Vet. Pathol. *12:* 239–263 (1975)

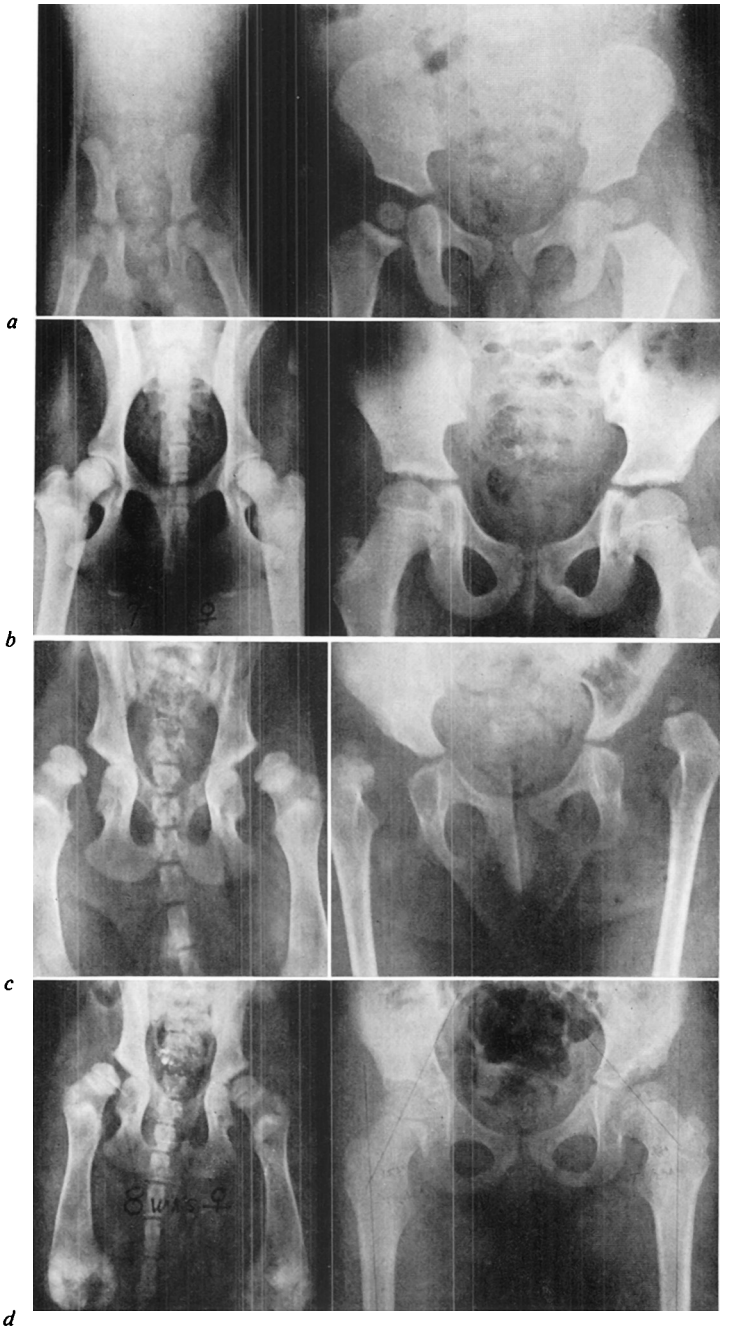
Observations and Research on Hip Dysplasia

Volumes have been written on hip dysplasia in the child, a comparable amount about the dog, and reports of the disease in other mammals are frequent. The literature is too extensive for a general review here. Only the pertinent observations and experiences will be summarized. The disease as it affects man and the dog will be compared where relevant from the viewpoint of prevalence and the genetic, environmental, and metabolic influences. The similarity of the radiographic appearance of both the normal and dysplastic hip joints of man and the dog is striking (fig. 1).

Prevalence

In the human infant in New York there is a frequency of hip dysplasia of 1.3 per 1000 births. This does not include those additional newborns with the Ortolani click as the only sign of hip dysplasia. An untreated population of Navajo Indians at Many Farms, Ariz., was estimated to have a prevalence of 10.9 hip dysplasias per 1000 births. In this Indian population it was highly significant that the occurrence of hip dysplasia decreased when the cradleboard was discarded and the children were diapered [44]. In Bantu tribes, hip dysplasia is relatively unknown [47].

Hip dysplasia has been observed in most breeds of dogs; however, it is a greater problem in some breeds [42]. Which breeds of dogs have the highest prevalence, and why is the disease more prevalent in some breeds? The true prevalence of hip dysplasia among breeds of purebred dogs is not known, but data from the Orthopedic Foundation for Animals on the first 36000 pelvic radiographs evaluated have given insight into answering these questions.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Differences in the genetic composition of the dog are important in predisposing its shape, size, and general characteristics [53, 56]. The genetic code also controls the metabolic life and, to a great extent, the diseases a dog will have, especially the clinical and metabolic variations that are based on inherited traits. This is not a new concept. In man and dogs, it is well known that there are exaggerated deviations in types and constitutions [48, 49, 53]. These body types influence skeletal, physical, and behavioral characteristics to a noticeable extent. These body types or somatotypes have been classified into three general categories: the ectomorph – light type of body build; the mesomorph – intermediate or average body build (athletic), and the endomorph – heavy type of body build (short, stocky, rounded). In dogs as well as in man, all breeds and individuals exhibit a mixture of variation of specific somatotypes but, within limits, the general classification can be characterized and distinguished [24, 53].

In our early work with canine hip dysplasia, it was discovered in some breeds (July Foxhounds) that almost all were dysplastic, whereas in other breeds (Greyhounds) hip dysplasia was rare. Between these extremes the prevalence of hip dysplasia varied [41]. There was also a strong correlation between body form, size, growth rate, quantity of subcutaneous fat, type of connective tissue, pelvic muscle mass, and the general body type of the different breeds and the prevalence of hip dysplasia [24, 45, 46, 53].

Recently we have identified certain general characteristics of a breed that increase the risk of hip dysplasia [42]. Such correlation was also suggested previously [24]. Certain somatotypes are associated with a high prevalence of hip dysplasia in the dog.

