Canine Hip Dysplasia

Canine Hip Dysplasia (CHD), a crippling disease of the coxofemoral joint (hip), was first observed and reported in 1945, by the late Dr. Gary Schnelle. Dr. Schnelle, who was the staff radiologist at Angels Memorial Hospital at Boston, Massachusetts, and reported his observations in The North American Veterinarian Journal and termed the condition Congenital Coxofemoral Subluxation. Though congenital, by definition, means existing at birth but not hereditary, Schnelle theorized the cause to be a recessive gene. Today, this condition is associated with the large breeds of canines. However, Schnelle's initial observations were in the smaller breeds, i.e., Cocker Spaniels, Terriers, etc.

During the 1960s, Dr. Wayne Riser, a veterinary pathologist, collaborated with a geneticist and postulated the concept that CHD was polygenetic with environmental overtones, and during this time frame, the term Canine Hip Dysplasia was born. There has been a string of professionals who have perpetuated the heredity theory even until the present. We have been encouraged to breed selectively, alter and, in some cases, euthanize the afflicted canines. This is what the veterinary experts have been insisting the dog breeders and fanciers do to prevent CHD for the past 52 years.

Is it possible we have been on the wrong track for more than a half century? One authority of a veterinary teaching institution in a personal correspondence to the author admitted the experts have failed to solve the CHD problem.

In 1976, the author published his observations, Chronic Subclinical Scurvy and Canine Hip Dysplasia, in his small animal practice through the administration of vitamin C to prevent CHD. Whether this concept has been accepted or not, it has sparked controversy and has some veterinarians reassessing the archaic heredity theory.

There are two unanswered relevant questions the experts have failed to answer to prove the heredity theory.

1. What is the action of the gene/genes and how do they create the disease?
2. How does the veterinary practitioner differentiate between a hip subluxation due to trauma (injury) from true CHD?

Since the mid-1970s, the author has been successful in preventing CHD through the administration of nutritional supplements to the pregnant female and within hours of birth to the newborn.

As earlier presented, the first canines observed with CHD were the smaller breeds; yet today the larger canines are most affected. The reason for this phenomenon is that the present day dog food
is considerably more nutritious than that being fed in the 1940s, i.e., more attention is now being
given to nutritional requirements. The requirements that have been established over the past four
decades have eliminated the CHD problem in the smaller breeds but these requirements are not
adequate for the large and giant breeds. One cannot expect the nutritional requirements for a
Chihuahua to be the same for that of a Great Dane. When these large and giant breeds of canines
are adequately subsidized through nutritional supplementation, the condition is prevented.

CHD must be more specifically defined. At the present, any abnormality in the coxofemoral joint
is considered hip dysplasia. The first photograph shows a dog who was diagnosed with unilateral
dysplasia. No consideration was given to the history of this patient. The fact of the matter is, this
subluxation was due to a falling bale of hay impacting the right pelvic region.

Bitches often sit on their newborn, causing subluxations which are not manifested for some
weeks later when the pups begin to walk. For this reason, the author, in his practice, does not
diagnose or recognize unilateral hip dysplasia.

The second photograph is without a doubt CHD. Both hips are subluxated with accompanying
osteoarthritis. The problem of CHD is directly associated with collagen synthesis. Inadequate
collagen synthesis will adversely affect osteogenesis (development and formation of bone),
chondrogenesis (development and formation of cartilage) and myogenesis (develop and
formation of muscle). These three physiological processes are dependent on good collagen
synthesis which is dependent on good nutrition.

Research biochemists during the ‘90s have established the involvement of good nutrition for
good collagen synthesis. This supports the author's concept set forth in 1976, that CHD is
nutritionally related rather than hereditary.

If it can be established that these alleged genes, in fact, inhibit bone, cartilage and muscle
formation, then it would be possible to control dysplasia through nutritional supplementation.
Some genetic tendencies can be overridden by other factors, such as nutritional supplementation.

The bottom line is if CHD can be prevented, be it hereditary or nutritional, there is more to gain
than to lose with the nutritional concept.

The author has developed a nutritional protocol for the prevention and control of CHD. The
protocol begins with the pregnant bitch. To ensure a healthy pregnancy, the female is
administered Mega C Plus. This supplement will aid in the maintenance of a good pregnancy and
a healthy litter. This vitamin/mineral compound will enhance immune function and, most
importantly, aid in the synthesis of collagen while the pups are developing in utero. After the
birth of the litter, Mega C Drops (pediatric formula) is administered to each pup within two
hours and is continued through weaning. Mega C Drops is a formula containing hydrolyzed
protein (collagen), vitamin C as sodium ascorbate, plus other essential nutrients that enhances
collagen synthesis. Post weaning, the pups are placed on a Mega C Plus regimen through two years of age.


There has been extensive research in recent years concerning collagen synthesis by many renowned biochemists that support the author's nutritional concept. This new textbook is targeting veterinary practitioners and educators and will be available September, 1997.