

# Canine Hip Dysplasia Literature Review

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6 November 2014

## Introduction

Over the last 50 years a lot of research has been done on what is widely perceived to be a serious problem in dogs: Canine Hip Dysplasia (CHD). What is clear is that the emphasis most veterinarians and those doing research on the subject place on the phenomenon is grossly exaggerated. This has led to a needless escalated awareness amongst the public buying purebred dogs in general and German Shepherd Dogs in particular who generally have a totally unrealistic viewpoint of the problem (*pers. obs.*).

Furthermore, and most importantly, it is apparent that the original intention, that of shielding the buyers of registered puppies from the trauma of prematurely having to put down a family pet due to clinical lameness as a result of CHD, has been forgotten (*pers. obs.*). The total lack of consideration of this aspect in the literature is testimony to the point.

Encouraged thereto by veterinarians and researchers in the field, many canine breed control authorities have over the years introduced Canine Hip Dysplasia (CHD) control schemes for a number of breeds under their control. Over the years these schemes have become stricter and diagnoses of CHD more detailed and pedantic in an attempt to eradicate the disease, without significant success.

“If you don’t understand the ‘why’, the ‘how’ does not matter” (Ulianich 2014). This statement comes from a source unrelated to CHD, but is as valid here as from whence it comes. The research been done on CHD over the last 50 years focussed almost exclusively on the ‘how’, *inter alia*: the details of running a CHD control scheme (Corley 1992, Dennis 2012, Fluckiger 2008, Hedhammar & Indrebø 2011, Hou YaLi et al. 2013, Leppänen & Saloniemi 1999, Verhoeven et al. 2012, Worth, Bridges & Jones 2011); how to position the dog for radiography (Broeckx et al. 2014, Ginja et al. 2008, Guilliard 2014, Sehic et al. 2012, Verhoeven et al. 2012, Worth, Laven & Erceg 2009); how to make the diagnosis (Fluckiger 2008, Ginja et al. 2010, Hedhammar & Indrebø 2011, Murray & Crim 2001, Worth, Laven & Erceg 2009, Zhang & Jordan 2008); how to refine the diagnosis (Ginja et al. 2010, Ginja et al. 2008, Guilliard 2014, Runge et al. 2010, Todhunter 2011, Verhoeven et al. 2012, Worth, Laven & Erceg 2009); how to grade various levels of severity (Banfield et al. 1996, Comhaire & Schoonjans 2011, Sehic et al. 2012, Verhoeven et al. 2009) and how to treat CHD once diagnosed (Bergh & Budsberg 2014, Kirkby & Lewis 2012, Remedios & Fries 1995). The ‘why’ seems to have been forgotten.

Even though CHD occurs in most breeds of dogs, the perception amongst the public and many veterinarians is that the German Shepherd Dog (GSD) is the worst affected (*pers. obs.*). This is untrue and, according to the Orthopaedic Foundation for Animals (OFFA), at least 38 other breeds have been identified in the United States of America (USA) as having worse problems with the manifestation of radiologically diagnosed CHD than GSDs. This perception probably arose because GSD registration authorities were the first, and for many years the only, to institute compulsory CHD control schemes, as well as the fact that research on the GSD is over-represented due to their popularity and large numbers in relation to other breeds.

An equally incorrect perception amongst the public is that cross or mixed breeds are less affected. Various studies refute this perception (Bellumori et al. 2013, Switzer & Nolte 2007, Wamberg 1963).

Not a single peer reviewed study on the emotional or financial impact of CHD control on the owners of dogs that fail a scheme could be found. One study on the impact of legal implications in Germany on breeders when puppies they breed fail a scheme pointed out the seriousness of the matter in this regard (Nolte 2013). This paper further points out that in Germany there is a two year “guarantee” period during which a breeder could be held accountable. From the paper it is also apparent that the German legislation does not consider environmental aspects whereby a dog owner may be the architect of his own demise through having provided an environment conducive for his pet to have developed CHD, or that a diagnosis may be wrong due to incompetence of the diagnosing veterinarian. This can only be due to the incorrect perception that the problem is wholly genetic and that the breeder is therefore responsible.

In South Africa the Consumer Protection Act (South African Government 2009) excludes animal breeders from liability regarding genetic diseases, but this may change in the existing escalating climate of consumer perception that it is the breeder that is responsible if his dog is, correctly or incorrectly, is diagnosed with CHD (*pers. obs.*). At least one paper directly encourages escalating awareness of consumers (Leppänen & Saloniemi 1999).