The data used were tabulated from the first 36 000 radiographs received by the Orthopedic Foundation for Animals, which serves as a registry and

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Fig. 1. a Pelvic radiograph of a 4-week-old Greyhound (left) and a 2-year-old child (right). Both have normal hips. *b* Pelvic radiograph of a 7-month-old Greyhound (left) and a 10-year-old child (right). Both have normal hips. *c* Pelvic radiograph of an 8-week-old German Shepherd (left) and a 2⅔-year-old child (right). The hip joints of both are subluxated and dysplastic. *d* Pelvic radiograph of an 8-week-old German Shepherd (left) and a 5-year-old child (right). Both have dysplastic hips. In the dog there is subluxation of the femoral heads and retarded development of the acetabulums. The hip joints are unstable and characteristic of early, severe hip dysplasia. In the child, there is subluxation of the left femoral head and a lack of development of the acetabulum. This is characteristic of hip dysplasia in children.

diagnostic service to identify normal pelvic phenotypes with respect to hip dysplasia in dogs 1 year of age or older. Dog owners from all 50 states and the provinces of Canada have participated in the program, with more than 90 breeds represented. Each radiograph submitted was reviewed independently by at least three diplomates of the American College of Veterinary Radiology. By consensus, each radiograph was classified in one of four categories: normal, near-normal, dysplastic, or repeat study advised. Only breeds represented by more than 100 individual pelvic radiographs were included in the analysis (table I).

The percentage of hip dysplasia is not a true representation of the prevalence of the disease in these breeds because radiographs depicting obvious dysplasia were screened by referring veterinarians and not submitted. Therefore, the overall prevalence of hip dysplasia is higher than that represented in the analysis (table I). The rankings of the breeds are consistent with those obtained previously from smaller populations [24]. All breeds were screened in a similar manner.

The rankings of the 38 breeds represented were based on tabulation of 35 285 radiographs in breeds with more than 100 individual pelvic radiographs (137–5351 for each breed; table I). The prevalence of hip dysplasia varied from 4.2 to 43.2%, a spread of almost 40% [42]. Neither the prevalence of hip dysplasia nor the rank of the breeds changed appreciably from previous studies when tabulations were published for the first 5000 radiographs [24, 42].

Why dysplasia was more prevalent in some breeds was difficult to assess, but information was obtained by comparing a group of breeds having the highest percentage of hip dysplasia with a group having the lowest percentage. The two groups were compared for body size, body type, and growth pattern (table II).

Fifteen breeds, nine with a low percentage and six with a high percentage of dysplasia, which represented the breeds at the ends of the binominal curve (outside ± 1 SD), were studied as two groups. The mean percentage of dysplasia was 19.2%, and one standard deviation was ± 9.43%. Statistically, 68.26% of the population (representing 23 breeds) fell within ± 1 standard deviation.

The two groups varied widely in appearance and body type but resembled each other in the three categories studied. In the high percentage group, the three breeds at the top and the one that ranked sixth were of the giant type, with various characteristics of acromegalia. The two hunting breeds in this group were long-legged and rangy but had a low muscle mass index, which gave them a high probability toward hip dysplasia. The nine breeds with the

Table I. Pelvic radiographic diagnoses[1]

Breed	Evaluations	Dysplastic %	Normal %	Near normal %	Repeat study %
Saint Bernard	1453	43.2	49.5	2.6	4.7
Newfoundland	338	39.9	50.9	3.2	5.9
Bullmastiff	278	34.5	60.8	1.1	3.6
English Setter	327	32.1	59.0	2.4	6.4
Gordon Setter	218	31.2	62.4	0.4	6.0
Old English Sheepdog	2311	29.9	65.1	1.0	3.9
English Springer Spaniel	611	27.3	67.8	1.8	3.1
Akita	251	27.1	68.5	0.0	4.4
Chesapeake Bay Retriever	300	25.7	68.7	2.0	3.7
Golden Retriever	3028	25.7	68.6	2.1	3.6
Norwegian Elkhound	660	25.6	69.8	0.4	4.1
Rottweiler	660	25.4	70.2	0.4	3.9
German Shepherd Dog	5351	25.1	67.5	2.9	4.5
Giant Schnauzer	215	24.2	70.2	2.3	3.2
Standard Poodle	772	23.6	73.7	0.6	2.1
Brittany Spaniel	824	22.4	74.0	0.8	2.7
Rhodesian Ridgeback	317	19.6	77.0	0.0	3.5
Alaskan Malamute	1121	18.5	77.5	0.5	3.5
Weimaraner	995	18.0	78.7	1.2	2.1
Irish Setter	1614	16.9	79.7	0.7	2.7
Samoyed	1974	15.7	80.7	0.9	2.7
Labrador Retriever	4089	15.5	80.7	1.3	2.5
Vizsla	531	15.2	82.1	1.1	1.5
Great Dane	1325	15.2	81.4	0.2	3.2
Keeshond	244	15.2	82.0	0.8	2.0
Puli	175	13.1	85.1	0.0	1.7
Great Pyrenees	336	12.8	85.7	0.0	1.5
Standard Schnauzer	374	12.6	85.6	0.5	1.3
Australian Shepherd Dog	161	11.2	88.2	0.0	0.6
Wirehaired Pointing Griffon	160	10.6	87.5	0.0	1.9
Doberman Pinscher	377	10.6	86.2	1.3	1.8
Collie	137	8.0	89.8	0.0	2.2
Dalmatian	168	6.5	91.7	0.0	1.8
Irish Wolfhound	235	6.0	93.2	0.0	0.8
German Shorthaired Pointer	839	5.8	93.2	0.0	1.0
Afghan Hound	867	5.3	93.6	0.0	0.9
Belgian Turvuren	171	5.3	93.6	0.0	1.2
Siberian Husky	867	4.2	93.9	0.0	2.0
Total	35 285	19.2	77.2	0.8	2.8

[1] Reproduced with permission from J. Am. vet. med. Ass. *165:* 79–81 (1974).

