

Controlling canine hip dysplasia in Finland

Minna Leppänen^{*}, Hannu Saloniemi

*Faculty of Veterinary Medicine, Department of Clinical Veterinary Science/Animal Hygiene,
University of Helsinki, PL 57, 00140 Helsinki, Yliopisto, Finland*

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Abstract

The aim of the study was to evaluate the Finnish Kennel Club's hip-dysplasia screening and control programs. As a retrospective study, records of hip-dysplasia screening of 69,349 dogs in 22 breeds that were born in 1988–1995 were analyzed and compared to data from prior to 1988. In most breeds, no significant changes in dysplasia prevalence could be found. In English cocker spaniels, golden and Labrador retrievers and Rottweilers a significant decrease – but in boxers, Dobermans, German Shepherd dogs and rough collies a significant increase – in prevalence was detected. In flat-coated retrievers overall prevalence increased – but the prevalence of severe hip-dysplasia decreased significantly during the study period.

The present control program has not resulted in fast progress. Selecting against hip-dysplasia cannot be expected to be very effective, when based only on mass selection on phenotypic observations. Predicted breeding values based on progeny testing would probably give better results. Also, breeders' compliance and commitment to programs is not always high and other selection criteria in breeding are thought to be more important.

Modern society has high demands for animal welfare and consumer issues, and breeders and kennel societies should pay more attention to health issues in breeding pedigreed dogs. © 1999 Elsevier Science B.V. All rights reserved.

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1. Introduction

Canine hip-dysplasia was reported over 60 years ago (Schnelle, 1935). Research on hip-dysplasia has been ongoing ever since and there is now wide consensus that hip-dysplasia

^{*} Corresponding author. Tel.: +358-9-70851; fax: +358-9-27476450
E-mail address: minna.leppanen@helsinki.fi (M. Leppänen)

is a quantitatively inherited trait. Variation in heritability estimates between breeds and in different studies has been discovered; reported heritabilities vary from 0.17 to 0.6 (Fisher, 1979; Lingaas and Klemetsdaal, 1990; Swenson et al., 1997; Tomlinson and McLaughlin, 1996). Environmental factors and their role in the development and severity of hip-dysplasia have also been widely researched (Aichinger, 1997; Brass, 1989; Schwalder et al., 1996; Swenson et al., 1997; Tomlinson and McLaughlin, 1996).

There are different opinions of the clinical importance of hip-dysplasia and possibilities of predicting the development of clinical signs from radiographs. Joint laxity is important in predicting hip-dysplasia and degenerative joint disease. Susceptibility for degenerative changes has important between-breed variation (Lust, 1997; Popovitch et al., 1995; Smith, 1997). Hip-dysplasia is commonly considered to cause important disability and to decrease ability to work. However, a recent study of military working dogs showed no significant decrease in working ability or time in dogs suffering from hip-dysplasia (Banfield et al., 1996).

Many countries and kennel organizations have established control programs for hip-dysplasia. All the present official programs are based on radiographic evaluation of the phenotype (Brass, 1989; Corley, 1992; Flückiger, 1994; Anon., 1994). Schnelle (1954) proposed the first classifying system in 1954; currently, there are many different systems in use. In Finland the Federation Cynologue Internationale's (FCI) five-scaled system is used (Appendix A).

The first official hip-dysplasia-control program in Finland was started for German Shepherds in 1963. Then, negative hip-dysplasia radiographs were required before a dog could gain a champion's title (Paatsama, 1978, 1979). The first of the currently used control programs were established in 1984, when breeding animals of retriever breeds were required to undergo official hip screening as a prerequisite for litter registration. Currently, 71 breeds have an official control program for hip-dysplasia under the Finnish Kennel Club and about 7500 radiographs are screened annually in this scheme (Liman, A., personal communication). For some breeds in the program, only screening for hip-dysplasia is mandatory – but for the others, there are threshold values for hip-dysplasia for breeding stock.

Control programs and their effectiveness in reducing hip-dysplasia have shown variable results. Flückiger et al. (1995) found in about 3700 dogs Swiss an overall dysplasia prevalence of 42%; however, significant between-breed variation was observed. In some breeds, prevalence decreased clearly – but in others, it increased during the study period. In most breeds, however, a decrease in severity of dysplasia was noticed. In that study also, some correlation was observed between breeding restrictions and the decrease of dysplasia prevalence: the milder the restrictions, the slower the decrease in prevalence (Flückiger et al., 1995). Also, English and American studies have found remarkable variation in prevalence and changes in different breeds (Corley, 1992; Willis, 1997). The best progress was found in Sweden, where disease prevalence decreased during the study period in all breeds investigated (Swenson et al., 1997).

