A study of debilitating orthopaedic conditions of working New Zealand Police German shepherd dogs.

A thesis presented in partial fulfilment of the requirements for a Doctorate degree in Veterinary Science at Massey University, Manawatu, New Zealand.

Andrew John Worth
2015

Abstract

This thesis explored the causes of retirement or loss from service of German shepherd Police dogs working in New Zealand. Abnormal development of the hip joint with subsequent development of osteoarthritis (hip dysplasia), and degeneration of the lumbosacral junction, were identified as the leading causes of early retirement of Police dogs due to an inability to meet the physical requirements of Police work.

Hip dysplasia is a multifactorial disease with moderate heritability and improvements in the phenotypic selection of dogs for breeding should improve subsequent longevity in future generations of Police dogs. Selection of dogs for breeding based on traditional phenotypic scoring, using radiographs of the hips in extension, was shown to be producing minimal improvement in hip status. When compared with distraction radiography, correlation between the two methods was low and they were not equivalent in terms of ranking dogs for susceptibility to hip dysplasia.

In the second part of the thesis, degeneration of the lumbosacral joint was reviewed and the role of surgical management of this condition was examined. A new method of computed tomographic volumetric analysis of the L7-S1 lateral intervertebral neurovascular foramen was described, which can be performed on anaesthetised dogs. This method was then tested on German shepherds, both normal and affected by lumbosacral degeneration. The dogs were imaged in extended, neutral and flexed positions of the lumbosacral junction. Extension results in marked narrowing of the L7-S1 foramina. Dogs affected by degenerative disease of the lumbosacral junction had smaller foraminal volumes than unaffected dogs, indicating that dynamic narrowing likely contributes to clinical signs. An ex-vivo experiment demonstrated that surgical resection of the dorsal annulus and partial L7-S1 discectomy (as commonly performed during dorsal decompressive surgery) may lead to further narrowing of the lateral intervertebral lumbosacral neurovascular canal. A prospective evaluation of a recently developed surgical procedure, dorsolateral foraminotomy, confirmed effective enlargement of the L7-S1 foraminal volume, but showed that by one year there was bone regrowth partially attenuating the effect. Finally, a novel method of dorsal stabilisation, which maintained the foraminal volume by fixation of the lumbosacral junction in a favourable position, was developed through a series of pilot studies, providing the basis for further development.

Table of contents

Abbreviations

ANOVA = Analysis of variance

BVA = British Veterinary Association

CHD = Canine hip dysplasia

CT = Computed tomography

DI = distraction index (PennHIP)

DLS = dorsolateral subluxation score

DLSS = degenerative lumbosacral stenosis

FCI = Federation Cytologique Internationale

GSD = German shepherd dog

KC = Kennel Club (UK)

L = lumbar (nerve or vertebrae)

LS = Lumbosacral

MRI = Magnetic Resonance Imaging

NA = Norberg angle (measured on VD radiographs of the coxofemoral joint)

NZ PDS = New Zealand Police Dog Section

NZVA = New Zealand Veterinary Association

OFA = Orthopedic Foundation for Animals (USA)

PennHIP = The University of Pennsylvania Hip Improvement Program

PL = pelvic limb

VD = ventrodorsal (radiographic beam direction or positioning for surgery)

A study of debilitating orthopaedic conditions of working New Zealand Police German shepherd dogs.

The New Zealand Police force, like many law enforcement agencies around the world, uses the German shepherd dog (GSD) for patrol duties, tracking criminals, apprehension of criminals and armed offender operations. The GSD is known for its intelligence and willingness to work, having been initially bred as a dog for herding cattle.

The GSD is a relatively modern breed of dog. Herding dogs were used throughout Europe in the 18th century with widespread phenotypic variation in their physical and behavioural characteristics. In each local farming community, shepherds selected and bred dogs that they believed possessed desirable characteristics for herding of domestic livestock. The Phylax Society was formed in Germany in 1791 with the intention of creating standardized dog breeds (Strickland *et al.* 1988). However, it was disbanded after only three years due to conflicts amongst members regarding the traits of dogs that the society should promote.

The standardization of a German shepherding dog began in 1899 by Captain Max von Stephanitz (http://www.suite101.com/content/origin-of-the-german-shepherd-dog). Having purchased a part-wolf herding dog (which he renamed Horand von Grafrath), he went on to establish a new breed through inbreeding the progeny of that founder sire. Von Stephanitz founded a breed society and named the breed *Deutscher Schäferhund*, literally translating to "German shepherd dog" (Strickland *et al.* 1988).

The direct translation of the name was adopted for use in the official breed registry. However, at the conclusion of World War I (WWI), it was believed that the inclusion of the word "German" would harm the breed's popularity. The breed was officially renamed by the Kennel Club in the United Kingdom as the "Alsatian Wolf Dog" which was also adopted by many other international kennel clubs (Strickland *et al.* 1988). Eventually, the appendage "wolf dog" was dropped. The name Alsatian remained in use for five decades, until 1977, when successful campaigns by dog enthusiasts pressured the British kennel clubs to allow the breed to be registered again as the German Shepherd Dog. The word "Alsatian" still appeared in parentheses as part of the formal breed name and was finally removed in 2010 (Strickland *et al.* 1988).

German shepherd dogs were valuable as military dogs during WWI. They served as messengers, sentries, and guards as well as locating wounded soldiers for the Red Cross. Military working dogs were deployed in World War II, Vietnam and the Middle

East conflicts, and have been valuable in police work, drug detection and search and rescue as well as being excellent guide dogs for the blind and assistants to disabled persons (http://en.wikipedia.org/wiki/German_Shepherd_Dog). German shepherd and Belgian Malinois dogs are specifically chosen for this type of work due to their characteristics (size and high level of drive) and reputation for endurance, speed, strength, courage, intelligence and adaptability to almost any climate (Strickland *et al.* 1988).

The modern German Shepherd dog has been criticized for traits and an appearance different from von Stephanitz's original vision for the breed: that German Shepherd dogs should be bred primarily as working dogs, and that breeding should be strictly controlled to quickly eliminate undesirable traits. Some critics of the breed have commented that careless breeding has promoted inherited disease conditions and anatomical defects.

The history of the New Zealand Police Dog Section (NZPDS) began in 1956 when the then Prime Minister Sir Sidney Holland recruited a police sergeant and his dogs from the English Constabulary of Surrey. A dog training centre was set up in Trentham in conjunction with the Police Training School. Whilst the Police Training School was relocated to Porirua in 1981, the dog training centre has remained at its original site (http://www.police.govt.nz/service/dogs/history.html). From those early beginnings the dog section became established and training of dogs for specialist police work began. The first drug-detection training course was held in 1976, which was closely followed by the introduction of explosive detection courses in 1977. More recent developments have seen the introduction of other specialised courses including the Armed Offenders Squad dog course (1992), Accelerated Detection (1997), and Search and Rescue (1998) courses. Currently there are 115 Police Dog Handlers with 90-95 operational dogs and 10-15 in training at any one time (Inspector Brendon Gibson NZPDS pers. comm. 2015)

The operational life of a NZ Police GSD is considered to be approximately eight or nine years (Sergeant Mark Sandford, Breeding Unit Manager, NZPDS pers. comm. 2010) but precise reasons for withdrawal of dogs from service in NZ have not been collated. Each Police dog represents a large financial investment (*circa* $25,000 NZD) and time commitment for breeding and initial training. An operational team of one dog and one handler requires an annual expenditure of $120,000 including salary, vehicles, equipment and training (Inspector Brendon Gibson, NZPDS pers. comm. 2012). In order to maximise a Police dog's active working life it is important to identify the major causes of loss and to target strategies to reduce their impact. This is the subject of

chapter one. Degenerative orthopaedic disease has been identified as a major cause of early retirement of working dogs from the United States Military (Moore *et al.* 2001). Specifically, appendicular degenerative joint disease and spinal cord disease were the two conditions most likely to lead to elective euthanasia of military GSDs in that study. Canine hip dysplasia (CHD) and secondary osteoarthritis of the coxo-femoral joints have been reported to be the primary reason for rejection of working dogs during procurement and the most common reason for death/euthanasia in Military working dogs (Olson 1971; Dutton and Moore 1987). As military dogs share many training practices and have similar working conditions to Police dogs, it is likely that any disease incidence will be similar in these two populations. However there is little worldwide data and no specific data from New Zealand on the causes of loss/retirement from work of civilian Police working dogs. Sensibly the NZPDS has employed breeding policies for many years based on international practice, intended to reduce the incidence of CHD.

Hip Dysplasia is a multifactorial developmental disorder with a polygenic mode of inheritance (Cook, Tomlinson et al. 1996) [reviewed in chapter two]. Multiple quantitative trait loci associated with CHD have been identified (Lust and Farrell 1977, Mäki *et al.* 2002, Janutta *et al.* 2006). An experimental SNP array, which enables interrogation of genetic variation, is now available but is not yet suitable for the purposes of breeding selection (CanineHD BeadChip, Illumina Inc., San Diego, California, US). Therefore selection of breeding animals in an attempt to reduce the incidence of CHD is currently based on the radiological hip phenotype. There are several schemes based on radiological phenotype in use internationally. Each is based on detection of radiological features of hip dysplasia such as subluxation and degenerative joint disease. The British Veterinary Association Hip Dysplasia Scoring system was introduced in the UK in 1984 and subsequently adopted by the New Zealand Veterinary Association (NZVA). The NZVA has maintained a national computerised database since 1989. Since 1990 the NZPDS has utilised the NZVA hip dysplasia scheme to determine the most suitable dogs to select for breeding. This scheme is based on assessment of a single hip-extended radiograph, using semi-objective scoring criteria (Gibbs 1997). Studies in the UK and the US show a lack of, or only minor, improvement in hip phenotype of successive generations when the standard hip-extended radiograph is used as a selection tool (Corley and Hogan 1985, Willis 1997, Hou *et al.* 2010). Analysis and objective interpretation of the NZVA hip scoring data is required to assess whether the current hip dysplasia screening methods

as used by the NZ Police are effective for selecting dogs with a superior phenotype. This question will be addressed in chapter three.

A new paradigm in the understanding of hip dysplasia has been established with the recognition that coxo-femoral joint laxity is predictive of later osteoarthritis (Smith *et al.* 1990, 1995). A new screening test, utilising distraction radiography (PennHIP), has been developed and the degree of passive laxity has been estimated to have a heritability higher than the radiological score from the hip extended phenotype (Smith *et al.* 1990, 1995, Kapatkin 2002, Janutta and Distl 2006). Despite adoption by the Seeing Eye and other working dog organisations, the PennHIP method had not been adopted as a national scoring scheme anywhere in the world*, and in Europe the Federation Cytologique Internationale (FCI) does not recognise the PennHIP method as a means of CHD assessment. Changes to the reporting of the NZVA scheme from 2003, with a subtotal score in addition to the total score, were proposed to be more useful in determining a dog's hip status (Burbidge 2003). The subtotal score incorporates those radiological criteria present on the hip-extended view associated with coxo-femoral laxity and therefore should be at least partially representative of passive hip laxity. Thus a comparison needed to be performed to determine if the NZVA hip-extended subtotal score identifies the same population of affected dogs as the PennHIP distraction method (chapter four).

In the study by Moore *et al* (2001), "spinal cord disease" ranked second as a cause of death or euthanasia of GSDs in the US Military program. The general term 'spinal cord disease' used in that study includes degenerative intervertebral disc disease, degenerative lumbosacral stenosis (DLSS) and degenerative (GSD) myelopathy. Experience gained at the Massey University Veterinary Teaching Hospital indicates that of these, DLSS is the most prevalent within the NZPDS working dog population. DLSS is characterised by degenerative disc disease and changes to the articulation of the L7-S1 spinal unit with resultant compression of the *cauda equina* (Meij and Bergknut 2010) [reviewed in chapter five]. It has been proposed that working GSDs are predisposed to DLSS due to abnormal anatomical characteristics and/or the particular work they are required to undertake. Subsequent failure of the supportive structures of the lumbosacral (LS) junction is thought to lead to narrowing of the L7-S1 lateral intervertebral neurovascular foramen and LS vertebral canal, with subsequent nerve root and *cauda equina* compression, and clinical signs of pain and neurological dysfunction.

* Until 2014 when it was recommended by the NZVA on the recommendation of the author.

Breeding of GSDs with a lowered propensity to develop DLSS could potentially reduce rates of retirement due to this condition. Whilst transitional vertebral anomalies have been shown to predispose a dog to DLSS (Morgan *et al*. 1993), it is not clear what, if any, more subtle anatomical variations may be contributory to the syndrome. If such predisposing anatomical variations are found and proven to be heritable then breeding selection could be changed to promote better LS conformation. A better understanding of the LS articulation, both anatomically and dynamically, would aid development of new treatment interventions.

Several surgical procedures are promoted as a means of resolving pain and disability in dogs with DLSS but the long-term efficacy of surgery in working dogs with DLSS has been questioned (Linn *et al.* 2003). There is no consensus as to the best strategy for surgical management, whether to perform decompression of the *cauda equina* or stabilisation/fusion of the LS articulation. In order to establish some reference data for future research the long-term clinical success rate of surgical intervention for DLSS in working dogs was investigated using measures of performance in a handler survey, as outlined chapter six.

The effect of angle of the LS junction on the three-dimensional volume of the L7-S1 lateral intervertebral neurovascular foramina will be investigated in chapter seven. Dynamic narrowing of the foramen is considered to be a cause of L7 nerve root compression that contributes to the clinical signs of DLSS. The effect of surgical annulectomy (removal of a section of the dorsal annulus of the L7-S1 intervertebral disc) will be investigated in chapter eight.

A new surgical procedure, lateral foraminotomy, which decompresses the L7-S1 lateral intervertebral neurovascular foramen (Godde and Steffen 2007) offers promise as an effective treatment for DLSS but has not been evaluated long-term. A cohort of dogs were followed to determine if foraminal enlargement is long lasting (chapter nine). Finally, dorsal stabilisation with screws transfixing the adjacent articular processes is widely used to treat DLSS in pet dogs (Slocum and Devine 1986). However, anecdotally, there is a high incidence of implant failure in working dogs. A refinement of trans-fixation of the articular processes, by the addition of pedicle screws, would be considered a novel approach and the success or otherwise of such a procedure should be evaluated. Biomechanical testing could also be used to show the potential strength and failure resistance of the new construct prior to its clinical application. A novel method of dorsal stabilisation is then proposed which uses computer aided design and rapid prototyping technologies to overcome some of the deficiencies of the existing surgical strategies, (chapter ten).

Acknowledgements

I sincerely thank those individuals and organisations that contributed their expertise, or provided data, in support of the investigations for this doctoral thesis.

Sergeant Mark Sandford and Inspector Brendon Gibson of the New Zealand Police Dog Section, Trentham, New Zealand assisted with the survey design and contacting and collating the dog handler's data for chapter one.

Rachel Stratton and Vicki Erceg assisted with the design of the questionnaire and had input into the discussion in chapter one.

Magdeline Soo, a master student under my supervision researched and co-authored the review of Canine Hip Dysplasia. Some sections from chapter two, used material from her Master thesis in Veterinary Medicine. Two co-authored articles from this thesis have been published in the New Zealand Veterinary Journal.

The New Zealand Veterinary Association granted access to their Hip Dysplasia database. From 2008 I have been the Convenor for the NZVA Hip and Elbow Dysplasia panel, an appointee of the Companion Animal Society. On the basis of research described in chapters three and four, and subsequent projects funded by Massey University and the Companion Animal Health Fund (Soo *et al. in press 2014*), the NZVA discontinued traditional hip scoring in favour of recommending PennHIP from 2014.

Richard Laven (Massey University) assisted with the statistical analysis and had input to the discussion for chapter four. Vicki Erceg also contributed to the discussion and was instrumental in the adoption of PennHIP by the New Zealand Police Dog Section. Funding for the study in chapter four was provided by Inspector Brendon Gibson of the Police Dog Service on behalf of the New Zealand Government.

Lauren Blume (BVSc student) generated the lumbosacral angle data from CT images and obtained data for chapter six by contacting police dog handlers.

Angela Hartman (Massey University) contributed to the development of the correct positioning of the dogs for the CT study and to the protocols for the foramen volumetric measurements. In addition she performed many of the 3D reconstructions used as images and assisted with interpretation of the MRI/CT images for chapters six and seven.

Janis Bridges (Massey University) provided assistance with the statistical analysis for all chapters other than chapter four.

The radiographers at the Massey University Veterinary Teaching Hospital, Nicki Moffat, Sonia Torwick, Sharyn Bray and Darryl Newsome are thanked for performing the CT scans. The nursing team of the MUVTH are acknowledged for day-to-day care of clinical cases.

I would like to thank my supervisors Professors Boyd Jones and Joe Mayhew for their many hours of editing and critique and for their guidance through the process.

Finally I would like to thank my wife Susan and my family for their support.

Funding sources

I sincerely thank those organisations that have provided funding in support of the investigations for this doctoral thesis.

The Building Research Capabilities in Strategically Relevant Areas (BRCSRA, NZ Government) Staff Development Fund contributed $20,000 to the cost of CT imaging which made the lumbosacral studies possible. This money was used to obtain the post-operative data from clinical cases, scanning of normal GSDs and Greyhounds and for the *ex-vivo* experiments in chapters six, seven and eight. Without BRCSRA funding this thesis would not have been possible.

The Massey University Working Dog Centre provided $11,000 in research grants for the development of the custom made lumbosacral implants described in chapter ten. In addition the Centre provided a travel grant that enabled me to visit the Military Working Dog (MWD) Program, Lackland USAF base in San Antonio Texas. I presented my research and discussed collaborative application of my research to dogs in the US MWD program.

Lauren Blume was supported by a Massey University Summer Scholarship scheme, and a grant from the Massey University Working Dog Centre.

Chapter One

Causes of loss or retirement from active duty of German shepherd dogs in service with the New Zealand Police-Dog Section.

Introduction

Like many civil enforcement agencies worldwide, the New Zealand (NZ) Police use German shepherd dogs (GSDs) for tracking, patrol and apprehension duties. Each dog represents an investment of approximately $25,000 NZ dollars in breeding and training costs alone before the dog undertakes formal duties (Inspector Brendon Gibson, NZ Police Dog Section (PDS), pers. comm. 2010*)* Therefore maximizing longevity of service is important for the financial viability of the PDS. In addition there is an animal welfare consideration implicit for any service animal. It is therefore important to identify consistent trends in the incidence of injuries and illnesses that have animal welfare implications or which lead to Police dogs being withdrawn from active duty. Once identified, management strategies may be possible to mitigate losses. It is only once accurate data is available and suitable analysis undertaken that appropriate strategies to improve the health and welfare of GSD Police dogs working in New Zealand can be investigated.

There is little published data on the causes of loss from service of Police dogs. Kippenes and Gondalen (1999) investigated the cause of early retirement of Norwegian Police Patrol dogs by comparing them to a control group of non-working (pet) animals. The study was performed between 1985 and 1995 on a population of 228 working dogs (208 GSDs). There were 97 dogs that were retired early (defined as before 10 years of age), 38 (39%) of which were retired due to skeletal disease. The police dogs had a higher risk of developing skeletal lesions than did the pet control dogs. Neoplasia was the second most common cause of early retirement (13 dogs).

In several countries, including New Zealand, GSDs and Belgian shepherd dogs (Malinois and Tervuren) are also used by the military for security, patrol, and detection duties. Military working dogs share many training and service characteristics with their civilian counterparts. A study by Moore *et al. (*2001) found the leading causes of death or euthanasia of military working dogs in the United States to be appendicular degenerative joint disease (19%), neoplasia (18%), spinal cord disease (16%), non-specific geriatric decline (14%) and gastric dilatation-volvulus (9%). German shepherds

were more likely to be affected by “spinal cord disease” (including lumbosacral disease) than Belgian Malinois dogs. In GSDs the three leading causes of death or euthanasia were appendicular degenerative joint disease (20%), spinal cord disease (19%) and geriatric decline (15%). The general terms ‘spinal cord disease’ and ‘spinal disease’ used in this and other studies likely includes degenerative intervertebral disc disease, degenerative lumbosacral stenosis and degenerative (GSD) myelopathy. The GSD has been reported to be predisposed to lumbosacral disc degeneration (Bergknut 2011). Insurance data from Sweden (Agria, 1995-2006) revealed a claims incidence of 27.9 claims per 10,000 dog years at risk for GSDs with lumbosacral degenerative disease, (95% confidence interval [CI] of 25.5 to 30.3 claims per 10,000 dog years) (Bergknut *et al.* 2012). This was the highest breed specific risk for all dog breeds in the study. The average number of claims for the insured population studied was 5.6 claims per 10,000 dog years at risk (95% CI of 5.4 to 5.9). The mortality rate was reported as 18.1 (+/- 1.1) deaths per 10,000 dog years at risk for GSDs, again the highest for lumbosacral degeneration as a cause of euthanasia. The aim of the study reported here was to determine the causes of loss or retirement from active duty amongst GSDs in service with the NZ PDS. A suitable archive of accurate medical records was not available for analysis; therefore data was collected from Police Dog Handlers using a questionnaire, which relied on their own recall.

Materials and Methods

Police dog handlers identified from the staff database of the NZ PDS, Trentham NZ, were sent a 3-page questionnaire by e-mail or post (appendix 1). The questionnaire had been prepared after discussion with senior PDS staff and two animal behaviourists familiar with Police work. Handlers were asked to complete a questionnaire for each of their current operational dogs and for any other police dogs they had worked with during their career as a dog handler. For each dog the handler was asked to complete demographic data (name, date of birth, district), the date when the dog entered service, whether the dog was still operational or had been retired from service, been euthanased whilst in service, died of a disease/illness whilst in service, or had been killed whilst in service. For retired dogs, they were then asked to record the major and secondary reasons prompting retirement from a provided list: behavioural problem; loss of tracking ability; inability to meet the physical demands of the job; other (specify). For euthanased dogs the handler was asked to pick from a list of common medical and surgical conditions or behavioural problems, or specify the reason for euthanasia, should it not be listed. Similarly where a dog had died of an illness the handler was

given a choice of medical and surgical problems or the option to specify the diagnosis. When a dog had been killed on operational duty the handler was asked to describe the circumstances. If arthritis was indicated as a cause of euthanasia or retirement then the handler was asked to specify the joint/region from a list. If back pain or spinal problems were indicated as a cause of retirement or euthanasia the handler was asked to indicate the region/area of the vertebral column affected.

Statistical analysis

The age of the dog at the time of loss was classified according to category (retired/euthanased/died/killed) and the overall and individual loss category mean/median/SD was calculated. A one-way analysis of variance was performed (R v2.8.1; R Foundation for Statistical Computing, Vienna, Austria).

The null hypothesis was that there would be no relationship between the response (age of dog at the time of loss) and the factor (loss category). This model contains K=4 regression parameters. If f_{obs} is the observed value of the test statistic and X is a random variable from an $F_{K-1, n-K}$ distribution then $P = P(X \geq f_{obs})$.

A Kaplan-Meier product limit analysis was performed using the time from birth to loss from active service. From this curve a 3, 5 and 7-year “still working” rate was calculated. A further curve was generated to depict any differences between loss categories and age at retirement/loss.

Results

One hundred and eighty two questionnaires were returned completed by 149 current dog section staff that were, or had previously been, dog handlers. Of the 149 staff, 119 were currently working with an operational Police dog, 13 were retraining a replacement dog and 17 were now supervisors who no longer worked a dog. The earliest records were from two dogs born in 1975. Data was collected in July 2011 restricting the analysis to dogs already in service before that date. The median year of loss for dogs in the survey was 2004; the mean year of loss was 2002 (SD = 6.76 years). Only eight of 182 dogs surveyed were lost from the PDS prior to 1990. There were only four bitches (all entire). The remaining dogs entered service as entire males though an undetermined number were castrated for medical reasons later in life. Causes of loss were categorised as either retirement, euthanasia whilst still in active duty, death from illness/natural causes, or being killed whilst on duty. Of 182 dogs with complete surveys, 48 dogs were still in service leaving 134 that had either been retired (94), had been euthanased (24), had died (11) or had been killed (5).

The proportionate cause of loss/retirement is depicted in Figure 1 and the individual categories of loss/retirement are stratified by known or suspected cause (from the handler surveys) in Table 1.

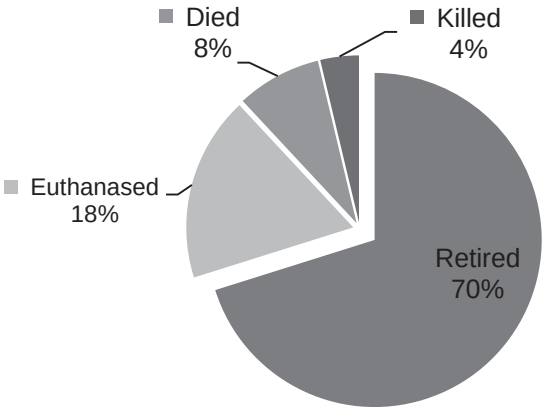


Figure 1. Cause of loss from active duty as a proportion of all known New Zealand Police German shepherd dogs lost from service between 1975 and 2011 (n=134).

Table 1. Reasons for loss from active service of 134 NZ Police Dogs, lost/retired between 1975 and 2011.

Category	Total dogs	%	Major reason	Number
Retired	*94*	*(70)*	*Inability to meet the physical demands of the job*	*61*
			Planned retirement due to age	*11*
			Loss of Tracking	*7*
			Other	*6*
			Behavioural problems	*5*
			Poor bite work	*4*
Euthanased	*24*	*(18)*	*Medical problem prompting euthanasia*	*19*
			Behavioural problem prompting euthanasia	*4*
			Unknown	*1*
Died	*11*	*(8)*	*Gastric dilatation/volvulus*	*4*
			Neoplasia	*3*
			Other	*4*
Killed	*5*	*(5)*	*Shot on duty*	*2*
			Motor vehicle accident	*2*
			Drowned	*1*

Of the 134 dogs with a known cause of loss, 10 records lacked sufficient information from which to calculate their age at loss from active duty. These 10 dogs were acquired from the public rather than bred by the PDS, and were approximately 9 – 24 months of age at acquisition; however they were deleted from the age analysis.

The mean age at loss for dogs no longer in service (n = 124) was 6.59 years (SD +/- 2.75, *df* = 120). The median age at loss for dogs no longer in service (n = 124) was 6.63 years. The mean and median for each of the categories of loss is shown in Table 2. The distribution of the age data is depicted in Figure 2. A one-way ANOVA showed there was a significant association between age at loss and loss category (*P* = 0.038), thus the null hypothesis was rejected (Multiple R-squared: 0.067, Adjusted R-squared: 0.044, F-statistic 2.89). The low R-squared value shows that category explains little of the variation in age at loss. Analysis between each category showed that euthanased dogs were lost from service at a significantly younger age than those dogs that retired (*P* = 0.007). The remaining category comparisons were not significantly different.

Table 2. Mean and median age at date of loss from service as a NZ Police Dog (n=124)

Category	Age at date of loss from service	
	Mean (years)	Median (years)
Retired (n=87)	7.0[a]	7.3
Euthanased (n=24)	5.4[a]	5.2
Died (n=8)	6.5	6.3
Killed (n=5)	5.5	6.0

[a] significant difference between mean ages by category of loss (*P*=0.007)

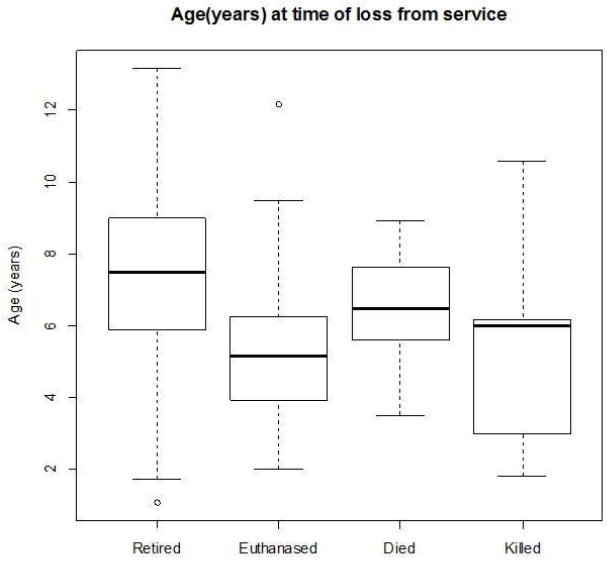


Figure 2. Box-and-whisker plots for age at loss from service of 124 New Zealand Police Dogs, classified according to whether a dog was retired, euthanased, died or was killed on duty. The bold line represents the median; the box is the first quartile above and below the mean and the whiskers represent the 95% and 5% limits of the population. Open circles represent outliers.

The Kaplan Meier curve is shown in Figure 3 and is almost linear showing a steady loss of dogs at all ages rather than an anticipated skew towards loss in older dogs. The 3-year still-working rate was 91%, the 5-year rate was 82% and the 7-year rate was 52%. The nominal age for planned retirement (8-years) was only reached by 40% of dogs. The Kaplan Meier curve depicted in Figure 4 shows that a trend towards a more rapid loss of dogs due to euthanasia at earlier ages than the other categories of loss.

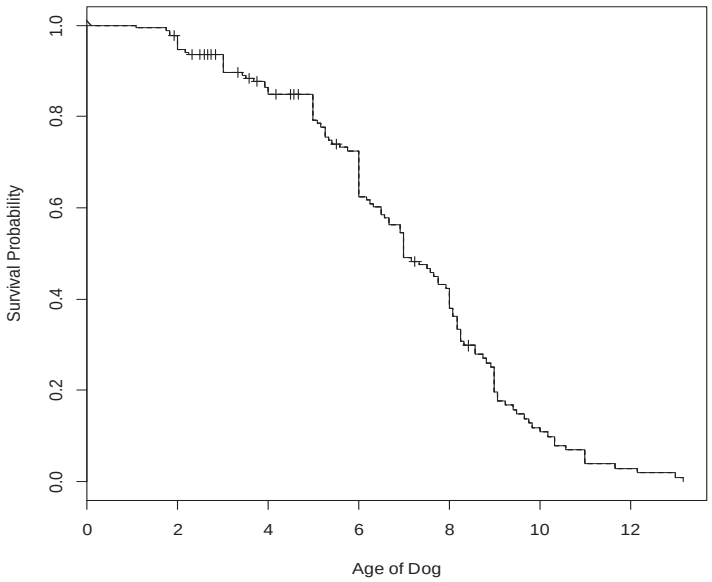


Figure 3. Kaplan-Meier analysis of 182 New Zealand Police Dogs from date of birth to date of loss from active duty. The survey was performed in July 2011. The earliest records were from two dogs born in 1975. Dogs still working on the survey date appear as censored observations on the 1.0 line at the top of the curve.

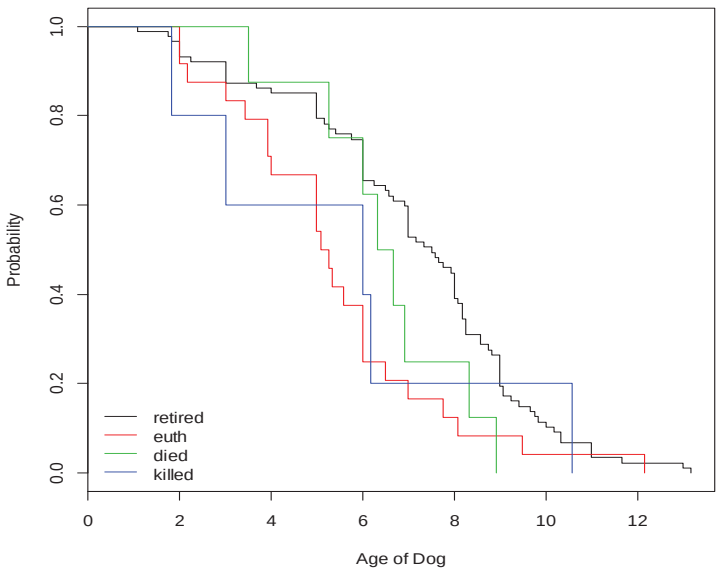


Figure 4. Kaplan-Meier analysis of 182 New Zealand Police Dogs from date of birth to date of loss from active duty. Stratified by cause or loss of retirement. Dogs still working on the survey date appear as censored observations on the 1.0 line at the top of the curve.

Reasons for retirement as stated in the survey.

The stated (primary) reasons for retirement are shown as relative proportions in Figure 5. The single most important cause of retirement was inability to cope with the physical demands of the job (61/94 dogs or 65%). Of these 61 dogs 25 were reported as having back/spinal problems affecting their ability to work and 24 had arthritis of one or more joints (seven dogs had concurrent arthritis and back/spinal problems). Overall 42/61 (69%) dogs were retired due to their inability to cope with physical demands of the job as a result of degenerative musculoskeletal disease (44% of all retired dogs). The remaining 19 dogs were classified as retired due to "old age".

Behavioural problems were the reason given for retirement of five dogs (two for aggression, one "scared dog", one with "lack of aggression", and one dog "lacking drive"). Loss of tracking ability sufficient to prompt retirement was reported in seven dogs. Loss of ability in bite work (grabbing and holding during bite training) resulted in retirement in four dogs. Loss of tracking ability, and loss of ability in bite work were cited as a secondary reason for retirement in three, and eleven dogs, respectively. Planned retirement due to age in accordance with PDS policy was the reason for retiring 11 dogs (12% of retirees).

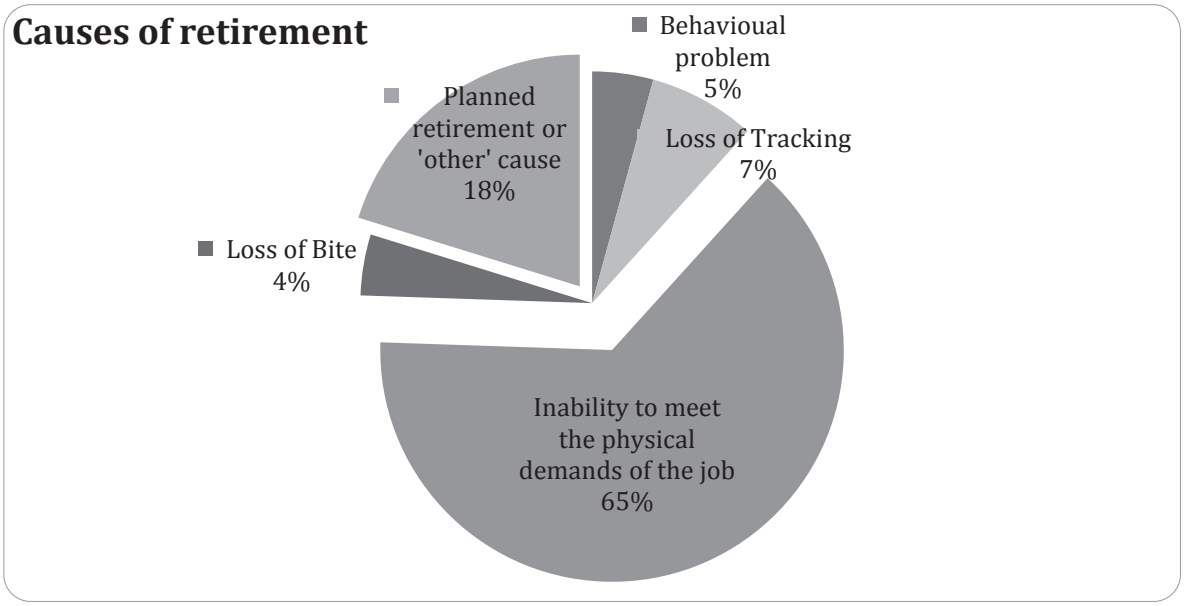


Figure 5. Reasons cited for retirement in NZ Police Dog Section German shepherd dogs (n=91) that were retired from active duty between 1975 and 2011.

Major causes prompting euthanasia

Of the 24-euthanased cases, four dogs (17%) were euthanased due to behavioural issues (three dogs had aggression problems and one dog was reported to be "environmentally inadequate" this was not defined further by the handler). A medical reason was given for 19 dogs. These conditions included back/spinal problems in eight dogs (33%), neoplasia in five dogs (21%) and arthritis in two dogs (8%). One dog was euthanased due to each of the following problems: blindness, gastric dilatation – volvulus (GDV), "stomach and intestinal disease", and chronic inflammatory bowel disease. The reason for euthanasia of one dog was not reported.

Minor causes contributing to euthanasia

Gastrointestinal disease and urinary tract disease were listed as secondary reasons prompting euthanasia in two dogs.

Thirty-three dogs that were retired or euthanased were reported to suffer from "back or spinal problems" (28%). Of these dogs, 24/33 had back pain/spinal problems involving the lumbosacral joint. The remainder were described as having a lumbar problem. The coxo-femoral joint was identified as the affected joint in 86% of dogs with arthritis, which prompted their retirement or euthanasia.

Natural causes of death

Of the 11 dogs that died when operational, four died as the result of GDV. Three died as the result of cancer (two from splenic haemangiosarcoma and one of multi-centric lymphoma). One dog died of each of the following problems; cardiac disease, suspected poisoning, pneumonia, unspecified infectious disease.

Dogs killed in action

Of the 5 dogs killed in action, two were shot and killed on duty, two died as the result of a motor vehicle accident, and one dog was drowned whilst tracking a suspect (possible malicious death).

Overall, the major primary causes of death/euthanasia or retirement, excluding planned retirement and death due to accident (n = 96 dogs), were musculoskeletal disorders (52 dogs or 54%), [including spinal disorders (33 dogs or 34%) and coxo-femoral arthritis (22 dogs or 23%)], behavioural problems (9 dogs or 9%), loss of tracking ability (7 dogs or 7%), GDV (5 dogs or 5%), and neoplasia (5 dogs or 5%).

Discussion

In this study the average age of a NZ Police German shepherd dog at the time of its death or retirement from service was 6.6 years. This retirement age is lower than the nominal retirement age of 8 years currently accepted by the NZ PDS, which was reached by only 40% of the study population. The 8-year goal represents a working career of 6.5 years assuming a dog enters service at approximately 18 months of age following training. According to NZ PDS policy, when a police dog reaches 7 years of age, its replacement is signalled and a new dog commences the training process. From 7.5 years of age onwards a dog is replaced according to logistical considerations related to the police district involved and the breeding/training program. An individual dog may work longer if a suitable replacement is not available. The analysis showed that the majority of dogs were lost from active duty prior to the planned retirement age.

A planned retirement policy confounds direct comparison of our data with other published reports on working dog longevity, which base their analysis on the date of death/euthanasia. In this study the date of loss from service was recorded for retired dogs not the ultimate date/time of their death/euthanasia. Many dogs live a considerable time in retirement as a family pet often with their old handlers. When retirements are excluded, the mean age that a NZ PDS dog died or was euthanased was only 5.65 years. This age is considerably lower than the 10 years reported for a large cohort of US Military working dogs by Moore *et al.* (2001). That study only considered dogs that were euthanased or died and dogs that retired were excluded. There are a number of reasons that could account for the difference between Police dogs in NZ and a US Military dog. There could be differences in: thresholds for euthanasia (cost, willingness to pursue necessary surgery), thresholds for selection into training, availability of dogs, access to veterinary care, genetic quality and breeding program success. As an example the NZ PDS has recently been considering a dog's lumbosacral disease status as a criterion for breeding selection. A US Military dog may also be redeployed to a less strenuous role such as a training or demonstration dog.

The US State Department maintains approximately thirty times the number of dogs as are used by the NZ PDS (Evans *et al.* 2007) and uses several breeds in addition to the GSD and Belgian Malinois. There are also hounds and sporting breeds, which are generally trained for detection work. When only GSDs were considered the mean age at death or euthanasia was still 10 years for the US dogs. The causes of loss were very

similar with appendicular degenerative joint disease, neoplasia and “spinal cord disease” being the most frequent (Moore *et al.* 2001).

In this study of NZ Police GSDs, the majority of dogs were retired (70%) rather than euthanased, dying of disease or being killed in action. The most common reason for retirement was inability to cope with the physical demands of the job. Of the retired dogs, 44% left service as a result of degenerative musculoskeletal disease, primarily attributed to lumbosacral disease and coxo-femoral arthritis. These findings are similar to findings in previous reports involving military working dogs, which share similarly physically demanding roles. In two other studies, canine hip dysplasia and secondary osteoarthritis of the coxo-femoral joints were reported to be the primary reasons for rejecting a dog during procurement evaluation and the most common reason for discharge of a military dog from active service (Dutton and Moore 1987, Olson 1971). However a study of 40 Police dogs (predominantly GSDs) found that the radiological progression of hip dysplasia did not correlate with the period of work, and had no influence on working ability, with no dog being retired primarily due to CHD (Zorko *et al.* 2008).

Of those dogs euthanased in our study, 33% were euthanased as the result of “spinal/back” problems and the average age at euthanasia was significantly younger than the average age of those at retirement. Spinal disease was the single most prevalent reason for euthanasia in NZ Police GSDs, whereas it was the third most common cause for early removal of dogs from the United States Military Dog Program between 1993 and 1996 (Moore *et al.* 2001). A later study of US Department of Defence dogs between 2000-2004 showed that “spinal cord disease” was the most common cause for discharge (60.0%). The next most prevalent cause of discharge from service was degenerative joint disease alone or in combination with spinal cord disease (26.3%) (Evans *et al.* 2007). The general terms ‘spinal cord disease’ and ‘spinal disease’ used in these studies likely encompass degenerative intervertebral disc disease, degenerative lumbosacral stenosis and degenerative (GSD) myelopathy. Our study relies entirely on handler knowledge and recall rather than confirmation of a diagnosis by a veterinarian or information from veterinary records or from post-mortem analysis and therefore the results should be interpreted with caution. Thirty-seven per cent of the dogs were retired or lost from service more than 10 years prior to the survey date. Therefore it is reasonable to assume some inaccuracy of recall by the handlers. The survey used the term ‘back/spinal disease’ and handler selection of the anatomical

region without further specification of diagnosis, as such the results provided should be considered indicative rather than precise.

The author's practice, the Massey University Veterinary Teaching Hospital (MUVTH) provides a referral service to the NZ PDS. Degenerative lumbosacral stenosis (DLSS) is the most prevalent disorder of the lumbosacral (LS) junction seen in working NZ Police GSDs presented to the MUVTH. In contrast degenerative myelopathy is rarely diagnosed in NZ Police GSDs during their working life. However, the inability to confirm degenerative myelopathy ante-mortem prevents accurate assessment of its prevalence within the population. In the author's experience there is compelling anecdotal evidence that the majority of the NZ Police GSDs that retired or were euthanased due to lower back/spinal issues in this study were indeed suffering from DLSS.

DLSS is characterised by degeneration of the lumbosacral articulation and subsequent compression on the *cauda equina* manifesting as caudal lumbar pain and pelvic limb disability (Meij and Bergknut 2010) see chapter five. It has a multifactorial aetiology, with the GSD having the highest breed incidence for degeneration of the lumbosacral disc (Bergknut 2011). A lesion resembling osteochondrosis has been reported to affect the dorsal end plate of the first sacral vertebra in the GSD (Hanna 2001, Lang 1992, Mathis *et al.* 2009) and to occur with increased incidence in dogs with *cauda equina* syndrome compared with dogs unaffected by DLSS (Hanna 2001). Affected dogs have ventral extradural compression associated with dorsal displacement of the osteochondral fragment from the edge of the first sacral vertebra. Only one dog with an osteochondrosis-like lesion was identified in this study. Many of the dogs included in the survey underwent investigation for DLSS and several underwent successful surgical decompression and/or stabilisation procedures (Worth unpublished data, chapter six). In a study based on pet insurance data from Sweden the GSD had the highest breed risk of disease related to degeneration of the lumbosacral intervertebral disc and a 7% lifetime prevalence of any disc degeneration related disease before the age of 12 years (Bergknut *et al.* 2012).

Dogs with transitional vertebrae have a higher risk of developing *cauda equina* syndrome (Morgan *et al.* 1993). Lumbosacral transitional vertebrae occur more frequently in GSDs than in other breeds (Morgan *et al.* 1993). Dogs with transitional vertebrae were eight times more likely to develop *cauda equina* syndrome than dogs without them, and GSDs were eight times more likely to develop *cauda equina* syndrome than other breeds, and at a significantly younger age (Fluckiger *et al.* 2006).

The incidence of transitional vertebrae in GSDs has been reported to range from 3.5 to 29%, and have a varying incidence within GSDs populations from different countries (Damur-Djuric *et al.* 2006, Scharf *et al.* 2004, Wigger *et al.* 2009). The incidence of transitional vertebrae amongst the dogs in the current study was not investigated. From approximately 2008 the NZ PDS began radiographic screening of breeding dogs for transitional vertebrae and osteochondrosis on the author's advice that dogs with transitional vertebrae should not be used as breeding stock.

All breed-associated diseases with proven high incidence in comparison to other dog breeds are suspected to have a genetic basis (Patterson 2000). Other than the elimination of dogs with transitional vertebrae or sacral osteochondrosis, suitable recommendations have not been developed to enable selection of dogs with a lower risk of developing pathology of the lumbosacral region. Future studies should seek to determine anatomical or work-related factors that may influence the development of DLSS. Studies into the characteristic stance and gait of the GSD and their relationship to conformation of the lumbosacral and coxo-femoral joints have not been undertaken.

Behavioural problems (mainly undesirable aggression) were the cited cause of loss in 7% of NZ PDS dogs. Undesirable aggression in an inappropriate situation may involve fear, or excessive (poorly controlled) aggression, whereas desirable aggression is seen as bold, non-fearful aggression shown in an appropriate operational situation. In comparison, other authors reported higher incidences of behavioural/aggression problems in working dogs as a cause of loss from service (Evans *et al.* 2007, Haverbeke *et al.* 2009). Behavioural problems were the most common cause of discharge for adult military working dogs <4 years old (Evans *et al.* 2007). The low level of undesirable aggression or of behavioural problems seen in this study suggests that effective breeding and selection of dogs, and appropriate training methods within the NZ PDS has largely avoided behavioural problems.

Police dogs are also selected for tracking ability and operational bite work. The loss of tracking ability was cited as a reason (primary or secondary) for the retirement of 11% of the retired Police dogs in this study. Successful tracking involves innate ability in addition to training and as such, reluctance or apparent inability to track may in part be due to physical inability or to behavioural issues. The loss of bite work ability was a secondary reason for the retirement of 12% of Police dogs and this loss may also have been due to either motivational/behavioural causes or a physical problem such as fractured canine teeth. Future analysis of the causes of tracking loss, bite ability loss

and aggression problems would be worthwhile to provide an opportunity to mitigate future losses.

Gastric dilatation - volvulus was a significant cause of death/euthanasia in this study. Police dogs are large breed, generally male dogs and for logistic reasons are often fed only once a day, all factors that are recognised as increasing the risk of GDV (Glickman *et al.* 1997). Feeding a large breed dog prior to exercise has been suggested as a potential risk factor for GDV (Lauten 2006), however such a situation is unavoidable in PDS dogs as they may be called on at any time to respond to an emergency. Theyse *et al.* (1998) found no association between the incidence of GDV and the interval between feeding and daily exercise in Great Danes and the role of post-prandial exercise in GDV remains controversial. Prophylactic gastropexy is an effective means of preventing GDV and is cost effective in some at-risk breeds depending on the local price differentiation between treatment and preventative surgeries (Ward *et al.* 2003). Given the high expense of breeding and training a Police GSD, prophylactic gastropexy may be cost saving to the NZ Government. A detailed cost-benefit analysis is warranted.

Study limitations

The NZ PDS does not have a searchable medical records system from which to generate data on loss/retirement from service. Therefore contacting handlers and asking them to provide the detail on their current and previous dogs was undertaken, and was the only way to obtain the data required. There are numerous deficiencies in conducting a survey in this manner. Police records were used to contact those handlers with addresses on file but only a subset of the total number of current or ex-dog handlers were contactable for this study by this means.

In this study considerable reliance was placed on handler recall thus introducing the possibility of recall error. The survey recruited data over 36 years and 4% of the dogs in the study were lost/retired from the PDS prior to 1990, with 50% of the dog's retired/lost before 2004. Our survey also relied on handler understanding of the medical state of the dog at euthanasia/retirement or the cause of death. For logistical reasons reported data could not be cross-referenced against veterinary or Police records. However, the handler/dog bond is strong and it is unlikely that each handler would be ill informed or otherwise unaware of the condition(s) affecting his or her dog at the point of loss from service. Previous studies of military working dogs in the United States (Dutton and Moore 1987, Moore *et al.* 2001) have published necropsy data and

are therefore likely to be more accurate regarding the pathological diagnosis than information from the present study. Unlike the US Military Working Dog program, the NZ PDS has no formal requirement for dogs lost to the program to be necropsied.

