorical traits with more than two classes have not proved to be superior compared with methods which assume normality (McGuirk 1989). Because the REML method uses more information of relatives than parent–offspring regression – which were used in many previous heritability studies – it is more reliable. The REML method takes into account the effect of selection through the relationship matrix and thus gives a reliable, unbiased estimate, if all information contributing to selection is included (Sorensen and Kennedy 1984).

The fixed effects that were included in the model were those that were found significant in preliminary data analysis. Significance of screening age and a panelist has been found also in previous studies (Distl et al. 1991; Stur et al. 1996; Smith 1997; Swenson et al. 1997; Willis 1997; Leppänen et al. 2000). Importance of the month of birth has been controversial, instead (Lingaas and Heim 1987; Distl et al. 1991; Hanssen 1991). Our study does not support the previous finding, in which the year of birth had no significant effect (Distl et al. 1991). No previous reports were found in which sires were divided according to their genetic background. It is, however, reasonable to assume that imported animals differ genetically from the original population, and thus have a significant effect (Leppänen et al. 1999).

It can be assumed that a breeder has stable feeding and management systems for pregnant bitches and litters. Nutritional factors during pregnancy and in early puppyhood as well as exercise and growth rate have been shown to effect the development of bones and joints Hedhammar et al 1979; Brass 1989; Smith 1997). Thus a breeder or a litter could be assumed to be a significant random effect. However, breeder and litter effects are partially confounded with maternal effects – so these effects are difficult to separate completely. Furthermore, in the present study, breeder and litter had only minor effects on heritability. They accounted only for 2 and 3% of the total variance, respectively, so the model which did not include either of them (Model 1a) can be considered valid.

The heritability estimates – from 0.306 to 0.354 – in this study and population were around average when compared with previous studies. Thus a low heritability value, leading to ineffective selection, cannot explain the poor performance of phenotypic selection in decreasing the prevalence of hip dysplasia. Nor does the low response to phenotypic selection prove low heritability. Other possible explanations for poor phenotypic progress

Table 3. **Distribution of dogs (%) in different categories of fixed effects**

Category		% of dogs
(a) Proportion of dogs screened by each panelist		
Panelist	1	13.0
	2	26.9
	3	16.6
	4	32.8
	5	10.6
(b) Proportion of dogs according to the birth month		
Month	January	10.2
	February	9.2
	March	9.7
	April	9.5
	May	8.7
	June	6.8
	July	8.6
	August	7.4
	September	7.0
	October	7.3
	November	7.6
	December	8.0
(c) Proportion of dogs in genetic groups according to imported versus nonimported sire		
Sire	Imported	55.1
	Nonimported	44.9
(d) Proportion of dogs screened at different ages		
Age (months)	12–16	29.8
	17–20	28.6
	21–24	15.7
	25–28	8.5
	29–32	4.8
	33–36	3.2
	37–42	3.0
	43–48	1.9
	49+	4.7
(e) Proportion (%) of dogs according to year of birth		
Year of birth	1979	0.1
	1980	0.2
	1981	0.1
	1982	0.2
	1983	0.4
	1984	0.6
	1985	1.5
	1986	4.6
	1987	8.5
	1988	10.3
	1989	11.6
	1990	12.3
	1991	12.2
	1992	12.3
	1993	11.2
	1994	9.8
	1995	4.0

Table 4. **Variances, heritability (h^2) estimates with their standard errors (SE) for hip dysplasia in German Shepherd Dogs, as well as effects of the breeder or the litter on heritability**

	Dog				Breeder effect			Litter effect		
Model	Residual effect	Variance	h^2	SE	Variance	Proportion of the total variance	SE	Variance	Proportion of the total variance	SE
Model 1a	0.915	0.502	0.354	0.018						
Model 1b	0.949	0.431	0.306	0.018	0.028	0.02	0.004			
Model 1c	0.918	0.450	0.319	0.018				0.043	0.031	0.006

could be changes in scoring scales or changes in nutrition and husbandry of breeding animals and puppies.

The phenotypic selection method can be very ineffective, because most dogs used for breeding are phenotypically equal (graded as nonaffected). Thus the selection intensity is very low, if only those few animals with poor phenotypes (grades D or E) are excluded from breeding. Furthermore, the increase of dogs phenotypically graded as C was found in our previous study (LEPPÄNEN et al. 2000). It is supposed that some of the dogs in this class are genetically close to dogs graded D phenotypically. Thus breeding with these dogs might increase the frequency of dysplastic dogs. On the other hand, breeding programmes in other species with respective heritabilities have made clear genetic progress also with mass selection. Here, the organization of and compliance with the breeding programme plays an essential role. Thus, it can be supposed that the current threshold values that exclude hip scores D and E from breeding, are not effective enough, or that many breeders have not selected their breeding stock effectively enough, in respect of hip dysplasia. Ineffective selection was also noted in this material (LEPPÄNEN et al. 2000). On the other hand phenotypic selection is greatly affected by environmental effects and thus fails to select the genetically best dogs for breeding. Instead dogs with good phenotype – but possibly poor genotype – are chosen from breeding. The accuracy of evaluation would increase, if the animal model BLUP was used in the evaluation of breeding values instead of pure phenotypic selection.

Acknowledgements

The data for this study was provided by the Finnish Kennel Club, who also financially supported this study. Technical assistance in programming and data analysis was provided by Mr VEIJO VILVA of the Department of Animal Science of the University of Helsinki.

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Appendix A

Scoring system for hip dysplasia in Finland according to the federation cynologue internationale (FCI)

CLASS	INTERPRETATION
A	No signs of dysplasia
B	Healthy, with slight changes in conformation
C	Mild dysplasia
D	Moderate dysplasia
E	Severe dysplasia