Table II. Analysis of phenotypes[1]

Low prevalence	High prevalence
Body size	
Weight and size of ancestral dog	Giant type, weight 2–3 times that of ancestral dog
Bones trim and small in diameter	Bones coarse and large in diameter
Head narrow and long	Head broad and oversized
Feet small and well arched	Feet oversized and splayed
Body type	
Slender ectomorphic (racing, hunting, fighting) type	Stocky acromegalic, endomorphic type
Thorax deep and narrow	Thorax barrel shaped
Skin trim and tight	Skin loose, thick, and wrinkled
Body fat limited	Body fat in excess
Muscles well developed and hard	Muscles deficient in quantity and tone
Joints stable, with well-developed ligaments and tendons	Joints unstable; ligaments and tendons weak
Gait well coordinated, fleet and light footed	Gait slow, awkward, heavy footed, poorly coordinated
Growth pattern	
Pups small at birth	Early rapid growth
Slow growing	Fat and heavy for age
Late physical and sexual maturity	Early physical and sexual maturity
Appetite good but self limiting	Appetite indulgent

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lowest percentage of hip dysplasia were similar in the three categories studied.

Body Size

The breeds with the lowest percentage of hip dysplasia were near the size of the ancestral dog. The bones were small in diameter and smooth, the feet were small and well arched, and the shape of the head was long and narrow.

The giant breeds with the high percentage of hip dysplasia were two to three times larger than the ancestral dog. Their bones were coarse and large in diameter, with prominent protrusions and depressions. The feet were large and splayed, and the head was wide and oversized.

Body Type

In general, the body conformation of the breeds with the lowest percentage of hip dysplasia was slender and trim. The skin was thin, smooth, and stretched tightly over the underlying tissues. The muscles were prominent, hard, and full-bellied. At dissection in these breeds, the skin and subcutaneous tissues and fascia rarely contained over 1–2% fat by weight. The joint ligaments were well developed; the fibers were coarse, closely packed, and relatively free of fat. The well-formed pelvic and thigh muscles were attached to broad, coarse tendons that were securely attached to the bones. This group of dogs is fleet-footed and well-coordinated in their movements.

Of the high-risk group, the four breeds of the giant type were not only two to three times the size of the ancestral dog, but their body conformation was heavy, round, and stocky. Acromegalic characteristics were present to some extent in all four breeds. The skin was thicker than that in the other group; it lay in folds over the head and neck. At dissection, the thickened skin was infiltrated with fat. Fat was also abundant in the subcutaneous and fascial spaces and commonly accounted for 5–10% of the weight of the soft tissues of the hindquarters. In comparison with the other group, the muscles were less prominent and less developed. Fat also was infiltrated into the tendons and ligaments. The fibers of these two structures were smaller in diameter than those of the low-risk group. The gait of the giant breeds was less graceful and slower than that of the smaller breeds.

Growth Pattern

The group of breeds with the highest percentage of hip dysplasia grew and matured more rapidly than did those in the low-risk group. We have observed this in several studies. Starting at birth, this group gained rapidly. The pups of these breeds were aggressive eaters, both as they nursed and as they began to take supplemental food. In one study, the growth of four young Greyhounds was compared with that of four young German Shepherds. The growth curves of the Greyhounds maintained a uniform scope for the first 14–16 weeks. By the age of 24 weeks, the German Shepherds had gained more than 60% of their adult weight. In another study involving 222 German Shepherds, we found that 63% of the dogs that weighed more than the mean of this group at 60 days of age were dysplastic at 1 year of age, whereas only 37% of those less than the mean became dysplastic. We have observed the same rapid rise in weight in other breeds of the high-risk group for dysplasia [37].

The 38 breeds, when ranked according to the highest prevalence for hip dysplasia, with few exceptions exhibited a gradual shift from the poorly muscled and poorly coordinated, acromegalic type giants at the top, to the lowest percentage of hip dysplasia at the bottom, characterized by the breeds that were sleek, tight skinned, highly coordinated and well muscled (table I, II). These correlations and observations support previous findings that the poorly muscled and coordinated breeds have a high percentage of hip dysplasia, whereas the well-muscled and highly coordinated types are relatively free of the disease [41].

As in man, in the breeds of dogs that have acromegalic characteristics, the muscular elements generally fail to keep pace in their development with the overgrowth of bone. Furthermore, some acromegalic breeds have long dense hair that conceals body conformation, and the lack of muscle mass in these dogs is not realized until comparisons are made with better muscled dogs.

Selection for acceleration in growth created dogs with excessive fat and weight at an early age. This has resulted in lowered dynamic and biomechanical efficiency of the hip joint [9]. The young dog that carries excessive weight runs the risk of over-extending the supporting soft tissues, and injury to these tissues results in pulling apart (subluxation) of the joint components. This results in changes that have been recognized as hip dysplasia. This is not a new concept. It was pointed out as long ago as 3 centuries that 'Muscles and bone are inseparably associated and connected, between muscle and bone there can be no change in one but it is correlated with changes within the other' [56, 57].

In 6313 pelvic radiographs representing 19 large breeds in Sweden, Finland, and Denmark, 2801 dogs (44.3%) were dysplastic [16].

Hip dysplasia has not been reported in the wild undomesticated carnivorous animals, such as wolves and foxes. A study of their pattern of growth found that the pups were slow-growing and late maturing. The young pups were whelped in dens. As newborns, their food came from nursing during the first few weeks. Then when more food was required, the mother killed rodents and either brought them to the den or ate the animal where it was killed and then returned to the den where the ingested rodents were regurgitated for the young to eat [43]. Young carnivores were quite mature and 6 to 10 months old before they began to hunt. The amount of food available for the growing members of a litter was limited. This caused the young to mature slowly and remain thin and light for their body size. Such an environment favored the completion of ossification and developmental maturity of

the joint before the hips could be subjected to possible injury, incongruity, or subluxation from excessive extrinsic forces (for example, excessive body weight) [41, 42].

Genetic Influences and Heritability

Few genes so far analyzed directly affect osseous structures [10]. The shape of bones reflects changes by biomechanical stresses [9].

Among the Navajo Indians, multigenetic factors in the etiology of the disease were decidedly influenced by associated child-rearing environmental methods [44]. Hip dysplasia in this tribe has decreased in the past 2 decades when the cradleboard was discarded and the newborn were diapered. These changes allowed the legs of the infant to be free, flexed, and abducted. These positions are favorable to hip joint congruity and stabilization [44].

