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Inheritance of Disc Calcification in the Dachshund

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With 3 figures and 4 tables

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Summary

The occurrence of intervertebral disc calcification was investigated by conventional spinal radiography in eight families of wirehaired dachshunds, with each family comprising one sire, two dams and one litter from each dam. Each offspring was examined radiographically once at 24–35 months of age. The occurrence of disc calcification was rated according to four different scales. A strong correlation was found in the occurrence of disc calcification between offspring and mean parent ($P < 0.001$) and between offspring and dams ($P < 0.005$) on an either/or scale. Statistically significant estimates of heritability of 0.60 and 0.87 were found based on the offspring–sire relationship using the total score and three-class scale, respectively. Higher correlation estimates were found based on the dam–offspring relationship than based on the sire–offspring relationship, suggesting an effect of maternal environmental factors.

Introduction

Intervertebral disc herniation (IDH) occurs in most breeds of dog, but the majority of cases are seen within a number of breeds with disproportionate dwarfism dominated by short, bandy legs and varying breed-specific involvement of the head and spine (Goggin et al., 1970; Gage, 1975). The breeds most frequently predisposed to IDH are the dachshund, Pekinese and French bulldog (Hansen, 1952; Goggin et al., 1970; Gage, 1975; Ball et al., 1982). In the USA, the relative risk for IDH was found to be 10–12 times higher for the dachshund than for all other breeds (Goggin et al., 1970; Priester, 1976) and the prevalence of IDH in dachshunds was estimated at 19 % (Ball et al., 1982). Traditionally, the term 'chondrodystrophia' has been used in veterinary nomenclature for this type of dwarfism, although the growth disturbance is hypochondrodysplastic (Verheijen and Bouw, 1982; Simpson, 1992). Considerable phenotypic variation is seen amongst breeds, with some of the syndromes resembling human hypochondroplasia and others resembling human achondroplasia (Jezyk, 1985). Histologically, achondroplasia is a severe hypochondroplastic disease (Rimoin et al., 1970). The human syndromes are codominant allelic abnormalities involving several alleles (Webster and Donoghue, 1997).

Pathogenesis

In the hypochondroplastic breeds, the predisposition for IDH is caused by an early degenerative process. The degeneration is preceded by early chondroid metaplasia ('chondroid metamorphosis') emerging from the perinuclear zone and affecting the majority of the nucleus pulposus and perinuclear annulus with profound matrix changes occurring within the first year

of life (Hansen, 1952; Braund et al., 1975; Ghosh et al., 1976a, 1977). Chondroid metaplasia becomes macroscopically visible within a few months of age, and histologically, an abnormal broad perinuclear zone consisting of immature fibrocartilage is already visible in the newborn hypochondroplastic dog (Hansen, 1952; Braund et al., 1975). So far, this specific type of metaplasia of intervertebral discs has been demonstrated in the dachshund, Pekinese and beagle (Hansen, 1952; Braund et al., 1975). The metaplasia apparently occurs throughout the vertebral column, but the severity of the degeneration varies between individuals and also within individuals between discs (Hansen, 1952; Havranek-Balzaretti, 1980). Necrosis often accompanied by intervertebral calcification (IDC) is seen in severely degenerated discs (Hansen, 1952). The occurrence of IDH is related to the severity of disc degeneration; IDH is common in dachshunds with calcified discs (IDH prevalence of 24–80 %, depending on age and follow-up period) but rarely occurs in dogs without disc calcification (IDH prevalence 0–6 %) (Havranek-Balzaretti, 1980; Stigen, 1996). One study suggested that the number of calcified discs may be a measure of the degree of intervertebral disc degeneration in the individual (Havranek-Balzeretti, 1980).

Chondroid metaplasia reflects a poor differentiation of the disc and may be considered a congenital malformation as the initial aberrations at a biochemical and cellular level are visible at birth (Hansen, 1952; Ghosh et al., 1976b, 1977). However, the aetiology of this abnormal differentiation is not known.

Inheritance

Disc herniation in the dachshund is a familial condition – with significantly varying incidences among families. A simple Mendelian dominant inheritance can be excluded (Ball et al., 1982). The authors (Ball et al., 1982) further excluded the possibility of IDH being a simple recessive trait or a sex-linked trait and suggested a genetic model involving the cumulative effects of several genes and environmental factors. The discontinuity in the occurrence may partly be accounted for by environmental factors. The low rectangular body conformation is not correlated to the predisposition (Hansen, 1952). For instance, the French bulldog is of a square body type but it is one of the most predisposed breeds (Hansen, 1952), while the basset hound with a low rectangular body is not among the highly predisposed breeds (Priester, 1976).