Hip-dysplasia control programs have been widely discussed since they have been in use. The slow progress in reducing the disease has been especially criticized; this has been stated to show futility of the programs and to justify giving them up. On the other hand, slow progress in decreasing dysplasia prevalence has also been considered a

justification for more-advanced measures and stronger restrictions on breeding animals. There has also been a lot of debate about the costs of the program to breeders and dog-owners. Hip-dysplasia screening and restrictive programs are also stated to cause a decrease in genetic variance (because of single-trait selection), which can lead to other more-severe problems (Bouw, 1982).

The objectives of this study were to evaluate the effectiveness of the Finnish hip-dysplasia-control program and changes in disease prevalence. The final aim of this study was to discuss the reasons for the effectiveness or futility of the program.

2. Materials and methods

2.1. *The data*

This study included hip-dysplasia records for 64 349 dogs in 22 breeds (Table 1). Nineteen of these breeds have had an official control program since 1993 and the number of registered puppies in each breed has exceeded 700 in the years 1993–1997. Rottweilers were included in the study; although they only joined the program in 1994, the breed club has campaigned strongly for voluntary screening for a long time before this. Saint Bernards were included as a model of a breed that is commonly thought to have a bad hip-dysplasia situation and for which the breeders commonly oppose the breeding restrictions. Although there were not >700 registered short-haired Saint Bernards during the study period, they were included, because in Finland the pedigree is open between both coat-types, and thus breeders can freely mate short- and long-haired dogs, although matings typically are between similar coat types. Also, in the same litter there can be dogs of both coat variations that are registered accordingly. Nine breeds in the study have established breeding restrictions, that only dogs whose hips are screened as better than the breed's threshold value are accepted for breeding and have their offspring registered in the Finnish Kennel Club. Because the German pointer became a restricted breed only on January 1, 1996, it is considered a non-restricted breed in the study.

All official hip-dysplasia radiographs are judged by panelists that have the authorization of the Finnish Kennel Club. There have been five panelists altogether. Until 1988, one panelist screened all the radiographs. In 1988–1995 three new panelists were appointed; since 1996, the fifth panelist has screened all radiographs. Each panelist had screened his share of radiographs individually.

This study consisted of the records of dogs that were born in 1988–1995 and were screened before January 1, 1997. The material was compared to the Kennel Club's dysplasia data that were recorded before 1988. To minimize the effects of annual random variation, breed-specific dysplasia prevalence was calculated from all dogs that were born in 1988–1995 and this was compared to the breed's prevalence in dogs that were screened before 1988. However, this material was available only divided according to the breed and screening year, so a direct comparison between years of birth could not be made. Also, the classification varied slightly (Appendix A). In some breeds, the number of screened dogs before 1988 was also very small (which in part makes comparisons difficult). The data do not allow any distinction according to the sex or dogs that were re-examined; neither was the age at examination included.

Table 1
Description of programs and of records available

Breed	Year		Threshold value	Total registrations from 1988 to 1995	% of registrations screened	Number of screened before 1988
	Joined the program	Restrictions begun				
Bearded collie	1989	1989	C	1550	22	545
Boxer	1988	1994	C	2798	40	1844
Bullmastiff	1989	1989	D	1525	34	243
Dalmatian	1991	NO	NO	1694	24	55
Doberman	1986	1993	C	3874	31	1025
English cocker spaniel	1990	NO	NO	5943	18	307
English springer spaniel	1990	NO	NO	2833	22	364
Flat-coated retriever	1984	1991	C	1418	64	426
German Shepherd dog	1986	1989	C	23 385	36	14 189
Golden retriever	1984	1991	C	16 414	32	3847
Irish setter	1986	NO	NO	1462	29	99
Labrador retriever	1984	1991	C	13 327	36	5682
Longhaired Saint Bernard	1992	NO	NO	3307	23	674
Nova Scotia duck tolling retriever	1984	1991	C	979	45	11
Rottweiler	1994	NO	NO	6672	17	1844
Rough collie	1986	NO	NO	10 708	26	1457
Samoyed	1988	NO	NO	1979	36	980
Shorthaired German pointer	1989	1996	C	1725	26	153
Shorthaired Saint Bernhard	1992	NO	NO	253	42	3
Smooth collie	1986	NO	NO	857	32	9
Welsh springer spaniel	1990	NO	NO	1130	31	233
Wirehaired German pointer	1989	1996	C	1977	22	227
Total				105 810		34 217

Threshold value = the most-severe grade of dysplasia allowed for breeding.
NO = no restrictions or threshold value, only obligatory screening before breeding.