In the dog there has been no clear-cut pattern of inheritance recognized [16–19]. This meant many genes were affected, and polygenic traits were subject to environmental modifications. New data have substantiated these findings [18].

The spread of hip dysplasia centers around the genetic transmission and heritability of certain body size, type, conformation, movement, growth pattern, and temperament. This conclusion is based on the facts that the prevalence of hip dysplasia is approximately the same in a number of breeds with similar body characteristics and there is no gene flow between these purebred breeds. Since these facts must be respected, biomechanical and environmental factors associated with certain body conformation and size must be considered as the causes [41].

Critical evaluation of the heritability of hip dysplasia has been made in the German Shepherd, in 244 individual offspring from 54 full sub-families. In this report 'heritability was defined as a property not only of the character (trait) but also the population and the environmental circumstances to which individuals are subjected. Heritability, because it represents the proportion of the total phenotypical variance, receives the attributes of a positive number which may range from 0 to 1.0 in magnitude' [18]. On this scale and based on evaluations of radiographs from 2-year-old dogs, the heritability was given an average estimate of 0.25. The conclusions were that canine hip dysplasia be termed a moderately heritable disease [19].

The heritability of canine hip dysplasia is in the same range as milk yield for dairy cattle, pig growing and maturing weight at 180 days, and egg production in certain varieties of chickens [18, 19]. It was concluded, therefore,

that individual selection be considered a suitable and practical method for controlling hip dysplasia [18, 19].

Effects of Breeding Programs

In a study involving 236 German Shepherds, it was demonstrated that the most reliable way to eliminate canine hip dysplasia was through the establishment of 'pedigree depth', that is, by the use of ancestral lines of dogs radiographically free of hip dysplasia [22].

Results of controlled breeding programs in Sweden further indicated that the prevalence of hip dysplasia in the German Shepherd was substantially reduced by mating only dogs with radiographically normal hips [6, 31]. Similar decreases in prevalence have occurred in another controlled breeding program in a colony of guide dogs (Seeing Eye, Inc., Morristown, N. J.).

In another account, with 584 progeny in a closed colony of German Shepherds, it was shown that the prevalence of hip dysplasia was noticeably reduced by selectively breeding dogs proved radiographically to have normal hips at 1 year of age or older. In 3 ½ years the incidence of hip dysplasia was lowered from 39% to less than 17% [38]. The male dogs in this colony had a wide variation in their ability to transmit normal hips to their progeny. For example, for one dog with radiographically normal hips at 2 years of age, only 8.7% of his progeny developed hip dysplasia, whereas for another dog with similar radiologic evaluation mated to the same bitches, 37.8% of his pups developed hip dysplasia [13].

In 1966 the Schäferhunde Club of Switzerland, cooperating with the veterinary schools of Berne and Zurich, developed a hip dysplasia control program for breeders. At the start, all dogs with greater than grade-2 dysplasia, and since 1970, grade-1 dysplasia, were refused breeding registration. During the control period, the number of dogs disqualified for breeding annually has declined appreciably [8].

In our work with breeders where small numbers of subjects were involved, every report, without exception, has indicated a decline in the number of affected dogs in successive generations when only dogs and bitches with normal hips were mated.

Environmental and Man-Made Influences

Embryologically, articular joints are differentiated as units *in situ* from a mass of skeletal mesenchyme [54]. Development progresses normally

in each joint as long as there is full congruity between the parts. The congruity remains as long as the supporting tissues are strong enough to withstand the mechanical or physiological factors that tend to pull them apart [51].

In man, intrauterine stress has been cited as contributing to hip dysplasia, particularly if the fetus is positioned with the legs in adduction and extension. In breech birth, 16% of neonates had an Ortolani click in one or both hips [33]. The practice of suspending an infant by its heels to induce breathing at birth may have contributed to stretching the supporting tissues of the hip. The Ortolani click was twice as frequent at birth in the first born as in successive siblings. In the first born, the uterus has greater muscular tone, and there is less placental fluid [47, 58]. The Ortolani click was less frequent when the birth was cephalic as such infants were carried in the uterus with the legs in flexion and abduction [58, 59]. The Ortolani click, when present, occurred unilaterally in 60% and bilaterally in 40% [33, 59].

Hip dysplasia in man was rarely associated with teratological abnormalities. Other hip abnormalities distinctive from dysplasia, however, were frequently associated with such deformities as club foot, hyperextension of the knees, spinal deformities, arthrogryposis multiplex, and chondroosteodystrophy [14].

In the young child, the position of the legs during infant care was found to be highly important to normal hip development [5, 45, 46, 49]. Abduction and flexion of the legs had a stabilizing effect on the hip joints. The square diaper favored greater abduction of the legs than did three-cornered diapers. The Bantu baby, who is carried with its front side bound to the mother's back with its legs in acute abduction and flexion, seldom has abnormal hip joints [45, 49]. In contrast, the Navajo Indian baby who spends its first years of life strapped to a cradleboard with the legs in abduction and extension has a high rate of hip joint instability [44].

Other factors such as femoral anteversion and spastic shortening of the psoas muscle have been shown to favor acetabular dislocation when the leg was extended [30]. These observations show that both environmental and hereditary influences are important [17, 29].

In the dog, the hip joints are normal at birth [27, 40]. The long bones of the pup are short during prenatal life, and mechanical stresses that bring about dislocation of the femoral heads are minimal.

Teratological abnormalities of the joints are rare in the dog, except for congenitally dislocated elbows and an occasional club foot deformity. Congenital malformation of hips is also rare.

