Estimates of genetic parameters for hip and elbow dysplasia in Finnish Rottweilers

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ABSTRACT: Data from 2,764 Rottweiler dogs born from 1987 to 1996 were analyzed with a Restricted Maximum Likelihood procedure using a mixed linear animal model to obtain variance component estimates for hip and elbow dysplasia. The data included 2,764 hip dysplasia and 2,278 elbow dysplasia records. Hip joints were scored as normal (0), borderline (1), slight (2), moderate (3), and severe (4, 4.5, and 5) hip dysplasia. Elbow joints were graded as normal or borderline (0), slight (1), moderate (2), and severe (3) elbow dysplasia. The mean for the hip scores was 1.07 and for the elbow scores .60. Environmental effects influencing hip dysplasia were age, birth year, birth year × season interaction, and experience of the veterinarian responsible for x-raying the dog. For elbow dysplasia, statistically significant effects were age, birth year, sex of the dog, and panelist responsible for each screening. Estimates of heritability for hip and elbow dysplasia were .58 ± .04 and .31 ± .04, respectively, with a genetic correlation of .37 ± .08 between the traits. Genetic improvement of almost one genetic standard deviation was observed in both traits during the 10 yr covered by the data.

Key Words: Dogs, Dysplasia, Elbows, Genetic Parameters, Hips

Introduction

Hip and elbow dysplasia are the most common heritable diseases of dogs (e.g., Studdert et al., 1991; Lust, 1997). Hip dysplasia was first diagnosed in dogs as early as 1935 (Schnelle, 1935), and malformations of the elbow joint were first studied in the 1950s. The syndrome currently known as elbow dysplasia, with four possible types of growth disorders, was first described by Olsson (1974).

Hip and elbow dysplasia are prevalent in large, fast-growing, and heavy dogs such as the Rottweiler (Guthrie and Pidduck, 1990; Distl et al., 1991). In severe cases dysplasia causes arthritis and the affected animals eventually become lame (Lust and Farrell, 1977; Guthrie and Pidduck, 1990; Grøndalen and Lingaas, 1991).

Most studies conclude that hip and elbow dysplasia are polygenic traits (e.g., Leighton et al., 1977; Swenson et al., 1997a), but in some studies hip dysplasia has been considered to be caused only by environmental factors (Torel, 1996). Heritability for hip dysplasia has been estimated to vary from .10 to .60 (e.g., Leighton et al., 1977; Distl et al., 1991; Swenson et al., 1997b) and for elbow dysplasia from .10 to .77 (e.g., Guthrie and Pidduck, 1990; Grøndalen and Lingaas, 1991; Studdert et al., 1991).

Attempts to reduce the incidence of hip and elbow dysplasia in Finland were started in the 1960s and late 1980s, respectively. In most breeds, selection to reduce the prevalence of hip and elbow dysplasia has been unsuccessful thus far. The proportion of phenotypically dysplastic dogs has remained high during the years the Finnish Kennel Club “Breeding against hereditary diseases of dogs” program has been in existence.

The objective of this study was to estimate genetic parameters for hip and elbow dysplasia in Rottweiler dogs in Finland. The genetic trend in prevalence of the dysplasias during the years the data covered was also estimated.

Materials and Methods

Materials

Data. Data and pedigree information were obtained from the Finnish breed association of the Rottweiler. The data consisted of the official hip and elbow dysplasia screening results of Rottweilers screened during the years 1988 to 1996. Included were 2,764 dogs screened.
for hip dysplasia, 2,278 of which were also screened for elbow dysplasia. In the Finnish Kennel Club’s (FKC) program “Breeding against hereditary diseases of dogs,” several breeds have requirements regarding various genetic diseases that breeding dogs must pass before their puppies can be registered by the FKC. For the Rottweiler, breeding dogs are examined for hip and elbow dysplasia and genetic eye defects, but the result of the examination does not influence registration of puppies.