The strong phenotypic relationship between chondroid metaplasia and hypochondroplastic dwarfism suggests that a major genetic factor for chondroid metaplasia and degeneration results from a pleitropic effect of the hypochondroplasia gene. It seems unlikely that the coexistence of the conditions is caused by two separate genes, as this would imply a specific founder effect in several separate breeds. Furthermore, abnormal chondrocyte differentiation is characteristic of both chondroid metaplasia (Hansen, 1966) and hypochondroplastic dwarfism.

The severity of the degenerative process varies significantly within the affected breeds (Hansen, 1952; Ghosh et al., 1975; Havranek-Balzaretti, 1980; Stigen, 1991). A continuous spectrum of disc degeneration is seen within and among breeds, suggesting a multifactorial disease. Occurrence of normal discs (i.e. absence of chondroid metaplasia) throughout the spine has been reported within a beagle family which also comprised beagles with the typical development of chondroid metaplasia (Ghosh et al., 1975). In the dachshund, large variation in the prevalence of IDC (severe degeneration) and IDH occurs among the dachshund hair coat varieties and even among families (Funkquist and Henricson, 1969; Havranek-Balzaretti, 1980; Stigen, 1991), indicating some degree of inheritance. This conclusion is supported by the occurrence of IDC in the offspring being significantly dependent on the status of the parents (Havranek-Balzaretti, 1980). Unidentified maternal environmental factors seem to influence the number of calcified discs (Stigen and Christensen, 1993) and recently a minor beneficial effect of mechanical loading (exercise) has been demonstrated (Jensen and Ersbøll, 2000).

The hereditary aspect of degeneration of the disc, particularly the variations within the breed, suggests that is might be possible to reduce the prevalence by selective breeding without changing the characteristics of the breed, in particular the low rectangular conformation of the dachshund.

The only available study of heritability of IDC was based on the occurrence in of IDC in half-sib groups, not including the parents (Stigen and Christensen, 1993). Grading of the degree

of degeneration by the number of calcified discs resulted in a higher heritability estimate ($h^2 = 0.22$) than grading as an all-or-none character ($h^2 = 0.16$). Both estimates were statistically non-significant, possibly due to an insufficient sample size ($N = 274$) (Stigen and Christensen, 1993). Another problem may have been the low age (mean = 14 months) of the dogs which may have caused low heritability estimates, as the number of visible calcified discs increases considerably up to 2 years of age in some dogs (Jensen and Arnbjerg, 2000). Stigen (1996) later demonstrated that the number of calcified discs increases by 70.1 % from 1 to 5 years of age.

The object of this study was to estimate the heritability of IDC in the dachshund by including both offspring and parents, the offspring being older than 2 years in order to optimize the accuracy of the evaluation and grading.

Materials and Methods

Sampling

The study was confined to standard wirehaired dachshunds, all registered in the Danish Kennel Club. Eight pairs of paternal half-sib litters and the parental generation, i.e. eight sires and 16 dams, were included. A total of 69 offspring were included, comprising at least three dogs from each litter (4.3 $\pm$ 1.1 offspring per litter).

For the offspring, a lower age limit of 2 years was used, because the majority of disc calcifications become radiographically visible at 6–24 months of age. The risk of IDH increases significantly from about 3 years of age. As suspicion of spinal disease may affect the owners' decision of whether or not to participate in the study, an upper age limit of 3 years for the offspring was defined to avoid biased selection within-litter. The mean age of the parents varied from 2.5 to 8.8 years (6.0 $\pm$ 1.5 years). None of the offspring had clinical symptoms relating to vertebral disease before or during the study. However, one dam had clinical IDH diagnosed prior to inclusion in the study.

The dogs included in this study were selected by multistage sampling according to the criteria listed in Table 1. All pedigrees for litters born between August 1994 and February 1996 and registered with the Danish Kennel Club (personal communication) were reviewed systematically. All litters meeting the criteria listed were included, with the exception of two families. The selection was not directly influenced by the occurrence of IDH, except for one or possibly both of the excluded families: one sire had been euthanized due to disc herniation and in another case the breeder was unwilling to cooperate for unknown reasons.