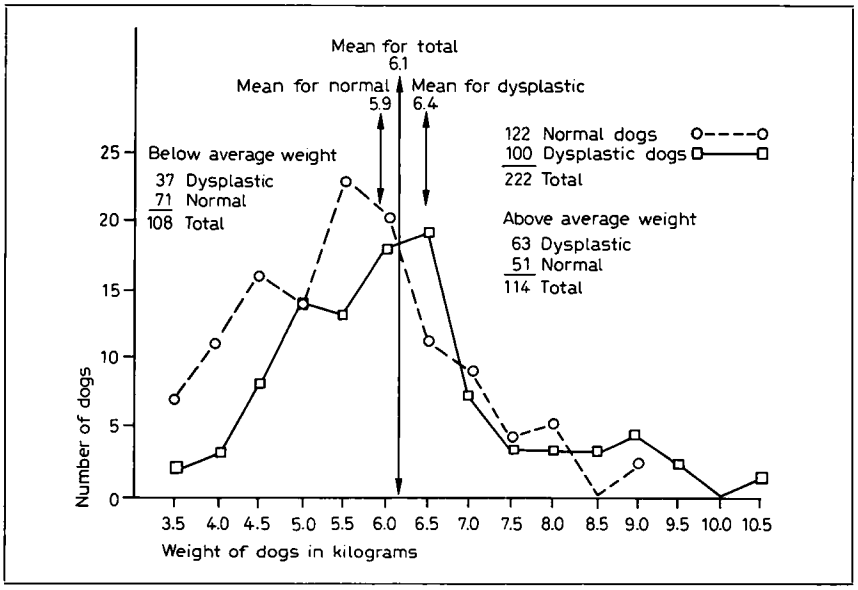

Fig. 2. Incidence of hip dysplasia in 222 German Shepherds. Data reproduced with permission from J. Am. vet. med. Ass. *145:* 661–668 (1964).

Extrauterine Influences

Early Weight Gain

In 222 German Shepherds born consecutively, 100 were dysplastic, and the prevalence of hip dysplasia at 1 year had a direct correlation with their weight at 60 days of age. The heavier dogs, that is, the heaviest males and heaviest females at 60 days of age, had the highest incidence of hip dysplasia at maturity (fig. 2) [37].

The 222 pups at 60 days had a mean weight of 6.1 kg; the mean weight of 100 dysplastic pups was 6.4 kg, and the mean weight of the 122 pups with normal hips was 5.87 kg (fig. 2, table III). This correlation was statistically significant ($P>0.04$). Despite the averages, 37 out of 100 dysplastic dogs were under average weight; only five out of 122 normal dogs were over average weight [37].

The mean weight at 60 days of age according to sex for all males was 6.29 kg (fig. 2).

The weight of dysplastic males averaged 6.59 kg compared with 6.08 kg for normal males. The weight of the dysplastic females averaged 6.29 kg

Table III. Average weight (kg) of normal and dysplastic dogs

Sex	Normal pups	Dysplastic pups	Average
M	6.08	6.59	6.29
F	5.60	6.29	5.94
Both sexes	5.87	6.44	6.12

compared with 5.6 kg for normal females. This indicated that the heavier pups at 60 days within each sex group had a significant tendency to become dysplastic. In some instances, normal males were heavier than dysplastic females. Therefore, it was not simply weight *per se* but weight within each sex group that was associated [37].

These data suggested a number of indirect genetic aspects influencing the rate of hip dysplasia. The aggressiveness in nursing may be inherited as well as the quality and quantity of the supporting tissues around the hip joint. It was concluded that when growth, gain in weight and nursing aggressiveness exceeded the strength of the supporting tissues, subluxation and hip dysplasia occurred [37].

The first subluxating stress on the hips occurs when the pup supports itself while nursing, and the hindlegs are in forceful adduction and extension [37]. The heaviest pups were the more aggressive, worked the hardest while nursing, and spent the most time feeding [37].

Pelvic Muscle Mass

Data indicate that there is a positive correlation between the amount of pelvic muscle mass and the prevalence of hip dysplasia. In three large breeds of dogs, the Greyhound is relatively free from hip dysplasia; over half of the German Shepherd Dogs are affected with hip dysplasia, and nearly all the July Foxhounds are dysplastic [41].

Ninety-five dogs (20 Greyhounds, 63 German Shepherds, 12 July Foxhounds) were radiographed, killed, skinned, and the pelves dissected to determine differences between these three breeds. These breeds were dissimilar in such characteristics as color, length of hair coat, and shape of ears, but similar in body type and skeletal structure, particularly in the hind quarters. All the dogs had attained most of their growth (aged 5 months to 10 years), were in good health and flesh, and none was extremely thin or fat.

At dissection, the right and left side of each pelvis muscle group was weighed separately in order to compare the total weight, the weight of the

Table IV. Dogs grouped by degree of hip dysplasia[1]

Group	Breed	Age range	Sex	Weight kg	Hip joints	Muscle mass index	
						minimum and maximum	mean
1	Greyhound	5 months to 10 years	12 F 8 M	12.7–29.5	normal	11.7–16.3	14.2±1.6
2	German Shepherd Dog	1 year to 3 years	4 F 3 M	17.7–30.0	normal	10.3–13.2	11.4±0.8
3	German Shepherd Dog	6½ months to 3 years	6 F 3 M	23.6–40.0	near-normal	10.3–11.4	11.0±0.1
4	German Shepherd Dog	5 months to 8 years	26 F 21 M	11.4–34.1	dysplastic	5.2–12.0	9.0±1.4
5	July Hound	6 months	6 F	13.6–19.1	dysplastic (subluxated)	7.4– 8.5	7.9±0.1
6	July Hound	6 months	1 F 5 M	13.6–17.3	dysplastic (luxated)	4.4– 6.5	5.6±0.2

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individual muscles, and the weight of each side of the pelvis. A 'pelvic muscle mass index' was then calculated for each dog from the following equation:

$$\frac{\text{Weight of pelvic muscles (kg)}}{\text{Total body weight (kg)}} \times 100 = \text{pelvic muscle index}$$

This equation took into account the differences in total body weight of each dog [41].

From the general observations it was determined that the Greyhound had a much larger muscle mass when compared with the German Shepherd, and, in turn, the pelvic musculature of the German Shepherd was considerably greater than that of the July Foxhound. The angles of the pelves and bone size and shape were similar in all three breeds. This suggested that significant differences lay in the muscle size and weight among these three groups [41].