Conclusion

The average age of at which a NZ Police dog was lost from service was lower than the nominal age of retirement currently expected by the NZ Police. Degenerative orthopaedic disease including appendicular osteoarthritis secondary to hip dysplasia and lower back pain presumed to be due to DLSS were the most important causes of loss from service in this study. The remaining chapters of this thesis are divided into two sections, those pertaining to canine hip dysplasia and those focussed on degenerative lumbosacral stenosis.

Chapter two will review canine hip dysplasia and the use of breeding selection based on hip phenotype. Research into the current effectiveness of breeding selection to minimize the incidence of hip dysplasia in the working population is warranted and is the subject of chapter three. A new strategy to improve selection of suitable genetic stock based on the use of distraction radiography will be the subject of chapter four.

Greater research effort should also be targeted at identification of the factors that lead to lumbosacral disease and methods of lowering its incidence in the working population. Such research will improve animal welfare and should lengthen the average working life of a Police GSD and prevent this debilitating and painful condition.

An abstract entitled “Causes of loss from active duty of German shepherd dogs in service with the New Zealand Police-Dog Section.” was presented by the author as a podium presentation at the WSAVA conference in Auckland March 2013 and as a poster at the International Working Dog Conference in San Antonio October 2013.

A peer-reviewed manuscript has been published in the Journal of Animal Welfare. *Causes of loss or retirement from active duty for New Zealand police German shepherd dogs. Author(s): Worth, AJ; Sandford, M; Gibson, B; Stratton, R; Erceg, V; Bridges, J; Jones, B. ANIMAL WELFARE Volume: 22 Issue: 2 Pages: 167-174 DOI: 10.7120/09627286.22.2.167 MAY 2013*.

Chapter Two

Canine Hip Dysplasia in working dogs – Literature Review

Co-authored by Magdeline Rui Shi Soo

Introduction

Canine hip dysplasia (CHD) is a developmental condition which is characterised by instability of the hip joint, leading to degenerative arthritis (Todhunter and Lust 2003). It primarily affects medium-sized and large-breed dogs. Canine hip dysplasia was first described in 1937 by Schnelle as a congenital subluxation of the coxo-femoral joint (Schnelle 1937). Hip dysplasia is initially detected radiologically as subluxation of the femoral head in young dogs. As the cartilage of the coxo-femoral joint degenerates, osteophytes develop on the margins of the acetabulum and femoral head, which are detectable on radiographs (Todhunter and Lust 2003).

Canine hip dysplasia is described as a heritable and multifactorial disorder, with its expression influenced by the effect of several genes (polygenic) and many, often unidentified, environmental factors (Hedhammar *et al.* 1979, Cook *et al.* 1996a, Bliss *et al.* 2002, Smith *et al.* 2006). Both diet and exercise have been implicated as significant environmental factors that influence the development of hip joint abnormalities (Hedhammer *et al.* 1974, Resnick 1974, Tvedten *et al.* 1977).

Canine hip dysplasia is characterised by pain from the coxo-femoral joint leading to lameness, stiffness and a progressive decline in function of the joint. It is a disease of particular importance to working dogs of susceptible breeds affecting their athletic performance (Kapatkin *et al.* 2002a). In severe cases clinical signs develop between 5 and 18 months of age. Lameness, a bunny-hopping gait and pain induced by manipulation of the hip joint are noted on examination.

Canine hip dysplasia is recognised as one of the most common orthopaedic diseases affecting companion animals. In the previous chapter, arthritis of the coxo-femoral joint was shown to be a major cause of early retirement of NZ Police GSDs that were unable to cope with the physical demands of their job. Therefore, it is important for the NZ PDS to reduce the incidence of CHD in their dogs. Recognition of the heritable nature of the disease and use of effective methods for selection of breeding stock is critical to achieve a reduction in the incidence of CHD. This chapter will summarise the current understanding of the pathogenesis, heritability and diagnosis of CHD, and outline criteria for selection of breeding stock with a favourable phenotype.

Pathogenesis of CHD

CHD is a developmental, rather than a congenital condition, since the coxo-femoral joints of dogs that later become dysplastic are normal and congruent at birth (Riser 1975c). The anatomical conformation of the coxo-femoral joint and its surrounding structures are genetically determined. During growth, correct development of the coxo-femoral joint is dependent on joint congruency and the balance of forces applied across the joint. An alteration in these factors may affect or interfere with development of the hip joint (Riser 1963, Riser 1975b, Frost 1989). Hip dysplasia is reported to affect both coxo-femoral joints in more than 90% of affected dogs (Fox *et al.* 1987, Fry and Clark 1992, Cook *et al.* 1996), however unilateral disease has been described by Cook and others (1996).

In the standing dog, approximately 30% of the body weight is distributed to, and carried by, the pelvic limbs (Prieur 1980). During normal weight bearing, forces are transmitted up the femoral shaft, through the femoral neck and head, across the coxo-femoral joint to the pelvic girdle and finally along the vertebral column. The transfer of the force generated by the pelvic limbs to the coxo-femoral joint creates pressure on the joint surfaces, which is uniformly distributed to the acetabulum in a normal dog (Prieur 1980). The actual load applied to the coxo-femoral joint is determined by body weight and the degree of physical activity.

The hip joint is stabilised by its joint capsule and the surrounding pelvic musculature. In 1966, Henricson, Norberg and Olson first described a link between early joint laxity and the later development of CHD. When laxity of the femoral head is present, the stabilizing structures are assumed to fail to restrain the head of the femur within the acetabulum. The femoral head shifts into a more lateral position during weight bearing, and force is concentrated on the dorsal acetabular rim resulting in higher stress (stress = force/area). Prior to 6 months of age, the dorsal acetabular rim in dogs is largely cartilaginous and very plastic (Riser 1975c). Concentration of weight bearing force on the latero-dorsal rim leads to micro fractures and modelling of the acetabulum (Riser 1963, Riser 1975c, Cardinet *et al.* 1997), the joint capsule is stretched and its attachments to the labrum can tear. After six months of age, changes in joint shape are only possible through the production of, or resorption of, bone (Riser 1993). Therefore, the phenotypic expression of CHD in genetically susceptible dogs may be preventable if coxo-femoral joint congruency can be maintained until ossification of the acetabulum is complete (Riser 1975c). The first 60 days of a puppy's life is considered to be the most critical period of development of the coxo-femoral joint, during which the joint is susceptible to modelling under abnormal stress loading (Riser 1963, 1975a). During

this period a puppy may be presented to a veterinarian because of pain from acetabular micro-fractures and traumatic synovitis (Soo and Worth 2015). Clinical signs often improve with conservative management (weight reduction, confinement, analgesics) and many dogs become free of clinical signs until osteoarthritis develops and progresses to the stage of full-thickness cartilage loss later in life (Soo and Worth 2015). In mild cases the degree of laxity and change in loading is insufficient to induce osteochondral lesions that result in lameness (Soo and Worth 2015). In more severe cases the trauma to the dorsal acetabulum caused by excessive loading leads to abrasion of the articular cartilage, inducing synovitis and effusion (Soo and Worth 2015). Changes to the composition of the synovial fluid within the dysplastic hip joint reduce the quality of lubrication of the cartilage leading to increased cartilage wear (Riser 1975a, Fox *et al.* 1987). As osteoarthritis progresses, joint effusion worsens the stability of the joint by a concurrent loss of hydrostatic pressure (Smith *et al.* 1990). These osteoarthritic changes further reduce stability of the joint sometimes to the extent of coxo-femoral subluxation or complete luxation (Riser 1975a).

The role of muscle development surrounding the coxo-femoral joint in CHD

Whilst the concept of laxity leading to subluxation is well accepted, the initiating cause of laxity is still uncertain (Soo and Worth 2015). The prime stabilisers of the coxo-femoral joint are the surrounding pelvic muscles, which actively hold the femoral head within the acetabulum. The role of muscle development in the pathogenesis of CHD has been investigated (Riser and Shirer 1967, Riser 1976, Ihemelandu *et al.* 1983, Fox *et al.* 1987, Cardinet *et al.* 1997). Riser and Shirer (1967) performed a study of 93 dogs representing three breeds, of which two had a high incidence of hip dysplasia (the GSD and July hound) compared to the control breed (the Greyhound, considered by investigators to be CHD-free). Pelvic radiographs were assessed as a measure of CHD, and the dogs were euthanased for dissection and the combined weight of specific pelvic muscles was expressed as a ratio to total body weight (pelvic muscle mass index). There was a higher incidence of CHD in GSDs and July hounds with a lower pelvic muscle mass index, independent of breed. Riser (1974) proposed that canine hip dysplasia represents a disparity between primary muscle mass, and too rapid growth of the skeleton. Inadequate muscle tone could lead to joint instability whereby the congruency of the femoral head and acetabulum would be lost during development. The growing hip is plastic and becomes deformed due to the abnormal loading resulting from incongruity. However, it was later argued that differences in pelvic muscle mass may be sequelae of CHD [i.e. disuse atrophy] rather than a contribution to the cause of CHD (Gustafsson *et al.* 1975).

Cardinet *et al.* (1997) performed a prospective breeding study to investigate the association between the pelvic muscle mass and hip dysplasia. Dogs from a dysplastic breed, the GSD, were mated with dogs of a non-dysplastic breed, the Greyhound, and both pure lines and crossbred dogs assessed for pelvic muscle mass and the presence of CHD. The Greyhounds had the greatest pelvic muscle mass, followed by the crossbred dogs, and finally the GSDs. GSDs affected with CHD had significantly reduced pelvic limb muscle mass compared to those GSDs that were classified as not having CHD. When compared to unaffected counterparts within the same breed, GSDs affected with CHD had significantly smaller pelvic limb musculature – in particular the muscles: quadriceps, sartorius, iliopsoas, adductor magnus et brevis, gracilis, pectineus, gluteus medius, semimembranosus and quadrates femoris; this is accompanied by a significantly larger abductor cruris caudalis (Cardinet *et al.* 1997, Soo 2013). A non-uniform reduction in pelvic muscle mass was also seen in CHD affected crossbred dogs. Reduction in pelvic muscle mass was significantly associated with a greater severity of grossly visible pathological changes within the coxo-femoral joints at necropsy (Cardinet *et al.* 1997). The authors proposed that the pelvic muscle mass is decreased in dogs with CHD as a genetically determined trait, making this disease a primary developmental abnormality of the pelvic musculature rather than a pattern of atrophy and hypertrophy secondary to hip dysfunction (Cardinet *et al.* 1997). A primary developmental neuropathy of the pectineus muscle was supported by a paucity of type-2 myofibres on histochemical staining of pectineus muscle sections with myosin-ATPase.

In 1968, Bardens and Hardwick proposed that excessive and prolonged contraction of the pectineus muscle had a role in the development of CHD. A necropsy study reported increased tension of the pectineus muscle, especially during abduction of the hip, as compared to other muscles of the hip in dysplastic dogs. Surgical transection of the insertion of the pectineus muscle was performed in one pelvic limb of bilaterally affected CHD puppies no older than 8 weeks of age, with the un-operated contralateral pelvic limb serving as a control. By eight months of age there was little evidence of hip laxity and there was a radiological improvement in the dysplasia grade of the hip joint of the operated limb. Pectineus muscle specimens from two of the dysplastic puppies revealed histopathological evidence of fibrosis and atrophy of muscle fibres. Subsequent studies later identified further pathological, functional and developmental abnormalities in hypotrophic pectineal muscles (Cardinet *et al.* 1969, Cardinet *et al.* 1982). Ihemelandu and others (1983) reported smaller type I and type II myofibres and a larger non-myofibrillar tissue component in the pectineus muscles of 2 month-old GSD puppies which went on to develop clinical signs of CHD by 24 months of age. The

authors proposed that the fibre composition of the pectineus muscle may be an important associative or causal factor in the development of CHD (Ihemelandu *et al*. 1983). However, subsequent clinical trials failed to support long-term success of surgical pectineal myotomy/tenotomy and radiological signs of CHD were shown to worsen after surgical pectineal myotomy (Bowen *et al.* 1972, Cardinet *et al.* 1974, Vaughan *et al.* 1975). By the mid-1990s pectineal myotomy/tenotomy was no longer advocated as a treatment option for CHD (Cook *et al.* 1996).

The role of hormonal influences in the development of CHD

The maternal hormone relaxin is produced by the corpus luteum of the ovary, the mammary glands and, during pregnancy, by the placenta. It has been suspected to have a role in the development of CHD due to a link between heightened sensitivity of collagen to relaxin and associated ligamentous laxity thereby predisposing to coxo-femoral subluxation in women (Wilkinson 1963). In dogs, Goldsmith *et al.* (1994) found that the serum concentration of relaxin was significantly higher and persisted for up to six weeks post-partum in dysplastic lactating bitches, compared to only the first fortnight post-partum in non-dysplastic lactating bitches. Maternal relaxin was identified in the foetal circulation prior to suckling [*in-utero* transfer] then sustained at a low concentration in suckling puppies for up to four weeks [oral absorption] (Goldsmith *et al.* 1994).

Experimental evidence has also implicated a role for oestrogen in the development of the canine coxo-femoral joint (Pierce *et al*. 1965). Exogenous administration of estradiol-17β to puppies at 2 or 6 months of age led to significantly increased urinary estradiol-17β only in dogs from dysplastic parentage but not in dogs from lines free of dysplasia (Pierce and Bridges 1967, Paatsama *et al.* 1968). Experimental studies by Steinetz *et al*. (2008) later showed that radiological indices of hip laxity were increased when non-dysplastic puppies were treated with oestradiol-17β and synthetic canine relaxin. Interestingly, the same study found no significant difference in either the milk or serum concentrations of both total oestrogen and relaxin in dysplastic and non-dysplastic dams. There was also no significant difference in serum relaxin concentrations between groups of dysplastic and non-dysplastic puppies. The authors suggested that when these maternal hormones are transmitted to the systemic circulation of puppies, they could still potentially induce laxity in the soft tissues supporting the coxo-femoral joints in puppies that were genetically predisposed to CHD. They proposed that hip joint laxity might result from premature or inappropriate expression of oestrogen receptors, relaxin receptors, or both, in the connective tissues of the hip joints of puppies susceptible to CHD (Steinetz *et al.* 2008). Genetic

predisposition to coxo-femoral joint laxity may be expressed through humoral signalling including a role for relaxin and oestrogen regulation and reception.

The role of synovial fluid volume in the development of CHD

In the healthy joint, synovial fluid forms a thin layer between adjacent congruent cartilage surfaces and a force is required to break the fluid coupling. With a normal synovial fluid volume any lateral displacement of the femoral head is countered by a hydrostatic pressure gradient. The joint capsule invaginates and there is a negative pressure effect that pulls the femoral head back into the acetabulum. This phenomenon has been termed the hydrostatic stability factor (Smith *et al.* 1990) and has been estimated to contribute approximately 50% of the constraint on lateral displacement of the femoral head. In absence of the hydrostatic stability factor, lateral displacement of the femoral head is only limited by the tensile properties of the joint capsule and the round ligament (Smith *et al.* 1990).

When the intra-articular fluid volume was increased by infusion of a small amount of either: isotonic saline, air or silicone into the coxo-femoral joint, the hydrostatic stability factor was eliminated with subsequent equilibration of pressure across the joint capsule. As a result, a proportional net increase in lateral displacement of the femoral head was seen (Lust *et al.* 1980, Smith *et al.* 1990). Conversely, after a small volume of synovial fluid was withdrawn from subluxated coxo-femoral joints, there was radiological evidence of reduced joint laxity and associated subluxation (Lust *et al.* 1980).

Olsewski *et al.* (1983*)* demonstrated that dysplastic dogs had increased synovial fluid osmolality and volume compared with dogs with normal hips. Synovial fluid osmolality is related to plasma osmolality and may be influenced by dietary anion gap. With the adjustment of dietary sodium, potassium and chloride concentrations in food fed to CHD-afflicted puppies, it was shown that feeding a diet with a decreased dietary anion gap significantly improved hip scores when treated dogs were re-radiographed at 2 years of age (Kealy *et al.* 1993). Further studies are needed to determine if joint effusion and synovial fluid osmolality are causal of or consequential to joint laxity and hip dysplasia (Soo 2013).

The role of nutrition in the development of CHD

Large and giant breeds of dogs that undergo rapid weight gain as puppies are predisposed to CHD (Riser *et al.* 1964, Kasström 1975, Riser 1975d). Kasström (1975) revealed that puppies with a pattern of weight gain above the standard growth curve expected for the breed had a higher frequency and severity of CHD than puppies with a pattern of weight gain below the standard breed weight gain curve. Rapid weight gain

in GSDs in the first 60 days of life has been associated with an increased incidence of CHD in adulthood (Riser *et al.* 1964). For German Shepherd dogs of the same gender, heavier males and females tended to be affected with CHD compared to their counterparts of a lighter body weight (Riser *et al.* 1964). In a lifetime study of Labrador retrievers, feeding a limited caloric ration significantly reduced the incidence and severity of CHD in puppies compared to littermates fed *ad libitum* (Kealy *et al.* 1992). Dogs that had restricted dietary intake were held to a lighter bodyweight throughout their life but were still above ideal bodyweight based on body condition scoring. Nevertheless, the incidence of CHD was reduced by 38% in the restricted feeding group based on the Orthopedic Foundation for Animals (OFA) grading system (Kealy *et al.* 1992). The effect of weight gain on the incidence of CHD was recently confirmed by a study reporting the relationship of body mass index (BMI) to the presence of CHD (Comhair and Snaps 2008). The BMI is the ratio of a dog's body weight to its size (body dimensions). Comhair and Snaps (2008) reported that breeds with a higher BMI have a higher prevalence of hip dysplasia.

These studies have led to the concept that 'over-nutrition' promotes the expression and determines the severity of CHD (Riser *et al.* 1964, Lust *et al.* 1973, Kasström 1975, Kealy *et al.* 1992). It is important to observe that over-nutrition in itself has not been suggested to cause CHD, just that over-nutrition maximises expression of the phenotypic trait in genetically susceptible animals (Lust *et al.* 1973, Hedhammar *et al.* 1974). Conversely, feeding a genetically susceptible individual a restricted caloric intake promotes the development of a phenotypically "normal" hip. Whilst restricted caloric intake may be beneficial to the individual animal, if phenotypically "normal" but genetically affected dogs are used for breeding their dysplastic genotype may be inadvertently added to the gene pool (Lust and Farrell 1977).

The role of dietary calcium in the development of CHD

Calcium plays an important role in early skeletal development, yet abnormalities in calcium metabolism are not explicitly implicated in the development of CHD. In the classic experiments of Hedhammer *et al.* (1974), Great Dane dogs were fed a high protein, energy dense diet *ad lib*, resulting in over-nutrition and excess calcium intake. Skeletal pathology included osteochondrosis and stenosis of the cervical vertebral canal but not classical CHD. The authors described reduced modelling of the femoral head and neck, and the development of a more obtuse angle between the femoral neck and shaft (angle of inclination). Laxity, or the development of degenerative joint disease was not reported. In later studies with controlled caloric intake, immature Great Danes fed a dietary excess of calcium showed significantly heightened absorption and

retention of calcium, with minimal demonstration of protective mechanisms against hypercalcaemia and clinical hypercalcitonism (Hazewinkel *et al.* 1985, 1991). Chronic hypercalcaemia in large breed dogs may be accompanied by concomitant hypophosphataemia. As a result of the imbalance in the calcium:phosphorus ratio, there is delayed bone maturation, modelling and endochondral ossification of the immature skeleton manifested as osteochondrosis, retained cartilaginous cores and radius curvus (Hazewinkel *et al.* 1985, Schoenmakers *et al.* 2000). It is recommended that excess dietary calcium supplementation be avoided in dogs genetically predisposed to developmental orthopaedic conditions (Richardson 1992, Fries and Remedios 1995).

The role of exercise in the development of CHD

Biomechanical overloading of the immature coxo-femoral joint in genetically susceptible dogs is considered to contribute to the development of CHD (Riser 1963, Riser 1975b, 1975c). Therefore strenuous exercise during the crucial first 60 days of post-natal development is believed to exacerbate the degree of joint laxity and enhance development of CHD in puppies. In 1963 Riser showed that severe exercise restriction in the form of cage confinement for prolonged periods at an early age had a protective effect on the architecture of the coxo-femoral joint, and decreased the incidence of the disease in susceptible individuals. It was thought that during cage confinement, a puppy is more likely to remain seated with the pelvic limbs in abduction for extended lengths of time, thus generating less stress across the coxo-femoral joint. It was estimated that if hip stress in a standing puppy is taken to be 1, stress increases by a factor of 1.5 at the walk and 3.5 when running. If the degree of stress exceeds the active constraints around the hip joint in the standing or walking animal, the femoral head can subluxate and abnormal load is then concentrated on the acetabular rim. In a puppy where the skeleton is still susceptible to plastic deformation, loading applied to the dorsal acetabular rim leads to permanent modelling of the acetabulum. To prevent this effect an abduction hip brace is used in human infants with hip dysplasia (Riser 1963). The abduction hip brace is designed to hold the legs in abduction so that the femoral heads are “driven” into the acetabulae (Soo 2013). Forces acting across the hip joint are more evenly distributed until ossification has progressed past the point where acetabular and femoral head modelling can occur. If CHD-susceptible puppies are confined in cages with more time spent seated, thus allowing the femoral head to be reduced into the acetabulum until ossification of the acetabulae is complete, then the degree of dysplasia will be minimised (Riser 1963).

Krontveit *et al.* (2012b) have challenged the recommendation for severe exercise restriction suggesting that developing muscle strength through exercise could positively influence normal development of the coxo-femoral joint. They performed a prospective cohort study in Norway to identify risk factors associated with the onset of clinical CHD. This study involved privately owned large dogs of four different breeds followed from birth till 9 years of age. The study revealed that controlled exercise from a young age may be beneficial in strengthening muscle mass and improving the range of motion of joints, including that of the hip, thereby delaying the time of onset of CHD-related clinical signs (Krontveit *et al.* 2012b). A similar study by Krontveit *et al.* (2012a) also showed that monitored "off-leash" exercise on soft ground (grass or non-slippery surface) from birth up till three months of age might prove beneficial for the development of their hip joints in puppies. However, the study also found that walking up and down stairs daily from weaning to three months of age might increase the risk of development of CHD. It was hypothesized that immature neuromuscular function and poorer coordination in the puppy, may induce stress on the developing hip joint (Krontveit *et al.* 2012a). Finally, while moderate exercise may be favourable to the developing young puppy, some authors have suggested that boisterous activity such as playing with other dogs, jumping for a ball and chasing sticks, should be avoided as such activity may exert excessive stress on the immature hip joints (Sallander *et al.* 2006, Krontveit *et al.* 2012b).

The genetic basis of CHD

Canine hip dysplasia has been accepted to be a heritable condition since the 1950s (Janutta *et al*. 2006). In 1966, the OFA established a registry of inherited orthopaedic traits in dogs, and CHD was the initial focus (www.offa.org/search.html). Original hypotheses that a single gene with a recessive or dominant Mendelian pattern of inheritance was later doubted and the concept of incomplete manifestation and variable penetrance were promoted (Janutta *et al.* 2006). By the 1970s, a multifactorial mode of inheritance, a polygenic inheritance combined with environmental influences, was considered to be the most probable genetic basis for CHD (Lust and Farrell 1977, Hedhammar *et al.* 1979).

In order to understand the concept of a multifactorial mode of inheritance the concepts of phenotype and genotype must be defined. The expression of a trait is known as the phenotype, which is the sum of the genetic and environmental effects, expressed as P = G + E [where P is the phenotypic value; G is the genotypic value and E is non-genetic, environmental deviation] (Nicholas 2010e).

An animal's genotypic value is the combined effect of the animal's genes at all loci affecting the phenotype of interest. For any measured trait, G is fixed at conception and E is the effect of all environmental factors influencing trait expression between conception and measurement of P. Canine hip dysplasia may be expressed in a variety of phenotypes, each determined and measured by one or a combination of radiographic methods (Breur *et al.* 2001). For a quantitative trait, it is possible for two dogs of identical genotypes to express very different phenotypes under differing environmental influences. It is also possible that a genetically-susceptible individual may have a "normal" hip phenotype if environmental conditions are favourable (Fox *et al.* 1987).

Multifactorial inheritance refers to inheritance of a phenotypic characteristic (trait) that is attributable to two or more genes with an unknown number of non-genetic (environmental) factors. In such multifactorial diseases the actual number of genes determining the manifestation of the condition is commonly unknown. CHD is considered by many to be a quantitative trait due to its continuous phenotypic expression from normal to abnormal (Lust and Farrell 1977). Quantitative traits are determined by the action of different genes at many quantitative trait loci (QTL) and the effects of each of the individual alleles are often not immediately distinguishable. Quantitative trait loci (QTL) analysis is a statistical method that allows identification and allocation of complex phenotypic traits to certain locations on chromosomes by the use of molecular markers such as SNPs (single-nucleotide polymorphism) or FISH (fluorescent *in situ* hybridization) markers (Verhoeven *et al.* 2012). Non-genetic factors also account for variation in these quantitative traits (Lust and Farrell 1977, Nicholas 2010e).

Whilst a polygenic mode of inheritance has been widely accepted, work in the last two decades has suggested the possible existence of major gene loci influencing hip dysplasia (Leighton *et al.* 1977, Maki *et al.* 2004, Todhunter *et al.* 1999, 2003). Heritability (h^2) estimates for hip laxity using quantitative trait loci (QTL) analysis are high (0.5-0.6) and indicate that the distraction index (see PennHIP section) may be determined by a major gene locus (Todhunter *et al.* 2003). Similarly, Janutta *et al.* (2006) demonstrated the existence of a major gene responsible for the development of CHD in addition to additional polygenetic effects. The study used FCI scores and pedigree data from 8567 GSDs representing 20 families comprising 3 to 4 generations and involved complex segregation analysis. The authors rejected a pure polygenic model in this breed and argued for a major single gene with an autosomal dominant pattern of inheritance (with variable penetrance/expression). Given the known association between laxity and the development of CHD, a major genetic locus for

laxity would be a valuable marker to determine a predisposition for hip dysplasia in dogs.

Several key steps have been taken to develop a genetic test for CHD (Zhu 2009). The canine genome has been mapped (Breen *et al.* 2004) opening the possibility that genes responsible for the expression of CHD may be identifiable (Guo *et al.* 2011). Mutations related to hip laxity and Norberg's angle (see radiological section) have been identified by mapping the chromosomes of cohorts of dysplastic Labrador retrievers and disease-free greyhound crossbred dogs (Todhunter *et al.* 2005). Twelve candidate (approximate chromosomal) locations for CHD were found. Further work on the GSD genome using QTL analysis revealed 19 candidate loci associated with CHD, located on nine different chromosomes, of which dog chromosome (CFA) number 9 was the strongest possible candidate (Marschall and Distl 2007). In another genome-wide association study, Pfahler and Distl (2012) identified three QTL for CHD in Bernese Mountain dogs harbouring significantly associated single-nucleotide polymorphisms (SNP). Three SNP were found to be significantly associated with CHD on dog CFA 14 and 37, with candidate genes of interest being paraoxonase-2 (PON2) on CFA14 and fibronectin-1 (FN1) on CFA37. The PON2 and FN1 genes were hypothesised to be part of the pathogenesis of CHD due to their involvement with bone mineral density and cartilage extracellular matrix, respectively (Pfahler and Distl 2012). Four SNPs associated with CHD and two SNPs associated with hip DJD were also identified in a separate study (Zhou *et al.* 2010). Friedenberg *et al.* (2011) identified an association between a mutation-deletion haplotype in the fibrillin-2 gene (FBN2) and CHD. The FBN2 is the first gene reported to be associated with four phenotypic markers for the presence of CHD (the Norberg angle (NA), distraction index (DI), dorsolateral subulaxation (DLS) score and the Orthopaedic Foundation for Animals (OFA) hip grade, see later). Dogs homozygous for the deletion FBN2 haplotype were found to have worse hip joint conformation (i.e. more severely affected by CHD) as characterised by having a lower NA value and DLS score, higher DI and poorer OFA hip grade (see later for more information on hip scoring). It was also found that dogs with incipient osteoarthritis at necropsy had an approximately 50% greater FBN2 mRNA in their hip joint capsule in contrast to non-osteoarthritic dogs. However, Friedenberg and co-workers (2011) stressed that the FBN2 locus did not explain all the genetic trait variation observed in CHD, indicating that other genes must contribute to the expression of the disease.

Whilst current DNA marker technology is not sufficiently refined to be used in the selection of breeding animals, it may be available to breeders in the future (Marschall and Distl 2007, Zhu *et al.* 2008, Zhou *et al*. 2010). Sánchez-Molano *et al.* (2013) have

suggested that employing DNA markers as part of a genomic selection scheme would be an alternative to phenotypic selection as a means of reducing the prevalence of CHD. A genomic selection strategy involves a genomic (DNA) test that provides information on a large number of genotype markers in a training population of phenotypically-scored dogs. A subset of markers are then produced via linkage disequilibrium with the genes associated with the disease. These markers are later used to calculate genomic estimates of the true breeding values (genomic estimated breeding values, gEBV), which are in turn used for subsequent breeding selections within the same breed. In the simulated study population, those authors showed that genomic selection could have achieved greater genetic progress compared to selection based on the phenotype alone (British Veterinary Association hip scoring scheme) (Sánchez-Molano *et al*. 2013). Compared to phenotypic selection schemes, genomic selection accelerates the rate of genetic progress due to higher selection accuracy and also allows breeding selection to be carried out earlier because the DNA tests can be performed on animals at a younger age (Sánchez-Molano *et al*. 2013). Unlike phenotypic selection schemes, genomic selection removes environmental biases (such as age at scoring) which may artefactually influence breeding selection. One potential drawback of this selection strategy is that due to the decay of the linkage disequilibrium between the genotype markers and causative loci, the accuracy of the genomic selection will likely decrease over time. Therefore, re-estimates (via phenotypic scoring) of the marker effects will still be necessary every few generations in order to maintain the level of accuracy (Sánchez-Molano *et al.* 2013).

Commentators predict that genetic information will likely be used in combination with phenotypic testing, rather than replace radiological assessment (Stock and Distl 2010, Zhou *et al.* 2010). A combination of a genetic test for a major gene locus in combination with phenotypic scoring would represent an important development in CHD screening and determination of such criteria is the subject of ongoing research (Zhu *et al.* 2009). Until DNA marker technology has been further refined for routine use in screening for CHD clinicians and breeders will have to rely on other modalities of breeding selection in the interim, as discussed in the following sections (Woolliams *et al.* 2011).

Diagnosis

Clinical signs of CHD include unilateral or bilateral pelvic limb lameness, reluctance to exercise, difficulty when rising, joint stiffness and overt pain expressed on palpation of the hip joints. Affected puppies may present lame from 4 to 12 months of age as a result of micro-fractures and modelling of the acetabulum, whereas adults present as being lame due to the pain and immobility resulting from hip osteoarthritis. The stride length is shortened and a dog may bunny-hop rather than canter. On physical examination there is typically pain on abduction and extension of the hip, with a decreased range of motion due to peri-articular osteophyte formation and/or capsular fibrosis (Fry and Clark 1992, Riser 1993). Crepitus may be detected during manipulation of the coxo-femoral joint which may indicate eburnation of cartilage of the acetabular rim (Rettenmaier and Constantinescu 1991). Subluxation of the hip may be palpable when a hand is placed on the greater trochanter as the dog walks.

Manipulative tests

Young dogs may be screened for laxity of the coxo-femoral joint using the Ortolani, Barlow and Bardens tests (Fry and Clark 1992, Kapatkin *et al.* 2002). The Ortolani and Barlow tests were originally described in the human medical literature as a diagnostic method for detecting coxo-femoral laxity in newborn babies (Barlow 1962, Ortolani 1976, Soo 2013), but these tests were later applied to and refined for use in the dog (Chalman and Butler 1985). The Bardens manoeuvre appears to have only been utilised in veterinary patients (Bardens and Hardwick 1968, Bardens 1972).

The Ortolani test is well described in the veterinary literature as a test of coxo-femoral laxity (Chalman and Butler 1985, Fox *et al.* 1987, Fry and Clark 1992, Cook *et al.* 1996). Heavy sedation or general anaesthesia is recommended, especially in dogs with overt hip pain, in order to reliably demonstrate the Ortolani sign. The dog may either be positioned in lateral or dorsal recumbency. With the pelvis supported in one hand, the other hand is used to position the pelvic limb perpendicular to the vertebral column while grasping the stifle firmly. To elicit dorsal subluxation of the femoral head, pressure is applied down the long axis of the femoral shaft, directed towards the coxo-femoral joint. While still maintaining a firm hold of the stifle, the pelvic limb is then slowly abducted. The subluxated femoral head will suddenly reduce into the acetabulum, which can be felt as a sudden change in position of the femoral head and/or heard as a “clunk”. This “clunk” phenomenon is known as a positive Ortolani sign (Soo 2013). The presence of osteophytes and cartilage loss may also produce crepitus during joint manipulation which should not be confused with a positive Ortolani

sign (Chalman and Butler 1985). The presence of an Ortolani sign has been shown to be associated with the development of CHD. Dogs with a positive Ortolani sign at 16 weeks or after 1 year of age were 2.5 and 4.4 times respectively more likely to develop radiological signs of CHD compared to those dogs with a negative test (Ginja *et al.* 2009a). Correct detection of the disease depends largely on proper technique in executing the test. False negatives may occur with application of an improper technique, inadequate depth of anaesthesia, large patient size preventing effective palpation, fixed luxation of the femoral head, and peri-articular fibrosis with destruction of the dorsal acetabular rim (Chalman and Butler 1985). The Ortolani test has 92% sensitivity, 79% specificity, 78% positive predictive value, and 92% negative predictive value (Ginja *et al.* 2008a). A high sensitivity indicates that a positive Ortolani sign identifies the majority of dogs that have or will develop CHD, however the lower specificity means that the test is not accurate at identifying animals without the disease. The relatively low PPV means that a positive Ortolani is not pathognomic and does not necessarily imply a dog will develop clinical signs of CHD. But a high NPV means that there is a high likelihood that dogs without an Ortolani sign do not have or will not develop CHD. The Barlow sign is essentially the first part of the Ortolani test, where axial pressure is directed down the femoral shaft to produce femoral head subluxation, which constitutes a positive test (Fry and Clark 1992).

The Bardens test was designed to detect coxo-femoral joint laxity in puppies at a very early age, reportedly as young as four weeks old (Soo 2013). As with the Ortolani test, sedation may facilitate palpation. To perform the Bardens test, the patient is placed in lateral recumbency. On the non-dependent pelvic limb, the clinician's thumb is positioned on the ischiatic tuberosity, index finger on the greater trochanter of the femur, and middle finger on the dorsal iliac spine. The pelvic limb is firmly grasped at the thigh with the other hand, and the thigh is lifted laterally and perpendicularly to the pelvis without abduction (Soo 2013). The degree of laxity is estimated by the amount of movement of the index finger away from the dorsal acetabular rim. Upward displacement of the operator's index finger of more than 1 mm is considered to be abnormal and determines the need for further diagnostic investigation. The subluxated femoral head may be reduced by briskly pressing down on the greater trochanter (Bardens and Hardwick 1968). A more objective quantification of the degree of coxo-femoral laxity elicited with the Bardens test can be made using a simple lever device to increase the accuracy of this diagnostic method, which has been described elsewhere (Wright and Mason 1977). A study in 2000 that evaluated over 200 hip joints of dogs at an average age of 7.3 weeks found that the Bardens test was predictive of DJD for the collective study population of Golden retrievers, Labrador retrievers and

golden/Labrador retriever crossbred dogs (Adams 2000). However, when the analysis was performed by individual breed, the Bardens test was only predicative of DJD in golden retrievers. A subsequent study found that there was no association between a positive Bardens' test and the clinical manifestations of CHD (Ginja *et al.* 2009a). Therefore the reliability of this technique remains questionable (Soo 2013).

Even though physical examination is useful in identifying coxo-femoral joint laxity, manipulation of the hip joint is not consistently reliable in detecting laxity and therefore as a predictor of CHD (Adams *et al*. 1998, Adams *et al.* 2000, Ginja *et al.* 2009a). The reasonable conclusion is that definitive diagnosis of CHD requires pelvic imaging (Adams *et al.* 1998, Adams *et al.* 2000, Ginja *et al.* 2009a).

Radiography

Features of CHD on plain radiographs vary depending on the age of the animal when it is radiographed and these features have been extensively documented by Riser (1975a). In affected young dogs CHD is initially detected radiologically on an extended ventro-dorsal view as subluxation of the femoral head. Subluxation represents the degree of coverage of the femoral head by the margins of the acetabulum. The femoral head is poorly seated within the acetabulum of dogs affected by CHD, increasing the width of the joint space. It is generally accepted that subluxation of the femoral head is usually the earliest, and sometimes only, radiological manifestation of coxo-femoral joint laxity (Henricson *et al.* 1966, Bardens and Hardwick 1968, Riser 1975a).

Early subluxation is followed by osteophyte development on the acetabular borders and femoral head and/or neck as a dog ages (Riser 1975a, Todhunter and Lust 2003). Tearing of the joint capsule at its attachment at the femoral neck results in caudo-lateral curvilinear enthesiophyte formation at that site, known as the "Morgan's line" (Witte and Scott 2011). The acetabulum may appear shallower due to the acetabular fossa becoming filled with new bone. The femoral head and neck then undergo extensive modelling in severely affected animals. There is continual osteophyte development over the life of a dysplastic dog, indicating that osteoarthritis associated with CHD is progressive (Smith *et al.* 2006, Smith *et al.* 2012). Similarly, increased age at the time of assessment is significantly correlated with degenerative radiological changes associated with hip dysplasia (Hou *et al.* 2010). These radiological signs are indirect markers of cartilage degeneration secondary to abnormal biomechanics of the hip joint. In the early stages, the radiological signs lag behind cartilage injury but as a dog ages, radiography becomes more accurate at identifying the changes in hip joint morphology (Smith *et al.* 2012).

When a dog is younger than five years of age, the radiological diagnosis of CHD is reliant on evidence of femoral head subluxation and/or DJD (Smith *et al.* 2012). Those authors found that no new cases of subluxation were diagnosed after 2 years of age. In older dogs, radiological identification of CHD may encompass the presence of a shallow acetabulum, coxo-femoral incongruity and DJD (Riser 1975a). After 6 years of age, newly diagnosed cases of CHD appear to be dependent on radiological evidence of coxo-femoral DJD.

Phenotypic selection of dogs for breeding by utilising radiological signs.

Due to the current lack of readily available genomic tests for routine clinical use, and the difficulty in objectively quantifying clinical signs of CHD, radiological methods have been developed in an attempt to allow dog breeders to select less affected stock for breeding (Lawson 1963, Smith *et al.* 1990, Fluckiger *et al.* 1999, Lust *et al*. 2001). There are several methods of radiographic screening currently in use around the world (Verhoeven *et al.* 2012). The majority are based on images of the hips and pelvis taken under deep sedation or general anaesthesia with the dog in ventral recumbency and with the pelvic limbs extended. The dog may require deep sedation or need to be anaesthetised in order to obtain a suitable hip-extended image. This position gives a consistent view of the sites at which osteophytes typically develop including the femoral head/neck and acetabulum. Hip laxity can be indirectly observed as subluxation on an extended VD radiograph (Figure 6, page 31) but dogs with laxity can have apparently normal hip joints without radiological evidence of subluxation, due to the inherent positioning of the limbs for radiography and the level of sedation. If the patient is incompletely anaesthetised or sedated, it will respond to extension of the pelvic legs by contracting it's pelvic muscles, improving the seating of the femoral head within the acetabulum, falsely lowering the degree of subluxation. Additionally, when the coxo-femoral joints are positioned with the limbs in extension, the joint capsule and ligament of the head of the femur are both twisted, tightening the tensile elements of the joint capsule (Smith *et al.* 1990, Heyman *et al*. 1993). As the tensile elements tighten the femoral head is forced deeper into the acetabulum in a phenomenon known as "screw-home" tightening. The term is borrowed from human medicine. In the human knee, during the last 10 degrees of extension, a combination of extension and external rotation tightens the ligaments and joint capsule and drives the femur into contact with the meniscal cartilages, increasing stability. In the canine hip this 'screw-home' tightening can lead to false negative grades given when dogs are screened by radiography at 1-2 years of age as osteoarthritis may not be evident on radiographs in younger dogs and the assessor is relying on the evidence for subluxation or its

presence as an indicator of the severity of the disease and hip score status of the dog. Furthermore, the extent of laxity seen on the hip-extended VD is affected by the accuracy of positioning of the dog and the method of holding the limbs in extension. An oblique pelvic position increases the extent of femoral head coverage of one hip joint whilst lowering it on the other. If the dog's legs are hand-held (rather than being strapped in position for radiography) then the holder can also influence the degree of subluxation and falsely "tighten" the joints (Rendano and Ryan 1985). Additionally, because there is subjectivity in assessment of subluxation on the extended-hip VD, the assigned hip score may vary between different assessors with differing experience in interpretation of radiographs of hip joints (Verhoeven *et al*. 2009).

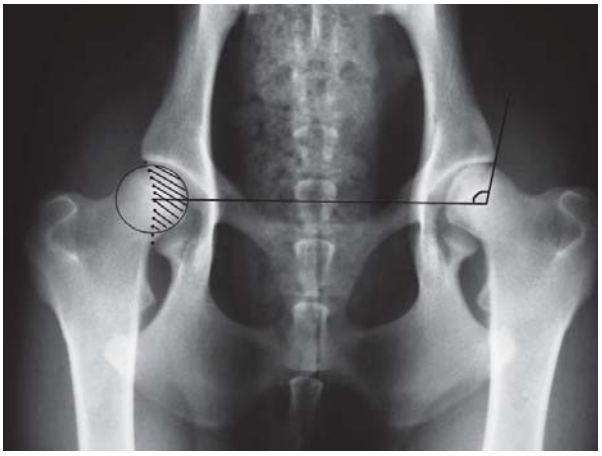


Figure 6. Norberg's angle (NA) [right hip pictured] and subluxation score (femoral head coverage): NA is the included angle between a line drawn connecting the centres of the femoral heads and another line from the centre of the femoral head to the outer limit of the cranial margin of the acetabulum in the ventro-dorsal radiographic projection. The degree of coverage of the femoral head (subluxation score) is determined by the area of the femoral head (lined region) within the acetabulum as determined by the position of the centre of the femoral head relative to the dorsal acetabular rim (dotted line) [left hip pictured]. (Soo and Worth 2015)

International scoring schemes for determination of CHD

The three most well utilized CHD radiographic schemes operate in the United States, Europe and the United Kingdom/Australasia (Fluckiger 2007).

In the United States the Orthopaedic Foundation for Animals (OFA) has been scoring hip radiographs of dogs in North America and Canada for CHD since 1966. Dogs must be over 24 months of age to be eligible for the scheme and a hip-extended ventro-dorsal view is taken under anaesthesia. In the OFA scheme there are nine anatomical areas of the coxo-femoral joint which are evaluated for osteophyte development: cranio-lateral acetabular rim, cranial acetabular margin, femoral head, fovea capitus, acetabular notch, caudal acetabular rim, dorsal acetabular margin, junction of the femoral head/neck and the trochanteric fossa. The degree of subluxation is subjectively assessed without measurement tools. The patient is then assigned to one of the seven phenotypes recognised under the OFA hip grading scale[1], which range from normal

[1] OFA: An Examination of Hip Grading http://www.offa.org/hd_grades.html

(excellent, good and fair) hip conformation to borderline, to dysplastic (mild, moderate and severe) hip conformation.

In Europe, the Federation Cytologique Internationale (FCI) is a cooperative of national kennel clubs which has been screening dogs for CHD for over 40 years (Flückiger 2007). Dogs must be over 12 months of age or over 18 months of age for the large and giant breeds. Accepted breed standards are listed on the FCI website (www.fci.be.nomenclature.aspx). The FCI scoring system combines subjective assessment of the degree of subluxation, congruity of the femoral head and acetabulum. and osteophyte development on a hip-extended ventro-dorsal radiograph with objective measurement of subluxation using the Norberg angle (NA, Figure 6, page 31). To calculate the NA on a hip-extended VD view of the pelvis, a line is drawn to connect the centres of the femoral heads. From each centre point a second line is drawn tangential to the most lateral margin of the cranial acetabular edge. The included angle is the NA. A normal NA is considered to be 105 degrees or greater (Morgan and Stephens 1985, Henry 1992). However, the upper limit of the "normal" value varies between breeds and should be interpreted with caution (Tomlinson and Johnson 2000, Culp *et al.* 2006). Additionally the NA is affected by 'screw-home' tightening, which masks subluxation and artificially increases the NA.

In the United Kingdom the British Veterinary Association (BVA) and The Kennel Club [UK] implemented a CHD screening program in 1965. Originally the program had a pass or fail system based on subjective assessment. The program was changed to a semi-objective point scoring system in 1984 using interpretation of a hip-extended ventro-dorsal radiograph and applying a numerical score to the indicators of subluxation, incongruity and degenerative joint disease (DJD) (Gibbs 1997). This scoring system was first developed in 1983 by Dr Malcolm Willis (Hunter 1986, Dennis 2012) and is often referred to as the "Willis" method. This technique of hip scoring is also utilised in Ireland, Australia and New Zealand (Flückiger 2007). Up to 53 demerit points are awarded per hip in 9 categories to yield a total score of 0 (best) to 106 (worst). The BVA system includes categories for NA, subluxation (femoral head coverage), (Figure 6, page 31), and cranial acetabular edge profile as measures of laxity and incongruency, (Table 3, page 33). The remaining six categories are the locations of typical osteophyte/enthesiophyte development. Dogs are eligible for assessment under this program from 12 months of age.

Table 3. British Veterinary Association / Kennel Club (UK) hip dysplasia scoring system components 1 to 3: Norberg's angle, Subluxation and Cranial acetabular edge, and their descriptors. Under the New Zealand Veterinary Association Hip Dysplasia Scoring System these three components constitute the "subtotal score". (Worth et al. 2009)

Score	*Norberg's angle*	*Subluxation*	*Cranial acetabular edge (CAE)*
0	*≥105°*	*'None': FHC well centred within the acetabulum*	*'Normal': Even, smooth curve of CAE parallel to the femoral head throughout*
1	*100–104°*	*'Slight': FHC lies within DAE, and the medial joint space increases slightly*	*CAE, as traced laterally, shows flattening, with only the lateral ¼ affected*
2	*95–99°*	*FHC overlies the DAE, and the medial joint space increases obviously*	*CAE flattened throughout most of its length*
3	*90–94°*	*'Moderate': FHC lies just lateral to the DAE, and approximately ½ the femoral head overlies the DAE*	*CAE is very flat, with slight cranial slope laterally (= slight bi-labiation)*
4	*85–89°*	*FHC is clearly lateral to the DAE, and ¼ of the femoral head overlies the DAE*	*CAE flat, and moderate bi-labiation*
5	*80–84°*	*'Gross': FHC is well lateral to the DAE but the femoral head touches the DAE*	*'S' deformity of CAE of moderate degree, with gross bi-labiation*
6	*≤79°*	*Total pathological luxation*	*Entire CAE sloped cranially*

FHC = femoral head centre; DAE = dorsal acetabular edge

The New Zealand Veterinary Association (NVZA) introduced the Canine Hip Dysplasia Scheme[2] in the mid 1980's (Hunter 1986) in cooperation with the NZ Kennel Club, with a national computerised database maintained from 1989. Dogs need to be at least 12 months of age to be eligible to be scored under this scheme. The three criteria deemed to be most indicative of joint laxity/incongruity are the Norberg angle grade, subluxation score, and the profile of the cranial acetabular edge (Gibbs 1997, Worth *et al.* 2009). The sum of the scores for these criteria form the 'subtotal score' of the NZVA CHD scheme, which was introduced as a means of highlighting the components of laxity and incongruity that are early indicators of CHD status prior to the development of DJD (Burbidge 2003).

[2] NZVA hip and elbow dysplasia http://www.nzva.org.nz/hip-and-elbow-dysplasia

These three criteria have been reported to constitute nearly 70% of the total scores in Labrador Retrievers graded using the BVA scheme in the UK (Wood et al. 2004). The subtotal score was introduced to the NZ Canine Hip Dysplasia Scheme to better guide breeders to select animals with no evidence of hip laxity for breeding. In 2003 a new recommendation was introduced, that a dog should not be bred from if it scored greater than 2 in either hip in the subtotal assessment section (Burbidge 2003). This recommendation replaced the previous recommendation that ideally only dogs with total scores ≤8 should be selected for breeding.