Radiographic examination

The dogs were radiographed in right lateral recumbency, and at least five lateral projections of each dog were made covering the vertebral column from the second cervical vertebra to the third sacral bone.

The X-ray equipment used was as follows: Polydores XL50 with Bucky-movable grid (Siemens A/S, Copenhagen, Denmark), 3 M Trimax T16 intensifying screens (3 M, Ferrania, Italy) and Fuji Super HR-L30 film (Fugi Photo Film Co., Tokyo, Japan); exposure: 55–60 kV, 9–16 mAs; focus-film distance: 1 m.

All radiological evaluations were blinded with respect to the evaluation of other members of the family concerned. Every set of radiographs was evaluated independently (blinded) twice by the same radiologist at an interval of at least 2 months. If differences were found, the discs in question were re-evaluated later for a definite diagnosis. The evaluations were performed with particular attention to

Table 1. Criteria for selection of litters

1.	Both parents and litters registered with the Danish Kennel Club (DKK).
2.	Litters aged 24–35 months.
3.	Several litters after the same sire.
4.	Sires unrelated (coefficient of relationship $\leqslant 1/16$).
5.	Unrelated dams within sires (coefficient of relationship $\leqslant 1/16$).
6.	All litters bred in different locations (kennels or private homes).
7.	At least three live offspring per litter.
8.	Both dams and sire alive within the examination period.
9.	Only litters reared on Zealand, that is, less than 2 h transport for examination.

Table 2. Definition of classification systems

Classification scale	Class	Definition of class
Either/or scale	0	No calcified discs visible
	1	$\geqslant$ 1 calcified disc
Three-class scale	Low	0–1 calcifications
	Moderate	2–4 calcifications
	Severe	$\geqslant$ 5 calcifications
Five-class scale	S0	No calcified discs visible
	S1	$\leqslant$ 2 calcifications; $\leqslant$ 1 calcifications outside T1–T9
	S2	$\leqslant$ 3 calcifications; $\leqslant$ 2 calcifications outside T1–T9
	S3	$\leqslant$ 6 calcifications; $\leqslant$ 3 calcifications outside T1–T9
	S4	$>$ 6 calcifications or $>$ 3 calcifications outside T1–T9
Score	Numeric (0–26)	Number of calcified discs (Continuous)

superimposition of procc. transversi, articulares and mammilares, extremitas vertebralis, capiti costae, bronchi or gastric content. Borderline cases were registered as normal.

Statistical methods

The occurrence of IDC was analysed using four different classification systems based on the number of calcified discs (Table 2). The association between the occurrence in parents and offspring was evaluated by χ^2-test based on the three-class scale and either/or scale. A linear model was also applied using the three-class scale, the five-class scale and the number of calcified discs as a continuous (numeric) variable ('score'). Parent–offspring regression was estimated and the heritability was estimated by means of regression, i.e. regression of offspring on mean parent or twice the regression of offspring on either dam or sire.

Results

A total of 368 calcifications were identified in eight sires, 16 dams and their 69 offspring. In the offspring group, 80 % (55/69) of the dogs were affected and the number of calcified discs varied with a mean $\pm$ standard deviation (SD) of 4.3 $\pm$ 4.0. All sires were affected (4.0 $\pm$ 3.4 calcifications) while 75 % (12/16) of the dams were affected (2.3 $\pm$ 1.9 calcifications). The distribution of disc calcifications in the vertebral columns of the progeny is shown in Fig. 1;

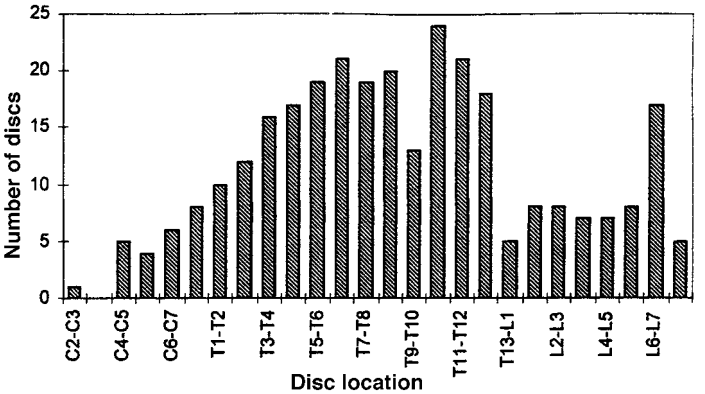

Fig. 1. Spinal distribution of disc calcification in 69 dachshund offspring, aged 24–35 months.