### **Clinical Manifestation of Canine Hip Dysplasia**

Attempts to correlate the clinical manifestation of CHD to radiologically diagnosed CHD are largely unsuccessful (Ginja et al. 2010) and no literature proving or even substantiating such correlation could be found. Substantial anecdotal evidence encountered all over the world by this investigator regarding the GSD is that very few GSDs diagnosed radiologically with CHD, even severe CHD, ever develop clinical symptoms or show any discomfort related to CHD until old age, which for the purposes of this study is regarded as older than nine years of age. This evidence is also substantiated in the literature and there is acceptance that the

vast majority of dogs afflicted with CHD show minimal to no clinical signs their entire lives (Ginja et al. 2010).

At least two studies points to the fact that the prevention of the development of clinical manifestation of CHD through selective breeding based on radiographic diagnosis of CHD is ineffective (Lewis, Blott & Woolliams 2013, Loeser & Shakoor 2003).

### **Diagnosis of Canine Hip Dysplasia**

Arthrosis in the joints in the joints may be painful, even if the affected animal does not show overt signs of discomfort (Vezzoni 2008). Accordingly the diagnosis of CHD based on the presence of arthrosis in the hip joints, particularly in bilateral occurrences, is not disputed (Zhang & Jordan 2008). Even so, the arthrosis may not be as per the definition of CHD as a “developmental disease up to two years of age” (Vezzoni 2008) and may be the result of other factors such as environmental consequences (Hedhammar 2008) due to *inter alia* trauma or unbalanced feeding (Richardson 1992). An indirect genetic predisposition for arthritis that may, as shown in humans, target all joints in certain individuals (Sandell 2012). One study pointed to coxarthrosis in the hip joint due to bacterial infection and described the condition in seven out of nineteen cases of CHD investigated (Benzioni et al. 2008). However, once arthrosis is present in the hip joints, the origin is nearly impossible to determine and a diagnosis of CHD is probably unavoidable.

No evidence could be found that subluxation within the hip joints without the presence of arthrosis could be painful. A diagnosis of CHD solely on the basis of subluxation is also not always supported and sometimes described as a “risk factor” rather than CHD (Ginja et al. 2010). At best it is a prediction that CHD may develop. Alarmingly some schemes such as the PennHIP scheme are actually based on measuring and even creating subluxation through manipulation and deep anaesthesia during the taking of the diagnostic radiographs (Guilliard 2014).

One study, while predisposed to finding new ways to diagnose CHD irrespective of clinical symptoms, did establish that laxity often reduced with the dog maturing (D'Amico et al. 2011). In line herewith, the vast majority of GSD's which fail the German Shepherd Dog Federation of South Africa (GSDF) scheme as a result of subluxation at 12 to 14 months of age with no arthrosis present in the hip joints, pass the scheme if radiographed a year later (*pers. obs.*). What is important is the position of the femoral head in the hip joint during the normal movement of the hip joint and not the amount of anaesthetic administered or the physical manipulation of overzealous radiographers.

At least one paper pointed out that CHD diagnosed at a relative advanced age could be due to normal aging and that this fact could not be disputed unless the CHD status at a young age was known as a comparison (Krontveit et al. 2012d).

(Loeser & Shakoor 2003) also contended that the development of perceived CHD may in some cases also be a process due to aging

Measurement of the Norberg angle may also not be a reliable indication of development of CHD (Smith et al. 1995).

### **Positioning for Canine Hip Dysplasia Radiography**

The correct positioning of the dog during the radiographic examination for CHD, particularly in order to avoid undue hip joint laxity, is critical and much has been written about it (Broeckx et al. 2014, Sehic et al. 2012).

However, almost all CHD schemes depend on veterinarians in private practice to take the radiographs which are then submitted to an official scheme to be evaluated by one or more official evaluators. Experienced breeders know who the veterinarians are that produce good radiographs that limit subluxation (*pers. obs.*). In such cases the eventual diagnosis of CHD due to subluxation may therefore be biased against radiographs produced by inexperienced veterinarians or pet owners that do not have the advantage of knowing which veterinarians to go to.

Private veterinarians that perform a large number of radiographic evaluations may have a conflict of interest in that to retain or gain clients, it is important that they produce radiographs that lead to as few failures as possible.

### **Changing Canine Hip Dysplasia Schemes by a Control Organisation**

When improvement by selective breeding fails to be significant, it is sometimes recommended that the diagnostic procedures be changed (Wilson, Nicholas & Thomson 2011). The organisation that does this is effectively discarding all results from before the change. What is important is monitoring progress, or lack thereof. If no discernible progress is apparent, it is perhaps advisable to question the very existence of the CHD scheme and not to try and find another method to diagnose something that is not really there or of little significance to the actual physical wellbeing of the dog before old age.