2.2. Statistical analysis

The $-\chi^2$ test (Statistix Version 4.1TM, Analytical Software, Tallahassee, FL, USA) was used in analyzing the heterogeneity between dogs screened before 1988 and dogs born in 1988–1995 and screened before 1997. Fisher's 2-tailed exact test with alpha 0.05 was used for comparisons between the breeds with breeding restrictions and no restrictions, as well as between the breeds that established breeding restrictions in 1991 or earlier, or the breeds that had restrictions since 1993.

3. Results

3.1. Disease prevalence

The breed-specific proportions of registered dogs that were screened varied from 18% (English cockerspaniel) to 64% (flat-coated retriever). Breeds varied greatly in dysplasia prevalence; it varied from 2% (smooth collie) to 80% (long-haired Saint Bernard) (Table 2). In every breed, annual variation in prevalence was also noticed. Figs. 1–3 show dysplasia prevalences according to the year of the birth in those breeds with significant changes. The figures for the year 1987 present the prevalences in all dogs of a breed that

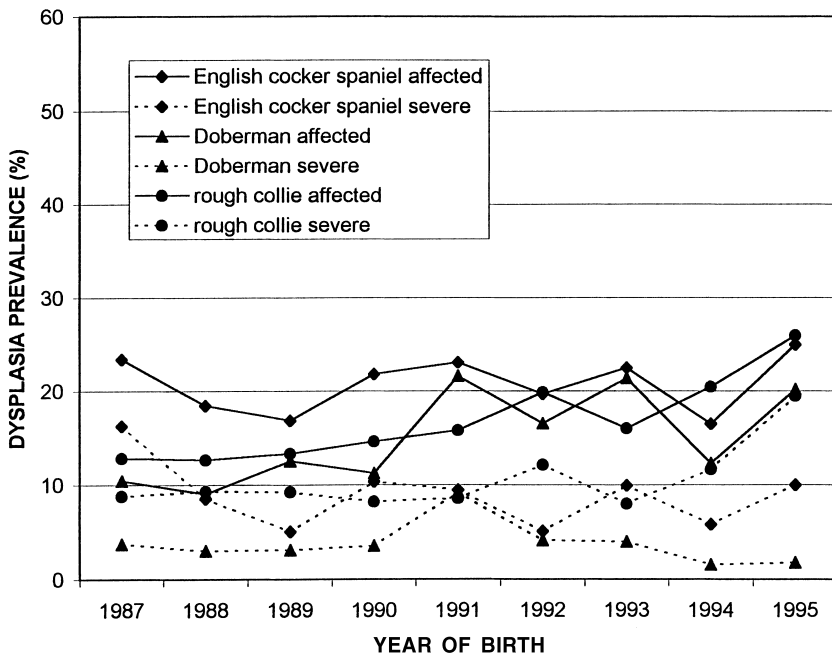


Fig. 1. Overall hip-dysplasia prevalence (straight line) and prevalence of severe dysplasia (broken line) according to the year of birth in English cocker spaniels, Dobermans and rough collies. Year 1987 represents the prevalence of all the animals in the breed screened before January 1, 1988.