Table 3. Chi-squared test of relation between offspring and parents in occurrence of intervertebral disc calcification

Source	Degrees of freedom	χ_{Yates}^2
Either/or scale		
Offspring–dam†	1	15.8**
Offspring–mean parent†	1	15.8**
Three-class scale		
Offspring–dam	4	17.6*
Offspring–sire	2	4.79
Offspring–mean parent	6	21.1*

Significance level: $*P < 0.005$; $**P < 0.001$.
†The relations offspring–dam and offspring–mean parent are identical as all sires are affected.

the majority of disc calcifications (72 %; 215/299) were located in the thoracic region (T_1–L_1), while 20 % (60/299) of the calcifications were located in the lumbar region (L_1–S_1).

Chi-squared test (either/or and three-class scales)

In the offspring group, the sample incidence (either/or scale) was 71 %. The occurrence depended significantly on the occurrence in the parents, with 91 % of the offspring affected when both parents were affected and 44 % affected when only one parent (sire) was affected (Table 3).

Within both classification systems, a highly significant parent–offspring correlation was found using a χ^2-test with Yates continuity correction. The strength of the relation between sire and offspring could not be estimated using the either/or scale as all sires were affected. All relations except the relation between sire and offspring were statistically significant using the three-class scale or the either/or scale (Table 3).

Linear model (score, five-class and three-class scales)

The relations between score in offspring and dams or sires are illustrated in Figs 2 and 3. The regression coefficients of offspring on parents are shown in Table 4. The regressions

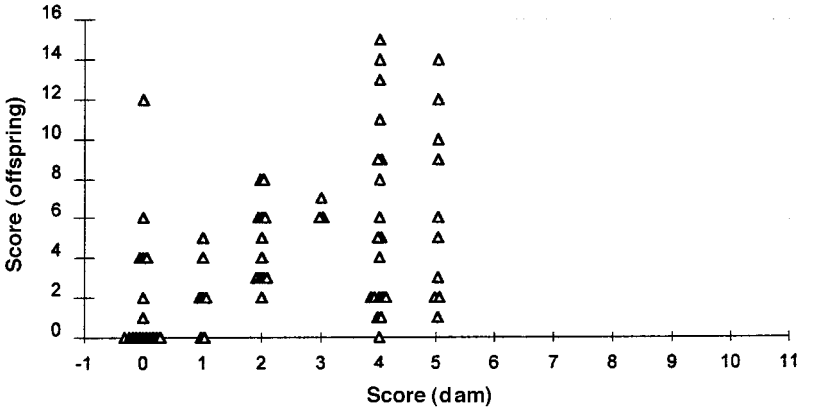

Fig. 2. Relation between dams and offspring in number of calcified discs. The individual score for every offspring (△) is shown.

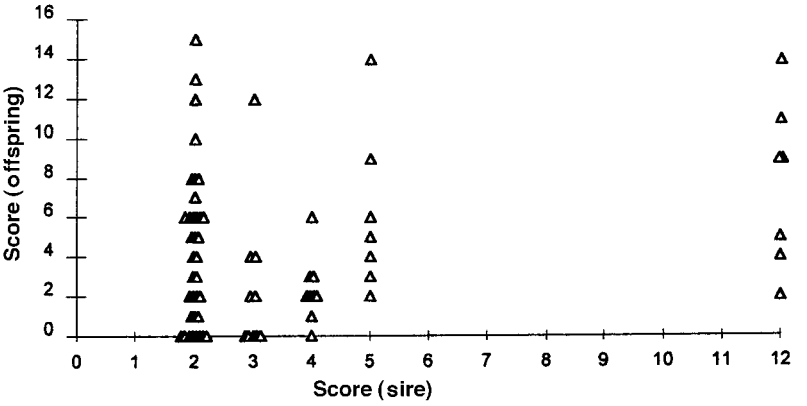

Fig. 3. Relation between sire and offspring in number of calcified discs. The individual score for every offspring (△) is shown.