The 95 dogs were first considered as a general population and secondly as allotted to six groups by breed and degree of hip dysplasia. The hips were classified as normal, near-normal, or dysplastic. Group 1 consisted of 20 Greyhounds. None was in racing condition at the time of the dissection, although 13 had raced, and seven had not raced or received training. All the hips were normal. Group 2 consisted of seven dogs, six German Shepherds and one half-breed Shepherd. Six dogs had received training as sentry and guide dogs (normal hips). Group 3 consisted of nine German Shepherds. These dogs had been selected for breeding but were discarded because of disposition. All had normal or near-normal hips. Group 4 consisted of 47

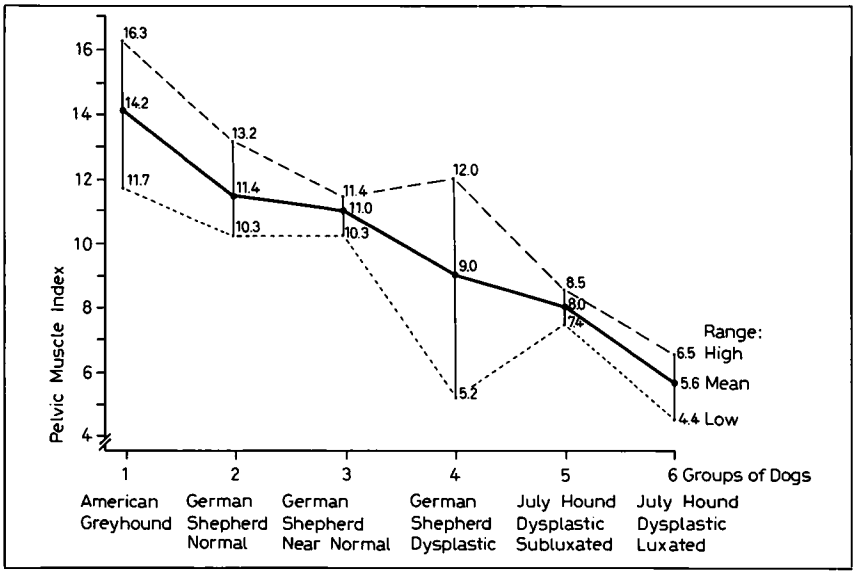


Fig. 3. Distribution of means and ranges of pelvic muscle mass indexes for six groups on the pelvic muscle mass index scale. Reproduced with permission from Am. J. vet. Res. *124:* 769–777 (1967).

German Shepherds. All were raised by us or given to the project, and all were dysplastic. Group 5 consisted of six July Foxhounds. All were raised in a kennel and were dysplastic (subluxated). Group 6 consisted of six July Foxhounds. All were raised in a kennel and were dysplastic (luxated). The data on these 95 dogs (six groups) are given in table IV and figure 3.

From these data, critical points on the pelvic muscle mass index scale have been established: a point above which hip dysplasia rarely occurs (10.89), and a point not far from this, below which hip dysplasia always occurs (9.0) (table V).

These data further emphasize that hip dysplasia encompasses biological height, weight and muscle bracing. The builder, before architecture was a science, learned that when the height of a structure was doubled, the bracing had to be tripled or the structure would fall of its own weight [56]. This basic rule pointed out many years ago strongly illustrates why a low foot stool fits solidly on the floor and the tall stool of the same area wobbles when supporting weight [56]. Similarly, it has been found that dogs less than 30.5 cm tall and less than 11.3 kg in weight (Dachshund) are relatively free from hip dysplasia. On the other hand, at least half the large dogs, 34 kg or more in weight and over 50.8 cm in height, are affected with dysplasia [39].

Table V. Expected incidence of hip dysplasia

Index	
14.2	All dogs have normal hips, and the disease was unreported
12.17	All dogs have normal hips, but the disease was reported in some siblings
11.63	Probability of 94% that dogs with this pelvic muscle mass would have normal hips
10.89	Probability of 86% that dogs with this muscle mass index would have normal hips
9.00	All dogs have some degree of dysplasia
8.00	All dogs have badly dysplastic hip joints
5.6	All dogs have hip joints that were luxated

The Greyhound is relatively free from hip dysplasia although he is over 50 cm in height and weighs from 22.7 to 34 kg. This dog was not bred specifically to have normal hips, but it has been rigidly selected for muscle power and speed for almost 3000 years. The Greyhound serves little purpose other than coursing, and those selected for breeding are those with the greatest pelvic mass and speed, less subcutaneous and fascial fat, and coarser tendon and ligament fibers. These are the ingredients that favor speed but also normal hips [39]. The lack of these qualities favors an unstable hip joint [24].

Muscle Myopathies

All newborn mammals, including human infants, undergo many metabolic changes during their transition from intrauterine to extrauterine life. The muscle tissues are relatively immature both anatomically and biochemically at birth. Lack of muscular maturation in the newborn influences the manner in which the newborn responds to function. This immaturity accounts for the failure of many mammals, for example, the child, dog, and cat, to walk at birth [59].

There is evidence that the wide range of acetabular and femoral changes occurring in hip dysplasia are the consequences of joint laxity. The possibility that this may be associated with or influenced by the rate of muscle maturation has not been explored. The rate of muscle maturation may be an inherited factor [7, 27]. Consequently, the degree of subluxation in the young may be influenced by subnormal muscular function. In man, the possibility of iliopsoas muscle spasm in the infant has been explored [28, 30].

In the adult dog, we have used the light microscope to examine histologically the individual pelvic muscles associated with hip joint motion. Evidence

of muscle disease was not recognized. In dogs with advanced hip dysplasia and associated osteoarthritis, atrophy of the pelvic muscles was present but changes such as muscular necrosis, inflammation, and extensive fibrosis were not found [39, 41].

One observer suggested that in young dogs with developing dysplasia, the pectineus muscles were in spasm and contained a degenerative lesion [4]. The pectineus muscle (an adductor) when in spasm was thought to favor forcing the femoral heads out of the acetabulums. This observer further suggested that if the pectineus were cut in the dog at an early age, the occurrence of hip dysplasia would be greatly lowered [4].

A causal relationship between the pectineus muscles and hip dysplasia could not be established in one experiment using the pelvic muscles from Labrador Retrievers, German Shepherds, Alaskan Malamutes, and Beagles [26]. Pectineus muscles in these dogs with both normal and dysplastic hips were examined and compared. The relationship between pectineus muscle abnormality and hip dysplasia remains undefined. The pectineus muscles from some young pups had both hypotrophic and hypertrophic changes. It was suggested that the alterations seen in the pectineus muscles of dysplastic dogs probably represented secondary manifestations associated with a disease of developing hip joints (hip dysplasia) [4, 7]. The available evidence does not support the concept that abnormal pectineus muscle behavior is a cause of hip dysplasia [25].