The data included information on the dates of birth and screening, the sex of the dog, the litter identification, the breeder, the x-raying veterinarian, and the panelist responsible for each screening. Litter size was calculated by assuming that all dogs in the pedigree registry of Rottweiler that were born in the same date for the same parents were littermates. This was assumed to be a reliable way to calculate the litter size, because in Finland the majority of breeders register the whole litter at the same time if they want any puppies registered from that litter. The litter size was calculated only for the dogs born in Finland, because imported dogs did not have littermates in the Finnish registry. The screening age of the dog and the age of the dam at the time the litter was born were also calculated. The age of the dogs screened varied from 8 mo to 8 yr. The average age at screening was 24.5 mo for hip dysplasia and 25.3 mo for elbow dysplasia. The majority (77.5%) of the dogs screened were from 18 to 30 mo old. The dogs in the data originated from 899 litters, 278 sires, and 342 breeders.

Hip Dysplasia Grading. The hip dysplasia grading had been done with two different systems in the data. From 1994 onward, the system used has been the present FCI (Fédération Cynologique Internationale) grading, with five classes from A to E. However, approximately half the dogs in the data were screened before the year 1994, and thus were graded according to an older system of the FCI, with two subclasses for each of the present system’s five classes (from A1 to E2). In the evaluation of hip radiographs the form of the femoral head and the acetabulum, joint space, and acetabular angle according to Norberg (Norberg angle) are checked (Figure 1), as follows (Brass and Paatsama, 1983):

- In a healthy joint (A, A1, and A2) the femoral head and the acetabulum are congruent and the craniolateral rim appears sharp and slightly rounded. The joint space is narrow and even and the Norberg angle is approximately 105° (as a reference).
- In a borderline joint (B, B1, and B2) the femoral head and the acetabulum are slightly incongruent and the Norberg angle is approximately 105°, or the center of the femoral head lies medial to the dorsal rim of the acetabulum and the femoral head and the acetabulum are congruent.
- In slight hip dysplasia (C, C1, and C2) the femoral head and the acetabulum are incongruent and the Norberg angle is approximately 100° and(or) there is a slightly flattened craniolateral rim. Irregularities or no more than slight signs of osteoarthritic changes may be present.
- In moderate hip dysplasia (D, D1, and D2) there is obvious incongruency between the femoral head and the acetabulum, with subluxation. Norberg angle is more than 90° (only as a reference). There is flattening of the craniolateral rim and(or) osteoarthritic signs.
- In severe hip dysplasia there are marked dysplastic changes of the joints, such as luxation or distinct luxation. Norberg angle is less than 90° and deformation of the femoral head (mushroom-shaped, flattened) or other signs of osteoarthritis can be seen.

The two grading systems were combined in the present study so that letters A, A1, and A2 were recoded with 0; letters B, B1, and B2 with 1; C, C1, and C2 with 2; and D, D1, and D2 with 3. Finally, letters E, E1, and E2, representing severely affected joints, were recoded as 4.5, 4.0, and 5.0, respectively. The letters E and E2 represent the most severely affected dogs, and the distance between them and the letter D is greater than the distance between other letters.

Elbow Dysplasia Grading. Elbow joints were graded according to International Elbow Working Group (IEWSG) protocol, with numbers from 0 to 3. The elbow score 0 represented dogs having normal or borderline.
A dog was classified as having minimal dysplasia (1) if there were one or more of the following findings (Figure 2):

- Osteophyte formation < 2 mm high seen on the dorsal edge of the anconeal process
- Minimal osteophyte formation (< 2 mm in any direction) on the dorsal proximal edge of the radius, the dorsal edge of the coronoid process, or the lateral palmar part of the humeral trochlea
- Obvious sclerosis in the trochlear notch of the ulna
- Notch and to the proximal radius.

A dog was scored as having mild dysplasia (2) if they were 2 to 5 mm and as having severe dysplasia if the osteophytes were 2 to 5 mm and as having severe dysplasia if they were > 5 mm.

For both hip and elbow dysplasia, the measures used in the statistical analyses were the means of the screening results for the left and right joints of each dog.

**Pedigree.** Pedigree information used in the analyses was the register of the Rottweiler, updated by the FKC but completed by the breed club. The register file included identification numbers of the dog and its parents, sex code, date of birth, and identification number of the litter. The total number of dogs in the register was 13,913. The pedigree in genetic analyses included 4,219 dogs, 399 of which were base animals with unknown parents.