Verhoeven *et al.* (2012) published a table which attempted to compare subjective grading schemes for CHD (Table 4). Whilst designed to help breeders compare the schemes such a comparison between scheme grades is highly speculative.

Table 4. Subjective comparisons between the OFA, FCI and BVA/KC scoring systems (modified from Voethoeven et al. 2012).

Subjective Grade Descriptor	*Grading System and Grade*			
	Federation Cytologique Internationale (Europe)		*British Veterinary Association (UK)*	*Orthopedic Foundation for Animals (USA)*
No signs of hip dysplasia	*A*	*NA >105°*	*0-4 (not > 3/hip)* *5-10 (not >6/hip)*	*Excellent* *Good*
Near normal hip joints	*B*	*NA ≤105°**	*11-18* *19-25*	*Fair* *Borderline*
Mild hip dysplasia	*C*	*NA 100°*	*26-35*	*Mild*
Moderate hip dysplasia	*D*	*NA >90° but <100°*	*36-50*	*Moderate*
Severe hip dysplasia	*E*	*NA <90°*	*51-106*	*Severe*

**Norberg angle 105° with slight incongruency, or <105° with congruency*

Radiological assessment of CHD by distraction radiography

Building on the link between early joint laxity and later development of CHD described by Henricson *et al.* (1966), Smith and co-workers at the University of Pennsylvania, Philadelphia, introduced the concept of passive versus functional hip laxity to distinguish between subluxation evident radiologically compared to subluxation induced during weight-bearing (Smith *et al.* 1990). They proposed that the joint capsule, round ligament of the femoral head and the hydrostatic stability factor are passive constraints to hip laxity and each play a role in maintaining congruency between the femoral head and acetabulum. These passive constraints restrict the amount of hip extension possible as well as creating a force that acts to drive the femoral head into the acetabulum, minimising laxity. If the constraints maintained by the soft tissue surrounding the hip joint do not function properly, the coxo-femoral joint will not receive and effectively transmit load during weight bearing. The inability or failure of these soft tissue passive constraints to limit the amount of lateral displacement of the femoral head in the relaxed dog is defined as passive laxity. Functional laxity is defined as the lateral displacement of the femoral head out of the acetabulum during weight bearing. Passive laxity is thought to be a pre-requisite for functional laxity, but passive laxity may not always correlate with an equal extent of functional laxity (Smith *et al.* 1990). Passive laxity is inducible by the radiographer and is not the 'cause' of functional laxity, the latter resulting from weight bearing, but passive laxity is hypothesized to be strongly correlated to the likelihood of subluxation occurring during weight-bearing.

A stress-radiographic method (distraction radiography) using a radiolucent fulcrum placed between the femurs was developed to quantify the degree of passive laxity in an anaesthetised or heavily sedated dog. Passive laxity has been reported to be highly correlated with the development of osteoarthritis and the clinical signs of hip dysplasia (Smith *et al.* 1990, Smith *et al.* 1995). The University of Pennsylvania Hip Improvement Program (PennHIP®) became commercially available in 1993 with the introduction of training courses for veterinary personnel in distraction radiography (http://research.vet.upenn.edu). The PennHIP evaluation comprises three ventrodorsal radiographic views: extended-hip, compression and distraction views. The EHV is used to assess the presence or absence of osteoarthritis and the compression view is used to assess the overall coxo-femoral congruency. The presence of osteophyte development on the EHV results in the dog being classified as having CHD (PennHIP 2013).

The PennHIP distraction index (DI) is a unit-less measure of passive hip laxity and is calculated by determining the degree to which the femoral head subluxates relative to the acetabulum. The adjustable radiolucent fulcrum is placed between the thighs of heavily sedated or anaesthetised dogs and by gripping the distal limb, the femurs are pulled together inducing femoral head subluxation at which point a radiograph (the distraction view) is taken (Figure 7). The linear distance between the centre of the acetabulum and the centre of the femoral head on the distracted radiographic view is divided by the radius of the femoral head to achieve a unit-less measure of laxity; the distraction index or DI (Figure 8).

Figure 7. The PennHIP method of distraction radiography. A radiograph is taken with the dog heavily sedated or anaesthetised and positioned in ventro-dorsal recumbency with the distraction device placed between the thighs. The operator uses the device as a fulcrum to achieve distraction of the coxo-femoral joints (passive hip laxity). (Soo and Worth 2015)

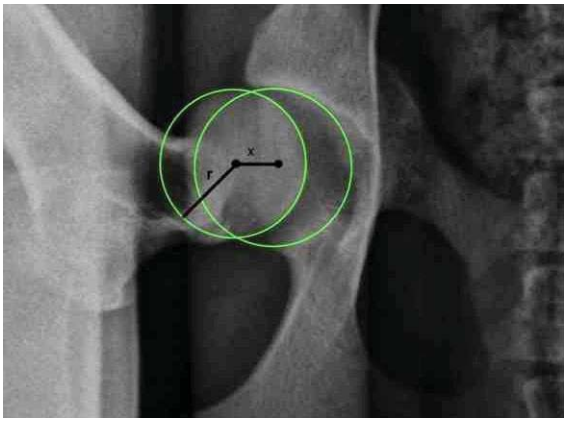


Figure 8. The PennHIP distraction index (DI) is the ratio of the distraction between the centres of the acetabulum and femoral head (x), divided by the radius of the femoral head (r), when a distraction force is applied to the sedated/anaesthetised patient with the pelvic limbs in a weight-bearing position [DI = x/r]. (Soo and Worth 2015)

Attempts have been made to measure functional laxity by mimicking load-bearing during radiography either by an operator manually attempting subluxation of the hip (Flückiger *et al.* 1998, 1999) or by the dorsolateral subluxation score (DLS, *Farese et al.* 1998). The DLS involves positioning the dog in sternal recumbency in a custom-made foam block with the dog's weight loaded through the point of the flexed stifles. The extent to which this procedure truly replicates physiological loading of the hip joint remains a subject of discussion (Verhoeven *et al*. 2012). On a ventro-dorsal radiograph, the percentage of coverage of the femoral head by the dorsal acetabular rim is calculated, with a high percentage implying a tighter and more normal hip. Dogs

with DLS scores > 55% are unlikely to develop CHD in later life, whereas dogs with a score < 45% have a greater chance of developing CHD (Lust *et al.* 2009). Other untested examples of stress radiography include the wedge technique, half-axial view (Henry 1992, Adams 2000), and the S-measurement derived from the dorsolateral subluxation examination (Ogden *et al.* 2012). None of these stress radiographic techniques have gained significant popularity nor been studied to the extent of the PennHIP DI or the DLS.

Effectiveness of the radiological scoring systems at lowering the incidence of CHD

Several authors have commented that despite over 40 years of radiographic screening the prevalence of CHD remains high and have questioned the efficacy of the schemes or programmes adopted by the OFA, FCI and BVA (Corley and Hogan 1985, Willis 1997, Leppänen 1999, Verhoeven *et al.* 2012). In 1992 a report on the effectiveness of the OFA program reported a decrease in the prevalence of CHD in 79% of breeds and an 88% increase in the number of excellent hip scores between the periods 1972-1980 and 1981-1988 (Corley 1992). A more recent evaluation of the OFA database has again shown a steady, albeit slow, increase in the proportions of dogs graded as excellent and good, whereas proportions of fair and mild/moderate/severe dysplastic grades significantly decreased between 1993-2003 (Kaneene *et al.* 2009).

However a report by Malcolm Willis in 1997, which looked at the BVA hip score trends in six different breeds, concluded that the expected progress towards lower hip scores had not been achieved. In fact, in 4 of the 6 breeds analysed the 4-year rolling mean BVA hip score had actually risen between the periods 1983-1986 and 1991-1995 (Willis 1997). In contrast, more recent (unpublished) data supplied by Dr Jeff Sampson of the Kennel Club (UK) showed a steady decline in the 5-year rolling mean hip score (improvement in hip phenotype) between 1992 and 2007 in virtually all breeds analysed (pers. comm. 2013). For the Labrador retriever breed the total BVA/Willis score dropped from a mean of 16.5 to 12.8 over that period. A smaller change was noted in the GSD with a drop in 5-year rolling mean hip score from 19.3 to 17.0 in the same period. This data has not been subjected to peer-review.

In countries, or in breeding colonies with mandatory scoring programmes, the use of extended ventro-dorsal radiological scoring methods has been reported to be more successful at lowering the incidence of CHD than in the UK or USA (Leighton *et al.* 1977, Hedhammer *et al.* 1979, Paatsama 1979, Leighton 1997). In Sweden it has been mandatory since 1984 for the hip joint status of both parents to be published before the Swedish kennel club will register the puppies of that mating. This open registration and

mandatory scoring may have contributed to the observed improvement in the median score for CHD of several breeds in Sweden (Swenson *et al.* 1997). In Finland scoring is mandatory and for some of the breeds affected by CHD threshold values for breeding are enforced (Leppänen and Saloniemi 1999).

Significant selection bias has been reported in the OFA system with radiographs of "normal appearing" hips being 8.2 times more likely to be submitted by veterinarians than those radiographs that showed the dogs were clearly dysplastic (Paster *et al.* 2005). When a breeder or veterinarian chooses to withhold radiographs of an obviously dysplastic dog rather then submit them for assessment and scoring this practice is known as pre-screening. The practice of pre-screening is a criticism of most CHD scoring schemes and skews the population of scored dogs by removing the worst affected individuals (Paster *et al.* 2005). Submission of radiographs is voluntary in all but a few countries whose kennel associations have mandated that scoring for CHD is required before puppies in a litter can receive official registration with the kennel association. In New Zealand there is no compulsion for veterinarians to submit all radiographs performed for the purpose of scoring and anecdotally pre-screening does occur. There is also no legal or NZKC requirement to hip score dogs that are to be used for breeding. Additionally, when a lame dog is diagnosed with CHD a radiological score is not captured by any database thus the published average hip scores are not a true reflection of the disease incidence or degree of severity within a given breed. Recently an "Accredited Breeders" program was introduced by the NZKC, which requires dogs that are to be used for breeding, to be scored under an approved scheme for any genetic disorders identified for that breed in order for the kennel to receive accreditation.

The quality of radiographs has a significant effect on the ability of any assessor to accurately determine a dog's CHD radiological phenotype. Verhoeven *et al.* (2009) concluded that the credibility of the FCI screening method for canine hip dysplasia (using the standard hip-extended radiographic view) as currently applied in most European countries, was questionable. An analysis of inter-observer agreement of the FCI system showed that assessment of the morphological characteristics of the hip joints and the final score are highly variable between observers, ranging from total disagreement to nearly full agreement (Coopman *et al.* 2007).

Improving the effectiveness of selection based on phenotype by calculation of breeding values

Analysis of the phenotypic status of a population over time is not the most accurate method of determining a genetic trend. Genetic trend (the effectiveness of the selection differential) can be better determined by estimated breeding value analysis, which relies on the availability of accurate pedigree information. An analysis of the OFA data based on estimated breeding values indicated that over 40 years the Labrador Retriever hip phenotype only reduced by 0.1 units, equivalent to a 5% decrease on a 1-7 ordinal scale (Hou *et al.* 2010). Hou *et al.* (2010) suggested that whilst the OFA system had been effective at reducing the number of severely affected dogs, expected progress towards reducing the overall impact of CHD in this breed has not eventuated.

Improving the effectiveness of selection based on phenotype by using the phenotype measure with the highest heritability

Genetic gain through breeding is a product of the heritability (h^2) of a trait and the selection pressure applied over generations. The selection pressure is related to the extent of phenotypic variation and the criteria for selection set by dog breeders. CHD scoring systems were introduced to help dog breeders select dogs for breeding with the goal of reducing the incidence and severity of hip dysplasia in future generations. The key assumption in any phenotypic scoring scheme is that successive generations will benefit from the selection pressure exerted against the trait in their parents and therefore, over time and for successive generations produce a more favourable phenotype.

Heritability measures the extent to which offspring resemble their parents for a certain trait (Nicholas 2010c). Estimation of heritability involves application of statistical techniques to determine the extent to which relatives resemble each other for a particular trait (Soo 2013). Heritability ranges from zero to one. If the heritability is zero, it means that relatives do not resemble each other at all. Conversely, a condition with heritability of one is completely genetically-determined. Traits with heritability less than 0.1 are considered lowly heritable; 0.2 – 0.3 moderately heritable and 0.4 – 1.0 to be highly heritable (Mackenzie 1985). The practical importance is that if the heritability is more than zero, then it should be possible to decrease the prevalence of a particular trait via selective breeding (Soo 2013). In general, the higher the heritability, the greater the influence of the genetic effects and thus the greater the potential response to breeding selection pressures (Mackenzie 1985, Nicholas 2010c).

Depending on the method of evaluation, the h^2 of CHD has been reported to be at least 0.22 to 0.25 (Leighton *et al.* 1977, Fox *et al.* 1987). Heritability estimates are performed using a variety of methods such as regression, Bayesian and/or Restricted Maximal Likelihood analyses. As a general rule, traits with a h^2 greater than 0.15 are considered to be under adequate genetic influence such that sufficient response will be seen with selective breeding (Wilson *et al.* 2011), thus decreasing the prevalence of the disease. Heritability estimates of previous studies performed on the OFA, FCI and BVA scoring systems have generally met or exceeded this level (Tables 5, 6, 7). To the author's knowledge, h^2 estimates for dogs scored under the NZVA CHD Scheme have not been published. However, because the NZVA CHD Scheme is based on the BVA hip scoring system, it is possible to infer the h^2 of the NZVA hip phenotype from the h^2 of the BVA hip phenotype (Table 7).

In the most comprehensive review of the veterinary literature to date, the heritability estimates of hip-extended VD radiology for CHD were reported to range from 0.06 to 0.74, with an overall weighted average (based on a total of 123,716 dogs) of 0.29 (Janutta and Distl 2006). An analysis of breeding value of the phenotype of 154,352 Labrador Retrievers, measured on extended-hip radiographs as reported by the OFA, resulted in a heritability estimate of 0.21 (Hou *et al.* 2010).

Table 5. Reported heritability for the Orthopaedic Foundation for Animals hip grading system, using the hip-extended radiographic view (subjective scoring system). (Soo 2013)

Breed	*h^2 (standard error)*	*Study*
Bernese Mountain dog	*0.30 (0.04)*	*(Reed et al. 2000)*
Chinese Shar-pei	*0.31 (0.05)*	*(Reed et al. 2000)*
English Setter	*0.17 (0.05)*	*(Reed et al. 2000)*
German Shepherd dog	*0.22 (0.06)*	*(Leighton et al. 1977)*
German Shepherd dog	*0.43*	*(Mackenzie et al. 1985)*
Labrador Retriever	*0.21 (0.006)*	*(Hou et al. 2010)*
Portuguese Water Dog	*0.30 (0.06)*	*(Reed et al. 2000)*
Pooled h^2 for 17 breeds	*0.76*	*(Zhang et al. 2009)*
Pooled h^2 for 74 breeds	*0.22 (0.002)*	*(Hou et al. 2013)*

Table 6. Reported heritability for the Fédération Cynologique Internationale hip grading system, using the hip-extended radiographic view (subjective scoring system). (Soo 2013)

Breed	*h^2 (standard error)*	*Study*
Bernese Mountain dog	*0.42 (0.03)*	*(Malm et al. 2008)*
Bernese Mountain dog	*0.31 (0.06)*	*(Lavrijsen et al. in press)*
Estrela Mountain dog	*0.38 to 0.43[a]*	*(Silvestre et al. 2007)*
German Shepherd dog	*0.31 to 0.35[a]*	*(Leppänen et al. 2000)*
German Shepherd dog	*0.24 to 0.26[a]*	*(Hamann et al. 2003)*
German Shepherd dog	*0.25 (0.01)*	*(Stock et al. 2011)*
Golden Retriever	*0.17 (0.03)*	*(Lingaas and Klemetsdal 1990)*
Golden Retriever	*0.18 (0.04)*	*(Lavrijsen et al. in press)*
Labrador Retriever	*0.44*	*(Ohlerth et al. 2001)*
Labrador Retriever	*0.38 (0.04)*	*(Engler et al. 2008)*
Labrador Retriever	*0.24 to 0.29[a]*	*(Vostrý et al. 2012)*
Labrador Retriever	*0.10 (0.03)*	*(Lavrijsen et al. in press)*
Rottweiler	*0.58 (0.04)*	*(Mäki et al. 2000)*
Rottweiler	*0.38 (0.02)*	*(Malm et al. 2008)*
Newfoundland	*0.0.26 to 0.28[a]*	*(Dietschi et al. 2003)*
Newfoundland	*0.23 (0.08)*	*(Lavrijsen et al. in press)*

[a] Heritability value varies with model and method of estimation.

Table 7. Reported heritability for the British Veterinary Association hip scoring system, using the hip-extended radiographic view (semi-subjective scoring system). (Soo 2013)

Breed	*h^2 (standard error)*	*Study*
Akita	*0.39 (0.053)[a]*	*(Lewis et al. 2013)*
Bearded Collie	*0.46 (0.048)[a]*	*(Lewis et al. 2013)*
Bernese Mountain dog	*0.36 (0.040)[a]*	*(Lewis et al. 2013)*
Border Collie	*0.44 (0.033)[a]*	*(Lewis et al. 2013)*
English Setter	*0.35 (0.049)[a]*	*(Lewis et al. 2013)*
Flat-coated Retriever	*0.74 (0.25)*	*(Wood et al. 2000a)*
Flat-coated Retriever	*0.28 (0.032)[a]*	*(Lewis et al. 2013)*
German Shepherd dog	*0.30 (0.02)*	*(Wilson et al. 2012)**
German Shepherd dog	*0.35 (0.015)[a]*	*(Lewis et al. 2013)*
Golden Retriever	*0.40 (0.017)[a]*	*(Lewis et al. 2013)*
Gordon Setter	*0.20 to 0.38[b]*	*(Wood et al. 2000b)*
Gordon Setter	*0.43 (0.062)[a]*	*(Lewis et al. 2013)*
Labrador Retriever	*0.34 (0.02)*	*(Wood et al. 2002)*
Labrador Retriever	*0.35 (0.016)[a]; 0.50 (0.018)[c]*	*(Lewis et al. 2010)*
Labrador Retriever	*0.35 (0.02)*	*(Woolliams et al. 2011)*
Labrador Retriever	*0.33 (0.012)[a]*	*(Lewis et al. 2013)*
Newfoundland	*0.49 (0.08)*	*(Wood et al. 2000a)*
Newfoundland	*0.46 (0.041)[a]*	*(Lewis et al. 2013)*
Rhodesian Ridgeback	*0.33 (0.048)[a]*	*(Lewis et al. 2013)*
Rottweiler	*0.39 (0.028)[a]*	*(Lewis et al. 2013)*
Siberian Husky	*0.48 (0.038)[a]*	*(Lewis et al. 2013)*
Tibetan Terrier	*0.34 (0.048)[a]*	*(Lewis et al. 2013)*

** Heritability estimate study is based on the Australian Veterinary Association hip scoring system, which is similar to the BVA system; [a] heritability estimate performed on hip scores transformed onto a logarithmic scale; [b] heritability value varies with model and method of estimate; [c] heritability estimate performed on original, untransformed hip score*

Distraction Radiography

While a thorough quantitative genetic analysis of the entire PennHIP database has not yet been published, a small number of h^2 studies on the DI have been performed. Todhunter *et al.* (2003) found the h^2 of the DI in 147 Labrador Retrievers across four generations to be 0.5. A larger study by Zhang *et al*. (2009) comprising of 2716 dogs across 17 breeds reported a h^2 of 0.61 for the DI.

Investigators from the University of Pennsylvania have reported the distraction index to have an estimated heritability of between 0.34 and 0.73, using various methodologies including analysis of full pedigrees and inter-class correlation coefficients (Kapatkin *et al.* 2002). Many of the reports cited by Kapatkin and co-authors were from non peer-reviewed sources and used small sample sizes. Commentators have expressed concern that despite more than two decades of accumulated data, the PennHIP database itself has not been subjected to wider analysis to determine the heritability of the DI. However, a recent genetic analysis of the DI in the Estrela Mountain dog, using a linear animal model, reported a very high h^2 of 0.83 (Ginja *et al*. 2008). This estimate was based on only 215 observations and has a standard error of 0.11. Since h^2 is unique to the phenotype and population in which it is estimated (Mackenzie 1985), this high h^2 from an atypical breed cannot be directly applied to other breeds.

Despite the wealth of scientific literature supporting distraction index as a superior method to traditional subjective extended VD radiology, no national veterinary association has adopted the PennHIP method as the basis for recommendations regarding the use of breeding stock. However, organisations that have adopted the PennHIP assessment such as The Seeing Eye (Inc.) in the US, have reported more rapid improvement in hip phenotype compared with selection based on traditional radiological investigation (Gail Smith, University of Pennsylvania, 2013, pers. comm.)

Relevance of radiological screening for CHD phenotype for the NZ Police Dog Section

The NZPDS has used the NZVA Hip Dysplasia scheme to score the phenotype of breeding dogs in the Police Dog breeding program since early in the schemes history. For each mating the hip phenotype, elbow phenotype (NVZA Elbow Dysplasia scoring scheme), behavioural characteristics, and work traits of the sire and dam are considered. Decisions regarding breeding any dog are reliant on the quality of the data provided by scoring schemes to make accurate predictions of a dog's genetic merit. If decisions are made for breeding on the basis of the hip extended VD phenotype it is imperative that the NZVA hip dysplasia scheme is accurate and reliable. Analysis of the NZVA data to determine any improvement in hip phenotype had not been performed prior to the author's appointment (AW) as convenor of the NZVA Hip and Elbow Dysplasia Schemes. In the next chapter data and analysis on the effectiveness of the NZVA scheme at reducing the incidence of CHD in the German shepherd breed from 1992 to 2008 is provided and discussed.

Two co-authored articles have been published in the NZ Veterinary Journal and are reprinted as appendices

Soo M, **Worth AJ**. Canine hip dysplasia: Phenotypic scoring and the role of estimated breeding value analysis. New Zealand Veterinary Journal 63, 69-78, 2015

Soo M, Sneddon NW, Lopez-Villalobos N, **Worth AJ**. Genetic evaluation of the total hip score of four populous breeds of dog, as recorded by the New Zealand Veterinary Association Hip Dysplasia Scheme (1991–2011). New Zealand Veterinary Journal 63, 79-85, 2015

Canine Hip Dysplasia – Determination of improvement in the hip phenotype of the German shepherd dog based on assessment of radiographs in accordance with the New Zealand Veterinary Association Hip Dysplasia Scheme

Introduction

In chapter one hip dysplasia was an important cause of early retirement in German shepherd dogs (GSDs) worked by the New Zealand Police Dog Section (NZPDS). Chapter two described the clinical features of CHD and discussed its mode of inheritance. Clinical signs of hip dysplasia are often only noted in mature dogs. However, there are financial and logistical costs in maintaining a dog through to breeding age, therefore decisions on using a dog in a breeding program are often required when the dog is still young. In the absence of a genetic test for CHD, radiographic screening programs are used as a means of selecting dogs with a superior hip phenotype for breeding in order to reduce the incidence of CHD in the offspring. Such schemes rely on the predictive value of the radiological characteristics for CHD. The New Zealand Veterinary Association (NVZA) introduced the Canine Hip Dysplasia Scheme in the mid-1980s in cooperation with the NZ Kennel Club (Hunter 1986) and a national computerised database was maintained by the NZVA from 1989. The NZPDS has utilised the NZVA CHD scoring scheme since 1990. As detailed in chapter two, the NZVA scheme uses the British Veterinary Association (BVA) scoring system that was developed by Malcolm Willis (Dennis 2012). The NVZA publishes biannually the running mean score for all dogs of each breed that have been scored by its scheme. However, to the author's knowledge, the NZVA data has never been analysed to determine trends that indicate any improvement in the phenotype for hip dysplasia over successive generations.

Aim: The aim of this study was to determine if the CHD scores for the extended hip radiographs in the GSD, as recorded in the NZVA database, have changed over time. Given the known heritability of the hip-extended ventro-dorsal radiological phenotype, the hypothesis was that if dog breeders have applied selection pressure for better hip conformation, there should be a trend towards lower total scores and lower grades for indicators of hip laxity. Scores from all the GSDs contained in the NZVA database were utilised as opposed to an analysis of scores from only police GSDs. Whilst limiting the

analysis to only Police GSDs would be more pertinent to the NZPDS, the small numbers of dogs involved would have provided a limited dataset. By pooling the data any interpretation of change in scores over time reflects the selection policies of both the PDS and of private breeders. The effectiveness of the PDS selection policies themselves cannot be accurately assessed.

Materials and Methods

The database of the NZVA CHD scheme was made available to the author by the NZVA and the NZKC for the purposes of this study. The NZVA records included all dogs scored from 1989 onwards, as prior to that date, data from dogs in New Zealand was held by Dr Malcolm Willis in the UK. The oldest GSDs present in the records were born in 1991 and scored between 1996 and 1998. There were several younger dogs scored in 1995. Data retrieval was performed using a final date of radiography set at 31st of December 2010, the youngest dogs in the analysis having been born in 2008. In order to assess the degree to which the sample population represented the registered GSD pedigree population, the New Zealand Kennel Club (NZKC) was contacted for their annual whelping data (number of puppies born to registered dams). This data was used to estimate the proportion of the total number of puppies born in a given year that were scored using the NZVA CHD scheme. Data consisting of each dog's date of birth, age at scoring, sex, total score and individual scores in the Norberg angle and subluxation categories were collated. Age at scoring (in days) was determined by subtracting the date of birth from the date radiographs of the hips were taken.

The median, mean and SD for total score, stratified by year of birth, were determined. The date of radiography was not used for stratification, as dogs may be radiographed for the purposes of NZVA scoring at any age over 12 months of age. The author was most interested in determining if there was any trend towards improvement in total score over time using the date of birth as the dependent variable. By using the date of birth as a continuous variable we sought to investigate a generational effect, with the younger dogs benefiting from the selection pressure applied to their forebears.

Statistical methods

Linear regression was performed using a model of total score against date of birth (as a continuous numerical scale), age at scoring (in days), and sex, and fitted to the data (R v2.8.1; R Foundation for Statistical Computing, Vienna, Austria).

The age at scoring and sex were placed in the models as variables to ensure confounding effects of the uneven age/sex distribution were taken into account.

If the age at scoring, and/or sex were non-significant, that variable was dropped from the regression model. The total score for each individual dog was assessed as a time point based on date of birth thereby allowing an assessment of the effectiveness of selection pressure over time on the investigated population. The null hypothesis was that the total hip score was not correlated with date of birth.
When a significant change over time was uncovered, ordinal logistic regression was used to model the probability of occurrence of individual scores in the Norberg angle and subluxation categories with date of birth (R v2.8.1; R Foundation for Statistical Computing, Vienna, Austria). The individual scores for the left and right hips were pooled.

Results

Data from 1,087 German shepherd dogs scored by the NZVA CHD scheme were included in the study. The proportion of dogs registered with the NZKC that received NZVA CHD scores is shown in Table 8 (page 48). Usage of the scheme by breeders increased from 1990 to 1997. The decline in the number of dogs scored with dates of birth from 2005 to 2008 may either be due to falling popularity of the NZVA scheme or be a reflection of the decision by breeders to not score young dogs, preferring to score their dogs at an older age. Median, mean and SD for the year groups are presented in Table 9. The data distribution, as grouped by year of birth, is presented in Figure 9. The median rather than mean total score is depicted due to the skewed distribution of the data set.

Total score linear regression showed a significant trend (Multiple R-squared: 0.015, Adjusted R-squared: 0.013, $p = 0.0003$) towards a lower total score over time, based on date of birth. However, although significant, the very low R^2 value indicates that the actual effect size (improvement in phenotypic score) was small. Indeed, date of birth explains just 1.5% of the variation in total score according to the fitted model. There was however strong evidence for linear dependence of total score on date of birth (negative linear association).
Age at scoring was not correlated but gender was positively correlated with the total NZVA HD score ($p = 0.009$). Examination of the dataset revealed that the proportion of female dogs compared to males scored increased during the study. Overall there were twice as many females as males scored during the study period.
The mean total score of the males was 10.2 and that of the females was 12.0.

Therefore it is likely that as the sex distribution per year shifted more towards females the effect was a slowing of the trend towards lower scores and an improved phenotype. Ordinal logistic regression on the Norberg angle scores indicated a significant effect due to date of birth (Regression coefficient = -0.05, SE = 0.01, p = 0.000). A plot of the fitted probabilities of the Norberg angle scores over time is shown in Figure 10.

Ordinal logistic regression on the subluxation scores also indicated a significant effect due to date of birth (Regression coefficient = -0.06, SE = 0.01, p = 0.000). A plot of the fitted probabilities of the subluxation angle scores over time is shown in Figure 11. These findings suggest that there was a trend towards hips with less laxity in the German shepherd dog breed, as scored by the NZVA between 1991 and 2008.

Table 8. Number of German shepherd bitches registered by the New Zealand Kennel Club that whelped each year (born), number of dogs scored by the New Zealand Veterinary Association Hip Dysplasia Scheme (scored) and proportion of dogs scored in year groups classified by year of birth (%).

	German shepherd dogs		
Year	Born	Scored	%
1990	1180	0	0
1991	1129	3	0.3
1992	1155	6	0.5
1993	1193	16	1.3
1994	1317	46	3.5
1995	1147	74	6.5
1996	1191	81	6.8
1997	1082	64	5.9
1998	1055	91	8.6
1999	1004	65	6.5
2000	1064	84	7.9
2001	849	76	9.0
2002	936	103	11.0
2003	718	71	9.9
2004	785	99	12.6
2005	823	74	9.0
2006	771	67	8.7
2007	810	41	5.1
2008	745	26	3.5

Table 9. Mean, Standard Deviation and median total hip score stratified by year of birth, as recorded by the New Zealand Veterinary Association hip dysplasia scheme for German shepherd dogs between 1990 and 2008.

Year of birth	German shepherd dogs (n=1087)		
	Mean	St.Dev	Median
1990			
1991	19.3	23.2	8
1992	7.7	4.5	6.5
1993	17.1	25.2	10
1994	14.7	15.7	8.5
1995	12.1	14.3	7
1996	13.9	15.7	8
1997	12	13.2	8.5
1998	10.9	11.1	7
1999	12.2	10.4	9
2000	10.8	10.3	9
2001	10.9	8.5	8
2002	11.9	15.1	8
2003	10.3	9.6	9
2004	10	9	8
2005	11.6	13.8	8
2006	9.6	10	7
2007	10.1	11.6	7
2008	8	4.7	7

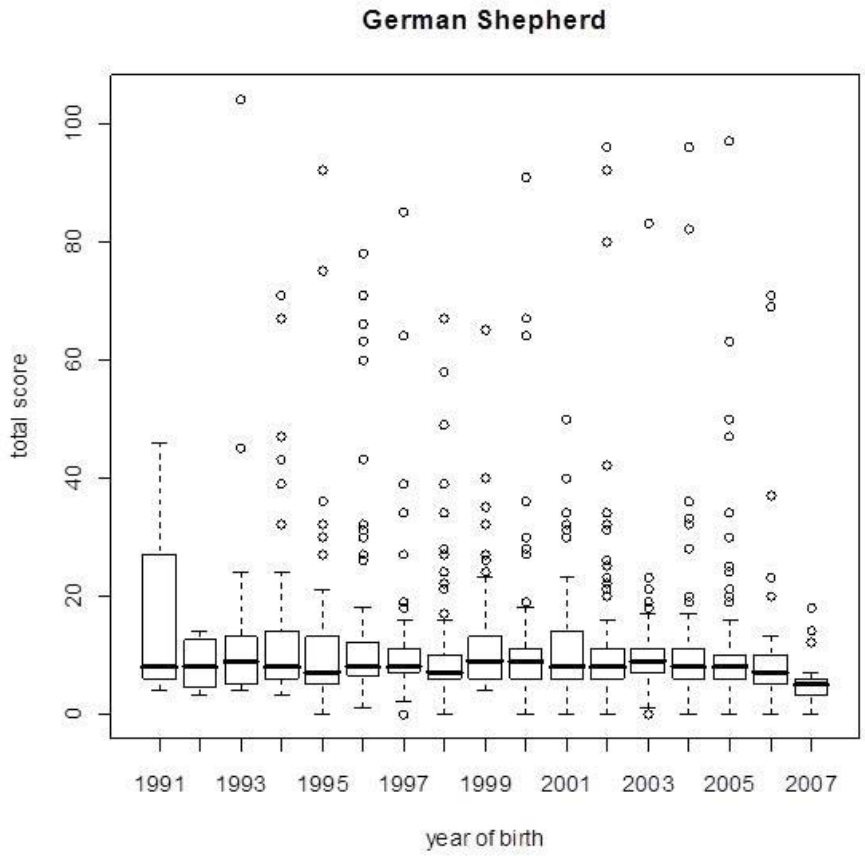


Figure 9: Distribution graph of the hip dysplasia scores for 1087 German shepherd dogs recorded by the New Zealand Veterinary Association and born between 1991 and 2008. Total scores are plotted by year of birth. The bold horizontal bar in each box plot shows the median total score. Boxes indicate the 25th and 75th percentiles and the whiskers show the maximum and minimum values excluding outliers. Outliers, shown as open circles, represent values more than 1.5 times the interquartile range. The 1991 data represents only 3 dogs and should be interpreted with caution.

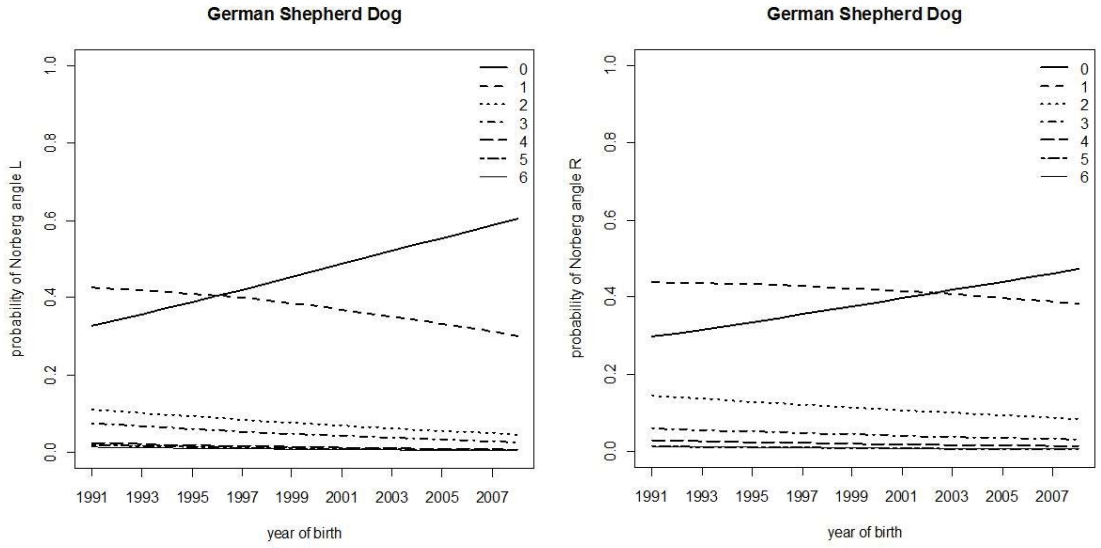


Figure 10. A plot of the fitted probabilities for the Norberg Angle score of the pooled data of 1087 German shepherd dogs as recorded in the New Zealand Veterinary Association hip dysplasia database. The Norberg Angle is scored from normal (0) to severely abnormal (6) using an ordinal scale. L=left, R=right hip.

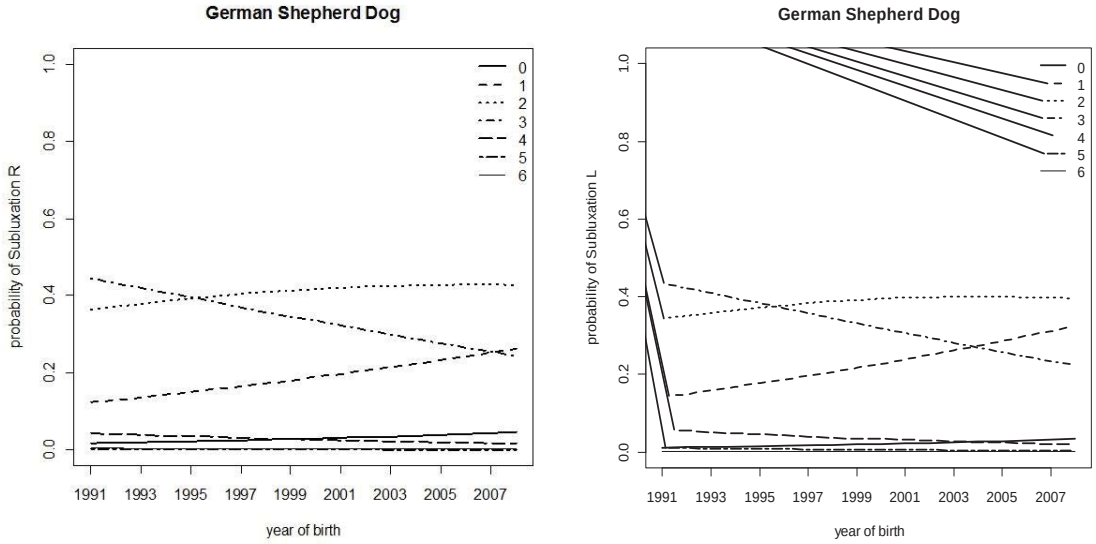


Figure 11. A plot of the fitted probabilities for the subluxation scores of the pooled data of 1087 German shepherd dogs as recorded in the New Zealand Veterinary Association hip dysplasia database. Subluxation is scored from normal (0) to severely abnormal (6) using an ordinal scale. L=left, R=right hip.

Discussion

Like similar schemes throughout the world, the NZVA CHD scoring system was introduced to help dog breeders select the best dogs for breeding with the aim of reducing the prevalence of CHD, a potentially crippling arthropathy. The key assumption of any scoring scheme based on phenotype is that successive generations will benefit from the selection pressure exerted against the trait in their forebears and therefore the offspring will have an improved phenotype. NZVA CHD data was analysed using linear regression to determine if the phenotypic score was negatively correlated to the date of birth of dogs as would be expected after selective breeding for a better hip phenotype. Using data from the sample population of German shepherd dogs scored by the NZVA between 1991 and 2008 there was a significant trend towards a better hip phenotype over successive generations. However the extent of the improvement was small, the median value improving from a score of 8 to 7, an improvement of 12% over 17 years, or 0.73% per year. The analysis revealed that the correlation between the date of birth and the score explained only 1.5% of the variation in the score, which means that the improvement in the median score was predominantly due to effects other than selection pressure.

Several authors have commented that despite over 40 years of radiographic screening, the prevalence of CHD remains high and have questioned the efficacy of the schemes or programmes adopted by the OFA, FCI and BVA (Corley and Hogan 1985, Willis 1997, Leppänen and Saloniemi 1999, Leppänen *et al.* 2000, Verhoeven *et al.* 2012). The OFA reported a decrease in the prevalence of CHD in 79% of breeds and an 88% increase in the number of excellent hip scores between 1972-1980 and 1981-1988 (Corley 1992). Kaneene *et al.* (2009) later reported a steady, albeit slow, increase in the proportions of dogs graded as excellent and good, whereas the proportions of dogs in the fair and mild/moderate/severely dysplastic categories have significantly decreased between 1993 and 2003. However, an analysis of the OFA data based on estimated breeding values indicated that over 40 years the Labrador retriever hip phenotype only reduced by 0.1 units, equivalent to a 5% decrease in the allocated grade on a 1-7 ordinal scale, or 17% of the phenotypic standard deviation (Hou *et al.* 2010). More recently, Hou *et al.* (2013) performed an analysis of the estimated breeding value (EBV) on 760,455 hip scores across 74 breeds listed in the OFA database to evaluate genetic trends between 1970 and 2009. The study found a genetic improvement of 0.1 units of hip score during the study period, which was equivalent to 16.4% of the phenotypic standard deviation. These values corresponded to a 0.52% decrease in incidence of CHD in the study population.

Lewis *et al*. (2010) conducted a genetic evaluation of the effectiveness of the BVA scoring system at reducing the prevalence of CHD in Labrador retrievers in the UK and reported a genetic progress of 0.376 untransformed (or 0.155 log-transformed) hip score units per annum. This translates to a 1.4% decline year on year or a 13% improvement in hip scores over the 10-year study period. The authors concluded that this was very minimal progress against CHD, and was equivalent to avoiding only 15% of the worst animals for breeding (Lewis *et al.* 2010). Lewis *et al.* (2013) performed an EBV analysis on 142,287 hip scores across fifteen breeds listed on the BVA CHD database to evaluate for any genetic improvement made when breeding selection was carried out on the basis of phenotypic scores. Regression of the EBV on the date of birth revealed that 14 out of the 15 breeds exhibited some genetic improvement during the study period. However, the extent of improvement was only small with a 0.13% to 1.98% decline in hip score units per year. This improvement was equivalent to excluding between 2% and 18% of the worst-affected animals from breeding, indicating a low selection intensity was employed throughout the study period (Lewis *et al*. 2013).

Nevertheless, the use of scoring systems based on the hip-extended radiological phenotype to allow application of selection pressure against CHD has been reported to be successful in countries or breeding colonies with mandatory scoring programmes (Leighton *et al.* 1977, Hedhammer *et al.* 1979, Paatsama 1979, Leighton 1997). Since 1984 it has been mandatory in Sweden for the hip joint status of both parents to be published before the Swedish kennel club allows registration of the puppies from a litter (Swenson *et al.* 1997, Genevois *et al.* 2008). This open registration policy and mandatory scoring scheme may contribute to the observed improvement in CHD median score of several breeds popular in Sweden.

Analysis of phenotypic trends of a population over time is not the most accurate method of determining any genetic trend. Genetic trend (the effectiveness of the selection differential) can be better determined by estimated breeding value analysis. Breeding value estimation relies on availability of accurate pedigree information. However, pedigree data was only entered in to the NZVA database from 1997 making breeding value determination impossible for the full period of this study. Estimated breeding value analysis of the NZVA CHD database from 1997 is the aim of a subsequent study undertaken by the author, the abstract of which is included as Appendix 2.

Genetic gain through selection of sires and dams for breeding is a product of the heritability of a trait and the selection pressure applied. The selection pressure is related to the phenotypic variation, and the criteria for selection. The NZVA CHD scheme is voluntary and provides information to allow best practice for selection of

breeding stock, but is not a genetic disease control programme *per se*. Scoring for the degree of hip dysplasia is not a requirement for pedigree registration by the New Zealand Kennel Club and anecdotally the NZVA scheme is used selectively by breeders in New Zealand. The NZVA has published recommended criteria for selection of breeding stock but neither the NZVA nor the New Zealand Kennel Club can impose on, or enforce any selection thresholds on private breeders and breed clubs. This lack of jurisdiction leads to breed clubs and individual breeders making decisions regarding the selection of breeding stock that may differ from ideal. The NZVA's criteria for selection advice includes: selectively breed from individual dogs with a total score below that of the breed mean; selectively breed from individual dogs with a total score of ≤8; and selectively breed dogs with a subtotal score of ≤2 per hip. Non-stringent selection criteria may have contributed to the small degree of improvement in the hip phenotype in this study (Nicholas 1987). In the United Kingdom, where the BVA system is also voluntary, 76% of matings in 1999 had at least one parent scored and 52% of matings had both parents scored (Wood *et al*. 2004a). Comparable analysis of NZVA data is unavailable. In New Zealand, a variable proportion of dogs born to NZKC registered dams were scored by the NZVA (Table 8) in the years covered by this analysis. Typically only dogs retained for breeding are scored and only a small proportion of a litter is maintained as a breeding population therefore many siblings remain un-scored. There is anecdotal evidence that members of several NZ GSD breed clubs prefer to send hip radiographs to private film reading services in Australia (affiliated with the Australian GSD Council), reducing the proportion of dogs used for breeding in New Zealand that are scored by the NZVA. Because of the variable uptake of the NZVA system it is impossible to determine if the data in this study is representative of the entire New Zealand GSD population. It is possible that inclusion of data (hip scores) from these private schemes could have shown a different result in the current study. Unfortunately, this data is not captured by the individual breed clubs or the NZKC in a manner that can be authenticated. Australian hip dysplasia data was reported by Karen Hedberg of the Australian National Kennel Club (ANKC) to a joint meeting between the Australian and New Zealand Veterinary Associations in 2010. Score data were compiled from the ANKC records (including scores generated by private schemes and the Australian Veterinary Association – both based on the BVA/Willis system) and represented dogs born between 1980 and 2008. Rolling median hip score for the GSD reportedly reduced from 9 in 1994, to 6 in 2005. A regression analysis was not performed and the results have not been peer-reviewed. For comparative purposes a 5-year rolling median was generated from our data, which showed that for the GSD the median increased from 7 in 1995, to 8 in 2005.

The unverified data would suggest that in Australia greater improvement in hip phenotype has been realised compared to the data reported in this study from New Zealand. One possible explanation for the disparity is the activity of the German shepherd Dog Council of Australia. The state GSD breed clubs are affiliated to the GSD Council of Australia, members are required to hip score dogs as a prerequisite for registration, and these scores are published. The results of matings between registered sires and dams are published in breed tables at regular intervals, and indicate the spread of hip scores of all progeny listed from each sire.

Failure of veterinarians and breeders to submit radiographs from obviously abnormal individuals (pre-screening) also biases the reported average score of populations measured by non-mandatory schemes (Paster *et al.* 2005). Pre-screening is not in the best interests of the breed as pre-screening falsely lowers the breed averages and is strongly discouraged by the NZVA. Indeed pre-screening could theoretically lead to conscientious breeders not using stock that have actual scores superior to the true median, if that median were known. Of greater concern is that there is no recording of un-scored pedigree dogs that are definitively diagnosed with clinical signs and evidence of CHD by veterinarians, leading to an under-estimation of the true prevalence of the disease. Due to the lack of compulsion to score, lost data from dogs scored overseas and pre-screening, the author cannot determine if the results of this study are a true and accurate reflection of the status of CHD in the GSD in NZ.

In a lifetime study of dysplastic Labrador retrievers a progressive increase in the scores for repeated hip-extended radiographs was noted over the life of the dogs, indicating that DJD is progressive (Kealy *et al.* 2000, Smith *et al.* 2006). Similarly, increasing age at first scoring was significantly associated with higher OFA scores and dysplasia in Labrador retrievers (Hou *et al.* 2010). Therefore, as a dog ages, its hip-extended score becomes more accurate at predicting its true hip status at end of its life. Whilst the OFA requires a dog being scored to be 2 years of age or older, a dog only has to be 1 year of age or older to be scored by the NZVA/AVA or BVA schemes. Scoring animals younger than 2-years of age likely leads to retention of some dogs for breeding that have less desirable genes. In the NZVA dataset we did not detect a significant effect of age on the total score, most likely due to the majority of dogs being between 1- and 2-years of age when they were scored. We therefore did not have a sufficient range of ages over which an effect could be assessed.

There was a significant effect of gender on total score due to a significant increase in the proportion of bitches scored in the later years of the current study. Ginja *et al.* (2008) also found an effect of sex on hip status with female Estrela mountain dogs having higher scores for hip laxity than males. This finding is in contrast to the results

of a much larger study in Labrador retrievers using the OFA scoring system, which found no significant effect of sex on their score (Hou *et al.* 2010). Whilst it has been proposed that endogenous oestrogen secretion can affect joint laxity, this hypothesis was shown not to be the case in a prospective study of bitches in oestrus (Hassinger *et al.* 1997*)*. The effect of gender on the model suggests that higher selection pressure is being exerted on a smaller number of male German shepherd dogs (lower scores) that are used as sires to service a larger numbers of bitches (higher scores).

Effective heritability of a phenotypic scheming program can be improved substantially by selecting using estimated breeding values (EBVs), which incorporate pedigree analysis rather than on individual phenotypes. Selection based on phenotype is as accurate as selection based on breeding value only when heritability of a trait is close to one (Hou *et al.* 2010). As suggested by Burbidge (2003), calculation of breeding values from pedigree information would provide a much better selection criterion for NZ dog breeders to use, independent of whatever scoring system is used to determine CHD status. An analysis of the NZVA CHD data determining estimated breeding values would be of value to NZ dog breeders. Such an analysis has recently been undertaken (Soo *et al.* 2015) and is presented in abstract form as Appendix 2.