Table 2
Dysplasia prevalences (%) within the study breeds

Breed	Dysplasia prevalence %			Prevalence % of severe dysplasia ^a		
	Before 1988	In 1988–1995	χ^2 (df=1) <i>p</i>	Before 1988	1988–1995	χ^2 (df=1) <i>p</i>
Bearded collie	22	24	0.69	11	12	0.39
Boxer	28	40	0.00	18	16	0.20
Bullmastiff	45	53	0.04	26	29	0.72
Dalmatian	16	20	0.52	5	4	0.59
Doberman	10	16	0.00	4	4	0.66
English cocker spaniel	23	20	0.21	16	8	0.00
English springer spaniel	28	29	0.58	14	14	0.95
Flat-coated retriever	16	20	0.00	7	4	0.03
German Shepherd dog	33	46	0.00	18	21	0.00
Golden retriever	41	36	0.00	21	18	0.00
Irish setter	33	40	0.02	14	19	0.49
Labrador retriever	32	29	0.00	22	17	0.00
Longhaired Saint Bernard	79	82	0.24	70	67	0.15
Nova Scotia duck tolling retriever	0	25	0.06	0	14	0.13
Rottweiler	45	38	0.00	31	20	0.00
Rough collie	13	16	0.00	9	10	0.00
Samoyed	36	34	0.11	23	19	0.06
Shorthaired German pointer	16	7	0.90	1	1	0.63
Shorthaired Saint Bernard	67	59	0.80	33	42	0.78
Smooth collie	11	2	0.06	0	0	
Welsh springer spaniel	41	35	0.19	20	17	0.29
Wirehaired German pointer	16	16	0.21	5	5	0.97

^a Severe canine hip dysplasia = grades D or E or >3.

were screened and that were born before 1988. No significant difference was found between the breeds with or without breeding restrictions ($p=0.20$), or between those breeds that started restrictions earlier and those that established restrictions recently ($p=1.00$).

4. Discussion

In Finland, approximately 95% of pedigreed dogs are registered with the Finnish Kennel Club. Within-breed proportions of dysplasia-screened dogs are quite stable every year, and in most cases the radiographs are submitted to the screening despite a bad preliminary evaluation. However, the presented frequencies of screened animals are only

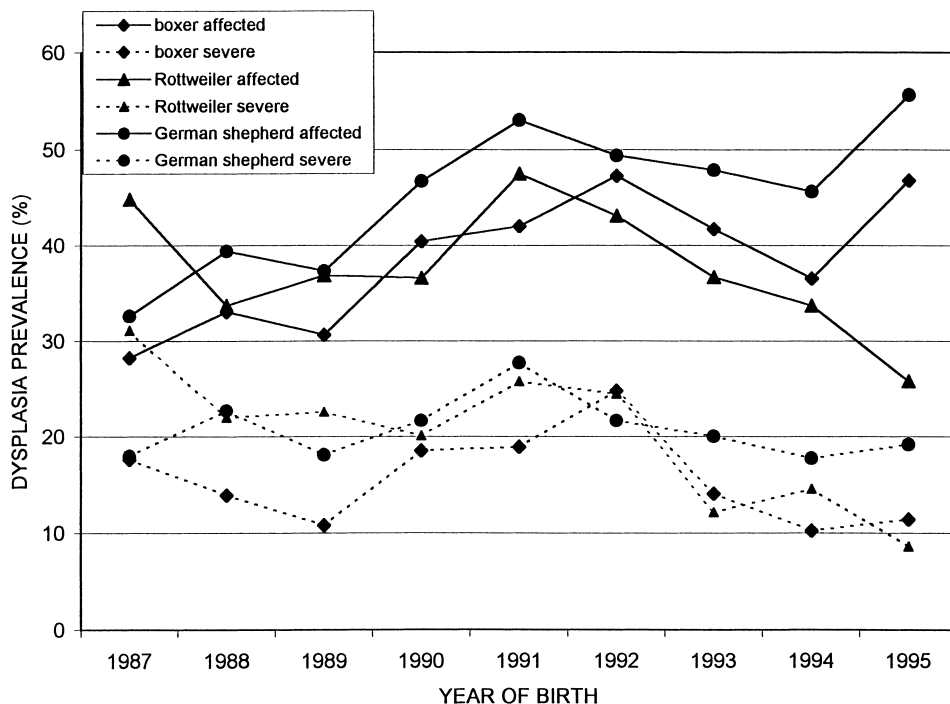


Fig. 2. Overall hip-dysplasia prevalence (straight line) and prevalence of severe dysplasia (broken line) according to the year of birth in boxers, Rottweilers and German Shepherds. Year 1987 represents the prevalence of all the animals in the breed screened before January 1, 1988.

estimations, because all dogs are not registered in the same year as they are born. At least all breeding animals – as well as a reasonable number of pet animals – in control-program breeds are screened. Thus the hip-dysplasia-control program gives a quite good overall picture of a breed's hip-dysplasia situation. Also, because almost all pedigree dogs are registered, registration restrictions for offspring of dysplastic parents are an effective measure that prevents breeding with affected animals. Slight bias might be caused by dogs that have been screened more than once (because then one dog has multiple results in the data).