Percentage of dogs screened was 48.4% for hip dysplasia and 37.1% for elbow dysplasia. This evaluation was based on all dogs born between 1988 and 1992 because they could have been screened during the years covered by the data. The percentage of dogs screened for elbow dysplasia had grown since 1992, and in 1995 it was very near the percentage of dogs screened for hip dysplasia.

**Statistical Methods**

Data editing and preliminary analyses were done with program package WSYS (Vilva, 1992). Variance components were estimated by Restricted Maximum Likelihood (REML) using REML VCE4 (Groeneveld, 1997). Environmental effects were tested using the F-test. Breeding values were estimated for hip and elbow joint status by Best Linear Unbiased Prediction (BLUP) using PEST (Groeneveld, 1990).

Effects of several factors affecting hip and elbow dysplasia were examined in preliminary analyses to determine the best models for genetic analyses. The factors studied included sex of the dog, birth year, birth season, birth year × birth season interaction, age of the dog at screening, breeder, litter size, age of the dam when the litter was born, experience of the x-raying veterinarian, and panelist responsible for each screening, as well as maternal and litter effects. Maternal and litter effects were considered random, and the other factors were fixed. Breeder, litter size, age of the dam, and x-raying veterinarian were tested both as fixed and random effects. Effects of breeder, litter size, age of the dam, and maternal effects were not statistically significant (P > .05) on studied measures and were therefore omitted from the final models.

The following model was assumed when estimating the variance and covariance components and the effects for fixed factors for hip dysplasia:

\[
y_{ijklmn} = \mu + \text{age}_i + \text{vet}_j + \text{year-season}_k + \text{ec}_m + a_n + \varepsilon_{ijklmn}
\]

where \(y_{ijklmn}\) is a screening result for hip dysplasia, \(\mu\) is overall mean, \(\text{age}_i\) is fixed effect of the \(i^{th}\) age class (\(i = 1\) to \(10\)), \(\text{vet}_j\) is fixed effect of the \(j^{th}\) veterinarian class (\(j = 1\) to \(7\)), \(\text{year-season}_k\) is fixed effect of the \(k^{th}\) birth year × season subclass (\(k = 1\) to \(32\)), \(\text{ec}_m\) is random effect of the \(m^{th}\) litter, \(a_n\) is random additive genetic effect of the \(n^{th}\) animal, and \(\varepsilon_{ijklmn}\) is random residual effect.

The distributions of \(\text{ec}, a,\) and \(\varepsilon\) were assumed to be multivariate normal with zero means and with Var(\(\text{ec}\)) = \(I\sigma^2_{\text{ec}}\), Var(\(a\)) = \(A\sigma^2_a\), and Var(\(\varepsilon\)) = \(I\sigma^2_\varepsilon\). Covariances between \(\text{ec}, a,\) and \(\varepsilon\) were assumed to be zero.

Elbow dysplasia was analyzed with the following model:

\[
y_{ijklmno} = \mu + \text{age}_i + \text{sex}_j + \text{year}_k + \text{panelist}_l + \text{ec}_m + a_n + \varepsilon_{ijklmno}
\]

where \(y_{ijklmno}\) is a screening result for elbow dysplasia, \(\mu\) is overall mean, \(\text{age}_i\) is fixed effect of the \(i^{th}\) age class (\(i = 1\) to \(10\)), \(\text{sex}_j\) is fixed effect of the \(j^{th}\) sex (\(j = 1\) to \(2\)), \(\text{year}_k\) is fixed effect of the \(k^{th}\) birth year (\(k = 1\) to \(8\)), \(\text{panelist}_l\) is fixed effect of the \(l^{th}\) panelist (\(l = 1\) to \(5\)), \(\text{ec}_m\) is random effect of the \(m^{th}\) litter, \(a_n\) is random additive genetic effect of the \(n^{th}\) animal, and \(\varepsilon_{ijklmno}\) is random residual effect. Distributions and covariances of the elbow dysplasia model were assumed to be similar to those in the model of hip dysplasia.