Selection differential (which is a function of the extent of variability and the extent of selection pressure applied) is equally as important as heritability in determining genetic gain from selective breeding programs based on measuring phenotypic variation. Even though the estimated heritability of the hip-extended radiological phenotype may be lower than that of the PennHIP DI, as outlined in chapter two, it is sufficiently high to expect genetic gain from selective breeding; assuming effective screening, sufficient phenotypic variation and appropriate selection pressure. Examination of Figure 9 shows that the hip-extended radiological scores of NZ GSDs have begun to cluster around the median with less variation present in the population. This reduction in phenotypic variation will make it difficult to drive selection pressure in future generations. Adoption of the PennHIP® distraction index as a selection criterion may be useful when clustering of a breeding population's subjective hip scores occurs (Leighton 1997) because it would introduce selection for a phenotype with a greater phenotypic variation.

In conclusion, between 1991 and 2008, the hip status based on the scores of hip-extended radiographs of German shepherd dogs has shown statistical evidence of improvement as measured by the NZVA CHD scheme, but the gains have not been dramatic. Failure to show a greater improvement may be due to a combination of factors including pre-screening by breeders and veterinarians, low uptake of the NZVA scheme by breeders, and failure to apply sufficient selection pressure by not following

recommended guidelines. However, individual breeders may have achieved greater genetic gain, which we cannot measure with the form of analysis used in this study. Greater improvement might be possible if use of the NZVA scheme is made a compulsory requirement for registration of pedigree breeding stock, and if stricter thresholds are prescribed and enforced before dogs are approved for breeding In addition, consideration should be given to a hip scoring method that is better able to detect passive hip laxity. Regardless of the test employed, the estimation of breeding values from phenotype and pedigree data would represent a major improvement in reporting of the CHD status and implementation of an open breeding value database would greatly assist dog breeders.

This study was performed to determine the effectiveness of the NZVA CHD scheme at lowering the incidence of CHD in GSDs in New Zealand. The subset of NZPDS GSDS scored by the NVZA CHD scheme was not separately analysed. Assuming, as is most probable, that selection pressures were more uniformly applied within the police dog breeding program than in the wider population studied here, greater improvement may have been achieved in improving the hip status of current Police dogs. Analysis of the genetic, rather than the phenotypic, trend may allow more accurate determination of the progress towards better CHD status by the NZPDS (Soo *et al.* 2015). Yet the perceived progress being made within the NZPDS was considered slow (Mark Sandford, NZPDS Breeding Centre Manager, pers. comm.). As a result of the apparent slow progress using the traditional scoring scheme the NZPDS sought an alternative means of scoring dogs for CHD status. PennHIP distraction radiography offered a phenotypic test for hip joint laxity with a reportedly higher heritability than the hip-extended radiological phenotype. While a thorough genetic evaluation of the PennHIP database has yet to be conducted, studies to date have shown the heritability of the DI to be higher than that of the BVA phenotype (see Table 7). The NZPDS funded the author to attend a PennHIP training course in 2003. The intention of supporting the PennHIP scheme was to score all breeding dogs using both the NZVA CHD scheme and PennHIP distraction index. A prospective trial was then planned to determine the extent of correlation between the two systems and then to better advise the NZPDS regarding selection of sires and dams in order to improve the hip phenotype of police dogs in the future. The conduct and results of this prospective study will be the subject of chapter four.

The data and figures from this study have been published - Worth AJ, Bridges, JP, Jones, G. Trends in the phenotypic hip status of selected breeds of dog as measured by the New Zealand Veterinary Association Hip Dysplasia scheme (1990-2008). New Zealand Veterinary Journal, 59: 2, 67-74, 2011.

An assessment of the agreement between the New Zealand Veterinary Association Hip Dysplasia Scoring System and the PennHIP Distraction Index in German shepherd dogs

Introduction

In chapter one, canine hip dysplasia was identified as an important reason for the early retirement of German shepherd dogs (GSDs) working for the NZ Police Dog Section (PDS). Prior to the initiation of this study, the NZ PDS used the New Zealand Veterinary Association (NZVA) CHD Scheme exclusively to score GSDs in its breeding program. Dogs with low total NZVA CHD scores were favoured, in addition to consideration of other traits in the selection of dogs for breeding. In chapter three the utility of the NZVA CHD scheme (which assesses hip extended, ventro-dorsal radiographs) at improving the hip phenotype in GSDs was investigated. Though statistically significant, there was only a minor improvement (approximately 0.73% per year) in median NZVA total score over the duration of the study. More importantly the year of birth across the time period of the study explained just 1.5% of the variation in the total score, suggesting a very small positive benefit of the selection pressure that was applied. However ordinal logistic regression analysis did demonstrate a significant lowering of the Norberg angle and subluxation scores of GSDs indicating some selection towards less hip laxity between 1990 and 2008.

The work of Dr Gail Smith and colleagues at the University of Pennsylvania has shown that passive laxity is better correlated with the development of DJD and clinical signs than the hip-extended OFA score (Smith *et al.* 1990, 1995), [see also chapter two]. The University of Pennsylvania Hip Improvement Program (PennHIP) measures passive laxity using distraction radiography to generate a distraction index (DI) (Smith *et al.* 1990). The program became commercially available through training courses for veterinarians in 1993 (www.pennhip.org) and consists of a network of trained veterinarians in 36 countries including Australia and New Zealand. Despite its promise no national veterinary medical association has yet adopted PennHIP as the “sole” hip screening system. It has been suggested that due to its greater sensitivity for laxity, the DI should be used for selecting dogs for breeding, whereas the high positive predictive value of the standard hip-extended-view, when DJD is present, is more appropriate for eliminating those dogs likely to show clinical signs of CHD (Ohlerth *et al.* 2003, Smith *et al.* 2006).

In an effort to increase the emphasis on the detection of hip laxity, the NZVA CHD system was modified in 2003 by the reporting of a subtotal score separate from the total score for each dog (Burbidge 2003). Rather than reporting the total score out of a possible 106, the individual score for each of three criteria deemed to be most indicative of joint laxity (Norberg angle, subluxation score and incongruity between the femoral head and cranial acetabular edge) were added to provide a subtotal score out of a maximum of 36. The revised NZVA CHD submission form read thus: "Whilst the ideal score (subtotal) is 0, a 2 or less is acceptable". The subtotal score is an attempt to indicate the degree of incongruity of the joint surfaces and provide an indication of subluxation. The remaining components of the overall total score are all measures of osteophyte development which, whilst a positive indication of CHD status, are age-dependent (Smith *et al.* 2006). The NZVA has published guidelines for breeders seeking advice on selection for improving hip phenotype. Over time the guidelines have been modified and have included: selectively breed from individual dogs with a score below the score for the breed mean; selectively breed from individual dogs with a total score of ≤8; and selectively breed dogs with a subtotal score of ≤2 per hip.

For the success of any screening programme using a single test, maximising identification of true positives and minimising the proportion of false negatives in the results is paramount. Empirically, dogs affected by hip dysplasia but scored by the NZVA at 1 year of age are less likely to receive scores for the presence of osteophytes compared to older dogs, as there has been less time for such lesions to develop (Smith *et al.* 2012). In consequence, the extent of subluxation and the Norberg angle (surrogates for the degree of hip laxity) may be the only indication of CHD status on radiographs from dogs scored at 1 year of age. Distraction radiography has been shown to more reliably determine the degree of hip laxity than the traditional hip-extended radiographic view. If the subtotal score does not identify all dogs with laxity then the positive predictive value of using hip-extended radiographs will be lower than using a distraction radiographic method. However, if the NZVA subtotal score correlates closely with passive laxity then recommendations based on the subtotal score could be used as an alternative to PennHIP to select dogs for breeding, with confidence that their progeny should have more favourable hip conformation.

The aim of this study was to determine the degree of agreement between the NZVA Hip Dysplasia Scoring Scheme and the PennHIP DI in NZ PDS GSDs, and to determine whether using the NZVA subtotal score or its components, improves agreement between these two different methods of scoring dogs for the presence of hip dysplasia.

Materials and methods

All GSD police dogs older than 1-year of age (the minimum age for NZVA CHD scoring) presented to the Massey University Veterinary Teaching Hospital between November 2003 and September 2007 for PennHIP radiographic evaluation were included in the study. Thirty-five dogs were radiographed for both PennHIP and the NZVA CHD scheme on the same day. In 10 dogs, the NZVA hip score had been previously determined 4 to 61 (median 37) months prior to radiographs for PennHIP assessment (older existing breeding stock). All dogs were presented for evaluation as potential breeding stock and were free of obvious orthopaedic disease as determined by clinical examination by the author.

All dogs were deeply sedated with a combination of 0.005 mg/kg medetomidine (Domitor; Pfizer Australia Pty Ltd, West Ryde, NSW, Australia) and 0.1-0.2 mg/kg butorphanol (Butorphic; Lloyd Laboratories, Shenandoah, Iowa, USA) I/V, or a combination of 0.01 mg/kg medetomidine and 0.1-0.2 mg/kg butorphanol I/M. A small number of dogs required additional doses of 0.005 mg/kg medetomidine I/V to obtain a sufficient depth of anaesthesia for manipulation of the hips. The standard NZVA radiographic view was taken with the dogs positioned in dorsal recumbency, then each dog was repositioned for the compression and distraction views according to the protocol for the PennHIP method (Smith *et al.* 1990). At the end of the procedure the dogs were given 0.025 mg/kg of atipamezole HCl (Antisedan; Pfizer Australia Pty Ltd), to reverse the effects of medetomidine anaesthesia.

For each dog a single hip-extended ventro-dorsal radiograph was submitted to the NVZA CHD Panel (Wellington, NZ) and the PennHIP radiographs were submitted to the PennHIP Analysis Center (Malvern PA, USA). Panellists scoring the radiographs were unaware of the aims of the study, and interpreted the radiographs as normal submissions without bias.

The NZVA CHD scheme semi-objectively grades subluxation/incongruity of the hip as well as the presence of osteophytes indicative of joint degeneration, from a hip-extended ventro-dorsal radiograph. The total score is the combination of the 6 categories of degeneration (different locations of osteophyte development) and the 3 categories that indicate subluxation and/or incongruity of the hip. The three categories for subluxation/incongruity are the Norberg angle score, the subluxation score and cranial acetabular edge (a score for any divergence of the joint at that location).

The Norberg angle is considered “normal” if ≥105°, receiving a score of 0. Deviation below 105° is scored from 1 to 6 on an interval scale, Table 3 (page 33). The subluxation score, also referred to as femoral head coverage, is scored by assessing the position of the centre of the femoral head in relation to the dorsal acetabular edge (Figure 6, page 31), and is graded 0 to 6 (Table 3, page 33). The cranial acetabular edge is graded 0 to 6 according to the descriptions presented in Table 3 (page 33).
The PennHIP method reports a DI as a unit-less ratio of the distance by which the centre of the femoral head is displaced laterally from the centre of the acetabulum relative to the radius of the femoral head when distracted by way of a fulcrum (Figure 7, page 36).

Statistical analysis

The frequency distribution of individual hip DI scores and NZVA total and subtotal scores were tested for normality using the Kolmogorov-Smirnov test. The relationships between these scores, and with the Norberg angle grades and NZVA subluxation scores (which were both interval scales) were assessed using measures of correlation. Pearson’s correlation was used when both parameters were normally distributed and Kendall’s tau-b was used when one or both parameters were not normally distributed. Correlation analysis was then repeated using the mean DI and the mean NZVA scores for each dog. Mean values were used to compensate for suboptimal positioning that can occur during the NZVA hip-extended ventro-dorsal view, which will increase the extent of subluxation on one hip, whilst reducing the extent of subluxation of the other. It was therefore important to average the scores to compensate for the effect of any oblique positioning during radiography. The 95% confidence limits were estimated for all correlations using the asymptotic calculated standard errors.
The relationship between the hip DI, and the Norberg angle and subluxation score components of the NZVA subtotal score was then assessed using univariate ANOVA, with DI as the dependent variable, and right or left hip and either Norberg angle scores or subluxation score as fixed effects. To ensure at least six dogs in each group, Norberg angle scores were divided into two groups (original score 0 or ≥1), as were scores for subluxation (original score ≤1 or ≥2). A univariate ANOVA was then repeated using mean DI, mean Norberg angle score and mean subluxation score. The Norberg angle scores were divided into three groups (mean score 0, 0.5, 1), while subluxation scores were divided into four groups (mean score ≤1, 1.5, 2 and 2.5) for analysis. The NZVA scores for these variables are whole number values, averaging the two hip scores can lead to a value midway between two full numbers, hence 1.5 and 2.5.

The DI scores were then dichotomised into either low risk, or increased risk of CHD. The threshold for the DI was set at 0.3. Dogs with a DI of ≤0.3 in both hips are considered non-susceptible for DJD (Smith *et al.* 1993). For the NZVA score three thresholds were used based on the most recent NZVA recommendation to selectively breed only dogs with a subtotal score of ≤2 per hip. The first threshold was a total score of >2, the second was a subtotal score of >2 and the third was a score of >1 in any individual category. The sign test was then used to determine whether each of the three NZVA threshold values identified the same proportion of 'at-risk' dogs as the DI threshold of >0.3. Where no significant difference in proportion of 'at-risk' dogs was identified, the Kappa test was used to estimate the level of agreement between each of the thresholds.

Also, the scores were re-dichotomised into either low risk, or increased risk of CHD using less restrictive thresholds. The threshold for the DI was set at 0.5 (>0.5 considered increased risk for DJD). The threshold for NZVA total score was set at 8 (>8 considered increased risk for DJD). A score of 8 on the NZVA/BVA system is equivalent to the lower limit of 'good' hip conformation" as defined by the OFA (Table 4, page 32). These less restrictive guidelines were accepted as criteria for breeding selections having previously been recommended by the NZVA.

All statistical analyses were undertaken using SPSS 16.0 for Windows (SPSS Inc., Chicago IL, USA).

Results

The 47 dogs, 16 male and 31 female, ranged in age from 12 to 84 (median 17.5) months and weighed 25.5 - 43.2 (median 30.5) kg. All dogs were being evaluated for inclusion in the NZ Police dog-breeding programme. Most dogs were bred in NZ but some dogs were imported.

In this group of dogs, the recorded DI of individual hips ranged from 0.21 to 0.65 (median 0.36). The median DI (for the group) of the worst (most lax) hip of each dog was 0.39. This is comparable to the median of 0.40 for all GSDs recorded in the PennHIP database (N=7810, accessed February 2008). The NZVA subtotal score ranged from 2 to 11 (median 6), and the NZVA total score ranged from 2 to 12 (median 6). This compares favourably to the rolling 5-year median of 8 for all GSDs tested by the NZVA method (calculated from the NZVA data by the author). Individual hip and mean DI and NZVA subtotal scores were normally distributed, as were mean NZVA total scores and the NZVA total score for the right hip. However, the NZVA total score for the left hip was not normally distributed (p=0.015).

The DI for the left hip and right hip in the same dog were significantly correlated (Pearson's r = 0.5; 95% CI = 0.28 to 0.73). However, for the NZVA scores there was no such correlation, with Pearson's r = 0.12 for the subtotal score (95% CI = -0.08 to 0.32), and Kendall's τ = 0.07 for total score (95% CI = -0.15 to 0.28). This lack of correlation was also true for the subluxation score (Kendall's τ = -0.09, 95% CI = -0.34 to 0.16) but for Norberg angle the relationship between the scores of the two hips was significant at the 5% level (Kendall's τ = 0.34, 95% CI = 0.07 to 0.6).

Comparing individual hip scores; the subtotal score and total score for each hip using the NZVA scoring system were significantly correlated (Pearson's r = 0.94 for right hip; 95% CI = 0.89 to 1; Kendall's τ = 0.87 for left hip, 95% CI = 0.76 to 0.99). Additionally, the left DI was significantly correlated to both left subtotal and left total score (Pearson's r = 0.53; 95% CI = 0.31 to 0.75 and Kendall's τ = 0.29 for left hip, 95% CI = 0.02 to 0.55 respectively). However for right hip scores there was no such correlation (Pearson's r = 0.24 for subtotal score; 95% CI = = -0.09 to 0.50; Pearson's r =0.24 for total score, 95% CI = -0.07 to 0.58). This lack of correlation is illustrated in Figure 12. Within each hip, the Norberg angle score and the subluxation score were significantly correlated (Kendall's **τ** for left hip = 0.47; 95% CI = 0.22 to 0.72 and 0.37 for the right; 95% CI = 0.22 to 0.72). However, when these parameters were compared to the DI of the same hip, the only significant association at the 5% level was that between the subluxation score and DI of the left hip (Kendall's **τ** = 0.35; 95% CI = 0.16 to 0.54). For the left hip Norberg angle score the association was almost significant (Kendall's **τ** = 0.24; 95% CI = -0.07 to 0.49) but for the right hip neither the subluxation score nor Norberg angle score were significantly correlated with the DI (Kendall's **τ** = 0.08; 95% CI = -0.18 to 0.33 and Kendall's **τ** = 0.09; 95% CI = -0.16 to 0.34, respectively).

For individual hips, the mean DI of hips with a Norberg angle score of 0 was lower than those with a Norberg angle score of ≥1 (p=0.063). For the subluxation score, hips that had a score of ≤1 had a significantly lower mean DI than those with a subluxation score of ≥2 (p=0.025). The relationship between the Norberg angle score, subluxation score and DI for individual hips is illustrated in Figure 13.

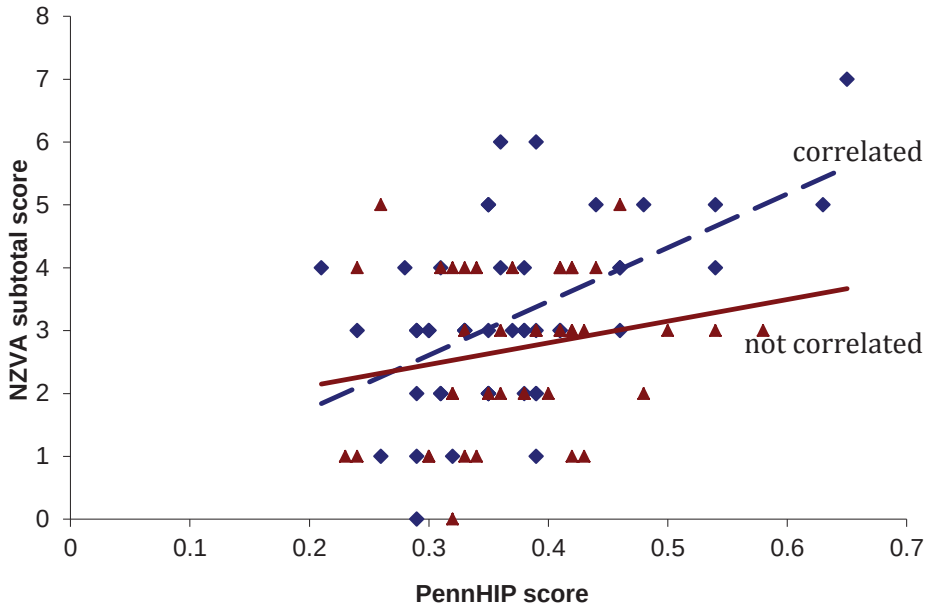


Figure 12: Correlation between the PennHIP score and the New Zealand Veterinary Association subtotal scores for individual (left ♦ and right ▲) hips in 47 German shepherd dogs. The dashed line is line of best fit for the left hip (r=0.507) and the solid line is line of best fit for the right hip (r=0.14).

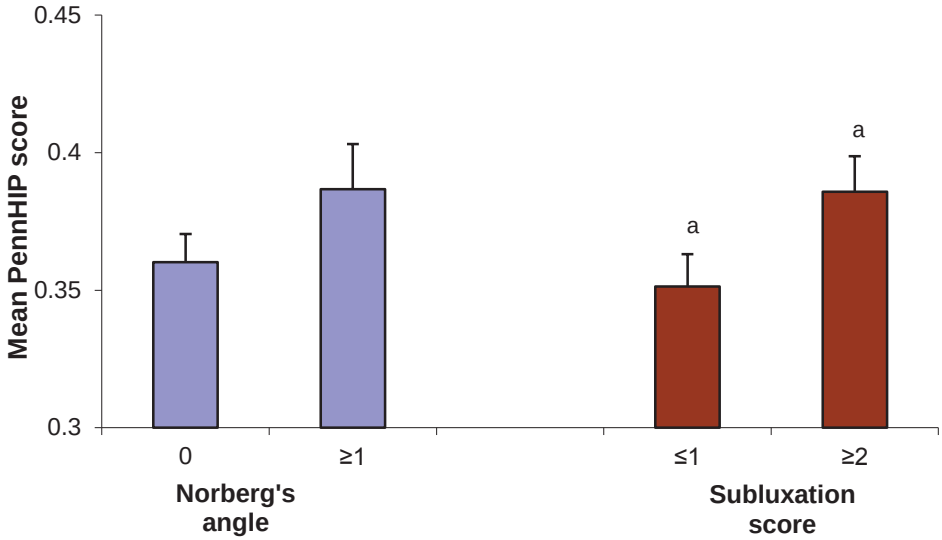


Figure 13. Relationship between Norberg angle score and subluxation score with PennHIP distraction index in individual hips from 47 German shepherd dogs. [a]Indicates a significant difference in mean PennHIP score between the two score categories(p=0.025).

In none of the ANOVA analyses was there a significant effect of whether the hip was right or left side or any significant interaction between sides ($p>0.1$). When the mean rather than the individual hip Norberg angle score was used, no significant association with the mean DI was found ($p=0.18$); for mean subluxation score the association tended towards significance at the 5% level ($p=0.06$).

The proportion of 'at-risk' dogs for each threshold is summarised in Table 10. A frequency table for the 'at-risk' dogs with a DI threshold of >0.3 and an NZVA subtotal score of >2 are shown in Table 11 (page 65).

The proportion of dogs identified as being 'at risk' of CHD by DI, subtotal score, total score and individual category score were similar for all comparisons when combined results from both hips were used ($p>0.5$ for all comparisons). However, when the results from individual hips were compared there were marked differences between left and right hips. For the left hip all comparisons identified a similar proportion of 'high-risk' hips ($p>0.45$), but for the right hip all comparisons were significantly different ($p<0.04$). Reducing the 'at-risk' threshold for the subtotal and total scores to >1 resulted in all comparisons for the right hip no longer being significantly different ($p>0.17$). Using this lower threshold for data for the left hip reduced the p-values for the comparisons but they remained above the 10% level. The threshold of >1 was thus used for further comparisons using data from individual hips.

Table 10. Table showing the number of dogs classified as high or low risk of developing hip dysplasia from a population of German shepherd dogs (N=47) of the NZ PDS. The risk of developing hip dysplasia was determined by using four separate thresholds, based on the Pennsylvania Hip Improvement Program (PennHIP) distraction index or the New Zealand Veterinary Association (NZVA) Canine Hip Dysplasia scheme.

Programme Criteria	**Risk**	
	High	**Low**
PennHIP Distraction Index[a]	44	3
NZVA subtotal score[b]	45	2
NZVA total score[b]	45	2
Categorised NZVA score[c]	41	6

[a] Distraction index >0.3 in either hip indicative of high risk of developing CHD

[b] Score >2 for combined score of both hips considered indicative of high risk of developing CHD

[c] Score >1 in any category or either hip indicative of high risk

Table 11. Frequency table showing the distribution of the NZVA and PennHIP scores of 47 German shepherd dogs when the scores are classified into high and low risk for development of hip dysplasia.

	Number of Dogs		
	NZVA total >2[a] (High Risk)	NZVA total ≤2[a] (Low Risk)	Total
DI >0.3 [b] (High Risk)	41	3	44
DI ≤0.3 (Low risk)	3	0	3
Total	44	3	47

[a] High risk if total NZVA score >2, low risk if total NZVA score ≤2

[b] High risk if DI >0.3, low risk if DI ≤0.3

Table 12. Comparison of agreement (Kappa values, and approximate significance/P-value) between the PennHIP distraction index (DI) and the New Zealand Veterinary Association Canine Hip Dysplasia score based on 'high' and 'low' risk thresholds for the development of hip dysplasia. Kappa values in ***bold*** *were significantly greater than 0 (P<0.05).*

	*Kappa (*confidence interval*)*		
PennHIP Distraction Index	NZVA CHD hip scheme		
	Total score[a]	Total subluxation score[a]	Categorised score[b]
DI worst[c]	-0.05 (-0.1 to 0)	-0.05 (-0.1 to 0)	-0.1 (-0.18 to 0.02)
DI left hip[c]	0.21 (-0.09 to 0.5)	**0.27** (-0.05 to 0.59)	0.22 (-0.08 to 0.53)
DI right hip[c]	**0.39** (0.06 to 0.74)	**0.44** (0.1 to 0.78)	0.12 (-0.11 to 0.34)

[a] Score >2 for total score indicative of risk of CHD, >1 for individual hip score

[b] Score >1 in any category indicative of risk of CHD

[c] Score >0.3 indicative of risk of CHD. For DI worst a score of >0.3 in either hip indicates risk

The Kappa results for the dichotomised comparisons are shown in Table 11. For the total values no agreement was found (Kappa value <0). In all cases, none of the dogs determined by the NZVA score as being low risk was scored as low risk by the PennHIP method, and *vice versa* (Table 10, page 64). The Kappa values were higher when results for individual hips were scored, however the values ranged from 0.12 to 0.44, which indicates only a low to moderate association between the NZVA and PennHIP systems.

Increasing the DI threshold to 0.5 for either hip identified an appreciably smaller proportion (5/47) of dogs as being at high risk of CHD. The proportion of dogs identified using an NZVA threshold of ≥8 was higher than that for the DI of 0.5 for both subtotal (11/47) and total score (13/47) (p=0.07 and 0.021, respectively). Increasing the NZVA threshold to ≥9 resulted in a smaller proportion of dogs identified as at 'high-risk' (5/47 and 10/47 respectively), which for both total and subtotal scores was not significantly different from that identified using the DI threshold of >0.5. The Kappa values using the ≥9 threshold were moderate for subtotal (0.55; 95% CI 0.16 to 0.94) and low for total score (0.31; 95% CI -0.02 to 0.63).

Discussion

Laxity of the hip joint has been shown to be a primary phenotypic factor in predicting a dog's susceptibility for developing coxo-femoral DJD (Smith *et al.* 1993, 1995, Popovitch *et al* 1995). The NZ PDS has been using the NZVA CHD scoring scheme to select dogs for favourable hip phenotype since the early 1990s. However the true extent of laxity of the hip joint can be underestimated from radiographs when a dog is positioned with the hips extended according to the NZVA/BVA method of scoring, (see chapter two). The heritability of the hip extended radiologic phenotype has been estimated to be 0.29 (weighted average) (Janutta and Distl 2006). The division of the NZVA score into the subtotal score, in addition to the total score, was intended to provide breeders with better guidance as to the primary phenotypic markers of CHD. The markers related to laxity, i.e. Norberg angle, degree of subluxation and the incongruity of the cranial acetabular edge, were grouped separately from those markers that related to osteophyte development and the presence of DJD. The subtotal score was intended to provide an indirect measure of the extent of coxo-femoral joint laxity. The PennHIP method was developed to objectively measure maximum passive hip laxity as a unit-less index (Smith *et al.* 1990). A recent report using the linear animal model estimated the heritability value of the DI using the PennHIP to be 0.83 ± 0.11 in a sample of 215 Estrela Mountain dogs (Ginja *et al.* 2008b).

However, it is more widely accepted that the heritability of the PennHIP DI is around 0.6 in most populations (Kapatkin *et al.* 2002).

In this study, the NZVA total and subtotal scores, and the components of the subtotal score, were compared to the PennHIP DI within a sample population of GSDs of the NZPDS. There was only limited agreement between either the NZVA subtotal or the total score, and the PennHIP DI. The DI is related to the risk of developing DJD (higher score = higher risk) (Popovitch *et al.* 1995, Smith *et al.* 1995). There is no data on the predictive value of the individual components of the NZVA subtotal score. Also, of the phenotypic radiological markers subjectively evaluated by the Orthopedic Foundation for Animals on a hip-extended radiograph, none has been shown to be statistically significant as a predictor of DJD, the ultimate outcome of hip dysplasia (Smith *et al.* 1993, 1995, Popovitch *et al.* 1995). The poor correlation with the DI suggests that an increased NZVA subtotal score above the analysed thresholds may not be linked to an increased risk of developing clinical CHD.

There was no association between passive laxity using the PennHIP DI and Norberg angle as determined using the NZVA scheme. A significant correlation between Norberg angle and the DI has been previously reported in German shepherd dogs (Culp *et al.* 2006) and Estrela Mountain dogs (Ginja *et al.* 2008a). Those workers compared the actual measured Norberg angle rather than a score on an interval scale. As the NZVA CHD system only reports the allocated score and not the angle measured, it is possible that if the Norberg angle were compared as a continuous variable rather than an interval scale the correlation would improve. The accepted threshold for Norberg angle for normal hip conformation as used by the NZVA is 105° (Lawson 1963) but use of this “cut-off” value has recently been questioned. In the study by Culp *et al.* (2006), 42% of Borzois tested had a Norberg angle of 99-105°. According to the NZVA scheme these dogs would have received a score of 1 for each hip for Norberg angle score. Yet the Borzoi is considered a non-CHD-susceptible breed, and in the same study all individuals had a DI of ≤0.32 Culp *et al.* (2006). Dogs with a DI ≤0.3 are considered to have normal (not lax) hips and are thus not susceptible to DJD (Smith *et al.* 1993, 1995, Popovitch 1995, Kealy *et al.* 1997). Using a 105° for Norberg angle as the cut off value may therefore increase the number of false positive dogs for CHD, i.e. lowering specificity for selection by this criterion.

In this study, there was a significant association between the DI and subluxation score but the overlap was large and the difference between means was small (0.02 on the PennHIP scale). A “hip-extended index”, derived in the same way as the PennHIP DI (difference between the centres of the acetabulum and femoral head divided by the femoral head radius) but taken from an OFA view without distraction, has been compared with the DI using the standard PennHIP method (Kapatkin *et al.* 2004). The hip-extended index would be similar to the NZVA subluxation score, except that the hip-extended index is a unit-less ratio and the NZVA subluxation score is an interval scale. In the study by Kapatkin and others, there was a significant correlation between a hip-extended-index and DI (r = 0.52) suggesting that both indices reflect degrees of laxity. However, those authors showed that if the threshold of normality (non-susceptible to DJD) was set at a hip-extended-index value of ≤0.24 (the highest value found in Borzois in the study), then most dogs of other breeds included in the study would have been classified as non-susceptible for DJD. From these data it can be extrapolated that the subluxation score alone, whilst specific for laxity and if high, a good indicator of laxity, is not as sensitive as the DI for determination of maximal passive laxity.

There was better agreement between the left and right hips using distraction indices than using the NZVA subtotal scores. CHD is known to be a bilateral disease (Smith *et al.* 1990), thus it is likely that the PennHIP results are more accurate. The disparity between the NZVA subtotal scores for the left and right hip of each dog is likely due to the effect of positioning of the dog for radiography. In order for the dorsal acetabular rim to project equally over each femoral head, the patient must be positioned symmetrically beneath the X-ray beam. Any tilting of the pelvis results in an oblique image and potential underestimation of the Norberg angle and subluxation score of one hip and over estimation of the other. For this reason the authors also compared mean Norberg angle and subluxation scores with the DI. Better agreement was found between the DI and mean subluxation score, than between the DI and the Norberg angle score.

Whilst the mean subluxation score may be a more representative criterion of laxity, in this dataset the improved agreement between DI and mean subluxation score may have been due to good correlation of the left hip masking the effect of poor correlation of the right hip. Veterinarians should carefully assess hip radiographs for symmetry of the obturator foraminae and the ilial wings prior to submitting hip-extended radiographs to the NZVA CHD panel. Radiography should be repeated until a symmetrical image is

obtained to avoid potential misinterpretation of evidence for subluxation. The radiographs analysed in this study were of high quality and accepted as suitable for scoring by the NZVA CHD panel, but despite this it is improbable that they were all exactly symmetrical.

The NVZA subtotal and total scores were strongly correlated. The development of DJD in CHD-susceptible dogs is linear into later life, rather than bimodal as previously thought (Smith *et al.* 2012*,* Smith *et al.* 2006). The total score includes those criteria that score the presence of DJD. Thus, older dogs (with CHD) would be expected to have greater disparity between total and subtotal scores than similarly-at-risk group of dogs radiographed at younger ages. In this population, 24/47 dogs were ≤18 months of age and could be expected to have lower scores for the presence of DJD even if affected by CHD. Therefore the total NZVA score is almost entirely determined by the components relating to laxity, which make up the subtotal score.

The statistical analysis undertaken for this study compared the NZVA hip extended radiological phenotype with passive hip laxity as determined by distraction radiography without assuming that either was the “gold standard”. All dogs were free of significant lameness at the time of examination, and longitudinal clinical data were not available. End-of-life and necropsy data would be required to conclusively classify dogs as dysplastic or non-dysplastic. If the NZVA score and PennHIP DI were equally effective as screening tools for CHD then the data from the same dogs should have shown a significant degree of agreement between the two tests, however this was not evident. Separate thresholds for NZVA score and DI were used to independently categorise the dogs as being at risk. Irrespective of the threshold chosen the proportion of high-risk dogs was very similar. Agreement between the two systems however was poor, as each system identified a different group of dogs as low risk when a lower threshold was used and a different group of high-risk dogs when a higher threshold was used. For the lower thresholds there was no agreement between either the subtotal or total NZVA scores and the DI. It is likely that the absence of dogs which were low risk as determined by both schemes may have skewed this analysis, and a more normally distributed dataset would be needed to confirm a complete lack of agreement. However, in this dataset, using the results for individual hips (doubling the dataset) only increased the degree of agreement to moderate, which suggests that even if a larger population were used, agreement would still be insufficient for either of the two schemes to be used interchangeably. Using a higher threshold improved agreement but there were still a considerable number of dogs that were identified as low risk with one test and high risk with the other.

To put our results into perspective we can use a simple comparative ranking analysis. Assuming the NZ PDS intends to breed the twenty dogs with the most favourable hip phenotype. If the 47 dogs are ranked by the NZVA subtotal score (lowest to highest) and then independently by DI (lowest to highest), of the top twenty dogs in each ranking, only 11 would be ranked in the top twenty by both methods.

For the original set of thresholds (NZVA ≤2, DI <0.3) the high proportion of dogs identified as 'at risk' meant that we gained an accurate assessment of how likely we were to identify a dog as at low risk of CHD using the DI or NZVA scale when the other test would suggest that it was high risk, but not how likely we were to identify a dog as low risk with one test when the other suggested it was not. The resulting dilemma can be illustrated using the data shown in Table 9. Forty-four dogs had a DI >0.3, of which two (5%) had an NZVA score of ≤2. Adding another dog to either category, changing this figure to 3/45 (7%), or 2/45 (4%), would not markedly change this percentage. In comparison, three dogs had a DI ≤0.3, all of which had an NZVA score >2. Adding another dog to either category would alter the ratio to 0/4 (0%) or 1/4 (25%), appreciably affecting the relationship. In the study by Culp *et al.* (2006), which analysed 350 dogs of 7 breeds, the Norberg angle had a positive predictive value (proportion of animals testing positive that are truly DJD-susceptible) of only 64% in the German shepherd breed.

In conclusion, the lack of correlation between the NZVA total and subtotal scores and the PennHIP DI is of concern as ranking dogs by each method gave disparate results. The advantage of a hip-extended radiographic view is its low cost and widespread availability, which would have financial benefits to the NZ PDS. However, the NZVA subtotal score can seemingly not be used as a surrogate for the DI. Comparisons between individuals may not be accurate due to poor sensitivity for laxity and the potential for false negative scores due to positioning artefact. Given the very high reported heritability of hip laxity as measured by the DI, and the relationship between laxity and the development of CHD, the NZ PDS should expect to make greater gains over fewer generations by selecting breeding stock from animals with a low DI.

The data and figures from this study have been published - Worth AJ, Laven RA, and Erceg VH. An assessment of the agreement between the New Zealand Veterinary Association Hip Dysplasia Scoring System and the PennHIP Distraction Index in German Shepherd dogs. New Zealand Veterinary Journal, 57 (6), 338-345, 2009

Lumbosacral disease in working dogs – pathogenesis, principles of diagnosis and investigative imaging.

In chapter one, the survey of police dog handlers showed that 34% of NZ Police dogs retired or lost from service were retired or euthanased due to conditions affecting their "back" or "spine," 73% of which involved the lumbosacral (LS) junction. Based on the author's experience with Police dogs at the Massey University Veterinary Teaching Hospital, degenerative lumbosacral stenosis (DLSS) is the most common cause of disease of the LS junction in GSDs of the NZ PDS. In this chapter the anatomy of the LS junction and DLSS will be discussed.

Anatomy of the canine lumbosacral junction (Figures 14,15 page 84)

The LS junction in the normal dog is the articulation between the seventh lumbar vertebra (L7) and the sacrum. L7 has a single spinous process (dorsally) and left and right transverse (lateral) processes. An intervertebral disc separates the vertebral bodies of L7 and the sacrum. Synovial joints (*juncturae zygapophyseales*) are present between the articular processes, which project dorso-laterally from the lamina. The articular surface (facet) of each articular process is lined by hyaline cartilage and faces the articular surface of the opposing process. The L7 paired cranial articular processes project laterally to the caudal articular processes of the sixth lumbar vertebra. The paired caudal articular processes of L7 project between the cranial articular processes of the sacrum (Evans 1993).

The orientation of the articular joint surfaces (facet angle) determines the biomechanical function of the articulation. The more sagittal the facets are orientated, the more satisfactorily the articulation will counter rotation and lateral translation. In the adult, the sacrum results from fusion of the foetal S1, S2 and S3 vertebrae. In the normal adult dog there are no spaces or intervertebral discs between the bodies of these three sacral vertebrae. The spinous processes of the first to third sacral vertebrae fuse to form the median sacral crest. The wing (alar process) of the sacrum articulates with the wing of the ilium via the sacro-iliac joint (*articulatio sarcoiliaca*), which has both a gliding synovial component and a restrictive fibro-cartilaginous component (*synchondrosis sacroiliaca*) (Evans 1993).

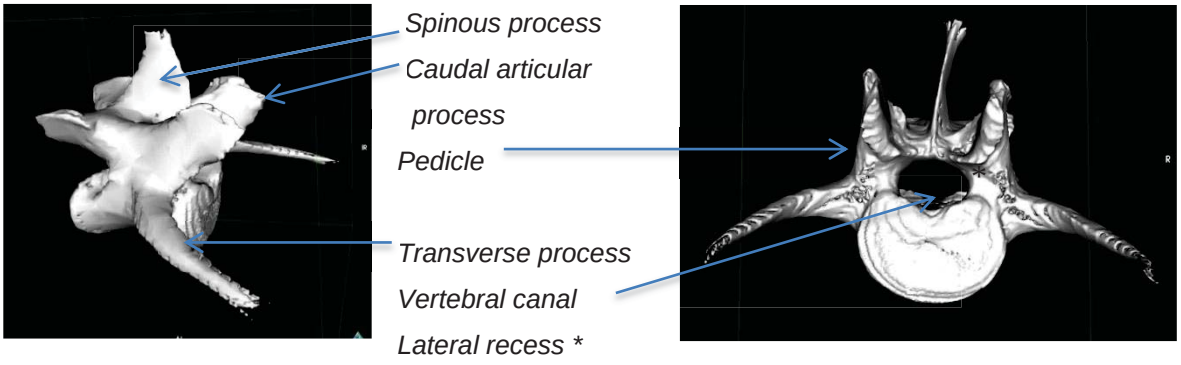


Figure 14. Normal anatomy of the canine seventh lumbar vertebra. Left image is a lateral oblique view, right image is a caudal view.

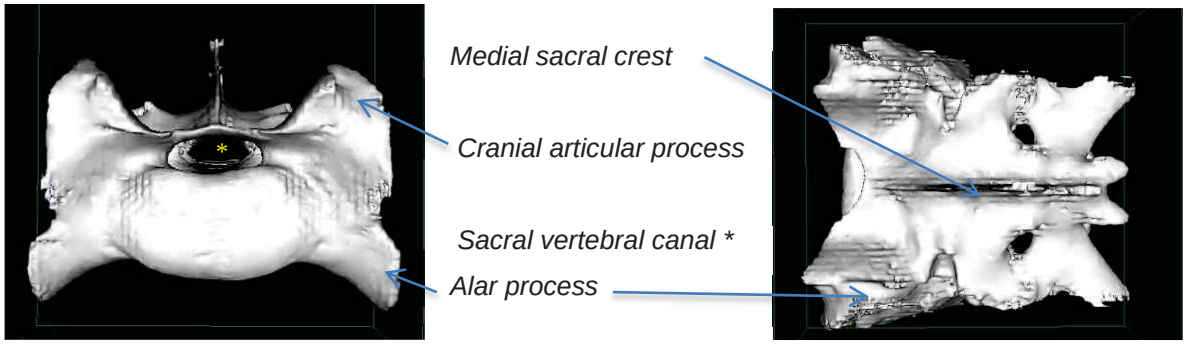


Figure 15. Normal anatomy of the canine sacrum. Left image is a cranial view, looking caudal, right image is a dorsal view.

Transitional vertebral anomalies of the lumbosacral junction

Variations of the normal morphology of the lumbosacral vertebrae are termed transitional vertebral anomalies (Damur-Djuric *et al.* 2006). They can be classified by their appearance and the symmetry of the transverse processes, by determining abnormal segmentation of the vertebral column, and/or the extent and nature of the articulation between the vertebrae and the ilial wing. In humans, lumbosacral transitional vertebrae have three classifications (Blumensaat and Clasing 1932).

1. Uni- or bilateral alteration of the transverse and/or costal elements
2. Uni- or bilateral alteration of the ventral alar element
3. Complete lumbarisation of S1 (8 lumbar vertebrae present) or sacralisation of L7 (6 lumbar vertebra present) with symmetrical formation

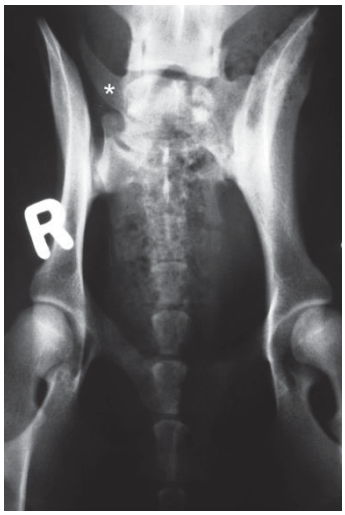
The terms lumbarisation and sacralisation have been used in the veterinary literature but may not properly reflect the true process of formation of a lumbosacral transitional vertebrae (Briet *et al.* 2002, Damur-Djuric *et al.* (2006).

Lumbarisation refers to an anomalous sacral vertebra with the morphological appearance of a lumbar vertebra. Sacralisation refers to an anomalous lumbar vertebra with the morphology of a sacral vertebra. Without radiographs of the entire vertebral column to determine the number and morphology of all the vertebrae, it is not accurate to label a transitional vertebral anomaly as lumbarisation or sacralisation.

In the veterinary literature, an alternative classification system has been proposed by Damur-Djuric *et al.* (2006) based on the morphological characteristics of the transverse processes of the lumbosacral transitional vertebrae:

1. Lumbar type transverse process (no attachment to the ilium)
2. Intermediate type transverse process (partly attached to the ilium)
3. Sacral type transverse process (broad sacral attachment)

Using Damur-Djuric ***et al.'s*** system the lumbosacral transitional vertebrae is classified with two numbers according to the morphology of the right and left processes (R/L). Symmetrical lumbosacral transitional vertebrae will therefore have the nomenclature 1/1, 2/2 or 3/3, whereas asymmetrical lumbosacral transitional vertebrae will be the remaining combinations (e.g. 1/2, 3/2). A type 3 of the Blumensaat and Clasing classification would be either a 1/1, 2/2 or 3/3, in the system proposed by Damur-Djuric *et al.* (2006).



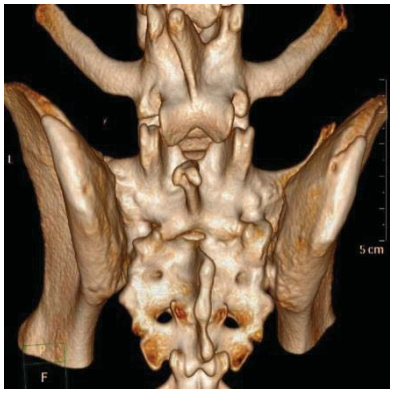


Figure 16. Transitional vertebral abnormalities in two German shepherd dogs. The ventro-dorsal radiograph left shows an asymmetrical transition anomaly of the lumbosacral junction. The transitional vertebral segment has attachment to the sacrum on the left whereas there is a transverse process on the right (asterisk) resembling a lumbar vertebra, type 1/3 (Damur-Djuric). There are seven normal lumbar vertebrae in this dog therefore assuming there are the seven cervical and thirteen thoracic vertebrae, the anomaly can also be termed asymmetrical lumbarisation of S1. On the right a 3-D CT reconstruction shows a symmetrical transitional LS vertebral anomaly with L7 attached to the sacrum on both sides.

Neuroanatomy of the lumbosacral junction

The lumbar spinal cord segments lie progressively more cranial than the same numbered vertebrae due to continued growth of the vertebral column after cessation of growth of the spinal cord. As a result the nerve roots of the caudal lumbar nerves

traverse down the vertebral canal for increasing distances before they exit at their respective lateral intervertebral foramina. The terminal lumbar, sacral and coccygeal roots exit the dura and run within meningeal tubes surrounded by epidural fat and collectively are known as the *cauda equina*. The dorsal root ganglia are located at the junction of the dorsal and ventral roots near each intervertebral foramen. Combined dorsal and ventral roots form a short main nerve trunk, which then divides into meningeal, dorsal, communicating and ventral branches. The ventral branches of *L4-7* and the sacral nerves combine to form the lumbosacral plexus from which the peripheral nerves to the pelvic limbs arise. The neurovascular bundle exiting the L7-S1 lateral intervertebral foramen is comprised of the dorsal and ventral *L7* nerve roots and the communicating artery and vein.

Degenerative stenosis of the lumbosacral junction in dogs

Pathogenesis

The term degenerative lumbosacral stenosis was first used by Chambers *et al*. (1989) to define an acquired narrowing of the vertebral canal, intervertebral foramina, or both, which results in compressive radiculopathy of one or more nerve roots of the *cauda equina* (Chambers 1989, DeRisio *et al*. 2001). The LS junction of large-breed dogs is prone to degenerative change, seemingly related to concurrent degeneration of the intervertebral disc, analogous to L5-S1 intervertebral disc degeneration in humans (Chambers 1989, Bergknut 2011). One or more nerve roots of the *cauda equina* become compressed by alterations of the surrounding soft and bony tissues associated with the vertebral column, coupled with or caused by suspected instability of the L7–S1 intervertebral motion segment (Chambers 1989, Godde and Steffen 2007). The motion segment of the LS junction is defined as the caudal lumbar and cranial sacral vertebrae and their soft-tissue connections, comprising the articular processes and the intervertebral disc, together with the associated joint capsules and ligaments (Godde and Steffen 2007).

Normally, vertebral stability is attained by passive and active components, which comprise a highly mobile neutral zone of physiological motion, and a more restricted elastic zone of low motion and high resistance. Passive stabilisation is provided by the vertebral bodies, intervertebral discs, longitudinal ligaments, *ligamentum flavum*, articular processes, and the joint capsules. The active stabilizers of the lumbosacral motion segment are the epaxial and hypaxial paravertebral musculature. Through proprioceptive input, appropriate muscular tone and contraction helps to limit motion and help prevent injury. Not only is the epaxial and hypaxial paravertebral musculature

involved in providing stability, but also the abdominal and truncal muscles restrict and support motion of the lumbosacral junction (Demoulin *et al*. 2007).