Table 4. Linear regression of occurrence of intervertebral disc calcification in offspring on occurrence in either parent or mean parent

Scale	Source	Regression coefficient	T (H = 0)	Heritability estimate ($h^2 \pm SD$)
Score	Offspring/dam	0.98 ± 0.23	4.17***	1.95 ± 0.46
	Offspring/sire	0.30 ± 0.14	2.09*	0.60 ± 0.28
	Offspring/meanparent	0.97 ± 0.24	4.11***	0.97 ± 0.47
Five-class scale	Offspring/dam	0.36 ± 0.13	2.97**	0.72 ± 0.26
	Offspring/sire	0.23 ± 0.16	1.43	0.46 ± 0.32
	Offspring/meanparent	0.62 ± 0.14	3.63**	0.62 ± 0.28
Three-class scale	Offspring/dam	0.52 ± 0.13	4.08***	1.0 ± 0.25
	Offspring/sire	0.44 ± 0.21	2.03*	0.87 ± 0.42
	Offspring/meanparent	0.99 ± 0.19	5.08***	0.99 ± 0.38

Significance level: * $P < 0.05$; ** $P < 0.005$; *** $P < 0.001$.

of offspring on mother and on mean parent were statistically highly significant ($P < 0.001$) in all classification systems. Using the three-class scale or score, the relation between sire and offspring was found to be statistically significant at the $P < 0.05$ level. The heritability estimates based on the linear regression model are shown in Table 4.

Discussion

In the present study, a number of heritability estimates were obtained based on different classification systems for rating the severity of IDC. In the three-class model, the setting of the boundaries was based on previous findings: (1) dogs with two or more calcified discs are highly predisposed to develop IDH (Havranek-Balzaretti, 1980) and (2) the number of calcified discs seems to be constant from 2 years of age in dogs with 0–1 calcifications, while occasionally new appearance or more commonly disappearance of individual calcifications occurs in dogs with many (> 4) calcified discs (Jensen and Arnbjerg, 2000). Thus, the degenerative process seems to be in some respect 'more active' in dogs with many calcifications, suggesting that dogs with many calcified discs are at higher risk of IDH. In the five-class scale, a qualitative measure of

clinical significance (location of IDC) is introduced in addition to a more graduated quantitative measure of IDC. In this study, the heritability estimates based on the five-class scale were lower than those based on the three-class scale and score.

The present study has demonstrated a strong relation for offspring–mean parent and offspring–dam in the classification systems used in the linear model (Table 4).

In comparison, Havranek-Balzaretti (1980) showed that the occurrence of IDC is significantly dependent on the status of both parents using the either/or scale: depending on whether none, one, or both parents were affected, 30.4, 56.4 or 83.3 % of the offspring were affected. This relation was confirmed in the present study ($P < 0.001$, cf. Table 3), in which 91 and 44 % of the offspring were affected depending on whether both or one of the parents was affected.

The heritability estimates based on the sire–offspring relationship ranged from 0.46 to 0.87 depending on scale. The higher estimates for the dam–offspring relationship suggest a maternal litter effect on the number of calcified discs, as proposed by Stigen and Christensen (1993). Their results indicated that these factors have a greater impact on the number of calcified discs than the occurrence according to the either/or scale. The present study supported the conclusion that the influence of maternal factors is greater when the absolute scale is used (relative to the three-class and five-class scales; cf. Table 4), indicating that the maternal litter effect influences the number of calcified discs. However, the selected groups of dams and sires were quite different (cf. Figs 2 and 3); in particular, none of the sires had less than two calcified discs.

The sire–offspring relationship is considered to give the best estimates because it is not influenced by environmental litter effects. In the linear model, two heritability estimates based on sire–offspring relationship were statistically significant ($P < 0.05$), namely the estimates based on score or three-class scale. Potentially, paternal environmental factors may occur, but this was probably not relevant in the present study, as none of the sires had been bred in the same kennels as their offspring and only two litters were bred in the kennel in which the sire was living.

The heritability estimates in the present study were much higher than those previously published (Stigen and Christensen, 1993). The reason may be that the occurrence of IDC was evaluated at an older age (i.e. giving better grading) in our study.