**Figure 2.** A line diagram of an elbow joint in a lateral view, showing the evaluated sites during the scoring procedure: 1 = dorsal edge of the anconeal process, 2 = dorsal proximal edge of the radius, 3 = dorsal edge of the coronoid process, 4 = lateral palmar part of the humeral trochlea, 5 = area caudal to the distal end of the ulnar trochlear notch and to the proximal radius.
Table 1. Distribution of dogs by hip and elbow scores

<table>
<thead>
<tr>
<th>Score</th>
<th>Left hip jointa</th>
<th>Left elbow jointb</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>0.0 (A)</td>
<td>1,147</td>
<td>41.5</td>
</tr>
<tr>
<td>1.0 (B)</td>
<td>757</td>
<td>27.39</td>
</tr>
<tr>
<td>2.0 (C)</td>
<td>456</td>
<td>16.5</td>
</tr>
<tr>
<td>3.0 (D)</td>
<td>350</td>
<td>12.66</td>
</tr>
<tr>
<td>4.0 (E1)</td>
<td>32</td>
<td>1.16</td>
</tr>
<tr>
<td>4.5 (E)</td>
<td>18</td>
<td>.65</td>
</tr>
<tr>
<td>5.0 (E2)</td>
<td>4</td>
<td>.14</td>
</tr>
<tr>
<td>Total</td>
<td>2,764</td>
<td>100</td>
</tr>
</tbody>
</table>

aScoring: 0.0 (A) = healthy, 5.0 (E2) = severe hip dysplasia.
bScoring: 0.0 = healthy or borderline, 3.0 = severe elbow dysplasia.
cNumber of observations.

When the maternal effect was studied, the random maternal effect of the dam replaced the random effect of the litter in both hip and elbow dysplasia models.

Classification of Fixed Effects

Age of the dog was classified into 10 categories (≤ 17, 18, 19, 20 to 22, 23 to 24, 25 to 26, 27 to 30, 31 to 35, and ≥ 36 mo) in both models. In the hip dysplasia model the veterinarians were classified according to the number of dogs x-rayed in the data set (1 to 10, 11 to 20, 21 to 30, 31 to 45, 46 to 65, 76 to 105, and ≥ 106).

Year-season subclasses include the effects of birth year and birth season, and the interactions between these effects. Number of seasons was four: January to March, April to June, July to September, and October to December. Because of the weather conditions in Finland, dogs born during spring or early summer (from April to June) were assumed to have had more outdoor exercise during puppyhood than dogs born in early autumn or winter (from October to December). Number of birth years was eight (1988 to 1995), which resulted in 32 year-season subclasses. The largest year × season subclass included 152 observations and the smallest 8 observations. In the data, there were four panelists until the end of November 1994. After that there has been only one panelist, who changed in January 1996. Total number of panelists was five.

Table 2. Effects of age on hip and elbow dysplasia, expressed relative to the scores in the class of dogs ≤ 17 mo old

<table>
<thead>
<tr>
<th>Age, mo</th>
<th>Hip dysplasia</th>
<th>Elbow dysplasia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>Dev.</td>
</tr>
<tr>
<td>≤ 17</td>
<td>186</td>
<td>0</td>
</tr>
<tr>
<td>18</td>
<td>129</td>
<td>.2</td>
</tr>
<tr>
<td>19</td>
<td>351</td>
<td>.17</td>
</tr>
<tr>
<td>20</td>
<td>275</td>
<td>.3</td>
</tr>
<tr>
<td>21–22</td>
<td>408</td>
<td>.26</td>
</tr>
<tr>
<td>23–24</td>
<td>346</td>
<td>.33</td>
</tr>
<tr>
<td>25–26</td>
<td>313</td>
<td>.46</td>
</tr>
<tr>
<td>27–30</td>
<td>316</td>
<td>.45</td>
</tr>
<tr>
<td>31–35</td>
<td>221</td>
<td>.52</td>
</tr>
<tr>
<td>≥ 36</td>
<td>216</td>
<td>.55</td>
</tr>
</tbody>
</table>

Means and Variation

The mean for the hip scores was 1.07 and for the elbow scores .60 (data not shown). Score 1 for hip dysplasia represents the borderline case. The mean elbow score was between normal or borderline (0) and slightly affected (1).