Developmental myopathy with type II fiber hypotrophy in the pectineus muscles of very young dysplastic German Shepherds has been described [7]. These investigators failed to establish a relationship between this muscle change, joint laxity, and dysplasia but have suggested this possibility. In their findings, the small fibers stained as type I (white) and the larger fibers as type II (dark) with an enzyme stain. They considered the differentiation between small and large fibers in *young* dogs to be a myopathy. No myopathies were present in either the normal or dysplastic *adult* dogs in their study [7]. This change in the young dog resembles muscle fiber hypotrophy, which follows the cutting of the nerve to a muscle. These hypotrophied muscles become functional again and the fibers become normal in size when the nerve unites and use is restored [23]. The appearances of the atrophied muscle caused by a cut nerve and of the immature muscle are similar [23].

Another investigator working with intercostal muscles of young normal rats has reported a similar muscle fiber hypotrophy. This hypotrophy was considered normal, and it was believed to be a delay in maturation that was within the expected natural range of development [23].

Influence of Inheritance and Selection on Pelvic Muscle Mass

The question has been asked repeatedly whether in the Greyhound the potential for having greater pelvic muscle mass was inherited or whether the pelvic muscle mass was acquired through exercise associated with training for coursing.

Seven of the 20 Greyhounds reported in table IV had never been trained or exercised outside of their pens. These had a mean pelvic muscles mass index of 13.67 as compared with a mean of 14.2 for the Greyhound group. One Greyhound raised by us was confined to a cage a cubic meter in size from 30 days old until killed at 5½ months. His pelvic muscle index was 14.9 (fig. 4) [41]. This is good evidence that muscle mass is inherited.

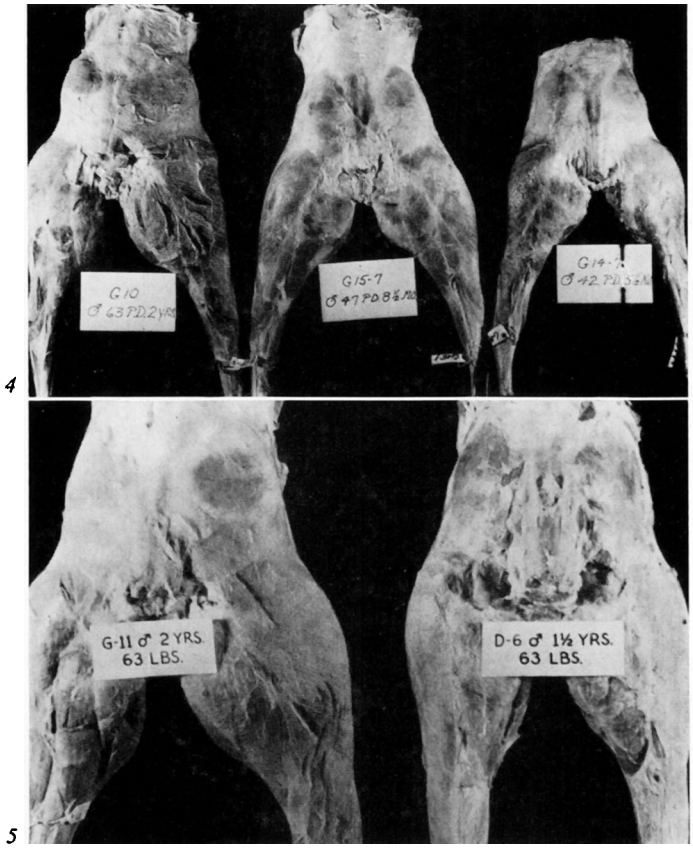
Two German Shepherds with normal hips were put through the Sentry Dog Training Program of the Armed Forces and were then killed at the end of training because of sub-standard performance. Their pelvic muscle mass indices were 11.7 and 12.0, respectively. This was above average for the German Shepherds with normal hips, 11.4, but was lower than the range of the Greyhound group that had received no training (fig. 5) [41]. Although the rigid training of both the German Shepherd and the Greyhound was about equal, the German Shepherd never acquired the muscle development maintained by the Greyhound.

A significant finding relative to differences in muscle mass was also observed in differences in the pelvic tissues. The size of the individual muscles determined by weight has already been discussed. In addition, the fascia of the Greyhound was thicker and heavier than that of the German Shepherd and July Foxhound. The fibers of the tendons and ligaments were also larger and coarser. Relatively little or no fat was present in the fascial planes and under the skin. The absence of fat was also observed in the Greyhounds raised in the cage and in the pen. The consistent finding of these differences in tissues of the pelves in instances where exercise and training could not have been an influencing factor shows that inheritance and selection contribute to the mass and structure of the tissues and hence to occurrence of normal hips. In the selection of the German Shepherds and July Foxhounds, there has been less and sometimes no regard for the musculature, speed, or hindquarter power [37, 39].

Metabolic Influences

Sex

In humans, the female was affected with hip dysplasia four to eight times as often as the male [14]. In the dog as many females as males were affected.



4

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Fig. 4. Pelves of three Greyhounds illustrating influence of heredity compared with that of exercise on the quantity of muscle mass. Pelvis on the left from a dog trained for racing. Other two pelves from dogs of racing Greyhound parents. Pelvis in center from a dog that weighed 21 kg at 8½ months. The dog had been restricted to a small pen and not trained during life. Pelvis on the right from a dog that weighed 19 kg at 5 months and had been restricted to a 3 × 3 × 3 meter cage the last 4 months of life and not exercised. Muscle mass is more dependent on heredity than training. Reproduced with permission from Am. J. vet. Res. *124:* 769–777 (1967).

Fig. 5. Pelvis from a racing-trained Greyhound (left), weight 29 kg and the pelvis of a German Shepherd (right), trained for Army sentry duty, weight 29 kg. The German Shepherd has a limited amount of pelvic muscle mass compared with the Greyhound. The training of both dogs was extensive and rigorous. Reproduced with permission from Am. J. vet. Res. *124:* 769–777 (1967).

The reasons for this difference have not been explained. Of 100 dysplastic German Shepherds at the Armens Hund Skula (Sweden), 49 were males and 51 were females [37].