In humans, and possibly in dogs, the degenerative process that leads to DLSS starts in the intervertebral disc as a result of prolonged overuse associated with activity and age, and by changes that occur from excessive strain beyond the normal physiological limits of function of the LS junction (Dupuis 1987). Radiological progression of DLSS has been reported by Steffen *et al*. (2007) in a cohort of Police GSDs. There was evidence of an increase in the degree of *spondylosis deformans*, a narrowing of the lumbosacral joint space, changes in the height of the vertebral canal, and an increase in the degree of mineralisation of L7-S1 discs noted over 3 years. It was suggested that faltering diffusion of nutrients into the *nucleus pulposus* leads to gradual change in the biochemistry and micro-architecture of the avascular regions of the disc (Dupuis 1987, Bray and Burbidge 1998a). The concentration of proteoglycan and water in both the *nucleus pulposus* and *annulus fibrosus* diminishes, and the disc becomes stiffer (Dupuis 1987). The eventual outcome is fibroid metaplasia. The once gelatinous nucleus begins to protrude through tears in the weakened annulus, resulting in a Hansen Type II disc protrusion, and rarely results in extrusion of the *nucleus pulposus* [Hansen Type I extrusion] (Bray and Burbidge 1998b). The degenerating annulus cannot absorb and distribute loading, and the resultant tearing of Sharpey's fibres leads to the formation of osteophytes around the periphery of the LS junction. The intervertebral disc space is narrowed and the *annulus fibrosus* protrudes dorsally reducing the dorso-ventral dimension of the vertebral canal. The resultant loss of intervertebral spacing, allowing the vertebrae to move closer together, reduces the volume of the lateral intervertebral foramina (Chambers 1989). The *L7* nerve roots, having left the dura at the level of the L6 vertebral body, lie within the lateral recess of the vertebral canal of L7 (Axlund and Hudson 2003) (figure 17). The caudal aspect of the dorsal body of L7 has a dorso-lateral notch that accommodates the *L7* nerve roots as they enter the intervertebral foramen. Thus the L7 roots lie cranial to the L7-S1 intervertebral disc.

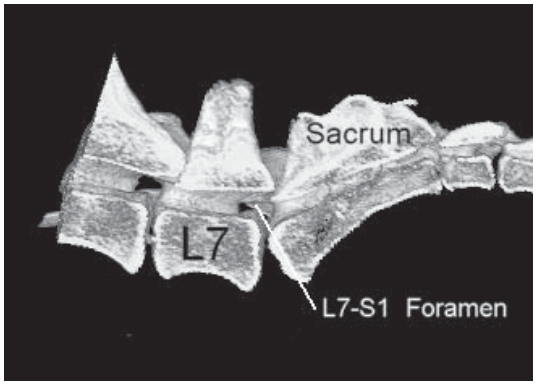


Figure 17. Reconstructed computed tomographic image showing the internal detail of the lumbosacral canal and L7–S1 lateral intervertebral neurovascular foramen of a dog. The L7 nerve passes caudally in the lateral recess before reaching the entrance zone of the lateral intervertebral foramen (labelled).

Each lateral intervertebral foramen is not a simple aperture but rather a tunnel, with an entrance, middle and exit zone in relation to nerve roots transiting from the vertebral canal to the periphery (Wood *et al*. 2004b). Protrusion of disc material in a dorsolateral direction can affect the entrance and middle zones of the foramen, while compression of the exit zone may be the result of instability of the LS junction, osteophytosis, or hypertrophy of the joint capsule of the articular processes (Godde and Steffen 2007). The degree of the resultant anatomical changes affects the nature and severity of the clinical signs detected. Central disc protrusion into the vertebral canal causes compression of the sacral and caudal nerves, potentially leading to faecal and/or urinary incontinence and abnormal carriage of the tail (Chambers 1989). In contrast, lateral (foraminal) compression of the L7-S1 neurovascular bundle leads to pelvic limb signs such as repetitive flexion of a hind limb, pain elicited on manual limb extension, intermittent to progressive lameness, and sciatic nerve deficits in severe cases (Chambers 1989). Whilst DLSS is considered likely to induce neuropathic pain (Mathews 2008), evidence of the induction of neuropathic pain has not been documented. Compression of the middle and exit zones was a feature of DLSS in 13/20 (65%) dogs in one study (Godde and Steffen 2007). The changes eventually lead to exercise-dependent compression of the nerve roots of the *cauda equina* and associated vasculature within the vertebral canal and/or its associated foramina (DeRisio *et al*. 2000).

Predisposing factors in the development of DLSS

Large-breed dogs, especially the German shepherd dog (GSD), retrievers, and active or working dogs (Police, military or sporting dogs) are commonly affected by DLSS (Chambers 1989, Ness 1994, Danielsson and Sjöström 1999, DeRisio *et al*. 2001). Affected dogs are generally heavier than 25 kg bodyweight, and mature. The mean age at the time of first diagnosis and treatment in the two largest studies to date were 5.5 and 5.8 years (Danielsson and Sjöström 1999, Suwankong *et al*. 2008). Males are over-represented, with a male: female ratio of >1.7:1 (Danielsson and Sjöström 1999, DeRisio *et al*. 2001, Suwankong *et al*. 2008). Whether breed, gender, weight, degree of activity, and specific use are independent or linked variables as risk factors for the development of DLSS has not been determined.

Undetected discospondylitis of the L7-S1 IVD may be associated with DLSS. Sterile swabs taken from the *nucleus pulposus* during partial discectomy when cultured recovered bacteria in 12/52 (23%) cases in one study (Suwankong *et al*. 2008). Some of the bacteria cultured were consistent with contamination of the sample,

e.g. *Bacillus* spp., whereas others were potentially pathogenic, e.g. *Staphylococcus pseudointermedius*.

Police dogs are trained to scale vertical walls, an activity that requires maximal LS extension, and to bite and hold an offender by the arm, an activity that requires maximal LS flexion. Both of these activities are thought to stress the LS junction, which may lead to degeneration of the LS intervertebral disc. In addition, there are congenital and developmental anomalies that may predispose some dogs to DLSS. A lesion resembling osteochondrosis has been reported, which affects the dorsal end plate of S1 in the GSD (Lang *et al*. 1992, Hanna 2001, Mathis *et al*. 2009) and is thought to occur with increased frequency in dogs with *cauda equina* syndrome compared with unaffected dogs (Hanna 2001). Ondreka *et al.* (2013) reported a 10.1% incidence of sacral osteochondrosis in GSDs, which was significantly (P=0.013) higher than that in non-GSDs (5.7%). Affected dogs may have displacement of the osteochondral bodies into the vertebral canal or LS lateral recesses causing nerve root compression (Figure 18). In addition the author has seen degeneration of the L7-S1 disc with subsequent protrusion after previously successful removal of the osteochondral lesion (n=1). The loss of normal end-plate architecture and damage to the dorsal annulus associated with removal of the lesion may predispose the dog to instability of the LS junction and further L7-S1 IVD degeneration. Sacral osteochondrosis has been reported to have moderate heritability (0.5) in GSDs (Ondreka *et al.* 2013)

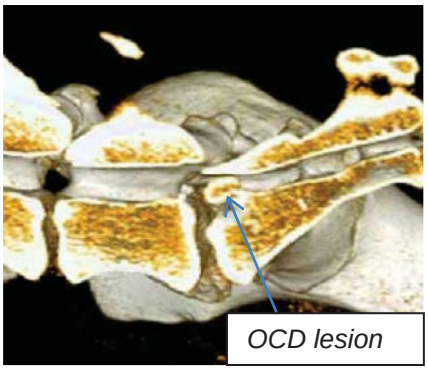


Figure 18. Three dimensional CT reconstruction image of a German shepherd dog with an osteochondrosis (OCD) lesion affecting the cranio-dorsal aspect of the vertebral body of the sacrum. The reconstruction has been sectioned along the sagittal plane and the left side has been removed to display the interior detail of the vertebral canal.

Lumbosacral transitional vertebrae occur more frequently in GSD than in other breeds (Morgan *et al*. 1993, Ondreka *et al.* 2013). The incidence of transitional vertebrae in the GSD population has been reported to range from 3.5 to 29%, and have varying prevalence within populations of GSD from different countries (Scharf *et al*. 2004; Damur-Djuric *et al*. 2006; Wigger *et al*. 2009). Dogs with a lumbosacral transitional vertebra (LTV) reportedly have a higher risk of developing *cauda equina* syndrome (Morgan *et al*. 1993), putatively due to abnormal rotational forces induced by

malalignment and malarticulation of the components of the LS junction. Dogs with transitional LS vertebrae were eight times more likely to develop *cauda equina* syndrome than dogs without abnormal LS vertebrae and GSDs were eight times more likely to develop *cauda equina* syndrome than other breeds, and at a significantly younger age (Fluckiger *et al*. 2006). Those authors hypothesised that an LTV accelerates degeneration of the disc cranial to the transitional vertebrae, or that the L7-S1 disc itself is dysplastic and therefore prone to premature degeneration.

If abnormal transmission of force through the LS junction predisposes a dog to intervertebral disc degeneration and DLSS, then asymmetrical LTV should induce more significant alteration of forces directed through the LS junction and thus be more likely to induce DLSS than symmetrical LTV. In one small case series of dogs with both LTV and clinical DLSS, all six had asymmetric anomalies of the LS junction (Steffen *et al.* 2004). The side with the sacral attachment was unaffected whereas the contra-lateral (lumbarised) side had intervertebral disc prolapse with foraminal involvement. In each case, decompressive surgery was deemed necessary to relieve nerve root compression and surgery ameliorated the clinical signs. However, a larger study (n=4000) found a higher proportion of symmetrical LTV amongst dogs affected by DLSS versus an almost equal distribution of symmetric versus asymmetric LTV in a control population of dogs with an incidental LTV but without clinical signs. Although the presence of a symmetrical LTV was a statistically significant ($P<0.01$) risk factor for development of DLSS, this determination was based on data from only 15 dogs with LTV and DLSS (Fluckiger *et al*. 2006).

When a lumbar transitional vertebra is present, the sacroiliac attachment may be asymmetric, which can result in rotation of the pelvis (around the transverse or dorsal planes) and potentially alter development of the coxo-femoral joint (Damur-Djuric *et al*. 2006). Those authors proposed that a rotational change in the sacroiliac attachment might predispose affected dogs to developing unilateral hip dysplasia. However, in a study of more than 4,000 pelvic radiographs of GSDs, there was no correlation between the presence of a LTV and a diagnosis of hip dysplasia (Wigger *et al*. 2009). The heritability for LTV is estimated to be between 0.2 and 0.64 (Wigger *et al.* 2009, Ondreka *et al.* 2013). In the latter study there was a mild negative correlation between LTV and sacral osteochondrosis. Thus is likely that LTV, sacral osteochondrosis and hip dysplasia are all inherited independently in the GSD and breeding strategies must rely on independent breeding evaluations for each trait.

Additional anatomical features investigated in association with DLSS

In addition to the possible roles of OCD–like lesions and LTV in DLSS, a number of authors have sought to correlate anatomical features of the LS junction with DLSS.

Dorsal narrowing of the vertebral canal by the sacral lamina

The sacral canal is defined dorsally by the inner cortex of the sacral lamina, which appears as a radiopaque line of varying length and clarity on lateral radiographs of the lumbosacral spine. An increased rostral projection of the sacral lamina decreases the size of the interarcuate space, which is a dynamic volume that varies considerably during flexion and extension (Henninger, Werner 2003a). It is the author's observation that some GSDs affected by DLSS have excessive "sacral overhang" resulting in a narrow vertebral canal at the level of the L7-S1 intervertebral disc (Figure 19). Dogs with this long sacral overhang may therefore be predisposed to developing clinical signs of DLSS earlier than dogs with normal sacral conformation as they are less able to tolerate annular protrusion as the disc degenerates. The correlation between sacral overhang and DLSS is being investigated in a study by Hartman and Worth (in draft). Preliminary results confirm the correlation between sacral overhang and DLSS in affected GSDs versus unaffected GSDs and greyhounds.

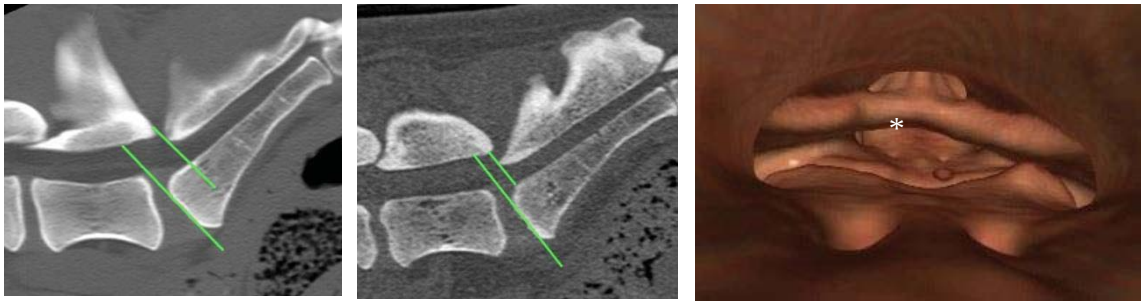


Figure 19. Sagittal CT images of the lumbosacral junction of a German shepherd with typical sacral overhang (left) compared to excessive sacral lamina overhang (middle). The image on the right is a 3-dimensional endoview of the lumbosacral canal showing the projection of the sacral lamina into the canal (asterisk). The image is windowed to exclude soft tissue so the inter-arcuate ligament is not visible.

LS junction angle

The LS junction angle can be measured from lateral radiographs of the LS junction using lines demarcating the vertebral canals of L7 and the sacrum (Figure 20). In a study of 93 normal dogs, the mean LS junction angle in a neutral position (hind limbs perpendicular to the vertebral column) was 159° (range 142-180°) (Mattoon and Koblik 1993). The mean LS junction angle in a flexed position (hind limbs pulled cranially) was

177° (range 163-190°). The mean LS junction angle in an extended LS junction angle was 150° (range (132-167°). Age and body weight had a significant effect on the LS junction extension angle, with older and heavier dogs having larger extension angles, indicating less ability to extend the lumbosacral junction. Dogs (n=26) with DLSS had a larger LS angle at full extension indicating that less extension was possible, a larger flexion angle indicating greater flexion was possible, and less total range of motion than dogs unaffected by DLSS. Schmid and Lang (1993) also reported a reduction in the overall range of motion of the LS junction in GSDs affected by DLSS. In contrast, other investigators have reported that GSDs affected by DLSS had a reduced flexion angle of the LS junction compared to normal GSD (Schmid and Lang 1993). The LS extension angle was the most significant parameter separating DLSS dogs from an age/weight-matched group of normal dogs (Mattoon and Koblik 1993).

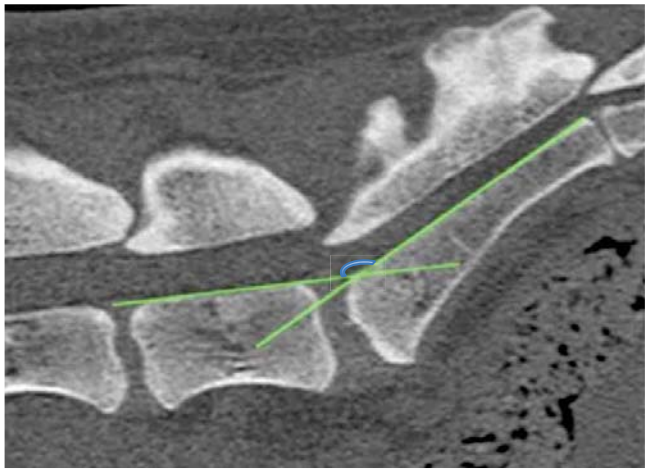


Figure 20. Lateral CT of a German shepherd dog in extended positioning of the LS junction. Lines demarcate the ventral margin of the vertebral canals of L7 and the sacrum. The angle formed by the intersection of the lines is defined as the lumbosacral angle (Mattoon and Koblik 1993).

These results suggest that as DLSS progresses the LS junction becomes less flexible due to hypertrophy of supporting soft tissues and/or osteophytosis. This is counter to the assumption made by many authors that DLSS is associated with instability of the LS junction.

Ventral displacement of the sacrum: step-lesion

In humans, lumbar pain caused by degenerative spondylolisthesis is associated with displacement of L4 anterior to L5, or L5 anterior to the sacrum. In dogs with DLSS, ventral displacement of the sacrum relative to L7 has been reported (Figure 21), which is the equivalent of retrolisthesis (posterior displacement of the proximal vertebra) in humans, which is less common than anterior spondylolisthesis. Ventral displacement of the sacrum relative to L7 is an inconsistent feature of DLSS in dogs and its absence does not preclude diagnosis of DLSS (Meij *et al.* 2007). The presence of a mal-alignment between the caudal vertebral canal of L7 and the cranial sacral canal due to

ventral displacement of the sacrum has been reported in 69% of dogs with DLSS (Suwankong *et al*. 2006). However, this “step-lesion” is also seen in healthy dogs free of clinical signs of DLSS (DeRisio *et al*. 2000). In a comparison of the morphology of the LS junction in the GSD (n=733) to dogs of other breeds (n=334), the presence of a step lesion between L7 and S1 was significantly more common in GSDs, a breed with a predisposition for DLSS (Ondreka *et al.* 2013). A step lesion that is exacerbated by extension of the LS junction is thought to indicate instability of the LS junction (Tarvin and Prata 1980, Slocum and Devine 1986). However, no difference was observed in the extent of ventral displacement of the sacrum from normal GSDs and those affected by DLSS in an earlier clinical evaluation (Schmid and Lang 1993). Mattoon and Koblik (1993) attempted to measure changes in the alignment of the LS junction induced by positioning for radiography. The distance between the locations of the intersection point defined by the LS junction angle in flexion versus extension was quantified. A greater absolute distance was defined as dynamic misalignment and was a feature of dogs with DLSS compared to normal age/weight matched dogs.

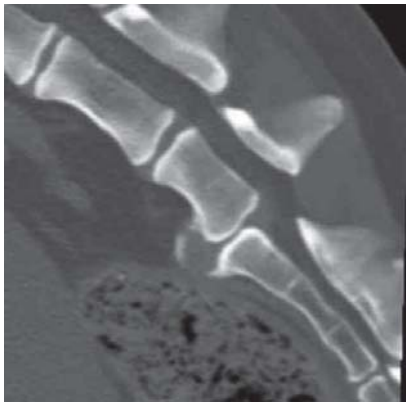


Figure 21. Computed tomographic image of a Labrador retriever showing ventral displacement of the sacrum relative to the L7. This would be termed retrolisthesis in human medicine. The presence of sacral displacement has also been termed instability or subluxation in the veterinary literature. This case represents an extreme example and the dog responded well to stabilisation surgery.

Vertebral canal stenosis

A reduced vertebral canal height throughout the entire length of the LS junction has been noted in GSDs compared to other breeds, using an adjusted ratio which took into account skeletal size (Ondreka *et al.* 2013). There was also a more abrupt drop in vertebral canal height between the caudal aspect of L7 and the cranial aspect of the sacrum. These findings suggest that the GSD may have a degree of primary LS stenosis which then predisposes dogs of this breed to compression of the *cauda equina* with even mild disc prolapse and degeneration.

Lateral intervertebral foramen narrowing and nerve root compression

Linn *et al*. (2003) found that narrowing of the L7-S1 lateral intervertebral foramina indicated a poor long-term prognosis for improvement. Superimposition of the articular processes of L7 and S1 over the lateral intervertebral foramina prevents accurate evaluation of the foraminal dimensions on radiographs, particularly in patients with degenerative changes of the articular processes, and those with spondylosis. Later investigators have sought to demonstrate the effect of DLSS on the size of the L7-S1 lateral intervertebral foramina by measuring the cross-sectional area of the foramen on sagittal CT images at the extremes of range of motion (Jones *et al*. 2008). Demonstration of an excessive dynamic reduction in lateral intervertebral foraminal cross-sectional area would be indicative of *L7* nerve root compression at full extension of the LS junction. In that study dogs with DLSS had decreased mean L7-S1 intervertebral foraminal area when positioned in LS extension compared to LS flexion. In dogs with clinical signs of DLSS (hind limb lameness and/or lumbosacral pain) there was a linear (negative) relationship between the LS junction angle and foraminal area, i.e. as extension increased, the L7-S1 intervertebral foraminal area decreased. In control dogs, unaffected by DLSS, there was no such relationship. Those authors hypothesised that one of the normal functions of the passive stabilizers of the LS junction is to maintain the dimensions of the lateral intervertebral foramina independent of the angle of the LS junction. However the percentage change in foraminal area from full flexion to full extension was not significantly different between affected and control dogs, a finding which did not support the hypothesis that DLSS should result in a greater decrease in the foraminal area. The measurements were made in sagittal orientation, rather that perpendicular to the curving path of the *L7* roots through the lateral intervertebral foramen, which may have affected the accuracy of these measurements. Orientating slices to be perpendicular to the nerve roots has been investigated by Higgins *et al.* (2011) but has not been reported in a clinical setting. Modern CT workstations can produce volume reconstructions of acquired anatomical data making it possible to measure the lateral intervertebral foraminal volume. Volume data may be a more accurate reflection of the dynamic changes to the foramen during motion. Measurement of foraminal volumes in affected and unaffected dogs is the subject of chapter seven.

Orientation of the articular process joint surfaces

It has been hypothesised that differences in the orientation of the articular process joint surfaces on the sagittal, transverse or dorsal planes could influence the type and degree of motion between two adjacent vertebral bodies, thus producing different

mechanical stresses on the interposed intervertebral disc (Seiler *et al*. 2002). Those authors measured the angles of the surfaces of the articular processes of 36 dogs relative to the dorsal and transverse planes, using CT (Figure 22). Angles measured for GSDs were more sagittally oriented at L5–L6 and L6–L7, and there was a larger angle of difference between the lumbar and lumbosacral articular surfaces compared with those of the other breeds assessed (Seiler *et al.* 2002). This data indicates a more rapid transition from upright articular process surfaces of L6-7 (which primarily limits rotation) to flatter L7-S1 articulations (which would limit dorsal subluxation of the sacrum) in the GSD compared to other breeds. Whether there is any causal relationship, between a rapid transition of the angles of the surfaces of the articular processes from L6-7 to L7-S and DLSS is still unknown.

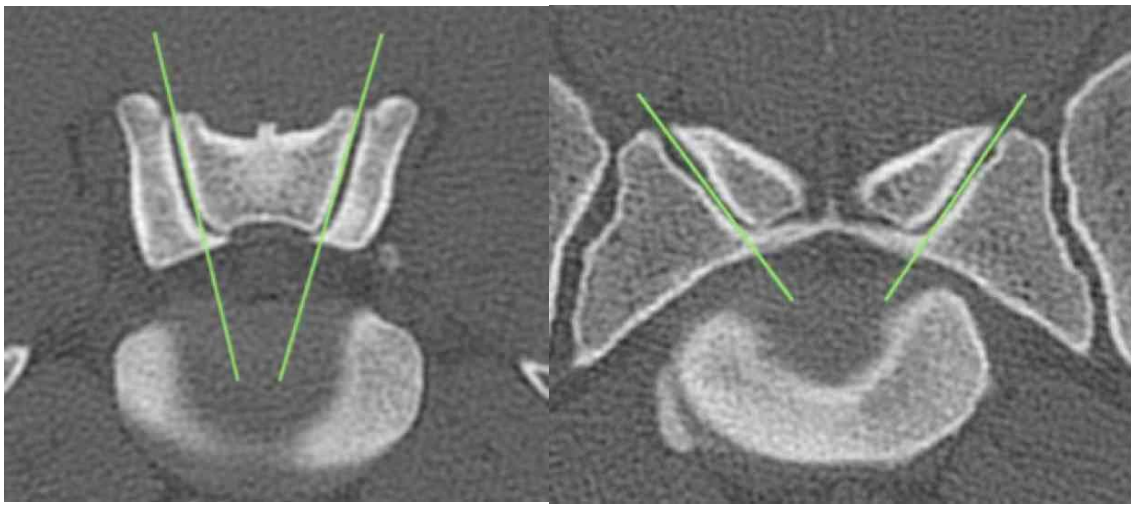


Figure 22. Axial computed tomographic images of a German shepherd dog demonstrating measurement of the angulation of the articular process joints. The image on the left is located at the mid-point of the articular process joint of the L6-7 articulation. The image on the right is at the mid-point of the articular process joint of the L7-S1 articulation. The angle of the articulation of the L6-7 process joints is more sagittal compared to the flatter L7-S1 angle.

Asymmetry of the left and right articular process surfaces of a single vertebra has been termed articular tropism (Rossi *et al.* 2004). Though trophism could alter the distribution of forces on the LS junction, with consequent asymmetric loading of the soft-tissue structures, an association between articular process trophism and IVD degeneration was not found by Seiler *et al.* (2002). Histopathology of the IVDs from the vertebral columns of cadavers, rather than clinical data, was used for the comparison. Later work by Rossi *et al*. (2004) using MRI found that tropism of the articular processes was associated with a greater severity of disc degeneration in dogs with clinical signs of compression of the *cauda equina*. Those authors also found a decrease in tropism with age, suggesting that the surfaces of the articular processes

are not static but undergo modelling over time, probably related to the type and degree of mechanical loading of the LS junction. However, a recent clinical study again found no association between articular process tropism and the severity of clinical signs of DLSS (Suwankong *et al*. 2008).

Summary of additional anatomical features investigated in association with DLSS

The aetiopathogenesis of DLSS is complex, and many questions still remain regarding the relationship between breed specific morphology, risk of work-related injury, the biomechanics of the LS junction, and the development of IVD degeneration. The individual abnormalities frequently associated with DLSS are summarised in Table 13. It is common for several of these abnormalities, components of lumbosacral degeneration, to be present in a dog with DLSS, each contributing to dynamic compression of either the vertebral canal or L7-S1 lateral intervertebral neurovascular foramen.

Table 13. Pathology recognised as a component of degenerative lumbosacral stenosis in dogs

- Hypertrophy of the ligaments stabilising the LS junction (dorsal longitudinal ligament ventrally and *ligamentum flavum* dorsally)
- Degeneration of the lumbosacral disc, with protrusion of the disc annulus
- Degenerative joint disease of the articular processes, with modification of the shape of the articular surface, peri-articular new bone formation, and hypertrophy of the joint capsule
- Lateral *spondylosis deformans* at the lumbosacral junction and sacroiliac joint, which can impinge into the exit zone of the L7-S1 intervertebral foramen, and compress the *L7* intervertebral neurovascular bundle
- Dynamic compression of the *cauda equina* caused by ventral displacement of the sacrum in relation to L7 (step lesion = retrolisthesis)
- Dynamic narrowing of the L7-S1 lateral intervertebral foramen during extension of the lumbosacral joint
- Congenital stenosis of the vertebral canal at the LS junction

Principles of diagnosis of DLSS

Clinical signs

The term '*cauda equina* syndrome' is not analogous to DLSS but rather refers to the complex of clinical signs resulting from a primary lesion involving the *cauda equina*, or secondary disease affecting the L5–L7, sacral or caudal vertebrae, or associated soft tissues, for which DLSS is one of the differential diagnoses (Morgan and Bailey 1990). Other diseases that can lead to *cauda equina* syndrome include; neoplasia, discospondylitis, epidural empyema, and epidural/para synovial cysts. These and other diseases must be ruled in or out by diagnostic investigations (Morgan and Bailey 1990, Palmer and Chambers 1991a).

The diagnosis of DLSS is based on dysfunction typically associated with the LS junction and the presence of pain inducible on LS junction motion or manipulation, combined with supportive imaging findings and the exclusion of alternative differential diagnoses. Chambers (1989) and Ness (1994) highlighted the importance of a high index of suspicion for the condition, and utilisation of appropriate manipulative tests to improve identification of dogs with DLSS. In a retrospective study of 131 client-owned dogs with DLSS, reluctance to jump, or purported evidence of pain when jumping or when rising from a prone position, or when climbing stairs (92%), and purported evidence of pain or stiffness during physical activity (86%) were the clinical observations most frequently cited by the owners (Danielsson and Sjöström 1999). On physical examination, hyperaesthesia isolated to the LS junction by induction of vocalisation or strong avoidance by the dog in response to dorsal pressure or manipulative tests (see later) is an important clinical sign of DLSS. Hyperaesthesia demonstrated during extension, and on direct digital palpation, has been reported in 98% and 85% of dogs affected by DLSS, respectively (Danielsson and Sjöström 1999). Pain may originate from entrapment of nerve roots (radicular pain), degeneration or tearing of the *annulus fibrosus* (discogenic pain), longitudinal ligaments, joint capsules, and periosteum, and irritation of the meningeal tube overlying the nerve root (Palmer and Chambers 1991a).

In humans with L5/S1 IVD degeneration, back pain can worsen acutely during exercise then subside with rest, although this finding is not consistent. One proposed mechanism for exercise-induced pain is increased pressure on the vascular supply of the L5 nerve roots due to dynamic compression, leading to ischaemia, and is termed 'intermittent claudication' (Chambers 1989). Intermittent pelvic limb lameness is reported in some dogs with DLSS (Ness 1994, DeRisio *et al*. 2001, Suwankong *et al*.

2008). Several NZ PDS GSDs have presented to the MUVTH for intermittent lameness induced by activity. They present without overt lameness at the walk and trot, but after jumping a wall will hold one hind limb flexed and be only partly-weight bearing for a short period thereafter. Surgical enlargement of the ipsilateral intervertebral foramen can resolve the clinical signs in these dogs, suggesting dynamic intervertebral neurovascular bundle compression as the cause.

Other reported clinical signs include pelvic limb weakness and ataxia, urinary and faecal incontinence, and a flaccid tail, which are all attributable to neurological dysfunction. Careful palpation may reveal atrophy of the gluteal or flexor muscles of the stifle (Palmer and Chambers 1991a). It is rare for dogs with DLSS to be unable to walk.

Physical examination

Physical examination of dogs suspected to have DLSS should include direct digital pressure applied dorsally over the lumbosacral space with and without extension of the hips; the 'tail jack', and the 'lordosis' tests (Figure 23) (Chambers 1989; Palmer and Chambers 1991a). Hyperaesthesia on dorsal LS junction pressure, vocalisation, aggression or avoidance behaviours on manipulation are all putative indications of a pain response from the patient. Signs of pain elicited on extension of the hip are non-specific for lumbosacral disease as they can occur with diseases of the lumbar vertebrae or coxo-femoral joint. Signs of pain on abduction or rotation of the hip are more specific for coxo-femoral joint disease. The perception of pain induced during extension of the hip with absence of pain on abduction or rotation of the hip is suggestive of lumbosacral or lumbar vertebral disease. Localisation of pain on manipulation to the LS junction is further reinforced by the examiner placing one hand between the dogs thighs to cradle the pubis, the other hand dorsally over the LS junction, then extending (lordosis of) the LS junction.

Figure 23. The lordosis test for the presence of lumbosacral pain in dogs. The test can be performed with the dog standing or in lateral recumbency. Either an examiner's hand (standing) or knee (recumbent) is placed as a fulcrum at the lumbosacral space, and the pelvic limbs and pelvis are firmly extended together.

Neurological examination may reveal depressed cranial tibial, peroneal, and withdrawal reflexes, and normal to exaggerated patellar reflexes. This ‘pseudo-exaggeration’ is the result of hypotonia of the flexor muscles of the stifle, which normally antagonise the quadriceps muscles in the patellar reflex. Thus the presence of an exaggerated response should not be confused with upper motor neuron disease (Chambers 1989; Morgan and Bailey 1990).

Diagnostic imaging

Plain radiography

Diagnostic investigation of *cauda equina* syndrome begins with plain radiographs of the lumbosacral region to rule out bone-associated neoplasia, discospondylitis, lesions from trauma, and vertebral anomalies (Morgan and Bailey 1990). For the lateral view the pelvis must be carefully positioned with pads, to ensure it is positioned parallel to the x-ray plate (Figure 24). The ventro-dorsal view should be taken with the pelvic limbs drawn forward, to position the lumbosacral disc space parallel to the beam (McKee and Dennis 2003) (Figure 25, page 88). Signs of DLSS on plain radiographs may include ventral or lateral spondylosis, narrowing of the intervertebral disc space, end-plate sclerosis of caudal L7 and cranial S1, and ventral displacement of the sacrum at the LS junction (Mattoon and Koblik 1993). However, plain radiography has poor accuracy due to both false-positive and false-negative diagnoses; the former due to presence of degenerative changes without clinical signs, the latter due to inability to image soft-tissue structures.

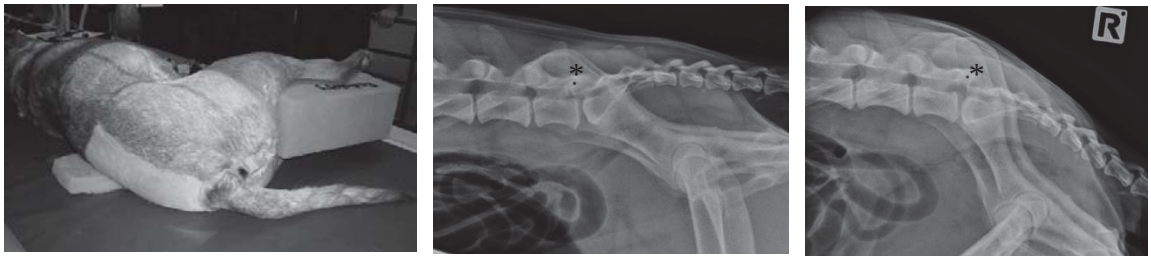


Figure 24. a) b) c)
Correct positioning for a lateral radiograph of the lumbosacral joint of the dog. (a) A small foam pad is placed under the lumbar vertebrae, and a foam block between the pelvic limbs. (b) Extended lateral radiograph of the normal lumbosacral joint of the dog, with minimal obliquity. (c) Flexed lateral radiograph of the normal lumbosacral joint of the dog, demonstrating the normal change in shape of the lumbosacral intervertebral disc space. Also note that the lumbosacral articular processes (asterisk), positioned dorsolateral to the vertebral canal at the lumbosacral intervertebral space, summate dorsally over the vertebral canal and should not be mistaken for a compressive bony lesion.

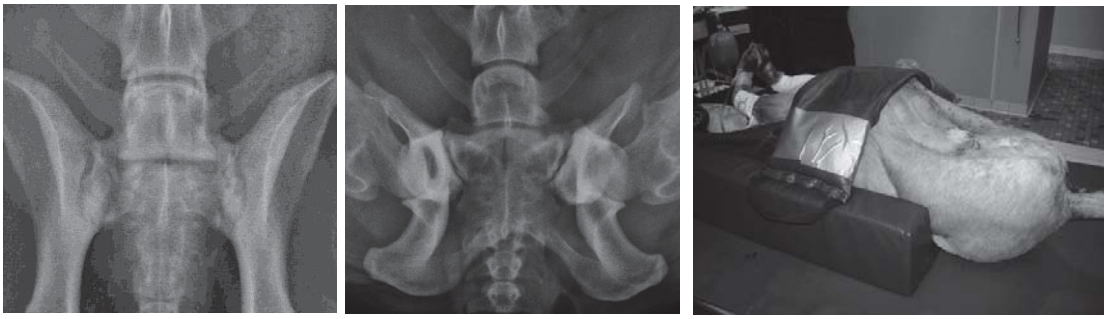


Figure 25. a) b) c)
Ventro-dorsal radiography of the lumbosacral joint in the dog. (a) Standard ventro-dorsal extended view of the pelvis, showing the typical inclined view of the sacrum. (b) Flexed ventro-dorsal view of the pelvis, as described by McKee and Dennis (2003). This view aligns the lumbosacral joint space parallel to the X-ray beam. Notice the clear view of the lumbosacral articular processes and sacral wings. This view is valuable when there is suspected trauma or neoplasia. (c) Correct positioning to obtain the view in (b). The pelvic limbs are drawn forward into maximum hip flexion, and the rump is raised.

In a study of active Swiss Police GSDs, radiological changes consistent with DLSS were found in 15 out of 21 dogs that had neurological signs and/or signs of back pain, and 18 of 36 dogs without clinical signs (Scharf et al. 2004). A correlation between neurological and radiological findings was not found. The width of the L7-S1 IVD space, LS junction angle in neutral positioning, and the presence of ventral sacral displacement were not significantly different from dogs with or without neurological signs. The positive predictive value of ventral spondylosis at L7–S1 was only 57% for the presence of concurrent clinical signs of lumbosacral disease. When dogs aged <5 or ≥5 years were compared, spondylosis/spondylarthrosis and sclerosis of the endplates of L7 and/or S1 were significantly more prevalent with increased age. Plain radiological findings were not predictive for the development of DLSS in a cohort study of working GSDs (Steffen *et al.* 2007).

hypere A radiolucency within the lumbosacral disc, called a vacuum phenomenon, is a well-recognised radiological sign of degenerative disc disease in humans, and has been seen in the lumbosacral disc in dogs with DLSS after positioning for radiography with the hind limbs xtended for radiography (Hathcock 1994; Schwarz *et al*. 2000).

Contrast radiography

Contrast radiography (myelography, discography, epidurography) was once the most suitable method to investigate the lumbosacral joint, but contrast imaging techniques have been superseded by advanced imaging methodologies (Ramirez and Thrall 1998;

DeRisio *et al*. 2000). An experimental comparison of myelography, intra-osseous caudal vertebral venography, and epidurography showed that in healthy dogs none of these techniques consistently produced images of sufficient quality to contribute to the diagnosis of DLSS. Epidurography yielded the largest number of positive diagnoses of a lumbosacral silicone mass simulating disc protrusion into the ventral vertebral canal. However, the sensitivity of the technique for detecting protrusion of the L7-S1 was <50% in the lateral, and 20% in the ventro-dorsal radiographic projections, respectively (Hathcock *et al*. 1988).

Myelography was initially thought unhelpful for detection of lumbosacral lesions in large breed dogs due to the dural sac reportedly terminating cranial to the L7-S1 intervertebral space in many dogs of large breeds. However, in a study of dogs with DLSS, the dural sac extended over the lumbosacral space in 30/36 (83%) dogs imaged (DeRisio *et al*. 2001), confirming an earlier report that the subarachnoid contrast column crossed the lumbosacral space in 19/22 normal and 21/26 affected dogs (Lang 1988). In dogs with DLSS, the contrast column was compressed and displaced dorsally during extension of the LS junction whereas it was centrally located and unaffected by flexion and extension in unaffected dogs. Myelography is thus likely to be useful for detecting dynamic extradural compression at the LS junction in many dogs with DLSS.

Discography involves the injection of contrast into the L7-S1 disc via a spinal needle inserted across the vertebral canal through the dorsal intervertebral space of L7-S1. Healthy non-degenerate discs resist injection of more than 0.1 ml of contrast agent. Leakage into the canal is diagnostic of a disc extrusion but as most dogs with DLSS have only a disc protrusion the position of the contrast is simply seen to project more dorsal than expected. (Sisson *et al*. 1992, Barthez *et al*. 1994). If CT or MRI are unavailable, myelography can be performed. Injection of contrast agent into the cisterna magna is preferred over a lumbar injection site to avoid inadvertent epidural injection of contrast. After cisternal injection of contrast the dog is re-positioned in lateral recumbency with the LS junction alternately in neutral, flexed and extended positions. LS junction flexion should precede extension to aid the flow of contrast into the caudal subarachnoid space (Lang 1988). If myelography is non-diagnostic then discography and epidurography are further investigative options. The most significant limitation of contrast radiography is its inability to reveal compression of the *L7* nerve root(s) within the LS lateral intervertebral foramina.

Computed tomography

Computed tomography [CT] offers many advantages over conventional radiology for imaging the lumbosacral region, including better contrast resolution of soft tissue and

transverse (axial) image orientation (Jones *et al*. 1995). Positioning the dog in flexion and extension of the LS junction allows changes in the dimensions of the lateral intervertebral neurovascular foramen and dorsal intervertebral canal to be demonstrated. Henninger and Werner (2003a) found that the L7-S1 lateral intervertebral neurovascular foramen and lumbosacral interarcuate (dorsal intervertebral) space appeared wider on flexion than on extension on sagittal images in normal dogs (Figure 23).

A further advantage of CT is the ability of the computer software to reformat dorsal and sagittal images from transverse planes. With the advent of multi-slice, helical scanning devices, the rapid generation of high-resolution volume datasets has improved the ability to reconstruct the lumbosacral region in three dimensions, which reduces the amount of volume-averaging artefact. CT imaging allows evaluation of the lateral recesses of L7, the lateral intervertebral foramina, the articular processes, and the extent of any bulge or prolapse of the dorsal annulus (Jones *et al*. 1995) (Figures 17 and 26).

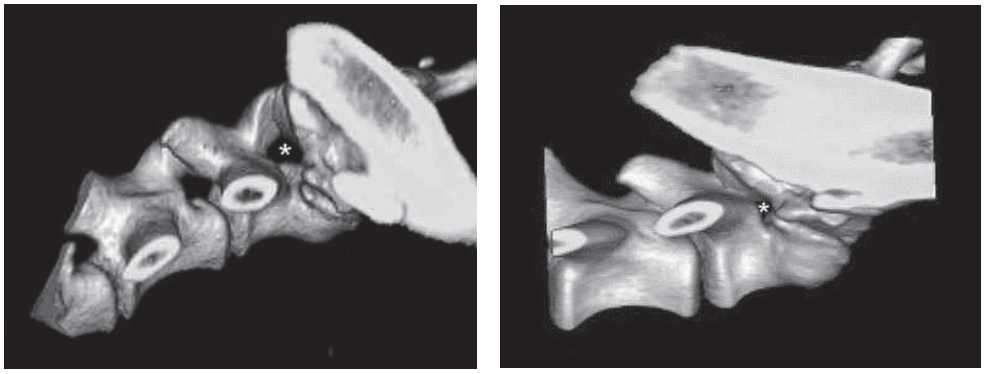


Figure 26. Three-dimensional reconstructions of computed tomographic data from a German shepherd dog with signs consistent with degenerative lumbosacral stenosis, showing changes in foraminal size (asterisk) during flexion (left), and extension (right). There is new bone formation on the vertebral endplates immediately ventral to the intervertebral foramen.

CT abnormalities which have previously been documented in dogs with clinical signs of DLSS have included loss of epidural fat, increased opacity of soft tissue in the intervertebral foramina, bulging of the intervertebral disc, spondylosis, displacement of the dural sac, a smaller L7-S1 lateral intervertebral neurovascular foramen, a narrowed vertebral canal, thickened articular processes, and osteophytosis of articular processes (Henninger and Werner 2003ab).

However, Jones and Inzana (2000) reported CT abnormalities in 5/6 large-breed dogs without clinical signs of lumbosacral disease. The amount of bulging of the dorsal disc as a proportion of the dorso-ventral dimension of the vertebral canal has been reported

to average 27% (20–43%) in normal Hound dogs (Axlund and Hudson 2003). Therefore care must be taken not to over-interpret the degree of disc protrusion in dogs with suspected DLSS. In patients with clinical signs consistent with *cauda equina* syndrome, compression should be suspected at the locations where there is an increase in opacity of peri neural soft tissue, together with the absence of epidural fat (Henninger and Werner 2003b).

Magnetic resonance imaging

Magnetic resonance imaging (MRI) is the most sensitive imaging modality for detecting degeneration of the *nucleus pulposus* due to fibroid metaplasia, which characterises disc degeneration in large-breed dogs. Though detectable in both T1- and T2-weighted images, the loss of signal due to dehydration of the disc is best seen in the latter (Karkkainen *et al*. 1993, Godde and Steffen 2007) (Figure 27).

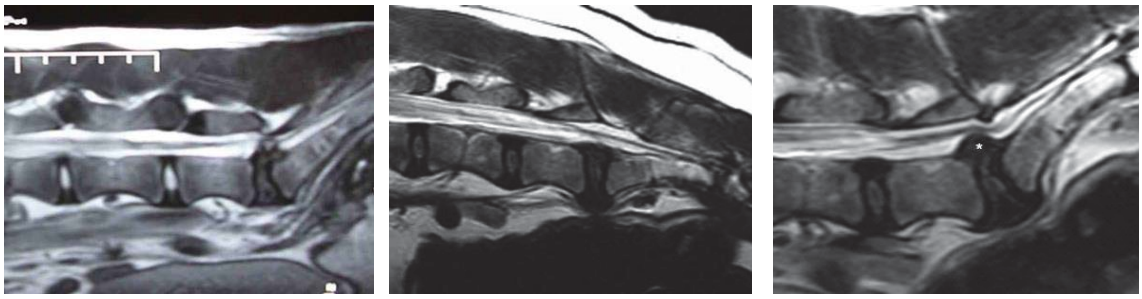


Figure 27. a) b) c)
Sagittal magnetic resonance images of a German shepherd dog with signs of degenerative lumbosacral stenosis. (a) On a T2-weighted image the signal from the nucleus pulposus of the L7–S1 *intervertebral disc is hypo-intense compared with the bright signal from normally hydrated discs; positioning the dog first in (b) flexion, then (c) extension demonstrates the dynamic nature of the* L7–S1 *articulation. Note the protrusion of the* L7–S1 *intervertebral disc into the ventral vertebral canal with extension (asterisk), and ventral compression of the nerve roots at this level; this compression is exacerbated by the bony reaction on the ventral face of the adjacent S1 articular process.*

Prolapse of an IVD, as well as attenuation of the normal epidural fat signal which is indicative of foraminal compression of nerve roots, can be detected by MRI (Adams *et al*. 1995; Godde and Steffen 2007) (Figure 28). Recognition of LS foraminal stenosis and compressive radiculopathy has increased greatly with the use of MRI, with up to 68% of dogs with DLSS reportedly affected (Adams *et al*. 1995; Mayhew *et al*. 2002). For accurate comparison of both foramina, meticulous positioning of the animal and a 2-mm slice protocol are important in order to avoid partial-volume artefacts and false-positive findings (Godde and Steffen 2007). The dog should be positioned in dorsal

recumbency with the aid of positioning troughs and sandbags. The patient must be orientated in the scanner gantry with the vertebral column parallel to the table in the longitudinal plane. Rotation around the vertebral axis must be avoided. Flexed and extended LS junction positioning is as previously described for dynamic radiography.

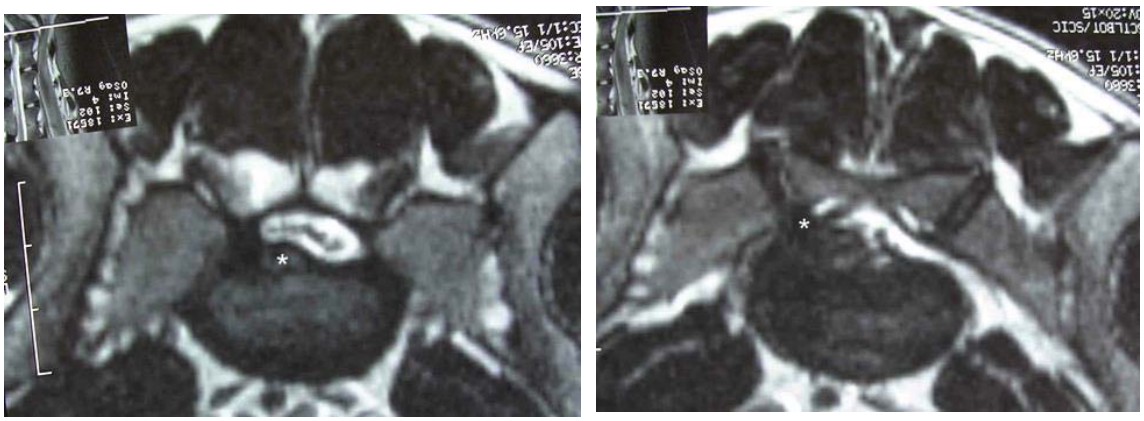


Figure 28. Axial magnetic resonance image of a German shepherd dog at the level of the L7–S1 *disc space (left image). There is protrusion of the annulus of the disc (asterisk), and (right image) loss of fat signal in the intervertebral foramen (asterisk).*

The link between the results of imaging studies and response to therapy

The results of imaging studies, including CT and MRI, may be less important than clinical or surgical factors in predicting an outcome for any dog affected with DLSS. No significant associations between imaging studies and post-operative outcome were identified in 12 working dogs that had been treated surgically for DLSS (Jones *et al*. 2000), although the low statistical power of that study should be noted. Similarly, no correlation between the degree of *cauda equina* compression and the severity of the clinical signs was shown in 27 dogs with DLSS (Mayhew *et al*. 2002). In that study, MRI scans were performed with the dog in a frog-leg position rather than with the lumbosacral joint in extension, a factor that may have reduced the degree of compression identified in some dogs. In another study, there was substantial agreement between CT findings and MRI findings with regard to the location of disc protrusion (Kappa=0.63), the degree of disc protrusion (Kappa=0.88), the position of the dural sac (Kappa=0.89), the amount of epidural fat (Kappa=0.72), and the amount of swelling of spinal nerve roots (Kappa=0.60). However, the degree of agreement between CT and surgical findings, and between MRI and surgical findings was only moderate with regard to the degree and location of disc protrusion (Kappa=0.44–0.56) and swelling of spinal nerve roots (Kappa=0.40 and 0.50) (Suwankong *et al*. 2006). In a later study, dogs with severe disc protrusion were 6.5 times more likely (P=0.013*)*,

and dogs with a lumbosacral step were 1.8 times more likely (P=0.012) to have ongoing clinical signs and fail to improve after surgery (Suwankong *et al*. 2008).