Coefficients of variation for the studied measures were high, 102 in hip and 118 in elbow dysplasia, because the screening results were not normally distributed (data not shown). Most of the results were normal or borderline cases, leading to a skewed distribution (Table 1). However, the overall proportion of dysplastic Rottweilers was considerable, especially for elbow dysplasia; 49% of males and 41% of females were affected to some degree. If the class of so-called normal dogs were divided further into two or three subclasses, the distribution of both dysplasia scores might be closer to normal. Even in the normal class differences exist among individuals in the conformation of the joints.

Systematic and Environmental Effects

The effect of sex was statistically significant only for elbow dysplasia ($P < .001$). Males had slightly worse elbow joints than females, but the difference was small on a practical level (.18 score points). This difference could be due to divergent hormonal levels of sexes, which among other things influence growth rates. Similar results of the effect of sex on elbow dysplasia have been obtained in other studies (e.g., Guthrie and Pidduck, 1990; Grøndalen and Lingaas, 1991; Swenson et al., 1997a). Similar to the present results, Leighton et al. (1977), Keller and Corley (1989), and Smith et al. (1995) concluded that sex did not influence hip dysplasia. However, there are also many studies in which the effect of sex has been statistically significant in hip dysplasia (e.g., Hedhammar et al., 1979; Lingaas and Heim, 1987; Distl et al., 1991), but in those studies Rottweilers were not included in the data.

Age was a highly significant factor for both types of dysplasia (Table 2). Young dogs had the best joints, and
Table 3. Effects of birth year on hip and elbow dysplasia, expressed relative to the scores of the dogs born in 1988

<table>
<thead>
<tr>
<th></th>
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<th></th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Hip dysplasia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n</td>
<td>297</td>
<td>349</td>
<td>442</td>
<td>365</td>
<td>442</td>
<td>426</td>
<td>339</td>
<td>104</td>
<td></td>
</tr>
<tr>
<td>Dev.</td>
<td>.00</td>
<td>.03</td>
<td>.12</td>
<td>.3</td>
<td>.32</td>
<td>.25</td>
<td>.35</td>
<td>.38</td>
<td>***</td>
</tr>
<tr>
<td>Elbow dysplasia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n</td>
<td>154</td>
<td>235</td>
<td>337</td>
<td>314</td>
<td>409</td>
<td>403</td>
<td>328</td>
<td>98</td>
<td></td>
</tr>
<tr>
<td>Dev.</td>
<td>.00</td>
<td>-.01</td>
<td>-.04</td>
<td>.11</td>
<td>.18</td>
<td>.16</td>
<td>.19</td>
<td>.12</td>
<td>***</td>
</tr>
</tbody>
</table>

^aNumber of observations and deviation from the class of comparison.
^bLevel of significance, ***P < .001.

the older the dog was at screening, the worse its joints were. This trend was logical because old dogs usually have at least some degree of arthritis in their joints even without being actually dysplastic. Popovitch et al. (1995) did not find the effect of age to be statistically significant in predicting hip dysplasia, but several other studies agree with the findings of this study (e.g., Distl et al., 1991; Swenson et al., 1997a, 1997b). Because of the age effect, all dogs should be screened approximately at the same age, or the records should be corrected for the effect of age.

The effect of birth year accounts for temporal changes in environment because the data were analyzed with the mixed model, which included genetic effects. Statistically significant differences were found between birth years in hip as well as in elbow dysplasia (Table 3). Dogs born in the 1980s scored better than dogs born in the 1990s. Reasons for the slight negative effect of the birth year might be, for example, changes in feeding conventions or in general care of the dogs during their growth period. Another possibility is that the panelists responsible for scoring the radiographs may have slightly changed the scale during the years the data covered. Distl et al. (1991) also reported that the effect of birth year was significant for hip dysplasia.

Only hip dysplasia was affected by the interaction effect of year and season (data not shown). The best subclass was winter (October to December) 1988 and the poorest winter 1995. The difference between the extreme subclasses was very large, 1.40 points. Effect of birth season alone was not statistically significant in either of the studied traits, which is supported by Leighton et al. (1977), Lingaas and Heim (1987), and Distl et al. (1991).