Potentially, sampling errors may contribute to variations in heritability estimates. In the present study, two families had to be excluded, one for unknown reasons and the other due to IDH and euthanasia of the sire. Exclusion from breeding or euthanasia due to occurrence of IDH may potentially cause bias leading to a lower prevalence of IDC in the parental generation compared to the population. However, the opposite seems to be the case in the present study with respect to the sires (100 % affected), while the occurrence in the dams (75 % affected) was similar to the population prevalence (77 %) (Jensen and Ersbøll, 2000). Also, the sample prevalence of IDC in the offspring (80 %) was in accordance with the population incidence (77 %), indicating that the sample is not biased in this respect. The spinal distribution of disc calcifications in the progeny showed a high frequency in the thoracic region (72 % of the calcifications), corresponding to the distribution found in a population sample of wirehaired dachshunds (69 % of calcifications in the thoracic region) and the distribution in Norwegian dachshunds reported by Stigen (1991) (60 % in the thoracic region). The proportion of calcifications located in the lumbar region (20 % of calcifications) was higher than expected from the population study (15 % of calcifications) but corresponded to the findings in the Norwegian study (20 % of calcifications in the lumbar region; Stigen, 1991).

In the present study, only obviously calcified discs as evaluated with conventional radiography were included. Other more sensitive methods (e.g. histology or CT imaging) may be used to detect less calcified discs, including some of the borderline cases in the present study. Disc degeneration is a continuous process, with gradual progression of chondroid metamorphosis, dehydration, necrosis and calcification (Hansen, 1952; Ghosh, 1990). The threshold for detection of degenerative changes in the disc depends on the method used. Only the last stage (calcification) is detected with CT and conventional radiography. Degenerative changes in the disc may be detected at an earlier stage with MR imaging, as MR is an effective,

sensitive method for studying water content and degenerative changes in the disc (Haughton, 1988; Levitski et al., 1999). The specificity of MR imaging in differentiating between different types or stages in the degenerative process within the disc remains to be explored; one significant limitation of MR is difficulty in differentiating between dehydration and calcification (Sether et al., 1990; Edelman et al., 1985). Chondroid metamorphosis and degenerative changes supposedly occur in all or nearly all discs throughout the spine, and this hypothesis may be tested by MR imaging. However, the differentiation of the more severe degenerative stage (disc calcification) is an advantage in heritability studies. CT imaging is relatively insensitive to the primary derangement of disc degeneration but is the most sensitive means of demonstrating disc calcification (Modic et al., 1988). If CT were used, the calcifications would be identified at an earlier stage and the peak in the number of calcified discs would probably be obtained at an earlier age. The use of CT instead of conventional radiography corresponds to setting another threshold for detection on a continuous scale. The major disadvantages of CT and MR imaging are the relative unavailability and high costs compared to conventional radiography, which would be a major issue in screening and breeding programmes. The setting of a lower threshold for detection of disc degeneration (with CT or MRI) is particularly relevant for selection in populations with a low prevalence of disc calcification.

As disc degeneration is a continuous and potentially self-perpetuating process (Holm and Urban, 1987), a higher degree of degeneration and IDC could be expected in the parental generation than in the offspring. Stigen (1995) reported that the relative risk of occurrence of IDC (either/or scale) in the parental generation is 1.9 compared to 1-year-old dogs (mean age of 14 months). However, this increase in the number of calcified discs seems to occur before 2 years of age (Jensen and Arnbjerg, 2000). Furthermore, disc calcifications may disappear (e.g. in connection with IDH) and a decline in the number of disc calcifications after 2 years of age has been demonstrated (Jensen and Arnbjerg, 2000). Thus, the number of calcified discs may be expected to be slightly lower in the parental generation than in the offspring, especially in families with many calcified discs. These minor aberrations in the number of calcified discs may have caused a lowering of the heritability estimates.

Like the estimates published by Stigen and Christensen (1993), the heritability estimates presented here are based on the assumption that IDC is a continuous variable. The existence of multiple (dominant) alleles at the locus for hypochondroplasia has been suggested based on variation in shortening (rhizomelia) and bowing of the limbs (Jezyk, 1985). In theory, the phenotypic expression of such alleles may potentially cause varying degrees of disc degeneration because hypochondroplasia is a major factor in chondroid metaplasia. However, variations in leg length are at least in part caused by minor genes, as many genes are involved in growth (Jezyk, 1985). Some variation in leg conformation is seen within breeds, but the dachshund breed is uniform with respect to hypochondroplastic dwarfism. In contrast, the phenotypic expression of disc degeneration and IDC seems to be that of a continuous variable within the dachshund breed. The assumption of a continuous spectrum is based on two findings: dachshunds without IDC are not free from disc degeneration (Hansen, 1952), and dogs affected by IDH and IDC seem to have severe disc degeneration throughout the spine, while dogs without IDH/IDC may have any degree of degeneration, even a few discs without signs of chondroid metaplasia (Havranek-Balzaretti, 1980). The continuous spectrum indicates that the degenerative process is multifactorial, influenced by multiple genetic and environmental factors. Assuming that the gene for hypochondroplasia is homozygotic in all dachshunds and there are not several alleles coding for hypochondroplasia within the breed, the possibility of varying pleiotropic effects of this major gene on the disc can be excluded; under this assumption, disc degeneration can be considered a multifactorial continuous character within the breed. However, the finding of a high heritability is also compatible with the existence of different alleles coding for hypochondroplasia and variable pleiotropic effects on the disc.