### **Correlation of Canine Hip Dysplasia with Anatomical Conformation**

Little or no studies have been conducted on the correlation of CHD to Anatomical Conformation and the one that could be found was inconclusive (Roberts & McGreevy 2010). It is clear that much needs to be done in this field.

### **Preventing Canine Hip Dysplasia through Selective Breeding**

It has long been accepted that the origins of CHD are multifactorial. Unfortunately, this term is taken by many to understand the origin as mainly polygenetic and thus controllable by selective breeding. This unfortunate perception has led to a number of role-players from all spheres of those involved with CHD ignoring or underestimating the influence of environment or possibly the existence of other

indirect factors such as a general predisposition for arthrosis in the joints on the development of CHD or the radiological diagnosis thereof. Similarly, the development of CHD as a normal aging process is also virtually ignored and only one study could be found in this regard (Loeser & Shakoor 2003). The study supported the premise.

Significantly, it is widely acknowledged that the reduction of CHD through selective breeding based on radiographic evaluation has been largely unsuccessful (Freeman, Evans & McEwan 2013, Ginja et al. 2010, Hou YaLi et al. 2013, Janssens et al. 2014, Krontveit et al. 2012c, Loeser & Shakoor 2003, Verhoeven et al. 2012, Wilson, Nicholas & Thomson 2011). It is further probable that positive manipulation of the rearing environment of puppies has significantly contributed to incorrect perceptions that improvement in CHD incidence was due to selective breeding (Fries & Remedios 1995, Krontveit et al. 2012b). There is also literary acceptance that lack of improvement through breeding selection is not limited to GSD's but prevalent amongst other breeds as well (Lavrijsen et al. 2014).

Examination of the OFFA results between 1992 and 2012, a period when the GSD was virtually the only breed subjected to widespread and compulsory CHD control, shows that the breed's CHD ranking did not improve in comparison to breeds that did not select against CHD (*pers. obs.*, Willis 1992). If breeding selection by GSD's against CHD was successful, the CHD ranking of the breed should have significantly improved.

Perceived improvements in CHD incidence have probably also been largely due to the wide spread phenomenon of pre-screening dogs before submitting their radiographs for official evaluation, and then not submitting radiographs of dogs likely to fail (Broeckx et al. 2014, Verhoeven et al. 2012). This pre-screening is the direct result of the negative impact on the reputation of breeders if animals of their breeding are diagnosed with CHD. The same applies to the owners of stud dogs that will do anything to protect the reputation of their stud dogs (*pers. obs.*). Some studies acknowledged the phenomenon of pre-screening and manipulation by examining veterinarians by not submitting radiographs of dogs likely to fail, and that these practices may bias their research (Broeckx et al. 2014, Verhoeven et al. 2012), but no study could be found that specifically studied the phenomenon and the impact thereof on the results of official schemes.

While there is a large body of evidence that CHD may be attributable to polygenetic or quantitative trait origins, this fact is not as yet totally proven. As Fels and Distl (2014) states in what must be regarded as the latest and most authoritative paper on the possible genetic origins of CHD: "There is strong evidence in support of a genetic predisposition to CHD in GSDs and many other dog breeds" and "In conclusion, this study identified and corroborated CHD-loci on three different chromosomes for GSDs and is (only) a further step towards elucidation of the genes underlying CHD. The three validated and significantly CHD-associated Single Nucleic Polymorphisms (SNPs) are located within or in

close proximity to genes which are involved in a joint network regulating bone formation, osteoclast activity, chondrocyte proliferation and differentiation”.

Unfortunately the creditability of this study was somewhat tainted by a press release based on an interview with Prof Distl which incorrectly announced to the world: “Canine hip dysplasia genes identified” (von Brethorst 2014), which was not the case. Meaningfully, the SNP’s they refer to also occurred in a significant proportion of their experimental animals that were free of CHD (Fels & Distl 2014). The presence of these SNP’s can therefore not be regarded as definitive proof that an animal will develop CHD if they are present.

### **Molecular Studies Connected to Canine Hip Dysplasia**

Most of the molecular genetic research on CHD is currently occurring in the Institute for Animal Breeding and Genetics headed by Prof Distl at the University of Veterinary Medicine, Hanover, Germany (Fels & Distl 2014, Marschall & Distl 2010). Their research is usually in conjunction with the Deutsche Schäferhundeverein (SV), the controlling authority for GSDs in Germany. The study referred to above contradicts some of the previous studies published by his other students and which he co-authored. The search for a specific gene or genes that may play a direct role in CHD is thus far from over.