Effect of the experience of the x-raying veterinarian was significant only for hip dysplasia (Table 4). The most experienced veterinarians, who x-rayed more than 105 dogs, had given on average better scores than the most inexperienced veterinarians, who x-rayed fewer than 20 dogs. However, the best scores were noted by veterinarians who x-rayed 31 to 45 dogs. The differences between the classes were very small and no clear trend could be seen.

Despite the given guidelines for scoring, the evaluation of the radiographs is always subjective, and it can be assumed that there are differences between the panelists. However, the effect of the panelist was statistically significant only for elbow joints (Table 5). The differences between the panelists in evaluating elbow joints were large. Nowadays there is only one panelist in Finland, so there is currently no need to correct the records for the effect of a panelist when comparing the dogs. Contrary to this study, Smith et al. (1996) and Stur et al. (1996) found significant differences between panelists in evaluating hip radiographs.

Genetic Parameters

Heritabilities. The estimate of heritability was high for hip dysplasia and moderate for elbow dysplasia (Table 6). The estimates indicate that there is much genetic variation that can be used in selection against hip and elbow dysplasia, even though phenotypic selection has already been practiced more than 30 yr in hip and 10 yr in elbow dysplasia.

Table 4. Effects of the experience of the veterinarian responsible for x-raying on scores for hip dysplasia, expressed relative to scores of veterinarians with ≥ 106 x-rayings

<table>
<thead>
<tr>
<th>No. of x-rayed dogs per veterinarian</th>
<th>1–10</th>
<th>11–20</th>
<th>21–30</th>
<th>31–45</th>
<th>46–65</th>
<th>76–105</th>
<th>≥ 106</th>
<th>p^b</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>664</td>
<td>484</td>
<td>388</td>
<td>304</td>
<td>224</td>
<td>277</td>
<td>419</td>
<td></td>
</tr>
<tr>
<td>Dev.</td>
<td>.17</td>
<td>.13</td>
<td>.15</td>
<td>-.08</td>
<td>.13</td>
<td>-.02</td>
<td>.00</td>
<td>**</td>
</tr>
</tbody>
</table>

^aNumber of observations and deviation from the class of comparison.
^bLevel of significance, **P < .01.
The heritability estimate for elbow dysplasia was in agreement with other studies (e.g., Grøndalen and Lingaas, 1991; Studdert et al., 1991; Swenson et al., 1997a). This would be expected, because those studies used the same kind of data and classification of defects we used. The estimates of heritability, .296 by Grøndalen and Lingaas (1991) and .34 by Swenson et al. (1997a), were also for the Rottweiler, as in our study. The heritability estimate for hip dysplasia was quite high compared to that from other studies performed with various methods and models (e.g., Leighton et al., 1977; Hedhammar et al., 1979; Distl et al., 1991). All of these studies used data from German shepherd dogs. However, a similar estimate for Labrador retrievers (.54) was reported by Swenson et al. (1997b), although it was derived by regression of offspring on parents.

The estimates for hip dysplasia presented in previous studies were computed for scores achieved by x-raying dogs in a position advocated by the Orthopedic Foundation for Animals (OFA), that has been a worldwide standard for more than 30 yr. The same position is also used in Finland. These radiographs are a ventrodorsal view of the pelvis with the coxofemoral joints fully extended, and the knees internally rotated (Figure 1).

A newly suggested, improved x-raying method, PennHIP (Smith, 1997), measures joint laxity in hip joints by a distraction index (DI) that predicts degenerative joint disease in hips. A corresponding method has also been developed in Switzerland (Flückiger et al., 1999). In a study using the DI measurements, the heritability estimate of hip dysplasia in German shepherd dogs was found to be between .42 and .65, and the upper limit for heritability (i.e., the estimate for repeatability of DI measurements over time) in Labrador retrievers was .92 (Smith, 1997). Because of the high heritability estimates, the PennHIP method is considered to be more reliable than the standard method. The estimate of heritability for hip dysplasia in this study is, however, of the same magnitude as in the PennHIP studies.