In conclusion, the present study has demonstrated a strong relation between the occurrence of IDC in parents and offspring. The heritability estimates based on the sire–offspring relation are in the range of 0.46–0.87 and the estimates based on total number of calcified discs ($h^2 = 0.60$) and three-class scale ($h^2 = 0.87$) were significantly different from zero. The most likely cause of the high heritability estimates compared to those presented in the literature is

the greater age of the offspring, which allows for a more reliable evaluation and rating of the individual dog. As the population prevalence influences the heritability, the high population prevalence of disc calcification is also expected to cause a high heritablity estimate in the present study. The Danish population prevalence of disc calcification was estimated at 77 % (Jensen and Ersbøll, 2000), compared to 52 % in the Norwegian wirehaired dachshund (estimated from Stigen, 1991, 1995).

With a heritability of around 0.6, the occurrence of IDC can be expected to respond well to selection. Estimation of breeding values based on many relatives may be expected to improve selection in the dachshund breed, particularly if this is based on a multiple threshold model with a graduation of the degree of degeneration (according to the number of calcified discs or possibly the three-class scale). At present, IDC is the only relevant parameter for predisposition to IDH, preferably rated by the total number of calcified discs (score). The total number of calcified discs seems to be the best parameter for selection, as this gives the strongest heritability estimate (score). Furthermore, the continuous scale is more likely to be closely related to predisposition. However, a close relation between the number of calcified discs and normally distributed predisposition for IDH (i.e. the severity of disc degeneration throughout the spine) remains to be demonstrated.

On the basis of current knowledge, breeders should choose their breeding animals from families with a low rate of calcified discs. The dogs should be radiographed at 2 years of age in order to select the individuals (and families) with the lowest numbers of calcified discs for breeding.

References

Ball, M. U., J. A. McGuire, S. A. Swam, and B. F. Hoerlein, 1982: Patterns of occurrence of disk disease among registered dachshunds. J. Am. Vet. Med. A **180,** 519–522.

Braund, K. G., P. Ghosh, T. F. K. Taylor, and L. H. Larsen, 1975: Morphological studies of the canine intervertebral disc. The assignment of the beagle to the achondroplastic classification. Res. Vet. Sci. **19,** 167–172.

Edelman R. R., G. M. Shoukimas, D. D. Stark, K. R. Davies, and P. F. J. New, 1985: High-resolution surface-coil imaging of lumbar disc disease. Am. J. Nucl. Rad. **6,** 479–485.

Funkquist, B., and B. Henricson, 1969: Kann die Frequenz von Bandscheibenprolaps beim Dachshund durch eugenische Massnahmen verhindert werden? [Genetical predisposition for disc degeneration in the dachshund]. Kleintier Prax. **14,** 219–223.

Gage, E. D., 1975: Incidence of clinical disc disease in the dog. J. Am. Anim. Hosp. Assoc. **11,** 135–138.

Ghosh, P., 1990: The role of mechanical and genetic factors in degeneration of the disc. J. Manual Med. **5,** 62–65.

Ghosh, P., T. F. K. Taylor, J. M. Yarrol, K. G. Braund, and L. H. Larsen, 1975: Genetic factors in the maturation of the canine disc. Res. Vet. Sci. **19,** 304–311.

Ghosh, P., T. F. K. Taylor, K. G. Braund, and L. H. Larsen, 1976a: A comparative chemical and histochemical study of the chondrodystrophoid and non-chondrodystrophoid canine intervertebral disc. Vet. Path. **13,** 414–427.

Ghosh, P., T. F. K. Taylor, K. G. Braund, and L. H. Larsen, 1976b: The collagenous and non-collagenous protein of the canine intervertebral disk and their variation with age, spinal level and breed. Gerontology **22,** 124–134.