Chemical and Hormonal Influences

Pelvic tissue relaxation is a well-known physiological phenomenon during the terminal phase of pregnancy in mammals. This reaction has been associated with the female hormone, estrogen. Experimentally, this reaction has been studied by injecting ovarian extracts into dogs to produce pelvic tissue relaxation resembling that seen at the termination of pregnancy. The specific polypeptide hormone that is commonly used is called relaxin. Male and spayed and virgin females when 'primed' with estrogen before relaxin was administered respond sufficiently to relax pelvic tissues on the hip joints [27, 36].

The urine of newborns was examined to see if there was a correlation of high estrogen levels and the unstable hip. From the first tests, it appeared that such a correlation existed, but the use of more refined tests failed to verify these findings [1–3, 55]. The present conclusion is that hormonal influence is not associated with the development of congenital hip dysplasia in man or animals [1–3, 45, 55].

In the dog it has been possible to increase the occurrence of hip dysplasia by giving relaxin to newborn pups, and to produce hip dysplasia in the Greyhound [11, 27, 32, 36]. 'It does not prove, however, that estrogens have anything to do with etiology and pathogenesis of spontaneously occurring hip dysplasia' [12]. There is no evidence that estrogen levels within the biological range have a relationship to the incidence of hip dysplasia in dogs [12].

Defective protein biosynthesis of collagen was suggested as a cause for increasing articular cartilage degradation in osteoarthritic joints. Soluble collagen was reported to be found in the acetabular cartilage of dysplastic dogs, while predominantly insoluble collagen was present in dogs with normal hip joints. It was not possible to relate these changes to hip dysplasia or to osteoarthritis [25, 26].

Inborn metabolic errors of chemical or hormonal origin have not been found in hip dysplasia of either man or the dog [25, 26].

Diet

A variety of nutritional and mineral supplements have been used in attempts to alter or prevent the course of hip dysplasia in the dog. Diet has not affected the occurrence or course of the disease other than the mechanical effect of increased or decreased weight upon the hip joint [39].

Production and Prevention

In addition to the production of hip dysplasia by the use of excessive doses of estrogens, hip dysplasia results from a number of mechanical and traumatic alterations.

In a group of mongrel pups, metal prostheses were substituted for the femoral heads; these were made in shapes that varied from normal femoral heads. Some were larger and round, some were larger and oval, some were larger and square. In each instance the acetabulum changed shape and size to accomodate the contacting femoral head. When the femoral head was removed the acetabulum cavity filled with new fiber bone [50].

Another group of investigators performed osteotomies on the femurs of young mongrel dogs and fixed the legs in extreme anteversion with intramedullary pins. The position of the femoral heads altered the congruity between the femoral heads and acetabulums by producing subluxation. This resulted in immediate remodelling changes and beginning degenerative disease that resembled hip dysplasia [15].

In newborn pigs, a hindleg was placed in extension and adduction with a plaster cast. Subluxation of the femoral head and dysplastic changes in the acetabulum resulted almost immediately. Dysplastic remodelling stopped, and immediate improvement in shape of the joint occurred when the plaster cast was removed and normal motion and position of the femoral heads was restored [15,49].

The same results occurred when a similar experiment was performed on young rabbits [58]. I have produced subluxation and luxation of the femoral head in both the rabbit and the dog by applying a cast to the leg that held the stifle in extension.

Traumatic coxofemoral luxation is followed by remodelling and degenerative joint disease. When healed, these changes are generally indistinguishable from spontaneous canine hip dysplasia. When traumatic coxofemoral luxation occurs the joint capsule and teres ligament are always ruptured. Even though the luxation is reduced, the joint never regains the former congruity and stability because of the torn supporting tissues. Fractures of the acetabulum, femoral head, and neck trigger similar remodelling and degenerative joint disease for the same reasons of incongruity and instability [39].

Acquired deformities of the femoral head bring about remodelling and degenerative joint disease, which when healed leaves changes indistinguishable from hip dysplasia [39].

In the child the development of hip dysplasia can be stopped, and the condition can be reversed to a stable normal hip if it is discovered early before

remodelling has begun. The key to treatment is the restoration of full congruity between the femoral head and acetabulum by placing the legs in an abductor-flexed position [50, 59].

In the young dog, confinement to a small cage (1 m^3) where the dog spends most of his time sitting on his haunches (abductor-flexed position) will prevent the development of hip dysplasia in the young dog genetically conditioned to develop hip dysplasia [39, 40].

Summary

Hip dysplasia affects man and all other domestic mammals. In man, 1.3 children in 1000 are affected. In dogs the prevalence may run over 50% in large dogs if control measures have not been practiced. Few data are available on the prevalence of hip dysplasia in other mammals, but it is thought to be low. The disease is undoubtedly rare in undomesticated animals.

No specific genetic pattern of inheritance has been demonstrated in this variable disease. It has been demonstrated that both genetic and environmental influences contribute to development, regardless of the species affected [5, 9, 20, 21, 26, 47, 50]. Consequently, the disease has been designated as polygenic or multigenic. There is no evidence that a primary defect of bone exists but rather the disease is a failure of the muscles and other soft tissues to hold the hip joint in full congruity [20, 21].

This is further supported by the fact that bony dysplasia can be increased, decreased, or prevented by controlling the degree of joint instability and incongruity [35]. No other malformations are associated with the disease [52], which signifies that hip dysplasia is 'many diseases that end up with common degenerative lesions of the hip joint' [51].

Inborn metabolic errors of chemical and hormonal origin are not evident [34, 58]. The quantity of hormones needed to produce hip dysplasia experimentally far exceeded those that could be produced by any biological system [34, 36, 57].

A causal relationship between muscles and soft tissue defects or pathological changes other than lack of muscle mass or strength has not been established [25, 26].

Experimentally, hip dysplasia may be produced in many ways [27, 36, 47, 50, 58, 59]. These include any circumstances that contribute to an unstable hip joint, that is, adductor forces, lack of muscle strength, chemical relaxation of the pelvic soft tissues, traumatic injury to the hip joint, and overload-

ing of the joint by weight. Hip dysplasia is a concentration of factors from a pool of genetic weaknesses and environmental stresses that fall into a programmed pattern of progressive remodelling and degenerative joint disease.

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