### **Environmental Causes of Canine Hip Dysplasia**

There is an wide acknowledgement that environmental factors such as feeding and injury play a significant role in the development of CHD (Comhaire & Snaps 2008, Distl et al. 1991a, Krontveit et al. 2012a, Lust & Farrell 1977, Worth et al. 2012) or alternatively in the development of arthrosis in the hip (Remedios & Fries 1995, Sandell 2012) which is then diagnosed as dysplastic. This is reflected in the various attempts to determine the heritability ( $h^2$ ) of CHD, with heritability figures differing significantly worldwide with  $h^2$  being quoted anywhere between 0.2 and 0.35. (Fels & Distl 2014, Hamann, Kirchhoff & Distl 2003, Janutta & Distl 2006, Wilson et al. 2012, Zhang et al. 2009). This implies that, depending on the study, the environmental component is estimated at between 65% and 80%.

Research is active in this field and there is acknowledgement that positive manipulation of the environment is perhaps the biggest contributor to progress that has been made in limiting CHD in dogs (Distl et al. 1991b, Fattahian et al. 2012, Krontveit et al. 2010, Worth et al. 2012). This is also the case in humans (Stevenson et al. 2009). Other than weight and feeding, no studies could be found on how owners may negatively influence environment and thus indirectly cause their dogs to develop CHD.

One study described coxarthrosis due to bacterial infection as a cause of unilateral CHD and accompanied clinical symptoms (Benzioni et al. 2008).

## **Unilateral Canine Hip Dysplasia**

It is widely recognised throughout the literature and elsewhere that unilateral CHD and cases of bilateral CHD, but where one hip is worse than the other, are common phenomena (Citi et al. 2005, Keller & Corley 1989). However, no research could be found to clarify this phenomenon except for a study that pointed to coxarthrosis as a result of bacterial infection (Benzioni et al. 2008). As the review article by Wilson, Nicholas & Thomson (2011) eloquently put it: "There is no evidence to support the idea that a dog with a poor hip and a good hip is genetically equal, on average, to another dog with two hips as poor as the poor hip of the first dog, yet they are scored equally. All schemes that use only one hip for evaluation, after scoring both hips, should, as a matter of urgency, seriously evaluate the consequences of ignoring half of their valuable data".

The assumption that in the case of unilateral CHD the affected hip, or in the case of bilateral CHD where one hip is worse than the other, the worst hip, is of genetic origin, is also highly likely incorrect. There is no recognised genetic mechanism whereby one hip is targeted to be dysplastic and the other not, or where one hip is more affected than the other. Any possible genetic predisposition should, through the manner in which genes function, target both hips equally. The difference in the respective hips is probably due to environmental influences.

Many CHD Schemes such as the Fédération Cynologique Internationale (FCI), an international federation of kennel clubs based in Thuin, Belgium, the SV and the GSD Federation of South Africa (GSDF) base their breeding recommendations on the worst hip. If it were to be accepted that any possible genetic predisposition was much more likely to be reflected in the best hip and that the worst hip was probably due to trauma or other environmental influences, a significant number of CHD failures would be avoided with momentous benefits to the people owning dogs that failed because of the evaluation of the worst hip. Research on CHD would also be redirected thereby and perhaps assist in arriving at the correct conclusions which are currently proving so elusive.

## **Impact of CHD Research and Schemes on Canine Organisations**

The original 'why' or purpose behind the institution of CHD controls and to prevent same through selective breeding was to save pet owners from the trauma of having to cope with premature clinical lameness due to CHD before old age. Because of the wide-spread implementation of such schemes all over the world and the tremendous publicity given to such control schemes, most pet owners regard a diagnosis of CHD, even in mild cases with no clinical symptoms present, as an absolute disaster and experience the same emotional traumas upon such a diagnosis as if their pet had in fact developed irreversible clinical lameness.

The fact that in the case of GSDs the animal is unlikely to experience clinical symptoms throughout its lifespan and likely to live a normal productive life, is

virtually ignored by the veterinary as well as the research communities (Ginja et al. 2010). It is thus not effectively communicated to the pet owner that his pet is unlikely to develop clinical symptoms from a source he or she trusts in order to reduce the emotional trauma experienced with a radiological diagnosis of CHD. In contrast, unnecessary surgical intervention at huge costs is more likely to be recommended by the veterinarian.

The fact that very few radiological diagnoses of CHD in GSDs ever lead to clinical lameness (Ginja et al. 2010) compounds this dilemma. This issue is further escalated by the large number of incorrect diagnoses made by non-specialist veterinarians. In the GSDF such incorrect diagnoses are made at a frequency of almost one per week (*pers. obs.*) and create enormous problems for the organisation, the breeders as well as the owners involved. Not a single peer reviewed article could be found that was based on research of how canine organisations, breeders and pet owners are negatively affected by CHD control.

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