Summary

Degenerative lumbosacral stenosis designates a clinical syndrome associated with acquired changes to the LS junction in large breed dogs. The pathogenesis of the disease suggests that morphological attributes of each breed combined with work-related factors lead to degeneration of the L7-S1 IVD. The disc is unable to fulfil its biomechanical function and the supporting soft tissue and bony structures of the junction undergo changes that ultimately contribute to the pathology by compressing the L7, sacral and caudal nerve roots. Dynamic compression of the neurovascular bundles at the level of the L7-S1 lateral intervertebral neurovascular foramina appears to have a significant contribution and warrants further investigation. Degenerative changes of the LS junction lead to clinical signs and, in working dogs surgery may be required to alleviate compression of the *L7* or sacral nerve roots for a dog to return to work. Surgical management of DLSS is reviewed, and the outcome of surgical intervention in working dogs with DLSS and treated by the author is the subject of Chapter 6.

Some of the figures from this review have been published - Worth AJ, Thompson DJ, Hartman AC. Degenerative lumbosacral stenosis in working dogs: Current concepts and review. New Zealand Veterinary Journal, 57(6), 319-30, 2009.

Long-term outcome and ability to return to work of German shepherd dogs after surgical management for degenerative lumbosacral stenosis.

Introduction

In chapter one, disease of the lumbosacral (LS) junction was established as a significant cause of retirement from active service in working GSDs of the NZ PDS. Thirty-three out of 118 dogs that were retired or euthanased had back or spinal problems (28%). Of these dogs, 24/33 (73%) had a back/spinal problem involving the LS junction. The data was derived from a survey of Police dog handlers, not from the medical records or ante/post-mortem veterinary examinations. Therefore a definitive pathological diagnosis for each dog was not available. There are several disorders of the LS junction that could result in back pain, and/or inability to perform work, including degenerative lumbosacral stenosis (DLSS), degenerative myelopathy, a compressive neoplasm, and advanced discospondylitis. However, based on the author's experience with the sub-population of NZ Police GSDs that have been examined at the Massey University Veterinary Teaching Hospital (MUVTH), DLSS is almost exclusively responsible for lower back pain and inability to work. Therefore the majority of the dogs with back pain attributable to the LS junction in the survey were likely affected by DLSS, the subject of review in chapter five.

Both surgical and conservative medical management have been advocated for DLSS. Conservative management, consisting of a reduction in activity and anti-inflammatory medication, has been recommended for those dogs with DLSS that present with pain only, and for those whose lifestyle can be modified to avoid strenuous exercise (Chambers 1989). Corticosteroids and non-steroidal anti-inflammatory drugs (NSAIDs) can provide effective temporary pain relief, but clinical signs typically return when medical therapy is discontinued. Ness (1994) reported that a good outcome was achieved in 8/16 dogs treated conservatively with 8 to 10 weeks rest and NSAIDs, although 25% of the dogs relapsed within the period of the study. In a recent study 17/31 dogs (55%) were successfully medically managed (restricted exercise in combination with anti-inflammatory and analgesic drugs), however 10 (32%) failed medical treatment and underwent surgical decompression, and 3 (10%) were euthanased due to progression of their clinical signs (De Decker *et al.* 2014). Others

have also reported that relapse is common after conservative therapy (Tarvin and Prata 1980), and some have noted that most if not all of the surgical candidates in retrospective studies have had unsatisfactory conservative management prior to surgical treatment (Jones *et al.* 2000, Godde and Steffen 2007). Delays in effective treatment can lead to irreversible neurological signs such as urinary and faecal incontinence, and paresis of the pelvic limbs (Linn *et al.* 2003). Nevertheless, repeated epidural infiltration of methylprednisolone reduced clinical signs in 30/38 (79%) dogs in a preliminary study, and 20 dogs were considered by their owners to be free of signs after a median of five injections (median follow-up 48 months) (Janssens *et al.* 2009). In the author's experience with NZ Police dogs, epidural corticosteroids are effective but relapses are common, and treatment with epidural corticosteroids should be reserved for dogs close to retirement. For working dogs that must continue to perform a strenuous physical working role, surgical management is thought to be more effective but more data is required to support this contention.

Surgical therapy for DLSS is directed at decompression of the *L7* or sacral/caudal nerve roots and/or providing stabilisation of the LS junction (Palmer and Chambers 1991). The most widely reported decompressive surgical technique for DLSS involves a partial dorsal laminectomy of L7 and the sacrum, with a dorsal annulectomy of the L7-S1 intervertebral disc to resect the dorsal *annulus fibrosus* that is commonly prolapsed (Denny *et al.* 1982, Chambers 1989, Ness 1994, Danielsson and Sjostrom 1999, Janssens *et al.* 2000). Other studies have used dorsal decompression without annulectomy, or dorsal decompression with disc fenestration and partial discectomy of L7-S1 (Klaveren *et al.* 2005, Suwankong *et al.* 2007). A dorsal laminectomy only provides access to the entrance zone of the L7-S1 lateral intervertebral neurovascular foramen, which may result in continuation of clinical signs in dogs with narrowing of the middle or exit zones of the neurovascular foramen (Godde and Steffen 2007). Techniques for enlargement of the lateral intervertebral neurovascular foramen include extension of the dorsal laminectomy beneath the articular process of L7, 'facetectomy', and a lateral approach to the exit zone of the foramen (Tarvin and Prata 1980, Chambers 1989, Godde and Steffen 2007). Facetectomy refers to complete removal of the caudal articular process of L7 to 'de-roof' the lateral intervertebral foramen (Palmar and Chambers 1991b). Facetectomy is no longer recommended due to the potential of this procedure to induce instability of the LS junction. The technique of lateral foramenotomy described by Godde and Steffen (2007) involves removing bone from the caudal pedicle of L7 directly over the lateral recess cranial to the foramen thus allowing decompression of the exit and middle zones of the L7-S1 neurovascular

foramen. This new approach offers a means for foraminal decompression, without significant destabilisation of the articular processes. However, the surgical approach required for this procedure is technically challenging to perform and has yet to find favour with a majority of surgeons (personal observation). In 2009 the author travelled to Tuttelingen, Germany to attend a surgical workshop delivered by Tomas Godde, at the Aesculap Centre. The author has since performed the lateral foraminotomy on 12 pet dogs and a smaller number of working dogs all identified with *L7* nerve root compression on CT. A comparison between lateral foraminotomy and any other technique for treatment of DLSS in working dogs has not been published.

An alternative approach to decompression of the LS junction is surgical stabilisation to prevent motion of the LS junction. In 1986 Slocum and Devine advanced a technique for bilateral fixation of the L7-S1 articular processes to permanently stabilise the LS junction in "normal alignment" (the ideal LS angle was not defined). Following a dorsal approach, a laminectomy spreader is used to expand the dorsal interarcuate space until the articular process alignment is subjectively "normal". Smooth or partially threaded bone pins are driven across the L7-S1 articular processes and a bone graft, harvested from the ilial wing, is placed above the articular processes to encourage dorsal fusion. The technique has since been modified by using screws rather than pins and attempting to remove the cartilage from the articular processes (Bagley 2003). Fracture, bending and loosening of screws have been observed, and concurrent dorsal laminectomy of the caudal aspect of L7 results in weakening of the articular processes, making them more prone to fracture (Moens and Runyon 2002, Sharp and Wheeler 2005, Hankin *et al.* 2012). For this reason some veterinary surgeons limit the extent of the dorsal laminectomy to the cranial aspect of S1 only (Kinsel *et al.* 2004) or do not perform a laminectomy in conjunction with fixation as originally described by Slocum and Devine (1980). A further modification is to place multiple pins or screws in L7 and S1 then bond them together with bone cement into a dorsal stabilisation, whilst the LS junction is positioned at a neutral LS angle (Beam *et al.* 2014). Other veterinarians have used specialised medical devices (Meheust 2000, Meij *et al.* 2007, Smolders *et al.* 2010) or the 'string of pearls' (SOP, Orthomed, Huddersfield, UK) locking plate for dorsal fixation/fusion (LG Carpenter[3], pers. comm.) by placing implants into the pedicle of L7. Pedicle screw fixation avoids loading the articular processes, thus allowing more aggressive laminectomy/dorso-medial foraminotomy, and decreased concern over weakening of the articular processes (Meheust 2000, Meij *et al.* 2007).

[3] LG Carpenter, Stryker Howmedica Osteonics. San Antonio TX, USA

Specialised medical devices are generally expensive and are not widely used in veterinary surgery for this reason. Recently, a novel spinal implant system has been described for LS fixation in dogs (Fitzpatrick 2014). The procedure is similar to posterior fixation procedures used in human neurosurgery where fixation screws are connected via polyaxial clamps to rigid rods (Fitzateur, Fitz-Bionics, UK). The Fitzateur implants are too large to be inserted into the pedicle of L7 therefore must be angled to engage the vertebral body, increasing the dissection and therefore morbidity of the procedure. The first instruction course for the Fitzateur was held at the 4th World Veterinary Orthopaedic Congress in Breckenridge Colorado 2014, attended by the author. The results of a small case-series of 34 dogs with DLSS treated with the Fitzateur device was presented by the inventor during the course. Good outcomes were documented but this study has not been published in a peer-reviewed journal at this time.

There are only limited studies reporting the outcome after surgical management of DLSS in working dogs (Danielsson and Sjöström 1999**,** Jones *et al.* 2002, Linn *et al*. 2003). At the MUVTH surgical management has been the preferred therapeutic option for working dogs when continued athletic function is required. The surgical technique performed varied with the advent of new techniques over time, and the decision as to which surgical procedure to perform was largely based on anecdotal information or experience extrapolated from the treatment of DLSS in pet animals. An assessment of the outcome following surgery of the cohort of working dogs operated at the MUVTH for DLSS has not been performed.

The aim of this retrospective study was to assess the outcome of surgical management of DLSS in a cohort of German shepherd working dogs treated by the author at the MUVTH. This observational data will provide a basis for future prospective evaluations of the management of DLSS in NZ Police dogs.

Materials and methods

A personal surgical case log (Fellowship of the Australian and New Zealand College of Veterinary Scientists credential) was used to identify cases of DLSS in working GSDs that had undergone surgical treatment by the author. In addition the medical records database of the MUVTH (RxWorks) was searched for Key words relating to spinal stenosis, spinal compression, and spinal instability of the lumbosacral region. The records were then cross-referenced against surgical theatre logs and medical records to determine those dogs that had been managed surgically with the author being the primary surgeon. Demographic data and clinical findings were tabulated from the medical records. Surgical logs and letters to referral veterinarians were referenced to document the type of surgery undertaken. Sixteen working GSDs that had undergone surgery at the MUVTH for DLSS between April 2002 and November 2013 were included in the study. Fourteen were Police dogs (NZ Police Dog Section) and two were Guide dogs (Guide Dog Services, NZ).

All dogs presented with a history of decreased ability to work, including reluctance to jump into vehicles or scale fences, decreased endurance, pelvic limb lameness or a handler‘s perception of pain in the dog during activity. In each case clinical signs were compatible with DLSS. All dogs underwent orthopaedic and neurological examination by the author, as far as the dogs’ temperament would allow. Aggressive dogs were sedated for further examination using 0.005 – 0.01 mg/kg medetomidine intra-muscularly. Concurrent coxo-femoral joint lesions were excluded by the failure to demonstrate pain on abduction of the coxo-femoral joints in the conscious dog or in a sedated (aggressive) dog that still reacted to other painful stimuli.
A dog was considered to have clinical signs localised to the lumbosacral area if there was one or more of the following:

- pain on deep palpation of the vertebrae and musculature dorsally over the LS junction
- pain or resentment with loss of range of motion in hip extension with a fulcrum placed at the LS junction
- pain on a standing or recumbent lordosis test (see chapter five)
- pain on forced tail extension

All dogs were sedated for survey radiographs of the lumbar vertebrae, pelvis, and LS junction, and sedated or anaesthetised for advanced imaging. Survey radiographs

were examined to rule out any dogs with discospondylitis, significant coxo-femoral osteoarthritis and obvious osseous neoplasia.

The first five dogs had magnetic resonance imaging performed at Mid Central MRI using a 1.5 Tesla magnet. Those dogs were positioned in sternal recumbency and no attempt was made to perform dynamic imaging. T1 and T2 sequences were performed without contrast enhancement. The remaining dogs were imaged using computed tomography at the MUVTH when a Phillips Brilliance helical CT unit became available (Philips Healthcare, The Netherlands). The imaging protocol was not initially standardised but evolved over time from experience with DLSS. The majority of dogs were imaged in dorsal recumbency using a foam trough as a support. Though not consistently performed at the start of the study, dogs were alternately strapped to the CT table with the pelvic limbs in neutral (perpendicular to the CT gantry), flexed (drawn forward beside the sternum), and extended (drawn backwards with the LS junction in lordosis) positions to demonstrate the range of motion (ROM) of the LS junction. A foam fulcrum was then placed beneath the LS junction for the extended views to increase extension of the LS junction. Soft tissue and bone algorithms were performed in each position and 3D renderings were created to study the L7-S1 lateral intervertebral neurovascular foramina at the extremes of ROM.

Criteria for a radiological diagnosis of DLSS included one or more of the following:

- significant annular protrusion of the L7-S1 *dorsal annulus*
- dorsal impingement of the vertebral canal by soft tissue associated with the *ligamentum flavum* or articular process joint capsule
- loss of fat signal within the intervertebral foramen
- osseous narrowing of the foramen due to dorsolateral extension of the cranial lamina of S1
- subjective dynamic narrowing of the foramen during extension
- narrowing of the foramen due to osteophyte development on the body or articular processes of L7 or S1

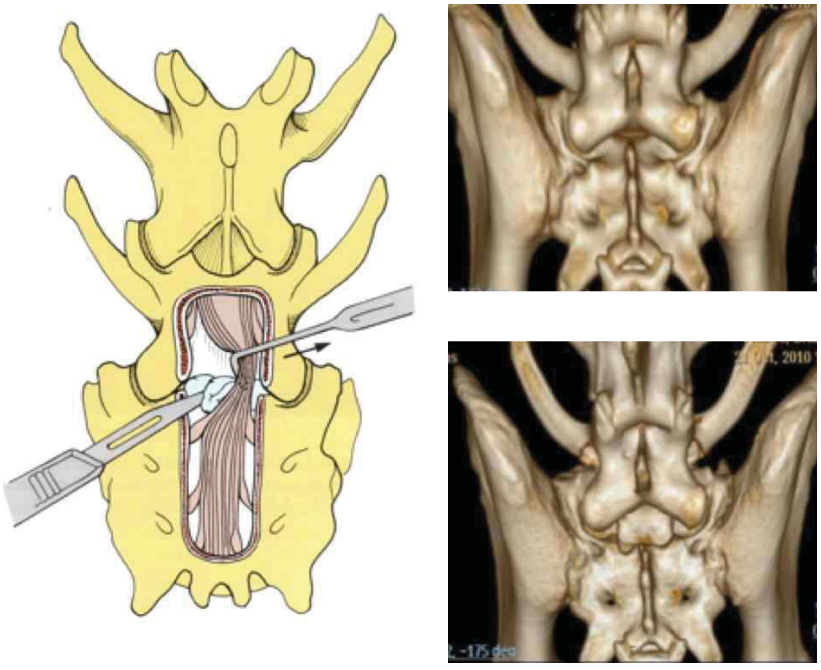
Surgical technique

Decision-making regarding the surgical technique to be undertaken in a dog was based on the presence or absence of:

1) ventral midline disc protrusion causing sacro-caudal nerve root compression
2) foraminal involvement consisting of lateral intervertebral disc protrusion, joint capsule or ligamentous hypertrophy, or dynamic alteration of the foraminal volume on extension of the LS junction
3) misalignment of the LS junction with ventral displacement of the sacrum

For surgery the dogs were positioned in sternal recumbency with the pelvis at one end of the surgical table. A rolled towel was placed under the pubis to elevate the pelvis. The pelvic limbs were pulled forward and held beside the flanks to provide maximum flexion of the LS junction. A standard dorsal midline approach to the lumbosacral space was performed in twelve dogs. In three dogs a paramedian approach was required to facilitate access for lateral foramenotomy (Godde and Steffen 2007), Table 14.

All dogs underwent a dorsal laminectomy with or without dorsal L7-S1 annulectomy (Chambers 1989, Meij and Bergknut 2010), (Table 14). In cases #1, #3 and #5, the dorsal laminectomy was extensive, removing the caudal half of the lamina of L7 and the cranial half of the sacral lamina as recommended by Meij and Bergknut (2010) (Figure 29a). In the remaining 13 cases the dorsal laminectomy was limited to a partial laminectomy of the sacrum, and the L7 lamina was not removed to preserve the base of the L7 articular processes (Kinzel *et al.* 2004), Figure 29b.



a) b)

Figure 29. a) Diagram showing a comparison of extent of dorsal laminectomy. On the left the diagram illustrates the extent of laminectomy as described by Meij and Bergknut 2010. On the right is a preoperative (top) and postoperative (bottom) 3D CT reconstruction of the LS junction in a dog that has undergone limited S1 dorsal laminectomy as described by Kinsel et al. 2004. Reproduced with permission from Meij, et al. (2007). Veterinary Surgery 36: 742-751.

An annulectomy was performed in seven cases (cases #1-6 and 8). A no.11 scalpel blade was used to cut a rectangular window in the dorsal *annulus fibrosus*. This window was cut in two phases, alternately holding the *cauda equina* to one side whilst directing the blade away from the nerve roots into the disc space. Sharp dissection was continued laterally to the level of the venous sinus in dogs with bulging lateral disc margins. A curette was used to remove the window of annulus and then loosen and extract *nucleus pulposus* (Chambers 1989). The decision to perform annulectomy was based on the presence of a large or asymmetric annular protrusion observed on

imaging and/or gross findings of a dorsally protruded disc annulus at surgery (asymmetric, palpably soft, not stretched out in the flexed position as employed for surgery).

Articular process fixation/fusion with bilateral screws (Slocum and Devine 1986, Bagley 2003) was performed in seven dogs after dorsal laminectomy with (2 cases) or without annulectomy (5 cases). During laminectomy cortico-cancellous bone chips from the S1 lamina were retained for possible use as a graft if that was required. With the dog positioned with the LS junction in maximal flexion, the joint capsules of the L7-S1 articular processes were removed medially and dorsally to expose the sacral articular surface. A small air powered burr was used to remove the articular cartilage from both the sacral and lumbar facet surfaces. The dog was then repositioned by bringing the pelvic limbs caudally to lie at approximately 90 degrees to the pelvis, in order to position the LS junction between a neutral and flexed position. The positioning of the LS junction was checked by reference to the location of the articular processes of L7 in reference to the sacral articular surfaces. A drill guide was placed on the dorso-medial aspect of the left L7 articular process and used to direct a 2.5 mm pilot drill hole through the L7 articular process into the S1 process and sacral wing. The angle of the drill required to avoid the drill bit entering the L7-S1 foramen necessitated removal of the tip of the L7 spinous process. The hole was measured and tapped for an appropriate length 3.5mm diameter stainless steel cortical screw (typically 40-45 mm length). The screw was placed as a position screw, not as a lag screw. The described process was repeated on the right side. The previously retained bone was placed as a graft within the joint and around the articular processes dorsally and laterally. Post-operative radiographs were taken to assess the position of the implant, Figure 30.

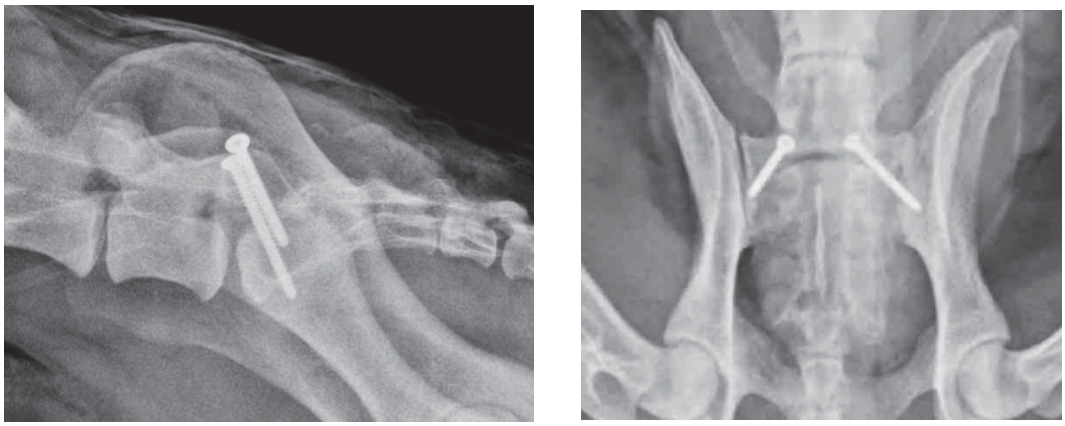


Figure 30. Post-operative radiographs (left lateral, right ventro-dorsal) of case #8 following bilateral articular process fixation of the lumbosacral junction with two 3.5mm screws.

Two dogs (cases # 7 and14) underwent dorsal laminectomy followed by dorsal fixation with “string of pearls” locking plates (SOP, Orthomed, UK) in a pedicle screw arrangement. In case #7 an extensive laminectomy was performed due to significant narrowing of the entrance zone of the foramina. A bilateral facetectomy *sic* was required to provide adequate decompression of the *L7* nerve roots, which precluded fixation of the articular processes with screws. Two 3-hole sections of 3.5mm SOP were used; one on each side of the vertebral column. The position of the L7 pedicle screw was determined by viewing the medial wall of the L7 pedicle from the laminectomy/facetectomy site. A drill guide directed a 2.5mm pilot drill hole through the lamina and into the pedicle, avoiding the medial wall of the pedicle. The SOP plate was then positioned using an appropriate length, self-tapping, 3.5mm screw. The need to contour the plate was assessed and then the plate was removed and contoured to fit. A second 3-hole section of SOP plate was contoured in a mirror image of the first. Each plate required both a ventral bow and a lateral twist to ensure appropriate contoured positioning on the screws in L7 and S1. The pedicle screw SOP construct was completed by reapplying the L7 screw then drilling and placing a screw in the sacrum with the use of the SOP drill guide. Post-operative radiographs were taken to assess the correct positioning of the implant, Figure 31.

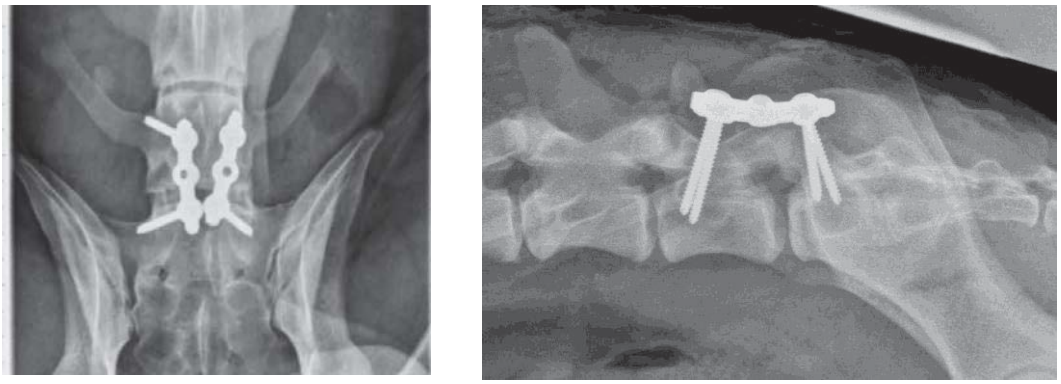
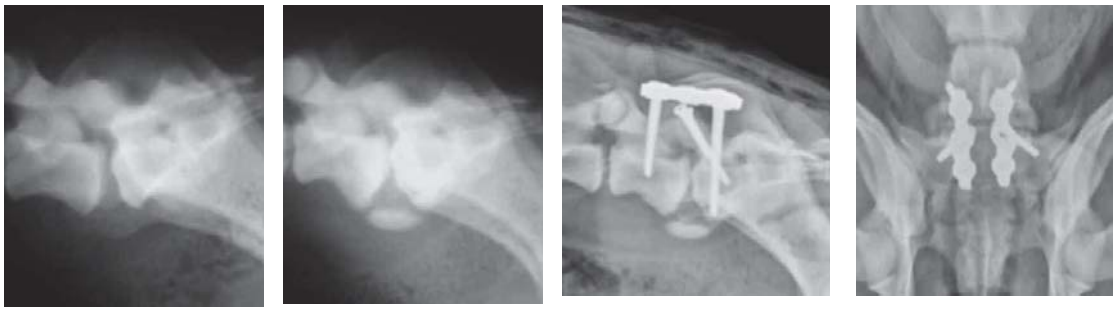


Figure 31. Post-operative radiographs (left ventro-dorsal, right lateral) of case #7 after bilateral pedicle screw fixation of a transitional lumbosacral junction with two 3.5mm SOP plates. This dog had undergone facetectomy to decompress the entrance zone to the lumbosacral lateral intervertebral neurovascular foramen on either side.

Case #14 was 38 months of age with mild clinical signs but significant radiological abnormalities. At 20 months of age, radiographs had been taken as part of a pre-breeding evaluation and had revealed a transitional LS junction (sacralised L7) without evidence of degenerative changes and no clinical signs were evident on physical examination (Figure 32 a). Within 18 months mild clinical signs had developed and

repeat radiographs revealed *spondylosis deformans* and disc wedging, Figure 32 b). CT evaluation showed dynamic narrowing of the lateral intervertebral neurovascular foramen with compression of the *L7* nerve root on extension of the LS junction. A combination of trans-articular screw fixation and dual SOP pedicle screw fixation was performed, Figure 32 c), d).

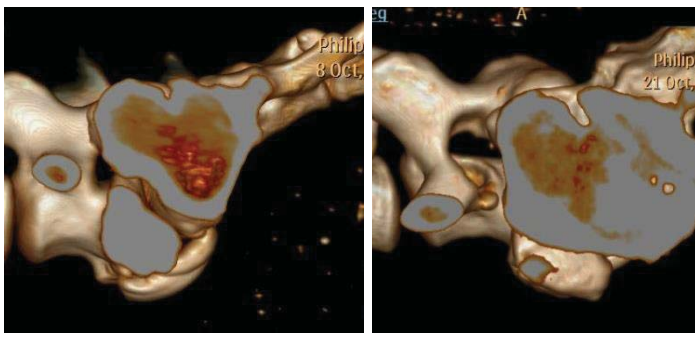


a) b) c) d)

Figure 32. Radiographs of case #14, a GSD diagnosed with degenerative lumbosacral stenosis. a) Radiographs at 20 months of age showed a transitional sacral anomaly but no degenerative changes. b) At 38 months of age evidence of degeneration was present and surgery was performed with combined trans-articular screw fixation and dual SOP plate pedicle screw fixation of the lumbosacral joint c) lateral radiograph, d) ventrodorsal radiograph.

Dog #11 initially underwent a standard trans-articular screw fixation but a screw fractured early in the recovery period, which was associated with intermittent lameness. The fixation was surgically revised with a single SOP plate as a pedicle screw fixation on the affected side.

Three dogs had a lateral foramenotomy (Godde and Steffen 2007) and dorsal laminectomy of S1. In each dog a separate dorsal laminectomy was performed first and then a paramedian approach was made to expose the dorsal and lateral aspect of the L7-S1 process joint and the foramen on either side. The foramenotomy was performed with a combination of pneumatic burrs and Kerrison-Smith rongeurs according to the technique demonstrated by Godde (Aesculapium, Tuttlingen, Germany March 2009), Figure 33, [see chapter nine for detailed description of surgical technique]. *Figure 33.*



Three-dimensional CT reconstructions of a dog, before (left) and after (right) lateral foramenotomy (Godde and Steffen 2007) has been performed. The grey/orange and yellow surfaces are the result of removing overlying bone by using ROI clipping tools.

Each dog was confined at the MUVTH for the first 3-5 days then returned to the handler. Discharge instructions were not standardised but included restriction from training and work for the first three months with only limited lead walking for exercise. The dogs were housed in runs and walked on a lead to defaecate and urinate. After three months an individual program of light training resumed. At 4-5 months dogs were cleared to return to full training according to a subjective determination of progress.

Outcome assessment

A survey was developed to gather data on pre-operative disability, clinical signs, post-operative disability and surgical outcome. The survey was based on that used in chapter one with appropriate additions (Appendix 3). The survey was trialled by a veterinarian who was familiar with service dogs acting as "a handler" (Vicki Erceg). Each PDS handler was then interviewed by telephone. An independent person conducted the interviews (Lauren Blume, final year BVSc student). The dog's handler was asked to recall the length of time from surgery the dog had been restricted in activity, the time taken to recover sufficiently for training to recommence, and the time taken until full activity was allowed. They were asked to score the dog for a range of clinical signs both prior-to, and after recovery from surgery and also rate the ability of the dog to return to work: able to perform full duties including bite work, tracking, scaling fences; or able to perform most duties but some allowances had to be made; or able to perform limited duties; or the dog required transferral to another role; or the dog was semi-retired but still kept in training and occasionally used. They were also asked to comment on any complications they observed, their impression of the outcome, and whether they would agree to surgery for an affected dog in the future. The outcome results were analysed according to the different methods of surgical intervention.

Results

Table 14 lists the demographic data and clinical signs of the study cohort. The dogs are listed in chronological order of the surgical intervention. There was one spayed bitch, the rest being entire male dogs. The median age of the dogs was 3.75 years with a range of 1.5 to 8 years. The mean weight of the dogs was 37.3 kg (range 29 – 44 kg). Table 15 lists the imaging modalities employed, the surgical procedure performed, the imaging interpretations, and findings at surgery. Table 16 lists the outcomes as determined from the survey. Fifteen dogs (94%) successfully returned to work. Twelve dogs (75%) were able to perform full duties, whereas two were able to perform most of the duties (12.5%) they were formerly capable of, and one could only perform limited duties and was semi-retired. Although dog #4 did return to full work following an

extended (12-month) rehabilitation period, it was retired 18 months later due to recurrence of clinical signs attributed to the LS junction. The handler was disappointed with the outcome and commented that in hindsight he would have preferred conservative management with epidural steroids and the expectation of keeping the dog in work, rather than the protracted stand-down period prior to only a limited period of further active duty.

One dog, case #8 did not return to active duty after surgery. This dog had undergone a limited S1 laminectomy and bilateral facet screw fixation. At three month follow-up there was mild pelvic limb lameness with ataxia and slight discomfort on palpation of the LS junction. Radiographs confirmed the implants were stable and in the correct position. Further confinement was recommended with a six-month delay before returning the dog to training. At six months the dog was incapable of performing the required duties and it was retired.

Median rating of the success of the surgery (Q11) was 9 (range 5 to 10), on a scale of 1, hopeless to 10, excellent. Fourteen of the sixteen handlers would agree to surgery on a working dog with DLSS if they had another dog with similar clinical signs (Q17).

Seven dogs were still working at the time of follow-up whereas nine dogs were euthanased (3) or had been retired (6) from working at latest follow-up (including #8). No dog was euthanased as the result of DLSS whilst still working. Of the six dogs that were retired, two dogs were retired due to progression of lumbosacral disease, two were retired for reasons unrelated to lumbosacral disease (blindness, loss of tracking/sharpness), and the remaining two dogs were retired due to old age. In one of the two dogs retired due to old age ongoing back pain and a limited ability to jump suggested that recurrence of DLSS might have been a contributing factor to these signs (table 16).

In the eight dogs that completed follow-up (returned to work but no longer in service [retired or died]) the median length of service after surgery was 2.6 years (range 1.5 - 4.75 years), the mean length of service after surgery was 2.98 years. The median time to return to training was 3 months (range 1 – 5 months). The median time to return to work was 4 months (range 2 – 10 months). The median time before the handler considered the dog was fully recovered was 6 months (range 2 – 12 months).

Table 14. Demographic data and clinical signs of 16 working German shepherd dogs with degenerative lumbosacral stenosis examined at the Massey University Veterinary Teaching Hospital between April 2002 and November 2013.

Name	Sex	Age[1] (yrs.)	Weight (kg)	Clinical signs at presentation
#1 (GDS)	M	5	44	Slightly slow placing reflex right pelvic limb, pain on right hip extension and pressure over right LS space dorsally. 1+ decreased endurance 1+ difficulty jumping into vehicle 3+ difficulty scaling walls, 3+ displaying pain, 3+ carrying leg after jumping
#2 (PDS)	M	3.5	38.5	Acute onset of pain associated with hyperextension leaping for a toy. Unable to work thereafter. 3+ difficulty jumping into vehicle, 3+ difficulty scaling walls, 1+ displaying pain, 1+ aggressive if handled, 3+ limp when walking/trotting
#3 (GDS)	FS	5	39.5	Pain on lordosis and extension of the left pelvic limb. 3+ difficulty jumping into vehicle, 1+ difficulty scaling walls, 3+ displaying pain, 2+ pain when handled 1+ limp when walking, 1+ carrying leg after jumping
#4 (PDS)	M	3	37	1/10 left pelvic limb lameness, pain on L hip extension and lordosis pain on left LS pressure. 2+ difficulty jumping into vehicles, 1+ difficulty scaling walls, 2+ displaying pain, 1+ pain when handled, 2+ lame when walking/trotting, 2+ carrying leg after jumping.
#5 (PDS)	M	1.5	39	2 weeks duration reluctance to jump into truck. Poor arm work, and reluctant to climb stairs. Ataxic RH gait, PL weakness, scuffing both PL nails. Bilateral PL muscle atrophy, proprioceptive deficits RH. Pain on L/S pressure. 1+ decreased endurance, 3+ difficulty scaling walls/jumping into vehicles, 2+ displaying pain/pain when handled
#6 (PDS)	M	5	40	Decreased work tolerance, low back pain. 2+ difficulty jumping into vehicle, 2+ difficulty scaling walls, 2+ displaying pain, ataxic pelvic limb gait
#7 (PDS)	M	5	35	2-3/10 lame, low and inactive tail carriage, scuffing left hind nails, pain in extension of L hip, pain on lordosis test and LS epaxial muscle pressure. 1+ decreased endurance, 1+ difficulty jumping into vehicle, 2+ difficulty scaling walls, 1+ lame when walking, limp tail
#8 (PDS)	M	5	31.5	Decreased work performance, abnormal tail carriage. Pain on LS pressure, pain on lordosis, 2+ poor tracking, 2+ decreased endurance, 3+ difficulty jump into vehicle, 3+ difficulty scaling walls, 2+ aggressive when handled, limp tail, scuffed nails
#9 (PDS)	M	8	39	Chronic PL stiffness and more plantigrade than normal, dribbling urine, LS pain, pain on lordosis, sciatic reflexes decreased. 3+ decreased endurance, 3+ difficulty jumping into vehicle/scaling walls, 2+ displaying pain, 1+ pain on handling, 3+ limp when walking, 3+ limp when running
#10 (PDS)	M	3.33	42	Intermittent RH lame after jumping. Pain on lordosis, hip extension, LS pressure and tail jack. Slow placing RH. 1+ decreased endurance, 1+ difficulty jumping into vehicle, 3+ difficulty scaling walls, 3+ displaying pain, 3+ carrying leg/limp after jumping.
#11 (PDS)	M	2.33	37	Pain on tail jack, mild resentment of lumbosacral pressure, resentment of lordosis and hip extension. 3+ difficulty jumping into vehicle, 3+ difficulty scaling walls, 3+ limp when walking/trot/running, 2+ lifting/carrying leg after jumping
# 12 (PDS)	M	4	38	Mild signs, no significant pain on LS pressure, resists lordosis and extension of the pelvic limbs
#13 (PDS)	M	2.25	40	Growls with any manipulation, tense and aggressive. Unable to confirm LS pain. Unable to work, 3+ difficulty jumping into vehicles, 3+ difficulty scaling walls, 3+ carrying leg/lame after jumping.
#14 (PDS)	M	3.2	29	2+ Difficulty, reluctance to jump into vehicles, 2+ difficulty scaling walls, 2+ lame all gaits, 2+ carrying the leg after jumping. Mild pain response to lordosis/hip extension.
#15 (PDS)	M	6.75	33.5	Pain on LS pressure, resents lordosis/hip extension. Lame LH, 3+ difficulty jumping, 3+ reluctance to jump into vehicles and scaling walls. 2+ carrying leg/lame after jumping.
#16 (PDS)	M	2	34	Pain on LS pressure, resents lordosis/hip extension. Decreased endurance 1+, difficulty scaling walls 1+. Displaying mild pain/when handled. 2+ limp when walking/trot/running, 2+ lifting/carrying leg after jumping

[1] Age recorded in clinical notes as age at surgery
PDS = Police Dog Section (NZ), GDS = Guide Dog Services (NZ), L = left, R = right, LS = lumbosacral, PL = pelvic limb

Table 15. Imaging modalities employed, surgical procedure performed and surgical findings in 16 working German shepherd dogs operated at the Massey University Veterinary Teaching Hospital between April 2002 and November 2013

Case #	Imaging	Imaging Findings	RFV in extn	LFV in extn	Surgical Procedure	Annul-ectomy	Complications
1	XR, MRI	DLSS			Dorsal laminectomy	Yes	None
2	XR, MRI	DLSS			Dorsal laminectomy	Yes	None
3	XR, MRI	Transitional vertebra, DLSS			Dorsal laminectomy, facet screw fixation	Yes	None
4	XR, MRI	DLSS, disc prolapse	108	98	Dorsal laminectomy, facet screw fixation	Yes	Recurrence of signs 9 months, screws broken, revised, partial resolution
5	XR, MRI	DLSS, disc prolapse			Dorsal laminectomy, right entrance (dorsal) foramenotomy	Yes	None
6	XR, MRI	DLSS, disc prolapse			Dorsal laminectomy, facet screw fixation	Yes	Infection, revised with PMMA beads ESF, complete resolution
7	XR, CT	Transitional vertebra, DLSS, disc prolapse	175	82	Dorsal laminectomy, bilateral facetectomy and dual SOP plate dorsal fixation	No	None
8	XR, CT	DLSS	61	36	Dorsal laminectomy, facet screw fixation	Yes	Recurrence of signs 3 months, screws intact, poor outcome
9	XR, CT	Transitional vertebrae, congenital LS stenosis, DLSS, fracture of lamina/facet base.	61	46	Dorsal laminectomy, bilateral lateral foraminotomies	No	None
10	XR, CT	Transitional vertebra, DLSS (son of #9)	103	107	Dorsal laminectomy, facet screw fixation	No	Recurrence of signs at 2 years, loose screw, 2 x revision surgeries
11	XR, CT	DLSS, synovial cyst	138	130	Dorsal laminectomy, facet screw fixation.	No	Recurrence of signs at 1 year, broken screw, revision with single SOP pedicle fixation
12	XR, CT	DLSS, disc prolapse	81	74	Dorsal laminectomy, facet screw fixation	No	Mild lameness at 2 months, screw breakage, not revised, improved. Cruciate disease.
13	XR, CT	Transitional vertebrae, DLSS	67	49	Dorsal laminectomy, dual SOP pedicle fixation	No	Seroma, resolved. Screw breakage 23 mths
14	XR, CT	DLSS, x-rays	44	37	Dorsal laminectomy, facet screw fixation, dual SOP pedicle fixation	No	Screw breakage on routine radiographs, not revised to date
15	XR, CT	DLSS, L-sided disc prolapse	121	73	Left lateral foraminotomy and S1 dorsal laminectomy	No	None
16	XR, CT	DLSS, sacral canal narrow			Dorsal laminectomy	No	None

XR = plain radiography, MRI = Magnetic Resonance Imaging, CT = Computed Tomography, RFV = right foraminal volume, LFV = left foraminal volume. DLSS = degenerative lumbosacral stenosis, LS = lumbosacral, SOP = string of pearls locking plate, S=sacral, ESF = external skeletal fixation, PMMA = polymethylmethacrylate

Table 16. Results of the survey of 16 working German shepherd dogs operated at the Massey University Veterinary Teaching Hospital between April 2002 and November 2013 for degenerative lumbosacral stenosis. Q 8 = "did the dog return to work". Q 11 "rate the success of surgery out of 10", Q 15 years remaining in service. Q 17 "Would you consider surgery for DLSS on another dog in the future?"

Case #	Q8	Time till the dog: (months)			Q10 Ability after return to work Q12 Clinical signs	Q 11*	Q 15	Reason left service / complications	Q17
		Resumed training	Resumed full work	Was fully recovered					
1	Yes	1	2	2	Able to perform full duty All former signs resolved	8.5	4.75	Developed urinary incontinence that was thought to be due to cauda equine syndrome but never confirmed. Work-up revealed metastatic haemangiosarcoma, euthanased	Yes
2	Yes	3	5	6	Able to perform full duty All former signs resolved	9	2.75	Developed seizures and progressive neurological signs, dx neoplasia became unable to work, euthanased.	Yes
3	Yes		3	3	Able to perform full duty All former signs resolved	10	3.5	Retired due to the development of blindness.	Yes
4	Yes			12	Able to perform full duty 2+ poor tracking	7.5	2.5	Retired due to poor work performance, loss of tracking, lacking sharpness. Only 1.5 years of service after initial 12month rehabilitation.	No
5	Yes	2	3	4	Able to perform full duty All former signs resolved	10	4	Retired at 11 years of age, no longer able to meet the physical demands of the job. Primary reason listed as retirement due to old age but was experiencing lower back pain.	Yes
6	Yes	4	6	6	Able to perform full duty 2+ difficulty jumping into vehicle, 2+ difficulty scaling walls	8	2.5	Euthanased after behavioural issues culminated in him badly biting his newly assigned handler.	Yes
7	Yes	3	4	12	Able to perform most duties, 1+ difficulty jumping, 1+ decreased endurance, 1+ displaying pain,	8.5	2.4	Retired at 8.5 years of age due to falling ability to cope with the demands of the job. Handler listed old age as the primary reason, but back pain and limited ability to jump suggest recurrence of DLSS contributed. Subject to euthanasia a year after retirement with lower back pain, urinary incontinence and inability to jump into vehicles.	Yes
8	No	6	not able		Retired, 2+ poor tracking, 2+ poor endurance, 1+ difficulty jumping/scaling walls,1+pain	5	0.5	Handler lost confidence in dog. Initially still kept in training and occasionally used in limited capacity. However 6 months post-op the dog was retired due to inability to continue to work.	No
9	Yes	5	6	8	Able to perform limited duties, 1+ difficulty jumping, 1+ difficulty scaling walls, 1+ lame at the walk	9	1.5	Used part-time, limited number of jobs, but same type of work. Later became an armed offender squad dog. In total 18 months back at work before retired due to inability to work related to lumbosacral disease. He was euthanased at 10 years of age after a period in retirement.	Yes
10	Yes	4	10	12	Able to perform full duty 1+ displaying pain, 1+ lame after jumping	10	SIS	At two years post-op developed mild intermittent exercise induced lameness, as prior to surgery. Revision surgery.	Yes
11	Yes	4	6	12	20-month follow-up after 2nd surgery. Able to perform full duty	8.5	SIS	Went lame again 9 months after surgery. Rested 3-months. Radiographs showed a bent facet screw. Revised with new screw and an SOP plate. Good result from second surgery returning to full duty.	Yes
12	Yes	3	4	5	10-month follow-up Able to perform most duties,	8	SIS	Developed right pelvic limb lameness one year following LS junction surgery. A cruciate ligament rupture and a pre-existing osteochondral lesion were diagnosed and treated surgically with dynamic stabilisation and cartilage replacement. The dog is currently back at work in a more limited role and a replacement is being trained.	Yes

Table 16 continued next page

Table 16 cont. Results of the survey of 16 working German shepherd dogs operated at the Massey University Veterinary Teaching Hospital between April 2002 and November 2013 for degenerative lumbosacral stenosis. Q 8 = "did the dog return to work". Q 11 "rate the success of surgery out of 10", Q 15 years remaining in service. Q 17 "Would you consider surgery for DLSS on another dog in the future?"

Case #	Q8	Time till the dog: (months)			Q10 Ability after return to work Q12 Clinical signs	Q 11*	Q 15	Reason left service / complications	Q17
		Resumed training	Resumed full work	Was fully recovered					
13	Yes	2	3	3	12-month follow-up Able to perform full duty	10	SIS	Post-operative seroma required repeated drainage. Moderate difficulty jumping into vehicles, mild difficulty scaling walls/fences. Mild lameness at 23 months, x-rays revealed screw failure.	Yes
14	Yes	3	4	4	15-month follow-up Able to perform full duty All former signs resolved	9.5	SIS	Six weeks after surgery routine follow-up radiographs showed implant failure despite the dog having been on restricted run rest. There were no clinical signs. Rehabilitation was delayed one month and Blaze returned to work successfully.	Yes
15	Yes	3	4	6	15-month follow-up Able to perform full duty All former signs resolved	10	SIS	Operational AOS and Patrol dog. Handler commented that should have had surgery earlier	Yes
16	Yes	3	3	3	12-month follow-up Able to perform full duty All former signs resolved	10	SIS		

* The survey asked for ratings 1 to 10, some respondents answered "8 or 9 etc." in which case the score was averaged hence the point five ratings. 1+ = mild, 2+ = moderate, 3+ = severe rating from the survey. SIS = still in service as at April 2014.

Results of dorsal decompressive surgery

All four of the dogs treated by dorsal laminectomy without stabilisation returned to full work without complications (2.75, 4, 4.75 years of further service, one ongoing). The first three cases had a concurrent annulectomy, the fourth and most recent did not.

Results of lateral decompressive surgery

Both of the dogs treated by lateral foraminotomy (and a limited dorsal laminectomy) returned to work and one was still on active duty as at April 2014. Case #15 was able to work without restriction but case #9 only returned to limited duties and was used in a limited capacity before being retired eighteen months after surgery. Six months after retirement, at the age of 10 years, case #9 was euthanased. Difficulty walking and ongoing back pain despite medication were cited as factors in the decision to euthanase the dog.

Results of LS junction stabilisation with trans-articular process screws

Of the seven dogs treated by trans-articular process screw fixation and limited S1 laminectomy, six returned to full work. At the time of follow-up three of these dogs were still in active duty. The other three dogs had between 2 ½ and 3 ½ years of further active duty as Police dogs after surgery to stabilise the LS junction. However, there were complications in five dogs that had screw fixation of the articular processes and further surgical intervention was required in four of them (Table 16). Complications included infection (1), breakage of a screw (3), and loosening and bending of a screw (1). Despite these complications all five dogs returned to work following management of the complications.

Results of LS junction stabilisation with SOP plate fixation

The first dog treated by facetectomy and bilateral SOP constructs returned to work without complications and could perform most of the duties it had been able to perform prior to surgery. The other two dogs with SOP constructs had breakage of screws but returned to work and were performing all expected duties at the time of the last follow-up (April 2014).

Individual complications

As stated in the results section Case #8 did not return to active duty after surgery (S1 laminectomy and bilateral facet screw fixation). At three month follow-up there was mild pelvic limb lameness with ataxia and slight discomfort on palpation of the LS junction. Radiographs confirmed the implants were stable and in the correct position. Further

confinement was recommended with a six-month delay before returning the dog to training. The dog was initially kept in training and used in a limited capacity, however, at six months the handler had lost confidence in the dog's ability and it was retired.

Case #4 returned to training but had episodes of apparent pain. The dog would vocalise and look around at its back. Radiography of the LS junction showed broken trans-articular screws. Nine months after surgery this dog was reoperated and a thick layer of scar tissue was present over the site of the previous dorsal laminectomy. This "laminectomy membrane" was carefully removed by enlarging the laminectomy and undermining its' attachment. The broken section of each screw within the L7 articular processes was removed. Case #4 did return to full work after a protracted recovery but his handler perceived him to have poorer work ability, possibly as the result of the time off work and loss of training. He was lacking in "sharpness" and had poorer tracking skills, problems that ultimately led to his retirement 2.5 years after surgery (1.5 years of further working life).

Case #6 developed a post-operative wound infection necessitating a second surgery to remove implants and re-stabilise the vertebrae with an external skeletal fixation device. The surgical site was packed with antibiotic-laden polymethylmethacrylate (PMMA) beads. The beads were aseptically prepared by mixing 1g of cefazolin in 20 g of PMMA. Despite the infection the dog returned to work for a total of 2 ½ years after the initial surgery.

