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How do hips become dysplastic? (<http://www.instituteofcaninebiology.org/blog/how-do-hips-become-dysplastic>)

12/23/2015

By Carol Beuchat PhD

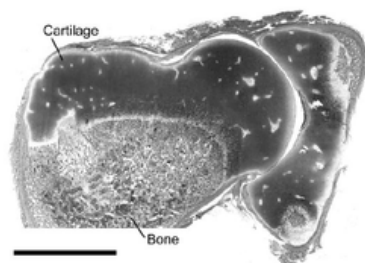
"Few students of biological science are trained to look beyond biochemistry and pathobiology to apply two principles fundamental to the architect and mechanical engineer; these are sound structure and sufficient bracing. The application of these principles seems essential to the investigation of hip dysplasia."

- Wayne H. Riser, DVM; Founder and first Program Director of the Orthopedic Foundation for Animals

Let's start with a point made in a previous blog ([The 10 most important things to know about canine hip dysplasia](http://www.instituteofcaninebiology.org/blog/the-10-most-important-things-to-know-about-canine-hip-dysplasia) (<http://www.instituteofcaninebiology.org/blog/the-10-most-important-things-to-know-about-canine-hip-dysplasia>)), that puppies are born with "perfect", normal hips. Of course, they're puppy hips and not adult hips, but they are quite remarkable.

A newborn puppy looks like it has no joints at all on an x-ray. This is because the ends of the long bones and many parts of the pelvis are soft cartilage at birth. Because cartilage doesn't show up on x-rays, a radiograph of a newborn puppy can look like an apparition of a spooky, disarticulated body. But this is just nature's way of providing enough support to be able to move around while the skeletal grows rapidly in the first few months of life.

The hip joints are also formed of cartilage at birth and are little more than a round ball at the end of the femur that sits in a depression in the pelvis where the hip socket will be.



As the puppy grows, the formation of the bony structures that will become the hip joint is not programmed by genes. Instead, the forces on the joint stimulate the deposition of bone in the right places to form an articulating ball and socket joint. As long as the head of the femur stays seated where it belongs in the developing hip socket, the hip joint should form perfectly.

This seems truly magical, but there's a catch. If, for some reason, the head of the femur is not tightly held in the hip socket, development will go awry. What results is "developmental hip dysplasia"; a malformed hip socket. In dogs, this is canine hip dysplasia.

Wayne Riser, who studied hip dysplasia in dogs for many years and was also the founder and first director of the

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Orthopedic Foundation for Animals, explained it this way (1975):

"In all mammalian embryos, the hip is laid down as a single unit from mesenchymal tissue, and it develops normally as long as the components are left in full congruity. The hip is normal at some time in the development of the mammal, and abnormal development occurs only when stresses pull the components apart.



In the dog, the hip is normal at birth. Intrauterine stresses are not sufficient to produce incongruity of the hip. The first time such forces are great enough is when the pup begins to take its position to nurse.

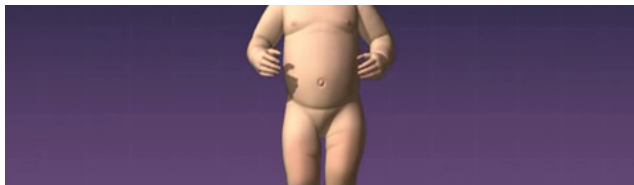
Observations of the disease in man, dog, and a number of other mammals for many years have culminated in the conviction that the bony changes of hip dysplasia, regardless of species, occur because the soft tissues do not have sufficient strength to maintain congruity between the articular surfaces of the femoral head and the acetabulum."

Riser is saying here that **muscles, ligaments, and tendons ("soft tissues")** normally keep the head of the femur properly seated in the developing socket. But if there are abnormal forces on the joint, the soft tissues might be inadequate to stabilize the joint, and malformation of the socket - hip dysplasia - will occur.

It might be hard to visualize what's going on, so here's a great video that shows how this can happen. It is about hip dysplasia in humans, but the anatomy is the same and the manual diagnostic techniques (Barlow and Ortolani tests) they demonstrate are used for dogs as well. The last 20 minutes or so are about examination of the human infant so you might want to skip that, and for some reason it's a 30 minute video that repeats, so it's not really an hour long.