In nine breeds included in this study – (boxer, Doberman, English cocker spaniel, flat-coated, golden and Labrador retriever, German Shepherd dog, rough collie and Rottweiler) significant changes in hip-dysplasia prevalence were detected. However in four breeds – boxer, Doberman, German Shepherd dog and rough collie – the prevalence increased. Our study could not support the previous studies which found significant overall decrease in disease prevalence (Brass, 1989; Swenson et al., 1997); instead, our findings support those of the studies that found distinct between-breed variation (Flückiger et al., 1995; Willis, 1997). The effectiveness of strong official breeding

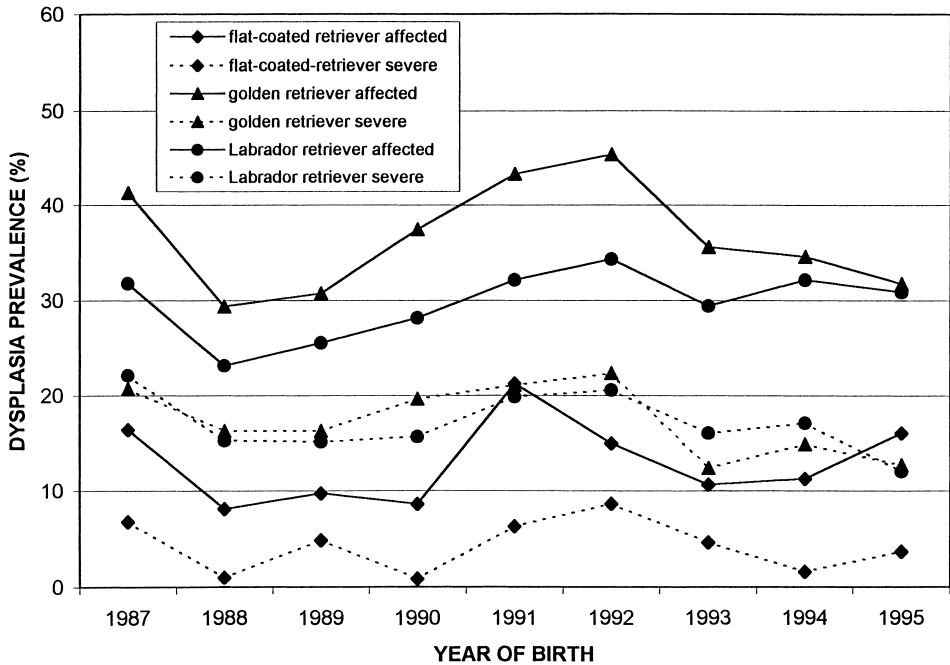


Fig. 3. Overall hip-dysplasia prevalence (straight line) and prevalence of severe dysplasia (broken line) according to the year of birth in flat-coated, golden and Labrador retrievers. Year 1987 represents the prevalence of all the animals in the breed screened before January 1, 1988.

restrictions is also questionable: German shepherds have had restriction the longest time and showed significant increase in dysplasia prevalence – but Rottweilers joined the program only recently and prevalence had decreased already before joining the scheme. Also, we could not show significant differences in effectiveness between the breeds with or without breeding restrictions. In another study (Leppänen et al., 1999), however, we reported that the number of German shepherd parent animals graded worse than excellent (B or C) had actually increased during the study period – which gives reason to doubt that the selection has been strong enough. Our current data set does not give information of parents' hip-dysplasia statuses, but at least the parents of all the dogs born after the breed has joined the program had to have been screened. Also, for most breeds involved, it was already a common procedure to screen at least breeding stock before the official program was begun – so it is justified to suppose that most animals in this study have screened parents. This conclusion is also supported by findings in the Swedish data (Swenson et al., 1997).

There are several possible reasons for detected minimal improvement in many breeds. It can be assumed that before joining the program, radiographs with dysplastic changes detected by veterinarians who took the radiographs were not forwarded for the official screening – and yet the parents still could have been used for

breeding – which may have biased earlier prevalences to look better than the real situation. Each breed club's own code of ethics as well as the authority a breed club had amongst the breeders will have had an influence on breeders and their actions. Also, various breeds have other selection considerations concerning health, conformation or performance abilities (which might dilute the effort to select against hip-dysplasia). Finland removed previous strict quarantine regulations for imported dogs in 1988. In many breeds (especially in those of German origin), this had led to a marked increase in the numbers of imported breeding dogs whose hip-dysplasia background is not known.