Case #10 successfully returned to work but intermittent lameness recurred two years after the initial surgery. Dynamic CT imaging confirmed that fusion of the LS junction had not occurred as evidenced by continued motion of the LS junction angle in flexed and extended positions. Reassessment of the immediate post-operative radiographs suggested that the articular process screw on the lame side crossed the sacroiliac joint. At re-operation the articular process screw on the side of the lameness was found to be loose so it was removed, along with a substantial laminectomy membrane. No attempt was made to promote further fusion. Six weeks after revision surgery the dog was re-examined because there was no improvement in clinical signs. Mild pain was shown on extension of the LS junction. The right pelvic limb gait was abnormal with a foreshortened stride and a flick of the hock as the forward motion of the tibia was arrested during mid-stride. A tight band of tissue was palpable from the groin to the proximal tibia, a sign consistent with contracture of the semitendinosus muscle. Semitendinosus muscle contracture is thought to be a form of compartment syndrome, occurring after an acute muscle injury, and unrelated to DLSS. A lateral foraminotomy was performed to relieve the LS junction pain, though the significance of foraminal narrowing to the clinical presentation in light of the semitendinosus contracture was

debatable. The dog was able to return to active duty without indications of pain when worked, however he is currently on limited duties due to the restriction of movement from the semitendinosus contracture.

Case #11 presented with an intermittent right pelvic lameness 12 months post-operatively and radiographs revealed bending of the trans-articular LS screw on the right side. Revision surgery identified a laminectomy membrane and revealed that the right LS junction articular processes had not fused. The loose and bent screw was removed and a 3-hole SOP plate was inserted and fixed with single L7 and S1 pedicle screws. After the second surgery the dog was able to return to work. There was a mild occasional limp and some difficulty jumping into vehicles.

Case #12 was three years of age when presented for mild intermittent pelvic limb lameness and scuffing of the nails to another veterinarian. There was no pain on palpation, extension of the LS junction, or tail jack. Methyl prednisolone acetate (Veta-cortyl, Ethical Agents, South Auckland) 1 mg/kg, was administered epidurally for four doses at monthly intervals. Eighteen months after the first presentation, there was recurrence of the clinical signs prompting referral to the MUVTH. Repeat CT examination confirmed DLSS, and S1 laminectomy and trans-articular screw fixation was performed. Initial rehabilitation was satisfactory, however at two months there was recurrence of mild intermittent lameness. Radiographs revealed the right trans-articular screw had fractured. Re-operation was not performed, and the dog's rehabilitation was extended by two months. At five months post-operatively he was deemed fit to return to work. He was subsequently diagnosed with stifle lameness on the ipsilateral pelvic limb one year following LS surgery. Stifle osteoarthritis was confirmed on radiographs and arthrotomy revealed a partial cranial cruciate ligament rupture with injury to the medial meniscus and an osteochondral lesion of the articular surface of the lateral femoral condyle. Tibial tuberosity advancement was performed to stabilise the stifle and an autogenous osteochondral transfer procedure was performed following removal of the osteochondral lesion by a fellow surgeon at the MUVTH. The dog subsequently returned to work. Case #13 developed a large wound seroma. The dog was anaesthetised on two occasions to drain the seroma.

Case #14. At six-week follow-up, radiography revealed broken screws in both of the L7 articular processes and both of the sacral pedicle screws. No clinical signs were reported and no further surgery was undertaken. The handler was advised to delay the dogs' rehabilitation by one month. He returned to work without any clinical signs of lumbosacral disease. Five months after surgery he was the leading dog in his section in the Christchurch regional trials and then went on to be top dog in the South Island Police Dog trials.

Discussion

In a recent veterinary literature review of DLSS, the authors stated that there is a paucity of high-quality evidence for the effectiveness of reported treatments and, more importantly, there has been no organized comparison of outcomes between different treatments given to specific categories of DLSS dogs (Jeffery *et al.* 2014).

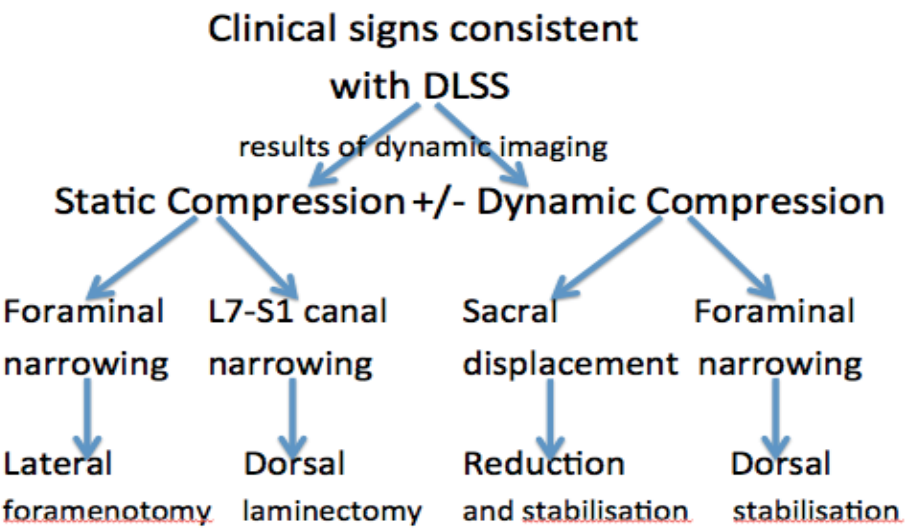
The two largest studies of the surgical management of DLSS are by Danielsson and Sjöström (1999) and Suwankong *et al.* (2008). Danielsson and Sjöström (1999) reported the results of 131 dogs with DLSS managed by dorsal laminectomy and annulectomy. Ninety-six dogs (73%) were rated as normal by their owners following surgery for DLSS, with an average follow-up period of 26 ± 17 months. An additional 20% were reported to be improved by the surgery. The authors considered that 57% (75 of 131 dogs) were active or working dogs (hunting, obedience-trained, Police, or security dogs), of which, 78% returned to previous activity/work following surgical treatment. When this data was combined with the results of several smaller case series in the veterinary literature, dorsal decompression and annulectomy yielded a mean total percentage cure of 55%, and improved the clinical signs in a further 25% of dogs of mixed breeds with DLSS (Janssens *et al.* 2000). More recently Suwankong *et al.* (2008), reported the outcome of 156 dogs with DLSS treated by dorsal laminectomy, with (50%) or without dorsal fenestration of the L7-S1 intervertebral disc. Eighty-three of 105 (79%) dogs showed improvement of the presenting clinical signs, however the outcome for working dogs was not assessed separately.

The data on the outcome of surgical management of DLSS in police or military working dogs is more limited and it has been suggested that the prognosis for surgical management of working dogs with DLSS may be poorer than for pet animals (Meij and Bergknut 2010). Jones *et al.* (2002) reported a successful outcome in 8 out of 12 military dogs affected by DLSS following dorsal decompressive surgery, of which 3 were GSDs. In a retrospective study, Linn *et al*. (2003) evaluated 29 military working dogs with DLSS, of which 18 were Belgian Malinois and 9 were GSDs. All dogs were treated by dorsal decompression via laminectomy with (n=18) or without (n=11) annulectomy, and eleven dogs had additional foramenotomy or facetectomy to free constricted L7 nerve roots. Outcome was categorised on the resolution of clinical signs and whether the dog could return to full or restricted duty, or if it failed to return to work (average follow-up interval 24 months). Twelve dogs (41%) returned to normal function, 11 (38%) improved, and 6 (20%) never returned to active duty. Eleven dogs (38%) were euthanased due to either ongoing clinical signs of DLSS after surgery (n=6) or recurrence of signs following an initial improvement after surgery (n=5) (Linn

et al. 2003). In the current study, 15/16 (94%) of working GSDs (Police and Guide) returned to work. The outcome after surgical management was good to excellent in the majority of cases (88%) and more favourable than results of surgery previously reported in working dogs.

The improvement in outcome in the present study over historical data may demonstrate the benefit of choosing a surgical intervention based on individual pathology. The author employed stabilisation of the LS junction and dorsal decompression with lateral foraminotomy in 12/16 cases and these procedures were not used in earlier studies of working dogs with DLSS. This result should be interpreted with caution as only 16 cases were analysed. Adding an additional dog with a poor outcome would reduce the percentage of good to excellent outcomes to 82%, which is still higher than the published success rate of surgical management of DLSS in working dogs. Over the ten years of this study the surgical procedures performed on the dogs were subject to evolution as the author's understanding of the variable presentation and signs of DLSS evolved through analysis of cases, and with evidence from dynamic CT imaging. The initial cases had MRI performed in neutral positioning only. Axial and sagittal images provided assessment of intervertebral disc protrusion and compression of the *cauda equina* as indicated by a loss of an epidural fat signal surrounding the exiting nerve roots. The initial cases were treated by decompressive dorsal laminectomy with good results. The subsequent acquisition of a CT scanner and use of a standardised CT protocol to position the dogs in flexed, neutral and extended LS positions allowed more exact and extensive evaluations. The dynamic images revealed the extent to which the L7-S1 lateral intervertebral foramen becomes narrowed during extension and led to a decision to treat these dogs by fixation of the articular processes of L7-S1 with screws to stabilise the joint. In addition, 3-dimensional volume rendering allowed subjective assessment of the size of the L7-S1 foramen, from both internal and external viewpoints. Dogs with degenerative changes at the exit zone causing static compression could be identified. In these dogs a dorso-lateral foraminotomy was thought to provide a better solution over fixation/stabilisation or dorsal decompression alone. The final case was treated successfully by dorsal decompression alone following a diagnosis of excessive overhang of the sacral lamina and dorsal narrowing of the L7-S1 canal with no foraminal involvement.

The author has since adopted the following algorithm for the surgical management of DLSS.



The only dog that did not return to work (#8) was treated by trans-articular stabilisation. Post-hoc volume analysis (using the method described in Chapter seven) revealed it had the smallest foraminal volumes of those measured in this cohort at 61mm^3 (right) and 36mm^3 (left) [Table 2]. The dog with the next smallest volumes (61 and 46 mm^3) was treated by bilateral dorsolateral foraminotomy with an excellent outcome and without complications. Given the degree of dynamic narrowing of the lateral intervertebral foramina, dog #8 may have benefited from the dorsolateral foraminotomy procedure rather than dorsal fixation/fusion.

The prognosis for DLSS is reported to decrease with age and presence of neurological deficits (DeRisio *et al.* 2001; Linn *et al.* 2003; Suwankong *et al.* 2008). Age at onset of clinical signs, or age at diagnosis/surgery for DLSS are significant in predicting outcome, with younger dogs tending to have better return to function after surgery (Linn et al. (2003). Due to the low numbers of cases in this study there is insufficient power to perform any appropriate analysis of age on outcome.

Six of 15 dogs with a poor outcome in one study had been incontinent, (faecal and/or urinary) for a median of eight weeks prior to surgery, and a significant correlation was found between the presence of urinary or faecal incontinence prior to surgery and a poorer outcome (DeRisio *et al*. 2001). When the duration of signs was considered, only urinary incontinence remained significantly related to a poorer outcome. In the current study, only one dog (case #9) had urinary incontinence referable to *cauda equina* compression prior to surgical intervention. The incontinence resolved and the dog had

a good outcome after bilateral lateral foraminotomy and dorsal laminectomy. The sacral nerve roots were likely decompressed by the dorsal laminectomy rather than from the LS foramenotomy.

Recurrence of LS pain and dysfunction following initially successful surgery has been noted in several studies. Danielsson and Sjöström (1999) reported recurrence of clinical signs resembling DLSS (as reported by the owner or diagnosed by clinical examination) in 18% of cases at a mean interval of 18 months post-operatively. Recurrence of signs of DLSS later in life was also reported in 37% of cases of DLSS treated using dorsal laminectomy and annulectomy by Janssens *et al*. (2000). The mean follow-up interval was 30 months. Similarly in a cohort of 36 dogs with DLSS treated by dorsal laminectomy +/- disc fenestration, 37% of dogs were reported to have pelvic limb lameness at 6-months post-op (Suwankong *et al.* 2006), and at a median follow-up of eighteen months 5/12 dogs treated for DLSS using dorsal decompression were no longer suitable for their actual or intended use prior to DLSS developing (Suwankong *et al.* 2007). These studies suggest that decompression alone is associated with a high rate of recurrence of clinical signs, a result that is likely to be concerning for working dog owners.

In the current study recurrence of back pain was noted at retirement (4 years post-op) in one of the three dogs treated by dorsal laminectomy alone. There were five recurrences in cases where a dorsal/fusion was performed. These recurrences appeared to be associated with implant failure (bending or fracture of screws) and resolved in four cases with (3) or without (1) surgical intervention to provide more stability. The overall recurrence rate of clinical signs was 43%, very similar to previous reports. Recurrence of clinical signs of *cauda equina* compression has been attributed to ongoing stenosis and the development of a laminectomy scar membrane (Danielsson and Sjostrom 1999; DeRisio *et al.* 2001) although the clinical significance of laminectomy membrane formation has been questioned by others (Janssens *et al*. 2000). Failure to relieve foraminal stenosis using only dorsal decompression, or ongoing instability and further disc space collapse with soft tissue hypertrophy, may contribute to recurrence of signs but neither has been properly investigated. In the current study broken implants were removed and additional stabilisation was attempted in combination with resection of the laminectomy membrane in four out of five of the dogs undergoing re-operation. Each dog improved but it remains unknown whether dorsal compression or instability were the primary cause of the recurrence of the clinical signs. In case #10, signs of LS junction pain recurred two years after trans-articular fixation after a bent and loose screw was removed without further stabilisation being attempted. Clinical signs remained after surgery therefore a lateral foraminotomy

was performed to relieve foraminal stenosis seen on CT. Six month follow-up indicated resolution of the clinical signs (principally no LS pain) and currently this dog is still working.

The indication for performing a dorsal annulectomy/disc fenestration is controversial. DeRisio *et al*. (2001) reported an excellent or good outcome in 54/69 (78%) dogs over a mean follow-up period of 38 ± 22 months. The procedures performed included dorsal laminectomy +/- annulectomy and/or foraminotomy. The decision to perform procedures in addition to dorsal laminectomy was based on findings of advanced imaging, and/or evaluation of the extent of annular prolapse during surgery. There was no significant difference in outcome between those dogs undergoing dorsal laminectomy alone and those that underwent an additional procedure. In the larger study by Suwankong *et al*. (2008) an equal percentage of dogs underwent dorsal laminectomy with or without partial discectomy. Dogs that underwent partial discectomy showed significantly less improvement than those that underwent dorsal laminectomy alone. Dorsal laminectomy and partial discectomy destabilises the LS junction in normal dogs (Early *et al.* 2013). Therefore it is likely that annulectomy or disc fenestration further destabilises the already degenerate LS junction in dogs with DLSS, and unless stabilisation of the LS junction is performed may induce greater collapse of the disc space. Disc space collapse could then lead to a further decrease in the L7-S1 lateral intervertebral neurovascular foraminal volume, potentiating compression of the *L7* nerve roots, and in turn may explain the high rate of recurrence reported. Despite concerns over continued annular protrusion, a dorsal annulectomy/discectomy may not be indicated in dogs undergoing fusion of the LS junction. Dorsal fixation/stabilisation may allow the hypertrophied soft tissues, including the dorsal disc annulus, to atrophy over time. The effect of annulectomy of the L7-S1 disc on the volume of the lateral intervertebral foramen has not been investigated and will be the subject of chapter eight of this thesis. In the current study annulectomy was performed early in the case series, in dogs undergoing extensive laminectomy without stabilisation. In the later cases that were stabilised or underwent a foraminotomy, annulectomy was performed only for markedly asymmetric protrusion of the L7-S1 annulus present on CT and confirmed at surgery.

Early attempts using dorsal stabilisation with trans-articular screws combined with standard L7-S2 dorsal laminectomy performed at the MUVTH for DLSS in pet dogs resulted in breakage of one or both L7 caudal articular processes. This result led to an initial reluctance to perform similar stabilisation procedures in working dogs because of a presumed risk of articular process fracture. After modification of the laminectomy to involve removal of only the S1 lamina (Kinzel *et a.* 2004), fracture of the articular

processes was eliminated. However, bending and breakage of screws was seen in this cohort of working dogs suggesting that the 3.5mm dual screw method is biomechanically weak in active working Police GSDs. The long-term results of dorsal fixation/fusion in a total of 56 dogs have been reported in four studies (Slocum and Devine 1986, Meheust *et al*. 2000, Hankin *et al.* 2012, Gorlini *et al.* 2014). Hankin *et al.* (2012) considered the outcome of trans-articular fixation of the LS to be good, with 85% of dogs reported by owners to be walking normally and free of lameness 6 months after surgery. Breakage of screws was present in 4 of 21 dogs evaluated radiologically at 6 weeks post-op and was not associated with any clinical signs. Loosening of screws in two dogs was associated with recurrence of LS pain at 14 and 17 months after surgery and both dogs required surgery to resolve the pain. Overall the rate of screw breakage or loosening was 28.5%. Gorlini *et al.* (2014) reported improvement in 76.5% of dogs, though 30% still had one or more episodes of stiffness, difficulty rising or lameness after physical activity. The successful outcomes included seven dogs described as herding or working, of which six returned to their previous level of activity. Breakage or "pull out" of screws was found in 5/17 (29%) of dogs, and two dogs required revision surgery. Notably, an assessment of the post-operative range of motion of the LS junction (flexion to extension) revealed that rigid LS fixation was not achieved and dogs had an average of 11 degrees of motion despite trans-articular screw fixation.

The high incidence of trans-articular screw failure led the author to develop and use the SOP technique on three dogs (two primary surgery and one revision surgery) in the current study. Despite what was perceived to be a very strong fixation, combining articular process screws and dual pedicle SOP plates, fixation failed within a few months in case #14. The optimal location points and angulation of the screws in order to avoid injuring the *cauda equina* and the utility of using 2 screws per vertebrae warrant further investigation. Biomechanical studies are needed to determine the relative strengths and resistance to fatigue and potential patterns of failure of SOP constructs in comparison to trans-articular screw fixation (Chapter ten).

The lateral foramenotomy procedure of Godde and Steffen (2007) was only performed in three dogs in this cohort therefore it cannot be determined if this procedure may represent a significant improvement in the management of DLSS in working dogs. In a retrospective study of 20 dogs with DLSS, reported by the pioneers of the technique, the outcome was good to excellent in 19 dogs at a mean follow-up of 15.2 months (Godde and Steffen 2007). The outcome was assessed with a combination of veterinary re-examination at 6 months post-operatively and a follow-up telephone discussion with the dog's owner (range 6-42 months). The improvement in outcome

over previous studies was attributed to the effect of either unilateral or bilateral lateral foramenotomy, which was performed on each dog as indicated by MRI findings. Osteophyte formation at the exit zone and proliferation of the articular process joint capsule were the most common findings at surgery. The utility of lateral foramenotomy has yet to be proven in a cohort of working dogs, and it remains to be seen if long-term narrowing of the L7-S1 lateral intervertebral neurovascular foramen will recur as a result of reformation of bone or from scar tissue after foramenotomy. Bone modelling around the intervertebral foramina has been observed in an experimental study of entrance- and middle-zone foramenotomy in normal dogs (Wood *et al.* 2004). Using CT, there was evidence of regrowth of bone in the entrance zone, reducing the foramen back to its pre-surgical size at 12 weeks. A prospective evaluation of modelling of the L7 foramen after foramenotomy is required before lateral foramenotomy can be recommended as a curative surgical procedure for working dogs with DLSS. However, the initial experience of lateral foramenotomy reported here in 3 dogs is favourable as all returned to work.

Recently there has been a call for studies of DLSS to be multi-centre with standardised inclusion criteria, randomisation to treatment groups and blinded evaluators using outcome measures (Jeffery *et al.* 2014). Limitations of this current study in NZPD GSDs include its retrospective nature and the small number of dogs per treatment group. Rather than random allocation to group, the dogs were treated based the author's evolving understanding of the factors likely to cause clinical signs in dogs with DLSS, introducing significant bias. The small number of dogs in each treatment group precludes a meaningful statistical evaluation. A comparison between this study and those previously reported is also problematic due to lack of standardisation of inclusion criteria across the studies and the subjective nature of most outcome assessment measures. Whilst this study does not provide high-level evidence on which to make treatment decisions for DLSS, it is at least evidence of the outcome following surgical intervention in a cohort of working dogs, something not well reported to date.

In summary the results of this analysis suggest that the outcome of surgical intervention for DLSS in NZ Police and Guide GSDs at the MUVTH is better than the results previously reported in working dogs. Dynamic CT imaging to decide the most appropriate surgical procedure may explain the improvement over previous reports in working dogs.

Dynamic alteration of the lumbosacral intervertebral neurovascular foramen as determined by volumetric analysis using computed tomography.

Introduction

The syndrome, degenerative lumbosacral stenosis (DLSS), was described in chapter five as an acquired narrowing of the vertebral canal, lateral intervertebral foramina, or both at the lumbosacral (LS) junction, which results in compressive radiculopathy of one or more nerve roots of the *cauda equina* (Chambers 1989, DeRisio *et al*. 2001). Components of DLSS, which are thought to contribute to compression of the *cauda equina* include, prolapse of the *annulus fibrosus*, hypertrophy of the interarcuate ligament, hypertrophy of the capsule of the articular processes, and dynamic narrowing or osteophytosis of the lateral intervertebral neurovascular foramen. The *L7* nerve roots, having left the dura at the level of the L6 vertebra, pass through the lateral recess within the vertebral canal of L7 (Axlund and Hudson 2003). They exit the lateral intervertebral neurovascular foramen cranial and lateral to the L7-S1 disc. The lateral intervertebral neurovascular foramen is formed by the pedicle of L7 cranially, the articular processes of L7 and S1 dorsally, the arch of the sacrum caudally, and the body of L7 and the *annulus fibrosus* of the L7-S1 intervertebral disc ventrally. The lateral intervertebral neurovascular foramen is not an aperture, but resembles a canal or tunnel with an entrance, middle, and exit zones. Dynamic narrowing of the lateral intervertebral neurovascular foramen is thought to occur during extension and potentially may be exacerbated by disc degeneration (Jones *et al.* 2008).

The LS junction is the most mobile of all the intervertebral articulations in healthy dogs, however, compared to other species the LS junction contributes proportionately less to overall vertebral mobility. In large herbivores the lumbar vertebral column is relatively inflexible and primarily acts as a supporting strut for the heavy abdominal compartment (Gal 1993). Therefore, in herbivores the LS junction is proportionately more mobile in comparison to the remaining vertebral segments than in carnivores. In carnivores the increased flexibility of the lumbar vertebral column allows for an increased stride length, which facilitates a faster gait when chasing prey (Gal 1993). The three-dimensional motion pattern of the caudal lumbar and lumbosacral portions of the vertebral column of dogs has been reported in two studies (Benninger *et al.* 2004, 2006). The normal LS junction permits bending (flexion/extension) in the median plane, lateral bending in the dorsal plane, and very limited rotation in the transverse plane.

The range of motion of the LS junction in the median plane was 32.8 ± 6.4° in 15 skeletally mature medium to large breed dogs (Early *et al.* 2013). There is evidence that as DLSS develops, motion of the LS junction is decreased by the changes that occur in the intervertebral disc and supporting structures (Jones *et al.* 2008). A decrease in the range of motion of the LS junction is in contrast to the concept that instability of the LS junction is a feature of DLSS. However, some dogs with DLSS do have ventral displacement of the sacrum relative to the L7 vertebral body (retrolisthesis or a "step-lesion"), a feature that is proposed as evidence of instability of the LS junction (Slocum and Devine 1986, Palmer and Chambers 1991a).

The angulation of the LS junction during activity alters the pathway of the nerves of the *cauda equina* and also affects the dimensions of the lateral intervertebral neurovascular foramina. The cross-sectional area of an intervertebral neurovascular foramen can be estimated *in-vivo* using computed tomography (CT). Using sagittal reconstructions of CT data, a region of interest (ROI) can be drawn around the bone margins of an intervertebral foramen and the area within that boundary calculated. A significant decrease in the mean radiographic foraminal area with the LS junction in an extended *vs.* flexed position was detected in dogs in sagittal CT images (Jones *et al.* 2008). Extension of the LS junction results in the cranial articular processes of the sacrum tipping rostrally into the lateral intervertebral neurovascular foramen. Whilst a significant linear relationship was found between a change in the foraminal area and the angle of the LS junction of dogs with hind limb lameness and lumbosacral pain, this relationship was not significant for dogs without these clinical signs (Jones *et al.* 2008). Those authors' hypothesised that one of the normal functions of supportive structures of the LS junction is to maintain the dimensions of the L7-S1 lateral intervertebral neurovascular foramen independent of the changes in LS angulation during flexion and extension. Counter-intuitively, the percentage change in the area of the L7-S1 lateral intervertebral neurovascular foramina from flexion to extension did not differ significantly between dogs with or without clinical signs attributed to degeneration of the LS junction. In the study by Jones *et al.* (2008) measurements of the foraminal area were derived from sagittal plane images, which were not orientated perpendicular to the path of the *L7* nerve roots. Sagittal orientation potentially over-estimated the true cross-sectional area of the foramen at its narrowest point. A double-oblique, parasagittal method of determining the foraminal area perpendicular to the path of the *L7* nerve roots has been reported by Higgins *et al.* (2011). However, this study method was determined to be unreliable due to inter-observer variability and the effects of positioning of the dog for CT slice orientation.

An alternative approach to quantifying the minimal cross-sectional area of a three-dimensional tunnel is to calculate its volume. The three dimensional volume of the L7-S1 lateral intervertebral neurovascular foramen has not been evaluated in dogs with or without DLSS. Measuring the volume of the L7-S1 lateral intervertebral neurovascular foramen might be a valuable clinical and research tool for testing the effects of motion on the LS junction. Establishing values for healthy dogs and those affected with DLSS may be valuable for diagnosis, help determine if surgical intervention is warranted, and help to determine which procedure should be recommended. With the advent of new therapies, many of which differ radically in concept (dorsal stabilisation versus lateral foraminotomy), it is vital that veterinarians have full understanding of the function of the LS junction in both health and disease.

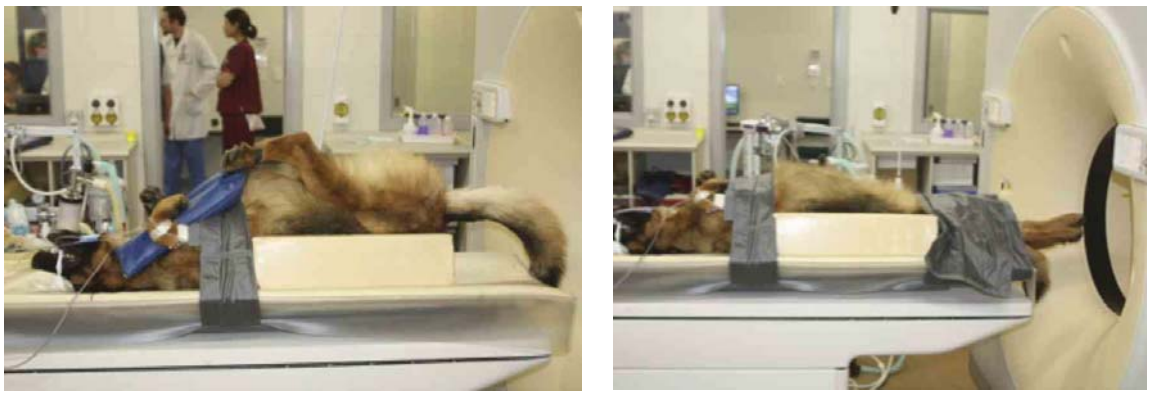
The aims of this study were to develop a method using CT images to determine the volume of the L7-S1 lateral intervertebral foramen and to quantify the effect of positioning of the LS junction on the volume of the foramen. Positioning the dog within the CT gantry with the LS junction in maximal extension, neutral positioning and full flexion was used to mimic the range of motion of the LS junction occurring during activity. The hypotheses were that extreme extension would significantly decrease the volume of the L7-S1 lateral intervertebral neurovascular foramen in both healthy dogs and dogs with DLSS, and that dogs' with DLSS would have a greater decrease in foraminal volume than unaffected dogs. In addition it was hypothesized that the Greyhound, a breed highly selected for athletic performance and less susceptible to DLSS, would have a lower ratio of L7-S1 lateral intervertebral neurovascular foraminal narrowing than the GSD.

Materials and methods

Three groups of dogs were compared. One group consisted of NZ PDS GSDs with clinical signs referable to the LS junction examined at the MUVTH between May 2009 and April 2013. All were confirmed to have DLSS (Abnormal GSDs = AbNGSD). Physical and neurological examinations localised the clinical signs to the LS region, and screening radiographs and CT scan findings were consistent with DLSS. The second group consisted of NZ PDS GSDs presented to the MUVTH in the same period for signs unrelated to lumbosacral disease (Normal GSDs = NGSDs). There were no historical indications of LS pain or dysfunction and clinical evaluations failed to confirm neurological or orthopaedic abnormalities. While sedated for diagnostic investigation of the primary complaint, each dog underwent LS imaging according to the study protocol. The third group consisted of healthy racing Greyhounds undergoing CT

scanning of the hock joints for an unrelated study on bone density of the central tarsal bone (NGH). An orthopaedic and neurological evaluation was performed and any greyhound with a hind limb gait abnormality or apparent pain on examination of the LS region was excluded. The imaging studies were identical for all groups. Each dog was deeply sedated with 0.005-0.01 mg/kg medetomidine and 0.1-0.2 mg/kg butorphanol administered intravenously (IV) as a single injection. A second dose of medetomidine was given IV to any dog that was insufficiently sedated. Computed tomography was performed using a Phillips Brilliance 16-slice helical scanner (Philips Healthcare, The Netherlands) with the dogs in dorsal recumbency supported by a high-density foam trough. Each dog was alternately strapped to the CT table with the LS junction in neutral, flexed, and extended LS positions. For the neutral LS position the femurs were allowed to rest at 90 degrees to the table, with the hocks supported on a foam block and the stifles taped around a block of foam at the width of the pelvis. For the flexed position the hind limbs were drawn forward and restrained beside the thorax using heavy sandbags. Flexion of the LS junction was maximised by ensuring the dog's hind-quarters were lifted off the foam trough (Figure 34A). For the extended views a foam block was placed between the stifles, which were secured with tape at the same width as the pelvis. The caudal edge of the foam trough was positioned directly beneath the LS junction as a fulcrum and the hind limbs were then extended caudally and secured in maximal extension of the coxofemoral joints using velcro straps which were secured to the CT table, (Figure 34B).

Figure 34. Positioning of the dog on the CT gantry for imaging of the lumbosacral junction.
A. Flexed CT positioning *B. Extended CT positioning*



Careful attention to detail was followed to ensure consistency of positioning at the limits of passive range of motion in flexion and extension of the LS junction. The acquisition window included the mid-body of L5 to the first caudal vertebrae and was positioned so that axial sectioning was parallel to the caudal endplate of L7. Axial images were acquired (120kV, 600MAS) with a 1 mm slice thickness and a 0.5 mm interval in both

bone and soft tissue algorithms (bone window 500 -1500 Hounsfield units, soft tissue window 40 to 400 Hounsfield units), using a matrix filter (768).

The CT images were interpreted by the author and a specialist veterinary radiologist. The extent of dorsal annular prolapse was assessed on sagittal and dorsal plane images. The extent of lateral disc annulus protrusion into the entrance zone was assessed on axial soft tissue sequences. The images were windowed and levelled to allow the *L7* nerve roots to be seen exiting the L7-S1 lateral intervertebral neurovascular foramen. A subjectively narrowed bony foramen, loss of peri-neural fat, and an increase in the size of the nerve roots were considered abnormal findings. The soft tissue window data were used to create 3-dimensional images of the LS junction including the lateral intervertebral neurovascular foramina. Clipping tools were used to remove the overlying ilial wings allowing visual assessment of the exit zones of the foramina. An inclusion ROI tool was used to divide the 3-dimensional reconstruction in half along the sagittal plane allowing subjective assessment of the entrance zones to the foramina. The 3-dimensional construct was rotated into a plane tangential to the expected path of the *L7* nerve roots. The foraminal size was subjectively compared between flexed, neutral and extended positions.

Dogs were classified as “affected” when the physical examination findings were consistent with the results of advanced diagnostic imaging (loss of perineural fat signal, annular protrusion, subjective foraminal narrowing). Based on these criteria, all dogs in the AbNGSD group, and no dogs in the NGSD or NGH groups, met the criteria for a diagnosis of DLSS to be made.

In order to quantify the volume of the L7-S1 lateral intervertebral neurovascular foramen the CT data were then manipulated on an Extended Brilliance Workstation (EBW, Phillips, the Netherlands). To isolate the foramina from the bulk data, a batch file was created from contiguous slices of the soft tissue windowing data covering the foraminal entrance, middle and exit zones. The batch file was converted into a 3-dimensional volume rendering, then a tissue segmentation protocol was applied with thresholds set to include the soft tissue and fat structures within the foramen but excluding bone and dorsal *annulus fibrosus* (centre -20, width 275 Hounsfield units), (Figure 35, page 126). The region of interest was defined laterally as the most abaxial extent of the pedicle of L7 just cranial to the lateral intervertebral neurovascular foramen. The cranial aspect of the foramen was identified and a 5 mm reference line was drawn from the most lateral extent of the pedicle across the width of the pedicle to the vertebral canal. A rectangular region of interest was created using an inclusion tool. This procedure defined a 3-dimensional foraminal volume with its cranial, caudal, dorsal and ventral limits determined by each dog’s anatomy (pedicles, sacral body,

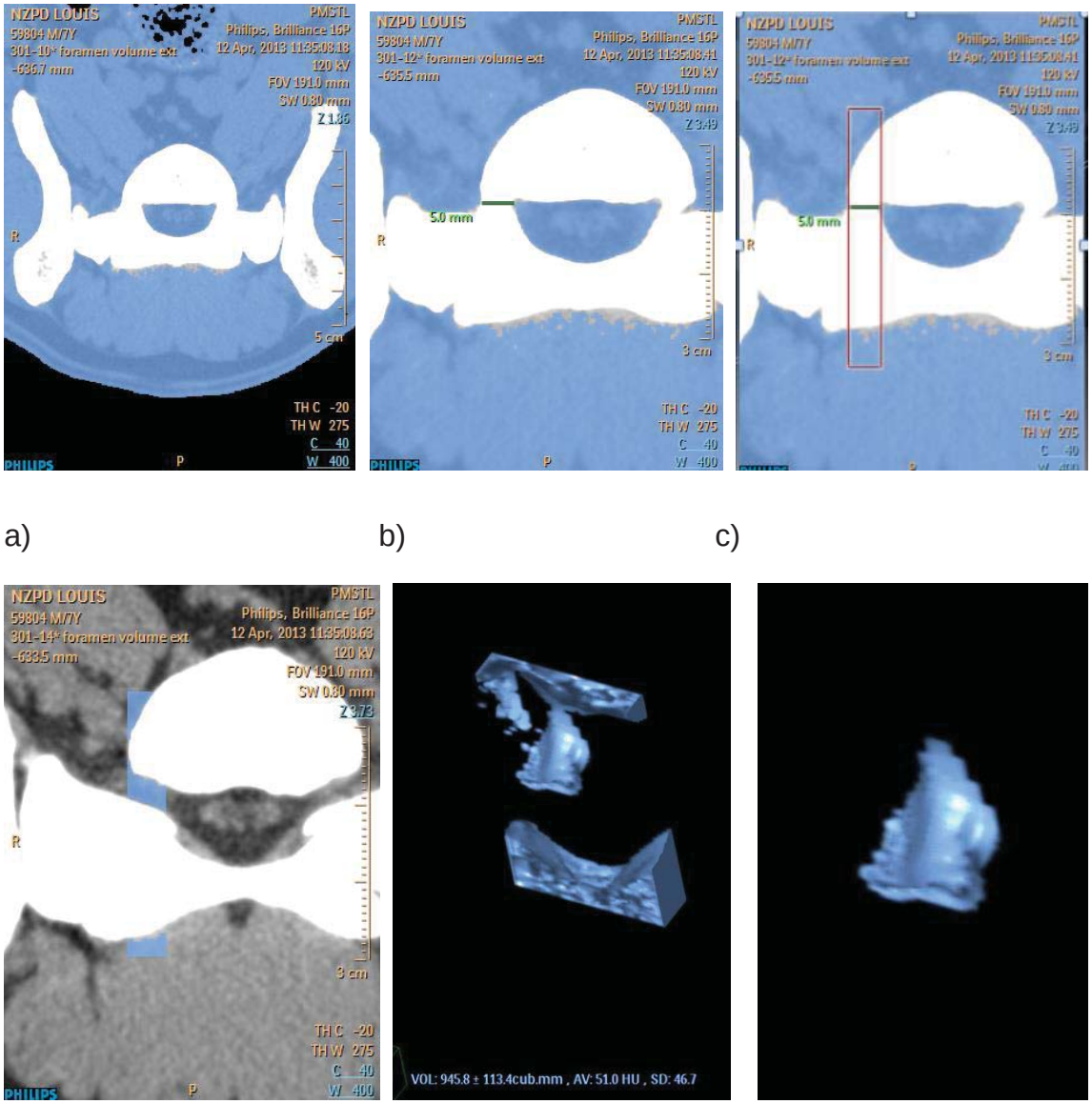
articular processes of L7 and S1 and vertebral body of L7 respectively), and the medial and lateral limits determined by the 5 mm reference line. The image of the foraminal volume was then magnified and manipulated to determine the extent of extraneous volume data outside the anatomical limits of the foramen. The volume was trimmed of any excess from beyond the foramen by visually checking sequential axial images and using a freehand exclusion tool.

The volume generated was expressed in mm^3 +/- a margin of measurement error. Each foramen was measured five times in each of the three positions creating 30 measurements per dog. If an outlier was noted it was dropped from the dataset and a sixth assessment performed. The five repeated volume measurements were then averaged.

In addition to the volume measurements, the longitudinal angulation of the LS junction was measured (LS junction angle). Using a sagittal reconstruction of the bone windowed sequence, a line was placed across the dorsal vertebral body of L7 and the dorsal sacral body representing the ventral floor of the vertebral canal of L7 and the sacrum respectively (Mattoon and Koblik 1993). Where the two lines intersected an angle was formed which was measured using digital imaging software (E-film). The length of the L6 and L7 vertebral bodies were measured between the end-plates of the respective vertebra at the mid-body height on mid-sagittal sections.

Statistical evaluation

All statistical analyses were performed using R (R Foundation for Statistical Computing, Vienna, Austria, 2013). The degree of intra-observer repeatability of the five repeated measurements for each volume was determined by calculating an interclass correlation coefficient. The age, weight, L6 and L7 length distributions of the groups were compared for significant differences with a linear model. The lateral intervertebral neurovascular foraminal volumes measured in neutral positioning for the NGH and NGSD dogs were pooled. The vertebral lengths of L6 and L7 were plotted against the respective dogs' foraminal volume in neutral positioning and a linear regression model applied to determine the relationship between animal size and foraminal volume. There was no statistical difference between L and R side, therefore 'side' was omitted from the model. A simple linear regression was used to compare the mean volume of the lateral intervertebral foramina in each position (combined left and right data) using the NGH volume data as the reference value. The linear regression was repeated with the NGSD volume data as the reference value. Statistical significance was set at $P<0.05$.



a) b) c)

d) e) f)

Figure 35. Axial CT images of the lumbosacral junction in a GSD, positioned at the pedicle just cranial to the lateral L7-S1 intervertebral neurovascular foramen. A tissue segmentation protocol has been applied with thresholding to highlight the soft tissue structures in blue (centre -20, width 275 Hounsfield units), a). A 5 mm width marker was placed on the pedicle using the lateral margin as the starting point b). A rectangular region of interest was created using an inclusion tool, which outlined the entire foramen c). The inclusion tool highlighted all the soft tissue within the lateral intervertebral foramen in contiguous slices, an example is shown in d). The contiguous slices where converted to a volume data file, which could be displayed in 3-dimensions and rotated and magnified e). Any volume from outside the actual foramen was removed with clipping tools leaving only the volume within the foramen itself f).

Results

There were 12 male GSDs with DLSS in the AbNGSD group.

There were 12 GSDs (8 male, 4 female) unaffected by DLSS in the NGSD group.

There were 10 Greyhounds (5 male, 5 female) unaffected by DLSS in the NGH group.

The age and weight comparisons for each group are shown in Table 17.

Table 17. Age, weight and vertebral length comparisons for three groups of dogs used to study the effect of lumbosacral positioning on the three-dimensional volume of the lateral L7-S1 intervertebral neurovascular as measured by CT. AbNGSD = German shepherds diagnosed with degenerative lumbosacral stenosis on the basis of clinical signs. NGSD = German shepherds with no clinical evidence of lumbosacral pain or dysfunction. NGH = healthy Greyhounds.

	AbNGSD	NGSD	NGH
Age - mean [SD] / median (years)	4.1 [2.0] / 4	4.35 [2.6] / 4	3.6 [0.9] / 3.5
Age – range (years)	1 – 7.5	1 – 8.75	3 – 5.9
Weight - mean [SD] / median (kg)	36.8 [4.8] / 35.3	33.0 [4.1] / 33.0	31.2 [2.2] / 31.0
Weight - range (kg)	31- 47	26 – 38	28 - 34.5
L6 length – mean/range (mm)	32.5 [31.0-35.1]	32.4 [30.7-35.5]	33.3 [32-34]
L7 length – mean/range (mm)	28.6 [26.2-30.7]	27.9 [27.0-29.7]	28.2 [27-30]

There was no significant difference in the age distribution between groups.

For weight, the difference between the AbNGSD and NGSD groups approached significance (P=0.058). There was a significant difference between the heavier AbNGSD and lighter NGH groups (P=0.002). The two heaviest dogs, 45 and 47kg, were both in the AbGSD group and represented outliers that somewhat skewed the distribution of this group. There was no significant difference in weight between the NGH and NGSD groups. Figure 36 (page 128) is a scatter plot of vertebral length versus foraminal volume. Vertebral length was not significantly different between any of the groups.

The interclass correlation coefficient was 0.89, 0.8 and 0.66 for the extended, neutral and flexed LS positions indicating good repeatability of the method in the extended and neutral positions of the LS junction, but not the flexed.

A linear relationship between L6 length and foraminal volume was not found (Adjusted R-squared: -0.05, P=0.84). A significant quadratic relationship between L7 length and the volume of the lateral intervertebral neurovascular foramen was found (Adjusted R-squared: 0.167, P=0.034).

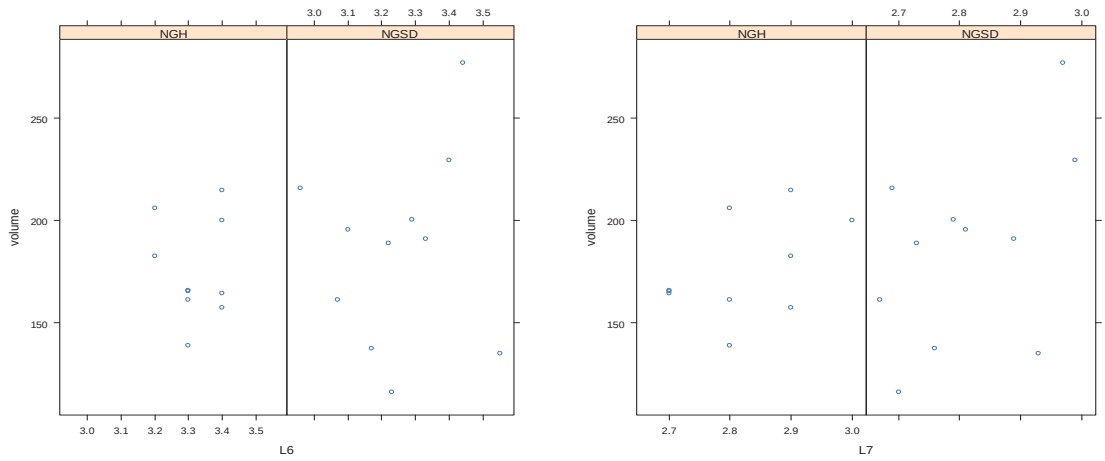


Figure 36. Scatter plot of the relationship between the length of the L6 (left) and L7 (right) vertebral bodies (cms) and the volume of the lateral L7-S1 lateral intervertebral foramen.

Although there was a significant relationship between L7 length and volume, since there was no significant difference in L7 length between groups, the absolute foraminal volumes were used in the further analysis without adjustment for L7 length.

The L7-S1 lateral intervertebral foramina were narrowest in extension, increasing in volume modestly in the neutral position and markedly in the flexed position. The individual data for left and right foramina by group are presented in Table 18. The pooled data (left and right foraminal data combined) is presented in Table 19 and the distribution patterns are shown in Figure 36.

Table 18. Determination and analysis of the L7-S1 lateral intervertebral neurovascular foramen volume in dogs using computed tomography, 3-dimensional reconstruction and an inclusion window region of interest. The volume data is in millimetres cubed (mm^3). AbNGSD = German shepherds with confirmed degenerative lumbosacral stenosis (N=12). NGSD = German shepherds thought to be unaffected by degenerative lumbosacral stenosis (N=12). NGH = Greyhounds thought to be unaffected by degenerative lumbosacral stenosis (N=10). Each value is the mean of the measurements for all the dogs in each group, each dog having been measured five times for each foramen.

		Mean Volume (mm^3)		**Standard deviation**		**Median Volume (mm^3)**	
Group	**Position**	**L**	**R**	**L**	**R**	**L**	**R**
NGH	Extended	127	144	24	23	128	142
NGSD	Extended	121	140	42	52	123	142
AbNGSD	Extended	84	87	32	36	75	82
NGH	Neutral	170	182	30	23	165	178
NGSD	Neutral	176	197	46	52	178	195
AbNGSD	Neutral	130	118	51	50	111	121
NGH	Flexed	512	525	90	74	501	524
NGSD	Flexed	644	600	87	76	646	602
AbNGSD	Flexed	589	618	110	132	566	621

Table 19. Volumetric analysis of the pooled (left and right) lateral intervertebral neurovascular foramina in dogs using computed tomography, 3-dimensional reconstruction and an inclusion window region of interest. AbNGSD = German shepherds with confirmed degenerative lumbosacral stenosis (N=12). NGSD = German shepherds thought to be unaffected by degenerative lumbosacral stenosis (N=12). NGH = Greyhounds thought to be unaffected by degenerative lumbosacral stenosis (N=10).

Group	Position	N=	Mean Volume (mm^3)	Standard deviation	Median Volume (mm^3)
NGH	Extended	10	135	25	135
NGSD	Extended	12	131	48	128
AbNGSD	Extended	12	85	34	77
NGH	Neutral	10	176	27	171
NGSD	Neutral	12	186	50	186
AbNGSD	Neutral	12	124	51	114
NGH	Flexed	10	519	82	517
NGSD	Flexed	12	622	84	622
AbNGSD	Flexed	12	604	122	607

Greyhounds experienced a 74% reduction in mean foraminal volume between flexion and extension, for GSDs unaffected by DLSS the reduction was 79% and for GSDs with DLSS the reduction in mean foraminal volume was 85%. When comparing neutral to extended LS junction positions, greyhounds experienced a 23% reduction, unaffected GSDs a 29% and affected GSDs a 34% reduction in mean foraminal volume.

The results of the simple linear regression model between position and group using the healthy Greyhound as the comparison group are shown in Table 20.

*Table 20. Statistical comparison between groups and position of the LS junction using Tukey multiple comparisons of means. AbNGSD = German shepherds with confirmed degenerative lumbosacral stenosis (N=12). NGSD = German shepherds thought to be unaffected by degenerative lumbosacral stenosis (N=12). NGH = Greyhounds thought to be unaffected by degenerative lumbosacral stenosis (N=10). (*statistically significant).*

	NGSD-NGH		AbNGSD-NGH		AbNGSD-NGSD	
	Difference	P value	Difference	P value	Difference	P value
Volume in extension	-4.89	0.90	-49.93	< 0.01*	-45.04	< 0.01*
Volume in neutral	10.50	0.73	-51.90	< 0.01*	-62.40	< 0.01*
Volume in flexion	103.68	< 0.01*	85.05	< 0.01*	-18.64	0.78

In the flexed LS position the GH group had significantly smaller L7-S1 lateral intervertebral foraminal volume than either affected or normal GSDs ($P<0.01$).

In the neutral LS position there was a significant difference between the foraminal volumes of AbNGSD and NGH, and AbNGSD and NGSD but not between NGSD and NGH. GSDs with DLSS had smaller foraminal volumes than the other two groups.