Litter Effect. The effect of the litter was quite small for both dysplasias (Table 6). The litter effect accounted for 4% of the total variation in hip joints and 6% in elbow joints. These percentages include all the environmental and genetic (dominance and epistasis, if they exist) effects common to the members of the same litter. The litter effect also contains some amount of the breeder’s effect, because breeder was not included in the models. Most puppies move to their new owners at the age of 7 or 8 wk, so the breeder does not have an effect on puppies during their major growth period. The effect of the dogs’ new environment could not be taken into account because there was no information available, and it is therefore included in the residual variance. Compared to our findings, Leighton et al. (1977) and Distl et al. (1991) reported slightly larger litter effects on hip dysplasia of German shepherds, 0 to 10% and 9.4%, respectively.

Maternal Effect. The maternal effect on joint configuration was almost negligible, 1.5% in hip dysplasia and 1.8% in elbow dysplasia (data not shown). As with the litter effect, it was not surprising that the maternal effect was so small. The puppies spend their first 6 to 8 wk with their dam and are separated and subjected to a wide range of environments thereafter. The finding with maternal effects on hip dysplasia is supported also by Lingaas and Heim (1987).

Correlations. The phenotypic and genetic correlations between hip and elbow dysplasia were positive and moderate, .24 and .37 ± .08, respectively. This finding would be expected, because both diseases are disorders in the growth of the bone and thus probably are influenced at least to some extent by the same genes and environmental factors. Due to the positive genetic correlation, selection against hip dysplasia also decreases the prevalence of elbow dysplasia, and vice versa. However, the rate of progress from indirect selection can be

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**Table 5. Effects of the panelists responsible for screening on elbow dysplasia, expressed relative to scores of the panelist number 1**

<table>
<thead>
<tr>
<th>Item</th>
<th>Panelist 1</th>
<th>Panelist 2</th>
<th>Panelist 3</th>
<th>Panelist 4</th>
<th>Panelist 5</th>
<th>Dev.</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>258</td>
<td>331</td>
<td>909</td>
<td>350</td>
<td>409</td>
<td>—</td>
</tr>
<tr>
<td>Dev.</td>
<td>.00</td>
<td>−.01</td>
<td>−.33</td>
<td>−.06</td>
<td>.16</td>
<td>***</td>
</tr>
</tbody>
</table>

*aNumber of observations and deviation from the class of comparison.

bLevel of significance, ***P < .001.
expected to be slow. Correlations between hip and elbow dysplasia have not been estimated in previously published studies, as far as we know.

**Genetic Trend**

The genetic trend in prevalence for hip and elbow dysplasia in Finnish Rottweilers during the years the data covered was studied by estimating breeding values for the dogs with respect to the two defects, and then comparing the means of the breeding values of the dogs born in different years. The breeding values were standardized ($\mu = 100$, $SD = 10$) so that the bigger the value, the better the joints.

Reasonable genetic progress had occurred in the studied population with respect to joint status in both hip and elbow joints (Figure 3). During the period of 10 yr that the data covered, a genetic improvement of almost one genetic standard deviation unit was observed in both traits. The effect of birth year was contrary to the genetic trend, showing worsened environmental conditions (Table 3). Phenotypic trend was nonexistent, so the positive genetic change has been equal to the negative effect of environmental factors, resulting in no change in phenotypes.

The realized genetic progress has been achieved by selection of breeding stock based on the phenotypic screening results only, because no estimated breeding values have been available for the breeders. Because of the high estimates of heritability, this kind of genetic progress can be expected to happen especially in hip dysplasia, but the genetic progress was almost similar in elbow dysplasia, too. This could be partly due to the positive genetic correlation between the defects.

However, because the influence of environmental factors on both types of dysplasia is large, dogs of normal phenotype can be carriers of some dysplasia genes and transmit these genes to their offspring. Therefore, the best method for the breeding evaluation of hip as well as of elbow joints would be the animal model BLUP method, which combines information for all available relatives and simultaneously accounts for differences in environmental effects.

**Literature Cited**


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<th>Trait</th>
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**Table 6.** Estimates of additive genetic ($\sigma^2$), litter ($\sigma_{e}^2$), and environmental variance ($\sigma^2$) for hip and elbow dysplasia, and estimates of heritability ($h^2$) and litter effect ($\text{ec}^2$) with their standard errors ($\text{SE}_h^2, \text{SE}_{ec}^2$).