Ghosh, P., T. F. K. Taylor, and K. G. Braund, 1977: The variation of the glycosaminoglycanes of the canine intervertebral disk with ageing. Gerontology **23,** 87–98.

Goggin, J. E., A. S. Li, and C. E. Franti, 1970: Canine intervertebral disc: Characterisation by age, sex, breed and anatomic site of involvement. Am. J. Vet. Res. **31,** 1687–1692.

Hansen, H. J., 1952: A pathological-anatomical study on disc degeneration in the dog. Acta Orthop. Scand. Suppl. **11,** 1–120.

Hansen, H. J., 1966: Pathogenesis of disc degeneration and rupture. In: Pettit, G. D. (ed.), Intervertebral Disc Protrusion in the Dog, pp. 21–50. Appleton-Century-Crofts, New York.

Haughton, V. M., 1988: MR imaging of the spine. Radiology **166,** 297–301.

Havranek-Balzaretti, B., 1980: Beitrag zur Aetiologie der Dackellähme und Vorschlag zur züchterischen Selektion. Inaugural Dissertation, University of Zürich.

Holm, S. H., and J. P. G. Urban, 1987: The intervertebral disc: Factors contributing to its nutrition and matrix turnover. In: Helminen, H. J., I. Kiviranta, M. Tammi, A.-M. Säämänen, K. Paukkonen, and J. Jurvelin (eds), Joint Loading. Biology and Health of Articular Structures, pp. 187–226. Wright (Butterworth Ltd), Bristol, UK.

Jensen, V. J., and J. Arnbjerg, 2000: Development of intervertebral disc calcification in the dachshund: A prospective longitudinal radiographic study. J. Am. Anim. Hosp. Assoc., in press.

Jensen, V. J., and A. K. Ersbøll, 2000: Mechanical factors affecting the occurrence of intervertebral disc calcification in the dachshund. A population study. J. Vet. Med. A, **47,** in press.

Jezyk, P. F., 1985: Constitutional disorders in the dog and cat. In: Newton, C. H. D., and D. M. Nunamaker (eds), Textbook of Small Animal Orthopaedics, pp. 637–655. J. B. Lippencott Company, Philadelphia, PA.

Levitski, R. E., D. Lipsitz, and A. E. Chauvet, 1999: Magnetic resonance imaging of the cervical spine in 27 dogs. Vet. Radiol. Ultrasound **40,** 332–341.

Modic, M. T., T. J. Masaryk, J. S. Ross, and J. R. Carter, 1988: Imaging of degenerative disc disease. Radiology **168,** 177–186.

Priester, W. A., 1976: Canine intervertebral disc disease – Occurrence by age, breed, and sex among 8117 cases. Theriogenology **6,** 293–301.

Rimoin, D. L., G. N. Hughes, R. L. Kaufman, R. E. Rosenthal, W. H. McAlister, and R. Silberberg, 1970: Endochondral ossification in achondroplastic dwarfism. N Eng. J. Med. **283,** 728–735.

Sether, L. A., C. Nguyen, S. Yu, V. M. Haughton, K.-C. Ho, D. S. Biller, J. A. Strandt, and J. C. Eurel, 1990: Canine intervertebral disks: Correlation of anatomy and MR imaging. Radiol. **175,** 207–211.

Simpson, T. S., 1992: Intervertebral disc disease. Vet. Clin. North Am. **22,** 889–897.

Stigen, Ø., 1991: Calcification of intervertebral discs in the dachshund. A radiographic study of 327 young dogs. Acta Vet. Scand. **32,** 197–203.

Stigen, Ø., 1995: Calcification of intervertebral discs in the dachshund: a radiographic study of 21 stud-dogs. Acta Vet. Scand. **36,** 329–334.

Stigen, Ø., 1996: Calcification of intervertebral discs in the dachshund: a radiographic study of 115 dogs at 1 and 5 years of age. Acta Vet. Scand. **37,** 229–237.

Stigen, Ø., and K. A. Christensen, 1993: Calcification of intervertebral discs in the dachshund: an estimation of heritability. Acta Vet. Scand. **34,** 357–361.

Verheijen, J., and J. Bouw, 1982: Canine intervertebral disc disease: a review of etiologic and predisposing factors. Vet. Q. **4,** 125–133.

Webster, M. K., and D. J. Donoghue, 1997: FGFR activation in skeletal disorders: too much of a good thing. Trends Gen. **13,** 178–182.