Willis (1997) found a smaller observed decrease in dysplasia prevalence than was expected, and suggested that heritability might be lower than estimated. Overestimation of heritability could have been caused by assortative mating or strong in-breeding. The heritabilities in that study were estimated with parental half-sib analysis. This type of error can be avoided with more-advanced methods for heritability estimation such as the restricted maximum-likelihood (REML) method (Patterson and Thomson, 1971; Groeneveld, 1997). Slow progress can also be explained by the fact that when selecting against a quantitative trait with moderate heritability – like hip-dysplasia – mass selection does not allow fast progress (Hutt, 1967). Advanced use of progeny results and individual breeding indices would give better reliability of selection criteria (Leighton, 1997; Lingaas and Klemetsdaal, 1990; Willis, 1997).

Because the present screening system is based on subjective classification of radiographic findings, panelist-dependent variation also could be possible. Studies have found significant within- and between- examiner variation in classifications (Smith et al., 1997; Stur et al., 1996). When dealing with large populations, good correlation of dysplasia results and classification variables between the examiners was, however, found (Stur et al., 1996).

Lately, there has been a steady increase in discussion and demands for advanced animal welfare (Anon., 1995, 1996). Discussions of pets' role in modern society and their meaning to human health has increased (Allen, 1997; Beaver, 1997; Glickman, 1991). Pets are often seen more as a family member than an also discussions animal. Thus, dog breeders and kennel societies have created health programs although direct economic benefits could not be shown. Also, Finnish consumer legislation treats animals as any other article and thus a producer – that is a breeder – is held responsible to compensate pet owners for possible production errors (such as inherited diseases). In spite of this, demonstrable progress in reducing the prevalence of canine hip-dysplasia is scarce in most breeds.

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Appendix A

Classification of hip-dysplasia

Present	Past	Interpretation
A	Normal	No signs of dysplasia
B	Borderline	Healthy, with slight changes in conformation
C	1	Mild dysplasia
D	2	Moderate dysplasia
E	3–4	Severe dysplasia

References

- Aichinger, O., 1997. Rassenrein aber krank – Hilfe bei Hüftgelenkdysplasie. *Tierärztliche-Umschau* 52(3), 153–154, 156.
- Allen, D.A., 1997. Effects of dogs on human health. *J. Am. Vet. Med. Assoc.* 210(8), 1136–1139.
- Anon., 1994. Joint WSAVA/ Kennel Clubs Meeting to discuss the control of hip-dysplasia in pedigree dogs. June 17, 1994, London.
- Anon., 1995. European convention for the protection of pet animals. Resolution on breeding in pet animals. Council of Europe.
- Anon., 1996. Animal welfare act 247/96. Finnish law.
- Banfield, C.M., Bartels, J.E., Hudson, J.A., Wright, J.C., Montgomery, R.D., Hathcock, J.T., 1996. A retrospective study of canine hip-dysplasia in 116 military working dogs. Part II: Clinical signs and performance data. *J. Am. Anim. Hosp. Assoc.* 32(5), 423–430.
- Beaver, B., 1997. Human-canine interactions: a summary of perspectives. *J. Am. Vet. Med. Assoc.* 210(8), 1148–1150.
- Bouw, J., 1982. Hip-dysplasia and dog breeding. *Vet. Quarterly* 4(4), 173–181.
- Brass, W., 1989. Hip-dysplasia in dogs. *J. Small Anim. Pract.* 30, 166–170.
- Corley, E.A., 1992. Role of the orthopedic foundation for animals in the control of canine hip-dysplasia. *Vet. Clin. N. Am.: Small Anim. Pract.* 22(3), 379–593.
- Fisher, T.M., 1979. The inheritance of canine hip-dysplasia. *Mod. Vet. Prac.* 60(11), 897–900.
- Flückiger, M., 1994. The standardized analysis of radiographs for hip-dysplasia in dogs. Objectifying a subjective process. *Eur. J. Comp. Anim. Pract.* V(2), 39–44.
- Flückiger, M., Lang, J., Binder, H., Busato, A., Boos, J., 1995. Die Bekämpfung der Hüftgelenkdysplasie in der Schweiz: Ein Rückblick auf die vergangenen 24 Jahre. *Schw. Arch. für Tierheilk.* 137(6), 243–250.
- Glickman, L.T., 1991. Implications of the human/animal bond for human health and veterinary practice. *J. Am. Vet. Med. Assoc.* 201(6), 848–851.
- Groeneveld, E., 1997. REML VCE – a multivariate multimodel restricted maximum likelihood (co)variance component estimation package. Version User's guide. Inst. Of Anim Husbandry and Anim. Ethology, Feder. Res. Center of Agric., Germany (Mimeo).
- Hutt, F.B., 1967. Genetic selection to reduce the incidence of hip-dysplasia in dogs. *J. Am. Vet. Med. Assoc.* 151, 1041–1048.
- Leighton, E.A., 1997. Genetics of canine hip-dysplasia. *J. Am. Vet. Med. Assoc.* 210(10), 1474–1479.
- Leppänen, M., Mäki, K., Juga, J., Saloniemi, H., 1999. The factors affecting hip-dysplasia in German Shepherds, prediction of breeding values and genetic change in breeding programmes. In press.
- Lingaas, F., Klemetsdaal, G., 1990. Breeding values and genetic trend for hip-dysplasia in the Norwegian Golden retriever population. *J. Anim. Breed. Genet.* 107, 437–443.
- Lust, G., 1997. An overview of the pathogenesis of canine hip-dysplasia. *J. Am. Vet. Med. Assoc.* 210(10), 1443–1445.