(Check out the shifty eyes on that baby. I think this might be the very same infant that went on to become Ally McBeal's famous "dancing baby" hallucination. If you missed that somehow, you can catch it [here](https://www.youtube.com/watch?v=rWYb93tiVQQ) (<https://www.youtube.com/watch?v=rWYb93tiVQQ>.)

Developmental dysplasia of the hip - e...  



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As Riser described, looseness or laxity of the hip joint is the prerequisite for the development of hip dysplasia. If the head of the femur is not positioned properly in the hip socket during development, the mechanical forces **that stimulate bone deposition** in the joint will be abnormal and the result will be a dysplastic hip.

In breeds like the Greyhound, which very rarely suffer from hip dysplasia, the muscles that support the hip are exceptionally well developed. Riser found that less pelvic muscle mass was associated with higher risk of dysplasia. This even held within a breed: in both German Shepherds and July Hounds, dogs with a higher pelvic mass index had better hips on average.

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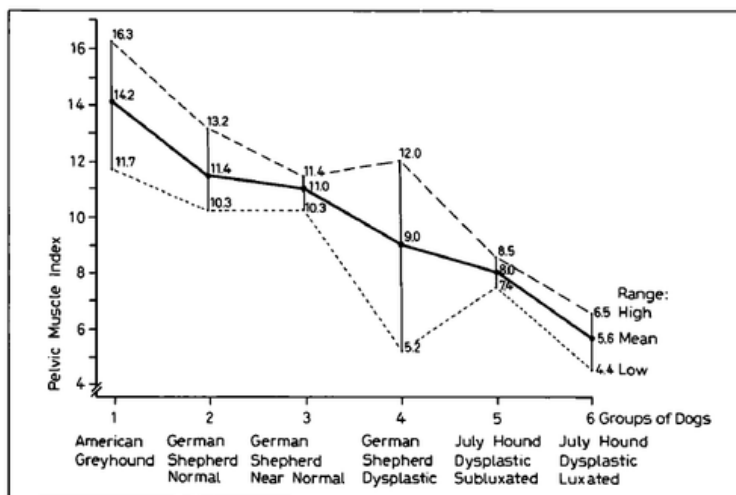


Fig. 3. Distribution of means and ranges of pelvic muscle mass indexes for six groups on the pelvic muscle mass index scale. Reproduced with permission from Am. J. vet. Res. 124: 769-777 (1967).

Riser 1975

The exceptional hip stability provided by the pelvic muscles of Greyhounds is evident even in newborns. Check out the one day old Greyhound puppies, standing and climbing to nurse, and looking like Ninjas compared to the desperately cute but nearly helpless baby Berners.

[Hungry One Day Old Greyhound Puppies](#)

[Bernese Mountain Dog puppies one da...](#)



Unfortunately, most breeds of dogs don't have the exceptional pelvic muscles of the Greyhound at birth, which are the result of selection over hundreds of generations to produce a dog with exceptional speed. The Bernese Mountain Dog, on the other hand, was bred to have the size, strength, and substance necessary for drafting (cart hauling). In fact, anatomical differences among breeds are reflected in propensity to develop hip dysplasia.

After studying radiographs of tens of thousands of dogs in dozens of breeds, Riser (1975) found that a pattern began to emerge.

There was...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. Recently, we have identified certain general characteristics of a breed that increase the risk of hip dysplasia.

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 a 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 fascia 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.

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. 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.

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. 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'.

Riser summarized these findings in a table:

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

You will recognize that many of these traits are features of breed type, and of course most are under direct genetic selection or are secondary to some other trait under selection. As it seems to go in the competitive show world, many of the traits on the "High Prevalence" list have become progressively more exaggerated through

artificial selection - the large size gets larger, heavy bone gets heavier, a broader head gets broader. And to make matters worse, the desire for dogs that "finish" (become champions) at ever younger ages drives selection for rapid growth, which overloads the immature skeleton and muscle systems. It seems that we are trying to select against hip dysplasia at the same time as we select for many traits that increase the risk.

The factors that cause hip laxity and the development of dysplasia occur in the first few months of the puppy's life. Sometime between birth, when the cartilagenous tissues of the hip joint have the capacity to become a perfectly constructed ball and socket joint, and when the puppy is four or five months old, the soft tissues of the pelvis fail to provide the support necessary to keep the femoral head properly seated in the hip socket. As a result, the joint doesn't develop properly, then abnormal biomechanical forces exacerbated by environmental factors such as weight and inappropriate exercise begin the cycle of damage and inflammation that result in dysplasia and osteoarthritis.

Wayne Riser showed 40 years ago that

In very young human and canine subjects with unstable coxofemoral joints, hip dysplasia can be prevented and the instability corrected if the congruity of the components of the hip joint is maintained and if femoral subluxation does not occur. If proper congruity cannot be maintained, the hip joint becomes malformed in a relatively short time. The changes in the bones and cartilage of the hip are thus the indirect result of failure of the soft tissues to support full congruity of the bony components of the hip.

Few genes so far analyzed directly affect osseous structures. [This is still true.] The shape of bones reflects changes by biomechanical stresses.

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.

According to Riser, the risk factors for hip dysplasia are related to the features of a dog and its breed that result in a mismatch between the forces necessary to maintain congruity of the hip joint and the support the soft tissues are able to provide during the critical first few months of life. By the time the puppy is 6 months old, the strength of the supporting tissues and ossification of the bones should be adequate to prevent the development of hip dysplasia under normal circumstances. Prevention of hip dysplasia will come both from reducing the genetic risk factors through selection and from avoiding situations that could result in joint instability or incongruity.

The literature review and report of our work presented here have revealed a basis for optimism in controlling and reducing the prevalence of hip dysplasia in both man and animals...In children, it is basically the test for hip laxity at birth, and for the dog it is restriction of breeding only those animals with radiographically normal hips. Both in children and dogs, hip dysplasia is, for the most part, a 'man-made' problem and can be controlled if man will use the tools at his disposal.

These findings uphold the validity of our two premises that (1) hip dysplasia occurs only if hip joint instability and joint incongruity are present in the young child or animal; and (2) the disease can be prevented if hip joint congruity can be maintained until ossification makes the acetabulum less plastic and the abductor muscles and supporting soft tissues become sufficiently strong and functional to prevent femoral head subluxation.

Genetic selection and management of environmental risks are both necessary to lower the frequency of hip dysplasia. For selection to be effective, we need to recognize that some of the traits that are valued as part of breed type are themselves risk factors for development of hip dysplasia. Selecting for faster growth and ever increasing size will confound efforts to produce dogs with better hips. Likewise, inadequate weight management of both puppies and adults is an environmental factor that dramatically increases the risk of developing dysplastic hips. Finally, we should use a little common sense about activities that are appropriate for puppies (see below...).

**You can learn more about hip dysplasia in ICB's 10 week course,
[Understanding Hip and Elbow Dysplasia \(http://www.instituteofcaninebiology.org/understanding-hip-elbow-dysplasia.html\)](http://www.instituteofcaninebiology.org/understanding-hip-elbow-dysplasia.html)
Starts 4 January 2016**

The papers by Riser referenced here were published in series as a collection in Veterinary Pathology 12 (4): pp.

234-334, in 1975.

Wayne Riser DVM was on the faculty of the vet school at the Univ. of Pennsylvania and was also the founder and first Program Director of OFA. In 1975, he published a collection of papers in the journal Veterinary Pathology that, taken together, represented a summary of what was known at the time about the disease of hip dysplasia in the dog. Much of what Riser had to say about anatomy, growth, development, and biomechanics forty years ago is just as relevant to breeders and dog owners today.

On a final note, I would like to highlight a few (apparently common) activities that are probably really, really bad for puppy hips. It should go without saying, but apparently not...

Puppies Vs. Stairs



Cute Puppies On Slides Compilation HD 1080



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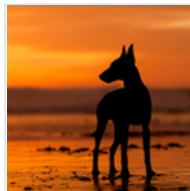
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