- Paatsama, S., 1978. Koiran lonkkanivelen kasvuhäiriön vastustaminen (Breeding against canine hip-dysplasia). Koiramme no. 6–7, 1–4.
- Paatsama, S., 1979. Hip-dysplasia in dogs: a controlled restrictive breeding programme in Finland. Twenty First World Veterinary Congress, Summaries, vol. 4. Sect. IX, pp. 42–43.
- Patterson, H.D., Thomson, R., 1971. Recovery of interblock information when block sizes are unequal. *Biometrika* 58, 427.
- Popovitch, C.A., Smith, G.K., Gregor, T.P., Shofer, F.S., 1995. Comparison of susceptibility for hip dysplasia between Rottweilers and German Shephard Dogs. *J. Am. Vet. Med. Assoc.* 206(5), 640–648.
- Schnelle, G.B., 1935. Some new diseases in dog. *American Kennel Gazette* 52, 25–26.
- Schnelle, G.B., 1954. Congenital dysplasia of the hip (canine) and sequalea. AVMA. Proceedings of the Annual meeting. Seattle, WA, pp. 253–258.
- Schwalder, P., Spreng, D., Dietschi, E., Dolf, G., Gaillard, C., 1996. Die Hüftgelenkdysplasie im Umfeld von sekundären Einflüssen und ektopischen Ursachen. *Kleintierpraxis*. 41(9), 625–638.
- Smith, G., 1997. Advances in diagnosing canine hip-dysplasia. *J. Am. Vet. Med. Assoc.* 210(10), 1451–1457.
- Smith, G.K., LaFond, E., Gregor, T., Lawler, D., Nie, R., 1997. Within- and between-examiner repeatability of distraction indices of the hip joints in dogs. *Am. J. Vet. Res.* 58(10), 1076–1077.
- Stur, I., Koppel, E., Schroder, K., 1996. Populationsgenetische Aspekte der Hüftgelenkdysplasie (HD) – diagnostik bei Hund – Bewertung unter Berücksichtigung differierender HD-Befunde. *Wien. Tierärztl. Mschr.* 83, 91–97.
- Swenson, L., Audell, L., Hedhammar, Å., 1997. Prevalence, inheritance and selection for hip-dysplasia in seven breeds of dogs in Sweden and a cost/benefit analysis of a screening and control program. *J. Am. Vet. Med. Assoc.* 210(2), 207–214.
- Tomlinson, J., McLaughlin, R., 1996. Canine hip-dysplasia: developmental factors, clinical signs, and initial examination steps. *Vet. Med.* 91(1), 26–33.
- Willis, M.B., 1997. A review of the progress in canine hip-dysplasia control in Britain. *J. Am. Vet. Med. Assoc.* 210(10), 1480–1482.