In the extended LS position there was a significant difference between the foraminal volumes of AbNGSD and NGH, and AbNGSD and NGSD. GSDs with DLSS had smaller foraminal volumes than the other two groups. There was no significant difference between the L7-S1 lateral intervertebral foraminal volumes of normal GSDs and normal GHs.

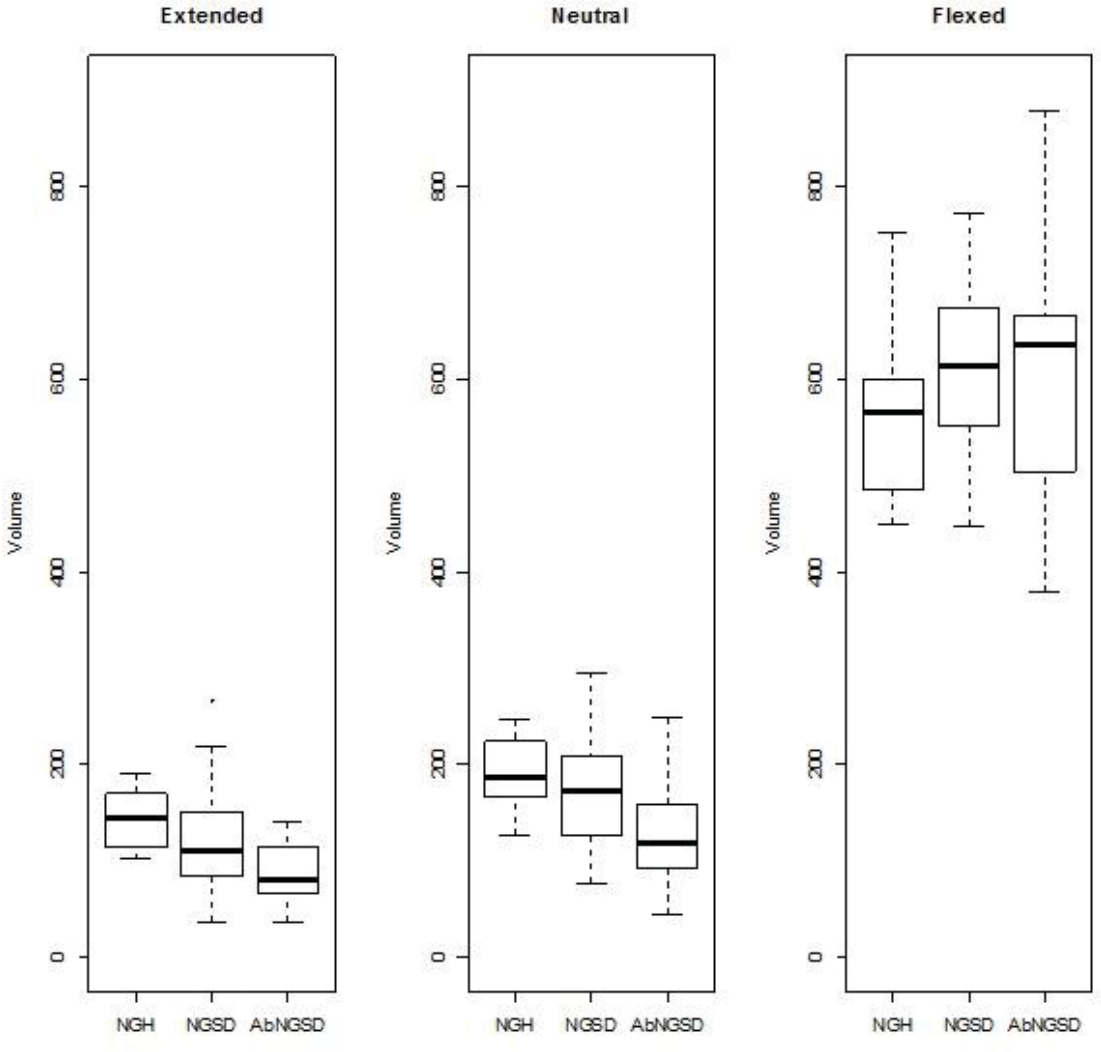


Figure 37. Box plot showing pooled volume data (L and R) of the L7-S1 lateral intervertebral foraminal volume as measured by computed tomography. German shepherds affected by degenerative lumbosacral stenosis (n=12) are labelled AbNGSD. German shepherds unaffected by degenerative lumbosacral stenosis (n=12) are labelled NGSD. Greyhounds unaffected by degenerative lumbosacral stenosis (n=10) are labelled NGH. Median (solid bar), 25th and 75th percentiles (box), 5th and 95th percentiles (whiskers) and outliers (dots).

Discussion

Quantifying the 3-dimensional volume of the L7-S1 lateral intervertebral neurovascular foramen from CT data has not been previously reported in the veterinary literature. The method described was repeatable in the author's hands and the findings correlated well with the expected effect of dynamic positioning of the LS junction. The high intra-observer repeatability supports this method of foraminal volume measurement as a useful tool in a research setting to assess the effect of therapeutic interventions. Volumetric analysis of the L7-S1 lateral intervertebral neurovascular foramen may be clinically useful when planning surgical intervention, however inter-observer repeatability requires investigation.

In all groups there was marked dynamic narrowing of the lateral intervertebral neurovascular foramina when the LS junction was in extension. Greyhounds had a smaller reduction in foraminal volume at full LS extension than GSDs. In comparison to a similar sized breed, GSDs may be predisposed to DLSS by having more marked dynamic narrowing of the lateral intervertebral neurovascular foraminal volumes prior to the onset of degeneration of the L7-S1 disc. Alternatively the GSDs assessed as normal/healthy in this study may have been sub-clinically affected by DLSS (asymptomatic) yet undergoing disc degeneration. Early disc degeneration may not be evident on CT images using conventional criteria; therefore some dogs classified as normal could subsequently develop DLSS. Volumetric analysis of the L7-S1 lateral intervertebral neurovascular foramina may be more sensitive than conventional radiographic assessment. A prospective study comparing volumetric measurement with measurements from 2-dimensional images, and comparison to cadaveric measurements of the foramen is required to fully validate the diagnostic accuracy of this method.

Germans shepherds with clinical signs and CT findings indicative of DLSS were shown (as a group) to have statistically smaller volumes of the L7-S1 lateral intervertebral foramina with the LS junction in an extended position than normal German shepherds or Greyhound dogs. These results support the hypothesis that GSDs affected by DLSS have greater dynamic narrowing of the lateral intervertebral foramen compared to GSDs without DLSS. Greater dynamic narrowing of the lateral intervertebral neurovascular foramen may result in mechanical compression of the *L7* nerve roots inducing pain and/or neurological dysfunction and hence could contribute to signs of DLSS.

Experimental studies have demonstrated that increasing degrees of mechanical compression of the *cauda equina* result in neurological signs and histological abnormalities of the compressed nerve roots (Delamarter *et al.*1990, Kim and Yang 1996). However, the relationship between the degree of compression of the *cauda equina* detected using advanced imaging, and the physical and surgical findings is more complex (Jones and Inzana 2000, Jones *et al.* 2000, Mayhew *et al.* 2002, Suwankong *et al.* 2006). The poor predictive value of advanced imaging might be improved by measuring the volume of the lateral intervertebral neuroforamen as an indication of *L7* nerve root compression in addition to standard radiological findings. An accurate, objective and repeatable, non-invasive method of measurement of the lateral intervertebral neurovascular foraminal volume may improve a clinicians' ability to diagnose DLSS in dogs.

A threshold value of 90 mm^3 was able to differentiate most of the affected dogs from the unaffected dogs. Using this threshold, a foraminal volume of less than 90 mm^3 in the fully extended LS position for either left or right lateral L7-S1 intervertebral neurovascular foramen would include 9 out of 12 of the GSDs with DLSS in this study, but exclude all of the 10 unaffected Greyhounds and 9 of 12 unaffected GSDs. This data can be expressed in a 2 x 2 table and the predictive value of the cut-off value calculated, Table 21.

Presence of DLSS	Foraminal volume < 90 mm^3	Foraminal volume > 90 mm^3	N =
Affected	9 (true positive)	3 (false negative)	12
Normal	3 (false positive)	9 + 10 (true negative)	22
N =	12	22	(34)

Positive predictive value = number of true positives / (number of true positives + number of false positives) x 100%

Therefore the positive predictive value of a foraminal volume <90m^3 = 9/(9+3) or 75%.

Negative predictive value = number of true negatives / (number of true negatives + number of false negatives) x 100%

Therefore the negative predictive value of a foraminal volume <90mm^3 = 19/(19+3) or 86%.

Therefore in a GSD with clinical signs compatible with DLSS, a foraminal volume of less than 90 mm^3 in the fully extended LS position provides 75% confidence that L7-S1 lateral neurovascular foraminal narrowing is associated with the dog's clinical signs. Conversely a dog with both lateral intervertebral foramina > 90 mm^3 has an 86% probability of being normal and not having DLSS. The box plots (figure 37) indicate significant overlap in the data, and the small number of dogs used to derive this threshold merit caution in its clinical use. A prospective study using a larger cohort of dogs with and without DLSS is needed to validate this threshold as a clinical tool.

A significant quadratic relationship between the length of the L7 vertebra and the lateral intervertebral canal volume was found. As the size of the L7 vertebral body increased the foraminal volume increased by the square of the length. Comparison to the length of vertebral bodies is commonly used to scale anatomical features between dogs of different size. Larger dogs would be expected to have greater lateral L7-S1 intervertebral neurovascular foraminal volumes. The AbNGSD group was significantly heavier than the NGH groups, due to two outliers that were the heaviest dogs in the entire cohort. However, the rest of the cohort was similar in weight to the NGSDs and NGHs. There was no difference between the correlation of vertebral length to foraminal volume between GSDs and GHs. Had there been disparate correlations then the volumes would have had to be scaled according to breed. A study including dogs of diverse breed and hence more varied in size might show dissimilar relationships between vertebral length and foraminal volume. Therefore whilst non-transformed comparison were appropriate for this study of GSDs and GHs of similar size, comparisons with other breeds requires further investigation of the effect of vertebral size on foraminal volume.

The volume of the lateral intervertebral neurovascular foramen measured in the neutral LS position chosen for this study was much closer to the volume of the extended position than that of the flexed position. Placing the hind limbs at 90 degrees to the table equated to an LS junction that was still significantly extended rather than halfway between full flexion and full extension. In future studies the femurs should be positioned at 90 degrees to the axis of the pelvis rather than 90 degrees to the CT gantry. There was greater variability in the flexed volume data of all dogs. Discerning the correct anatomic point to define the 5 mm window was less repeatable in this position. Small changes in centering the ROI led to larger volume variances than in the other two positions.

Greyhounds had consistently smaller foraminal volume in flexion than GSDs. Measurement on the LS angle (Mattoon and Koblik 1993) has shown than GSDs have a greater degree of LS flexion (larger LS angle in flexion) than greyhounds (Angela Hartman unpublished data).

There are several limitations to this novel method of measuring the volume of the lateral intervertebral neurovascular foramen. The method as described will tend to underestimate the extent of lateral intervertebral foraminal narrowing in dogs with an hourglass like narrowing in one zone of the foramen. An hourglass conformation of the neuroforamen could have the same volume as a more uniform foramen and yet have more significant compression at the narrowest cross-section. In addition the windowing presets exclude the soft tissues of the synovium of the articular process joints and any perineural fibrosis. Thus the bony perimeter of the foramen is delineated without the inclusion of any soft tissue structures. Hypertrophic soft tissues can also be responsible for nerve root compression; therefore this method potentially over-estimates the *in-vivo* volume of the neuroforamen. Three-dimensional CT measurements are based on the voxel size of the data. For the Phillips Brilliance system each voxel represents a volume of 0.8 mm^3. When measuring the width and height of the lateral intervertebral neurovascular foramen the addition or subtraction of a single voxel to the measurement can have a marked effect on the dimension recorded (Smith *et al.* 1993). Measurement of lateral intervertebral neurovascular foraminal dimensions using CT consistently underestimated the true measurements made from human cadaver specimens (Smith *et al.* 1993). This finding suggests that volumetric measurement as described could be a useful research tool and serve as a guide for comparing different surgical interventions but may not be an accurate measurement tool for determining precise anatomical parameters. The methods used were developed on a Phillips Brilliance 16-slice CT scanner and replication of our methods would be subject to differences between CT machines and manufacturers. It is likely that volume calibration would be required in order to ensure accuracy of diagnosis.

Whilst the method described for measurement of the volume of the L7-S1 lateral intervertebral foramen by one observer was repeatable, an assessment of inter-observer repeatability is required before this method can be recommended as a diagnostic method for general use by veterinarians in practice.

Conclusions

Positioning a dog with the LS junction in extension significantly decreases the volume of the L7-S1 lateral intervertebral neurovascular foramen. GSDs with DLSS had greater dynamic narrowing of the L7-S1 lateral intervertebral neurovascular foramen than GSDs without evidence of DLSS. The changes noted with positional CT imaging under sedation could mirror the range of motion resulting from activity. Surgical management of DLSS in dogs should consider the degree of foraminal narrowing as indicated by dynamic CT studies of the LS junction. In GSDs, a threshold of 90mm^3 had a positive predictive value of 75% and a negative predictive value of 86%. This threshold may provide greater diagnostic confidence in dogs in the early stages of degeneration of the LS junction. In addition, dynamic narrowing of the lateral intervertebral foramen suggests that fusion of the LS junction in a neutral to flexed position should be an effective form of therapy for DLSS.

An abstract entitled “Dynamic alteration of the lumbosacral lateral intervertebral canal and its potential clinical relevance, as determined by CT volume analysis.” [Worth AJ, Bridges JP, Hartman A] was presented by the author at the World Veterinary Orthopaedic Conference, Breckenridge, Colorado, 2-7th March 2014

Assessment of the effect of dorsal laminectomy and dorsal annulectomy with partial discectomy of the canine L7-S1 intervertebral disc on the volume of the lateral intervertebral neuroforamina in dogs when the lumbosacral joint is extended.

Introduction

In chapter seven it was demonstrated that motion of the lumbosacral (LS) junction in the sagittal plane resulted in alteration of the volume of the lateral intervertebral neurovascular foramina. German shepherd dogs with degenerative lumbosacral stenosis (DLSS) had significantly smaller foramina during extension than unaffected GSDs and healthy Greyhounds. Relative reduction in foraminal volume is most likely the result of degenerative changes first affecting the L7-S1 intervertebral disc. Disc degeneration has been demonstrated in large-breed dogs with DLSS and such degeneration can lead to stenosis of the lumbosacral vertebral canal and the lateral intervertebral neurovascular foramina (Bergknut 2011). Compression of the *L7* nerve roots by dynamic foraminal stenosis has been implicated as a cause of clinical signs such as intermittent hind limb lameness (Godde and Steffen 2007, Meij and Bergknut 2010). Surgical decompression of the *cauda equina* via dorsal laminectomy without stabilisation is a commonly performed surgical procedure for patients with chronic clinical signs associated with L7-S1 disc degeneration (Danielsson and Sjöström 1999, Suwankong *et al.* 2008). Dorsal annulectomy with partial discectomy at the L7-S1 site is often performed concurrently with dorsal decompression when pre-operative imaging confirms disc protrusion is present (Chambers 1989, Palmer and Chambers 1991b). Whilst effective at reducing LS pain in the majority of cases, recurrence of clinical signs is frequently reported after dorsal decompression (Danielsson and Sjöström 1999, Janssens *et al*. 2000, DeRisio *et al.* 2001, Suwankong *et al.* 2008). Two studies have questioned the benefit of annulectomy and partial discectomy in addition to dorsal decompression for DLSS. DeRisio *et al.* (2001) reported no significant difference in outcome between dogs undergoing dorsal laminectomy alone (n=15) and those that underwent concurrent annulectomy and dorsal foraminotomy (n=54) for DLSS. Suwankong *et al.* (2008) found that dogs that had undergone dorsal laminectomy and partial discectomy (n=78) showed significantly less improvement than those that had undergone dorsal laminectomy alone (n=78). Jones *et al.* (2008) proposed that a key function of the normal intervertebral disc is its ability to maintain the dimensions of the L7-S1 lateral intervertebral neurovascular foramina independent of the angle of the

lumbosacral junction. Dorsal laminectomy and annulectomy in cadavers of healthy dogs increased the range of motion of the LS junction in the median plane (Early *et al.* 2013). The extension and flexion angles were both increased as a result of destabilisation of the LS junction by the surgery. In a separate study, stability of the LS junction of the cadavers of healthy dogs was negatively affected after dorsal laminectomy and nucleotomy when tested in flexion-extension, lateral bending and torsion (Smolders *et al.* 2009). An increased LS junction extension angle will result in greater cranial motion of the sacral articular processes, which form the caudal and dorsal margins of the lateral intervertebral neurovascular foramina. In dogs with DLSS it is possible that annulectomy with partial discectomy further destabilises an already degenerate L7-S1 disc, and induces greater collapse of the intervertebral disc space. Decreased intervertebral disc space would result in a further reduction in the lateral intervertebral neurovascular foraminal volume, potentially worsening dynamic compression of the *L7* nerve roots. In a study of the LS junction in cadaver dogs determined to be unaffected by LS junction degeneration by radiography, dorsal laminectomy alone did not significantly affect the stiffness of the LS specimens compared to un-operated cadaver specimens when subjected to four-point bending in the median plane (Smith *et al* 2004). However, when partial discectomy was also undertaken there was a 33% decrease in stiffness when the LS junction was flexed but no significant decrease in stiffness of the LS junction in extension. The partial discectomy was performed by insertion of a power burr though a small hole in the dorsal *annulus fibrosus*. The *nucleus pulposus* was macerated but the *annulus fibrosus* was left intact. Partial discectomy through a small window in the annulus is potentially less destabilising to the LS junction than an annulectomy, where in a rectangular window of the protruded dorsal annulus is removed. Dorsal annulectomy is commonly undertaken by veterinary surgeons as a treatment for DLSS (Chambers 1989, Palmer and Chambers 1991b, Danielsson and Sjöström 1999, Janssens *et al*. 2000, DeRisio *et al.* 2001).

The aim of this study was to determine if a dorsal annulectomy and partial discectomy together, achieved via a partial dorsal laminectomy, have any effect on the volume of the L7-S1 lateral intervertebral neurovascular foramina in dogs when the LS junction is extended.

Materials and Methods

Isolated sections (L7-S3) of the vertebral columns of cadavers were obtained from 10 large breed dogs without known disease of the LS junction. The epaxial musculature was dissected and removed but the joint capsules and ligaments associated with the LS junction were left *in-situ*. The ilial wings were disarticulated from the sacrum by cutting through the sacro-ilac joint using a bone saw. The cadaver sections were frozen at minus 18-20° C until required for testing.

A custom jig, designed to fit within a CT gantry, was manufactured from wood so as to be radiolucent. The jig held each LS section rigidly via a clamp and locating pin system across S2-3. An upright mount behind the clamp acted as the locating point for a loading device located above the LS junction, Figure 38. On the day of testing the sections were thawed to room temperature, mounted in the jig and kept moist with normal saline. A 3.2 mm hole was drilled in the centre of the endplate of L7 parallel to the long axis of L7. A 4.0 mm 316L stainless steel malleolar screw (positive profile, partly threaded) was screwed into the hole with the non-threaded section of the screw projecting from the endplate. A small incision was made though the interarcuate ligament and an aqueous gel (K-Y jelly) was injected into the epidural space to expel any air to prevent CT artefact within the vertebral canal and the foramina. Computed tomography was performed to image the jig mounted LS section using a 16-slice helical scanner (Phillips Brilliance, Philips Healthcare, The Netherlands). The LS section that was clamped at the sacrum was then loaded with a cantilever bending force via a linkage from the L7 vertebral body screw to a digital weighing scale mounted on the jig, [see Figure 38]. A turnbuckle linked between the actuator of the weigh scale and the L7 screw allowed the tension to be adjusted. Following a short period of acclimation, each LS section was loaded in extension using a standardised load of (6.8kg) indicated on the weigh scale.

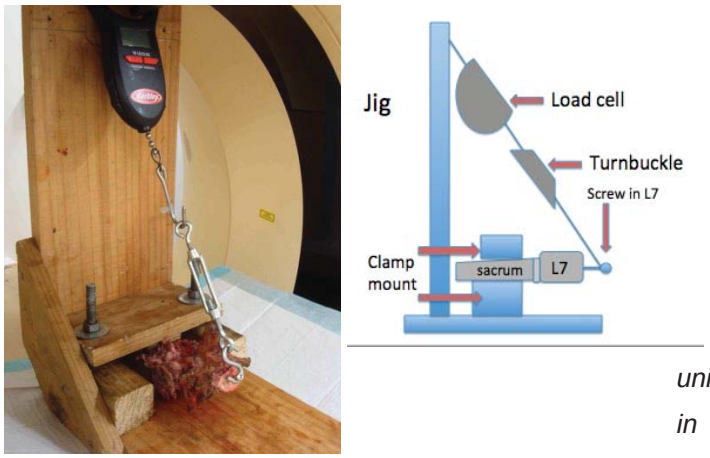


Figure 38. Lumbosacral specimen mounted in a custom jig and placed within the CT gantry (left). The sacrum is secured within the clamp section of the jig. A turnbuckle connects the screw in the vertebral body of L7 to the jig. The digital scale is used to create uniform tension (6.8kg load) resulting in extension of the LS junction as shown in the diagram (right).

The test rig was removed from the CT gantry. The LS segment was then unloaded and demounted, the dorsal intervertebral (interarcuate) ligament was removed and a minimal dorsal laminectomy of S1 was performed using an electric burr. Only a small area of lamina was removed, sufficient to allow a rectangular dorsal annulectomy of the L7-S1 disc to be performed with a no. 11 scalpel blade. A rectangular excision was performed, with lateral margins medial to the ventral vertebral venous sinuses. As much of the *nucleus pulposus* was removed as possible using a 2 mm Lambert curette to complete a partial discectomy. The vertebral canal and lateral foramina were injected with K-Y jelly to expel any air and the segment was remounted in the test rig. The specimen was loaded in extension with the same 6.8kg force and placed in the CT gantry and re-imaged.

The CT images were analysed for indications of evidence of degeneration of the LS junction. Specimens were classified by the degree of degeneration of the L7-S1 intervertebral disc according to the criteria in Table 22.

Table 22. Criteria for classifying the extent of degenerative change of the L7-S1 intervertebral disc evident in 10 large breed dogs by radiological signs. Specimens were classified as unaffected, marginally affected or mildly affected according to these criteria:

Specimen classification	**Criteria for classification**
Unaffected	No radiological changes.
Marginally affected	Intra-discal mineralisation Dorsal protrusion of the *annulus fibrosus* greater than 25% of the height of the vertebral canal
Mildly affected	Evidence of spondylosis. Intra-discal mineralisation Dorsal protrusion of the *annulus fibrosus* greater than 25% of the height of the vertebral canal

The LS junction angle was measured on CT by determining the angle between lines demarcating the floor of the L7 and sacral vertebral canal (Mattoon and Koblik 1993) using an angle tool (EFilm Digital Laboratories, California). The LS junction angle was measured with the specimens loaded in extension, both before and after dorsal laminectomy/annulectomy with partial discectomy. The volume of the lateral intervertebral neurovascular foramen was measured according to the method described in chapter seven. Briefly, the foraminal volume was calculated using tissue segmentation algorithms and creating a region of interest outlining the foramen with a standardised 5 mm width. The pre-sets excluded bone and dense annular material. Volume measurements (mm^3) were performed five times for each foramen then averaged. The foraminal volumes with the LS joint in an unloaded position and with the

load applied in extension both before and after annulectomy, were compared using a linear mixed-effects model (R Foundation for Statistical Computing, Vienna, Austria, 2013). The ratio of foraminal volume in extension post-annulectomy to foraminal volume in extension pre-annulectomy was expressed as a percentage.

Results

The mean sagittal lumbosacral angle of the 10 specimens was 169° in the unloaded position and 150° and 148° in the extended and the extended-post-annulectomy positions respectively. There was no significant difference between the mean sagittal LS junction angle pre and post annulectomy (p=0.10). The volume data is presented in Table 23 with the specimens ranked by degree of radiological evidence of L7-S1 disc degeneration. The mean volume of the L7-S1 lateral intervertebral neurovascular foramina (n=20) was 381mm^3 in neutral positioning and 137 mm^3 when loaded in extension. Following annulectomy the mean volume was significantly (p<0.01) reduced by a mean of 28% to 98 mm^3. Reduction in the volume of the lateral intervertebral neurovascular foramen occurred in 19 of 20 foramina, with a 3 to a 69% reduction in volume (median 28%). Based on CT images the reduction of the foraminal volume was significantly greater in the dogs with L7-S1 intervertebral discs designated mildly degenerate (33%) compared to those designated marginally degenerate (18%) (p<0.01), but not in those that were designated non-degenerate (28%).

Table 23. Lateral intervertebral neurovascular foramina volume data from cadaveric LS specimens (n=10) measured using CT under a 6.8kg load in cantilever bending (extension), prior to and following annulectomy/partial discectomy of the L7-S1 disc. The foraminal volume data is expressed in mm^3. The foraminal volume post annulectomy is also expressed as a percentage foraminal volume in extension prior to annulectomy. The dogs were stratified by the presence of any signs of L7-S1 disc degeneration. Dogs 1, 2, 3 and 8 had no indication of L7-S1 degeneration, Specimens 4, 5 and 6 had marginal signs and specimens 7, 9 and 10 had mild signs according to the criteria in Table 1. R = right. L = left, Extend or ext. = extension.

Dog	R extend Foramen	R post Annulectomy	% of ext.	L extend Foramen	L post Annulectomy	% of ext.
1	174	166	95	144	132	92
2	165	90	54	230	72	31
3	137	80	59	56	54	96
8	133	95	72	125	90	72
		average redn	70		average redn	73
4	74	64	87	67	80	120
5	249	194	78	158	154	97
6	204	133	65	203	90	45
			77			87
7	59	54	92	40	16	40
9	206	137	66	128	108	85
10	75	34	45	108	72	67
		average redn	68		average redn	64

Discussion

In this cadaver model, dorsal annulectomy with partial discectomy of the L7-S1 intervertebral disc resulted in a significant reduction of the volume of the lateral intervertebral neurovascular foramen when the L7-S1 articulation was loaded in full extension.

This study suggests that a dorsal annulectomy with partial discectomy may induce further dynamic collapse of the L7-S1 articulation in dogs. This finding has significance when a decision is made to perform dorsal annulectomy/partial discectomy in a clinical case of DLSS. Post-operatively the sacral articular processes could further compromise the L7-S1 intervertebral neurovascular foramen during extension of the LS junction thus leading to persistence or recurrence of clinical signs of nerve root compression. Post-operative reduction in the size of the L7-S1 lateral intervertebral neurovascular foramen may partly explain the high rate of recurrence of clinical signs after dorsal laminectomy with discectomy noted in previous studies (Danielsson and Sjöström 1999, Janssens *et al*. 2000).

When dorsal laminectomy plus annulectomy with partial discectomy was compared to dorsal laminectomy alone for the management of DLSS in dogs there was no apparent advantage of the former (DeRisio *et al.* 2001). Indeed, one study even reported a poorer outcome for those dogs that had disc surgery (Suwankong *et al.* 2008). The positive results seen with a dorsal laminectomy alone suggests that when performed on dogs with vertebral canal stenosis due to significant dorsal annular protrusion, the removal of the roof of the vertebral canal at the LS junction is sufficient to reduce compression of the *cauda equina*. Based on the present study in cadavers, the author recommends that excision of the dorsal annulus of L7-S1 is limited to only those cases with an asymmetric protrusion into the entrance zone of the *L7* foramen that is evident at surgery even when the LS junction is in a flexed position during surgery.

In this cadaver study a simple jig applied a cantilever bending moment to the LS junction. Previous investigations of the range of motion and stiffness of the LS junction have utilised 3 or 4-point bending in material testing devices (Smith *et al.* 2004, Early *et al.* 2013). The limitation of the bore diameter of a CT unit, and their metallic nature precluded the use of a traditional materials testing machine. A jig to hold the specimen within the CT gantry was therefore fabricated from wood. To counter any elastic deformation the turnbuckle was gradually tightened until the scale read a steady 6.8 kg. The weigh scale had a displayed accuracy of +/- 0.05 pounds. The specimens were

only tested in extension (incorrectly referred to by some authors as dorsi-flexion of the LS junction). During flexion the foramina enlarge therefore this position was not considered relevant to the study.
There was considerable variation in the degree of narrowing experienced by the left and right foramina in the same dog. This may be the result of asymmetry of the remaining support from the dorsal *annulus fibrosus* following partial annulectomy. Alternatively the screw position within the L7 vertebra was not standardised with respect to the true sagittal plane and may have resulted in an asymmetrical force inducing some LS lateral bending in addition to LS extension under load.

The dogs in this current study were not examined prior to euthanasia for clinical signs of DLSS but overt disease of the LS junction was not noted on dissection. The radiological signs of degeneration were mild and were unlikely to have been associated with clinical signs. When data from specimens with mild and moderate radiological evidence of degenerative changes were pooled, there was no difference in the mean extent of the reduction in foraminal volume versus those dogs without evidence of LS degeneration.

Conclusion

A dorsal annulectomy with partial discectomy may induce further dynamic collapse of the L7-S1 articulation in a dog with DLSS. Post-operatively the resultant narrowing of the L7-S1 lateral intervertebral neurovascular foramen may lead to persistence or recurrence of clinical signs of nerve root compression.

An abstract entitled “Dorsal annulectomy of the canine L7-S1 intervertebral disk may reduce the three dimensional volume of the L7-S1 lateral neuro-vascular canal during extension of the lumbosacral junction. [Worth AJ, Bridges JP] was presented by the author as a poster at the World Veterinary Orthopaedic Conference, Breckenridge Colorado, 2-7th March 2014

Long-term outcome and CT assessment of lateral foraminotomy at the lumbosacral junction in dogs with degenerative lumbosacral stenosis.

Introduction

As reviewed in chapter five, the diagnosis of DLSS is usually made on the basis of clinical signs and demonstration of compression of the *cauda equina* using CT or MRI. Narrowing of the sacral canal may be caused by a prolapsed L7-S1 intervertebral disc annulus, or hypertrophied joint capsule and/or *ligamentum flavum*. Narrowing of the lateral L7-S1 intervertebral neurovascular foramen may be static from osteophyte development or dynamic as the result of extension of the LS junction. Imaging with the dog's LS junction positioned in extension can detect dynamic displacement of the sacrum or dynamic narrowing of the L7-S1 lateral intervertebral neurovascular foramen. Surgical management of DLSS is currently directed at decompression of the affected *cauda equina* and/or nerve roots (Palmer and Chambers 1991) or stabilisation of the LS junction (Slocum and Devine 1986, Meheust 2000, Bagley 2003). The most widely reported decompressive surgical technique involves dorsal laminectomy of L7 and S1 combined with dorsal annulectomy to remove any prolapsed disc annulus (Denny et al. 1982, Chambers 1989, Ness 1994, Danielsson and Sjostrom 1999, Janssens et al. 2000). Other surgeons have performed dorsal decompression alone, or with concurrent disc fenestration and partial discectomy (Klaveren et al. 2005; Suwankong et al. 2007). More recently dorsal stabilisation techniques have been reported to have similar success rates to dorsal decompression (Slocum and Devine 1986, Hankin *et al.* 2012, Golini *et al.* 2014).

Guidelines as to which surgical procedure is the most appropriate for an individual dog have not been well defined (Jeffery *et al.* 2014). Dorsal compression of the *cauda equina* due to the sacral lamina and hypertrophied interarcuate ligament when the joint is in extension may resolve after decompression by dorsal laminectomy. Ventral compression of the *cauda equina* by protrusion of the dorsal annulus of the L7-S1 disc has traditionally been an indication for dorsal disc annulectomy and discectomy (Palmer and Chambers 1991b). The presence of ventral displacement of the sacrum relative to L7 and dynamic narrowing of the L7-S1 lateral intervertebral neurovascular foramen on extension, are considered indications for distraction and stabilisation (Slocum and Devine 1986, Bagley 2003, Hankin *et al.* 2012). The presence of clinical signs indicative of *L7* nerve root compression and narrowing of the L7-S1 lateral

intervertebral neurovascular foramen with loss of fat signal around the *L7* nerve roots is reported as an indication for nerve root decompression with a lateral foraminotomy (Godde and Steffen 2007). However, prospective comparative studies on which to base treatment decisions in regards to DLSS in dogs have not been undertaken (Jeffery *et al.* 2014).

The L7-S1 lateral intervertebral neurovascular foramen is not a simple aperture but is a complex three-dimensional tunnel. In humans the L5-S1 neurovascular foramen is comprised of entrance, middle and exit zones which have been defined according to their location in regards to the pedicle and articular processes of L5 and S1 (Lee *et al.* 1988). Whilst this terminology (entrance, middle and exit) has been applied to dogs (Wood *et al.* 2004, Godde and Steffen 2007), the canine pedicle differs anatomically from that of humans, being thinner and orientated at a more oblique angle with a longer and narrower lateral recess (Wood *et al.* 2004).

The prognosis for a successful outcome following traditional surgical management of DLSS ranges from 55 to 79% depending on the method of reporting and study population - pets versus working dogs (Janssens *et al.* 2000, Suwankong *et al.* 2008, Meij and Bergknut 2010).

Recurrence of lumbosacral pain and dysfunction following initially successful dorsal decompressive surgery ranges from 19 to 37% (Danielsson and Sjöström 1999, Janssens *et al.* 2000, Suwankong *et al.* 2006). Suwankong *et al.* (2007) found 5/12 dogs treated for DLSS using dorsal decompression were no longer able to perform their intended activities at a median follow-up of 1.5 years. In a study of 29 military working dogs, 38% were euthanased due to either continued signs of DLSS after surgery (n=6) or recurrence of signs following an initial improvement after surgery (n=5). All dogs had undergone dorsal laminectomy with (n=18) or without (n=11) annulectomy (Linn *et al*. 2003).

Recurrence of clinical signs of lumbosacral pain following decompressive surgery has been attributed to the development of a laminectomy scar membrane dorsally, leading to *cauda equina* compression (Danielsson and Sjostrom 1999, DeRisio *et al.* 2001), although the clinical significance of development of a laminectomy membrane has been questioned by others (Janssens *et al.* 2000). In humans, unrecognized foraminal stenosis not relieved by surgery is thought to be a significant cause of the “failed back surgery syndrome” (Fritsch *et al.* 1996, Stambough 1999). Failure to recognise and treat lateral intervertebral foraminal stenosis has been suggested as a cause of recurrent clinical signs in dogs following dorsal decompression alone (Godde and Steffen 2007).

The lateral foramenotomy procedure of Godde and Steffen (2007) may represent a significant improvement in the management of DLSS as it allows greater decompression of the L7-S1 lateral intervertebral neurovascular foramen in the dog. To date there has only been a single publication relating to the outcome of lateral L7-S1 foramenotomy in dogs (Godde and Steffen 2007). The developers of the technique undertook a retrospective evaluation of 20 dogs using a combination of veterinary re-examination six months post-operatively and a telephone interview with the owners of the dogs at final follow-up. Unilateral or bilateral lateral LS foramenotomy was performed on each dog based on MRI findings of *L7* nerve root compression. A limited dorsal laminectomy (Kinsel *et al.* 2004) had been performed concurrently in 11 of the 20 dogs. The decision to perform concurrent dorsal laminectomy was based on MRI evidence of LS vertebral canal stenosis. The outcome was good to excellent in 19 dogs (95%) at a mean follow-up of 15.2 months (range 6-42 months) (Godde and Steffen 2007). One dog was euthanased following progressive neurological decline attributed to a degenerative myelopathy. This improvement in outcome over traditional dorsal decompressive surgery alone was attributed to more effective decompression of the *L7* nerve root pathway. Recurrence was not noted in any of the 19 dogs that responded to surgery.

The study by Godde and Steffen (2007) did not evaluate whether re-growth of bone occurred at the foraminotomy site. Bone re-growth at the site of a dorsal laminectomy has been reported to occur in humans (Chen *et al.* 1994). In an experimental study in normal dogs, bone regrowth occurred at the site of an endoscopically assisted foraminotomy performed at the entrance and middle zones. By 12 weeks there was no significant difference between foraminal area measurements of the control and operated sides despite significant enlargement noted immediately post-operatively (Wood *et al.* 2004).

Assessment of the effect of foraminotomy requires an accurate method of measuring the size of the L7-S1 lateral intervertebral neurovascular foramina *in-vivo*. MRI is cited as the diagnostic modality of choice for the diagnosis of DLSS but it has poorer spatial and textural resolution of bone than CT (Grumme and Bittl 1998).

Computed tomography allows data reconstruction into high-resolution 3-dimensional images which can be manipulated and viewed from any angle. Volume rendering is a process by which regions of interest can be defined in 3-dimensions and a numerical volume determined. Both parasagittal foraminal area and volumetric analysis have been reported in the medical literature for determining the size of lateral intervertebral

foramina (Smith *et al.* 1993). Parasagittal CT images have been used to measure cross sectional L7-S1 foramen area in dogs (Jones *et al.* 2008) and recently a bi-oblique method of area measurement was reported (Higgins *et al.* 2011), but to the author's knowledge volumetric analysis has not been described in dogs. The author has developed a method for volumetric measurement of the L7-S1 lateral intervertebral neurovascular foramen as described in chapter seven.

The aim of this prospective study was to assess the outcome of lateral foraminotomy in a cohort of dogs with degenerative lumbosacral stenosis (DLSS) and confirmed narrowing of one or both L7-S1 lateral intervertebral neurovascular foramina. CT volumetric analysis was used to measure the foraminal volume both immediately post-operatively and at least 12 months after foraminotomy. The author sought to determine if bone regrowth occurs at the site of a lateral foraminotomy and could become a potential limiting factor for the technique's long-term success in dogs with DLSS.

Materials and methods

Dogs diagnosed with DLSS that underwent lateral LS foraminotomy by the author at the MUVTH between May 2009 and December 2011 were included in a prospective clinical trial with informed owner consent. All dogs entered into the study had clinical signs localised to the lumbosacral joint on physical and/or neurological examinations and had imaging findings consistent with *L7* nerve root compression. Plain screening radiographs were performed to rule-out vertebral neoplasia and discospondylitis. A CT scan was performed under anaesthesia or deep sedation using a standard protocol (Phillips Brilliance helical 16-slice, Philips Healthcare, The Netherlands). Dogs were imaged in dorsal recumbency supported in a foam trough. Each dog was alternately strapped to the CT table with the pelvic limbs perpendicular to the table (neutral LS junction position), maximally flexed cranially (flexed LS junction position), and maximally extended caudally (extended LS junction position), [chapter seven]. For the extension position the edge of the foam trough was then placed beneath the LS junction to aid full extension of the LS junction. Soft tissue and bone algorithms were performed in each position and 3-dimensional renderings were created to study the foramen at these extremes of range of motion.

The author and a specialist veterinary radiologist interpreted all CT scans. The extent of dorsal annular prolapse was determined on sagittal images. The extent of lateral annulus disc protrusion into the entrance zone of the LS lateral intervertebral neurovascular foramen was assessed on axial soft tissue sequences. The images were windowed and levelled to allow the *L7* nerve roots to be seen exiting the foramen.

The extent of perineural fat and the size of the nerve roots were subjectively assessed. Volume measurements of the L7-S1 lateral intervertebral neurovascular foramina were performed according to the protocol described in chapter seven.

The surgery was performed as demonstrated by Thomas Godde in a training session attended by the author at the Aesculapium, Tuttlingen, Germany, 20th-21st March 2009. Briefly, dogs were positioned in sternal recumbency at one end of the surgical table. A rolled towel was placed under the pubis to elevate the pelvis. The pelvic limbs were pulled forward and held beside the flanks to maximally flex the lumbosacral space. If a dorsal laminectomy was to be performed as part of the procedure a dorsal midline approach to the lumbosacral space was performed through a skin incision from L6 to S2. After sagittal muscular and fascial division the interarcuate ligament was grasped and sharply dissected taking care to not lacerate the *cauda equina*. In dogs requiring dorsal decompression either a traditional dorsal laminectomy (Chambers 1989) including the caudal L7 lamina, or a limited S1 dorsal laminectomy (Kinsel *et al.* 2004) was performed. The limited laminectomy completely preserves the L7 lamina whereas the cranial margin of S1 is removed with a pneumatic burr and Kerrison-Smith rongeurs. Laminectomy was continued caudally until reduction of sacral overhang was sufficient to alleviate compression of the *cauda equina* against the sacral lamina by the L7-S1 disc annulus. In dogs requiring a unilateral foramenotomy the skin incision was curved cranially if more exposure was required. In dogs requiring bilateral foraminotomy without dorsal laminectomy separate skin incisions were made on each side. During foraminotomy the table was tilted 20 degrees away from the side being operated to facilitate a lateral approach between the wing of the ilium and the vertebral column (Godde and Steffen 2007). The superficial truncal fascia was retracted with Gelpi retractors to expose the deep truncal fascia which was incised midway between the ilial wing and the vertebral spines of L6 to S2. Blunt dissection was used to create a cleft between the *multifidus* and *sacrocaudalis* muscles. Long-armed Gelpi retractors were utilised to create a working space for deeper dissection. Sharp dissection removed the muscular attachments to the articular processes caudal to L6-7 and cranial to L7-S1 (Godde and Steffen 2007). A periosteal elevator was used to bluntly dissect the remaining muscle and perineural tissue to expose the L7-S1 intervertebral foramen. In some dogs partial resection of the *quadratus lumborum* and *longissimus* muscles was required to see the transverse process and pedicle of L7. A pneumatic burr was then used to perform the foraminotomy at a site defined by the transverse process ventrally and the articular process of L7 dorsally. After breaching the outer cortical bone, a nerve hook determined the depth of the pedicle and defined the

direction of the foramen. Burring proceeded cautiously until the inner cortical bone was thin and unstable. Kerrison-Smith rongeurs and an angled curette were then used to break through the inner cortical bone into the lateral recess. The nerve hook was used to probe the foraminotomy and the surgery continued until the *L7* nerve moved freely to assume a relaxed position exiting more cranio-laterally than prior to surgery. At various times the nerve was protected from the instruments using the nerve hook as a shield. Head loupe magnification and a headlamp improved observation in what is a deep dissection with a narrow field of view. The truncal fascia was closed in a continuous suture pattern, and the subcutaneous and skin closure was routine.

Seven dogs were included in the study, of which the last three were re-CT scanned immediately after surgery and at follow-up using the same protocol as previously described. To minimize gas artefacts on CT the surgical site had been flushed with saline prior to closure. Due to financial limitations the other four dogs were only re-scanned at follow-up, not immediately post-op.

Post-operative instructions were standard and included a requirement to restrict the dogs to a small run or a cage for the first six weeks. In weeks 1-2 the dogs were allowed 5-minute leash walks for urination and defecation. This limited exercise increased to 10 minutes leash walking in week 3 and 4, and 15 minutes leash walking in weeks 5 and 6. Dogs were prescribed carprofen (Rimadyl, Pfizer Animal Health) 2 mg/kg twice daily for 7 days to decrease inflammation and provide analgesia.

All dogs were re-examined by the author six weeks after surgery. The owners were then interviewed by telephone in March 2012. They were asked to describe their dog's current mobility and relate that to the dog's clinical signs prior to surgery. Following the interview, all seven dogs were re-evaluated by the author at greater than 12 months post-operatively, and a repeat CT scan was performed, Figure 39. The dogs were scanned under sedation with medetomidine (0.005mg/kg IV) and butorphanol (0.2mg/kg IV), with the same CT protocol as outlined above. All scans were performed with the LS junction in extension a previously described in chapter 7.

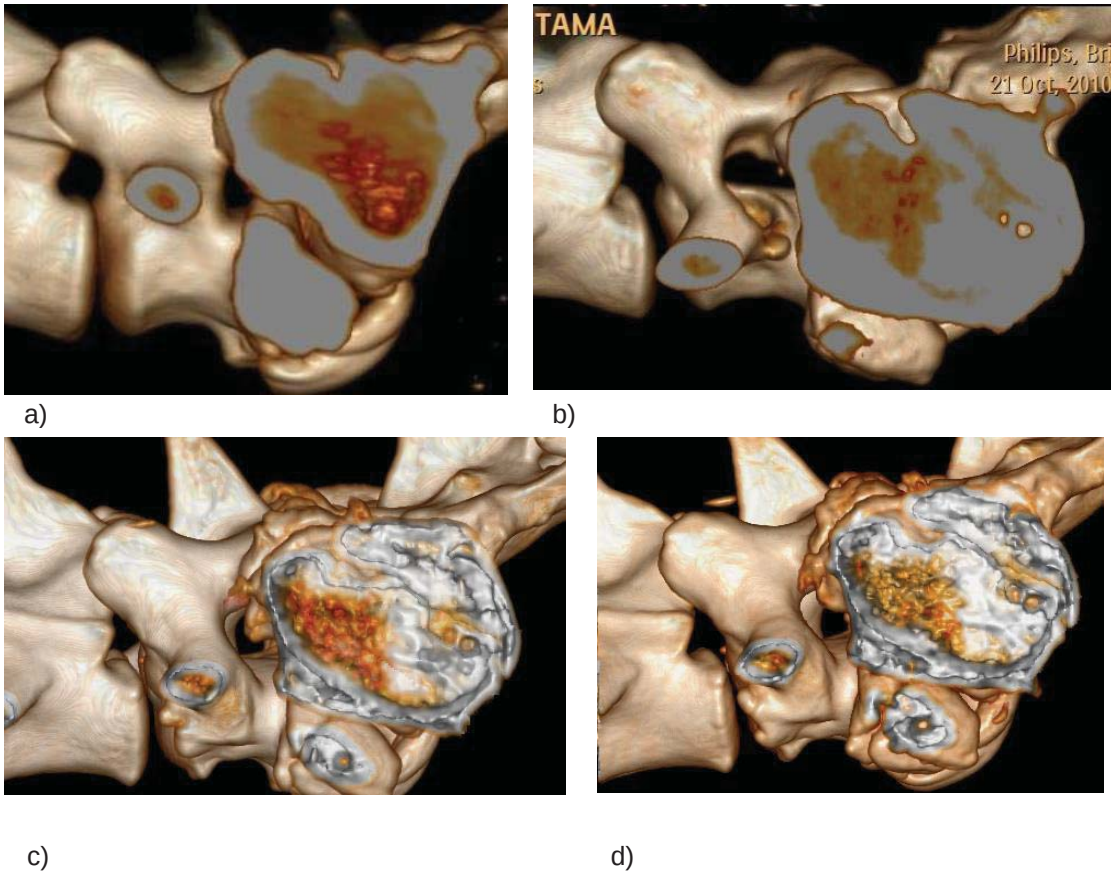


Figure 39. Reconstructed 3-D CT images of the left L7-S1 lateral intervertebral neurovascular foramen of dog #7 a) pre-operatively, b) immediately post-operatively following lateral foraminotomy then c) 14 months and d) 26 months post-operatively.

Results

Demographic and historical data of the seven dogs are listed in Table 24. One was a GSD Police dog in active duty, the remaining six were pet dogs. The median age of the dogs was 7 years (range 3 – 10 years). The median weight of the dogs was 31 kg (range 14.5 – 65.5 kg). Clinical signs had been present for a median of two months prior to presentation, but there was a considerable range (24 hours to 7 years). Clinical signs included pain on pressure over the LS junction, hind limb lameness, difficulty jumping, stiffness on rising, and reluctance to exercise (Table 24).

The imaging findings, surgical treatment and outcomes are listed in Table 25. Two dogs had bilateral lateral foraminotomy without dorsal laminectomy for bilateral neuro foraminal narrowing without sacral canal compression of the *cauda equina*. Two dogs underwent bilateral lateral foraminotomy with a limited S1 laminectomy for bilateral neuro foraminal narrowing combined with sacral canal compression of the *cauda equina*. Two dogs had unilateral bilateral lateral foraminotomy combined with traditional L7-S1 dorsal laminectomy for sacral canal compression of the *cauda equina* combined with unilateral neuro foraminal narrowing. One dog had bilateral lateral foraminotomy

combined with traditional L7-S1 dorsal laminectomy for sacral canal compression of the *cauda equina* combined with bilateral neuro foraminal narrowing. The volumetric measurements pre-operatively, immediate post-operatively (three dogs) and at 3-36 months (seven dogs, median 23 months) follow-up are shown in Table 26.

Table 24. Data from seven dogs diagnosed with degenerative lumbosacral stenosis that underwent lateral foraminotomy.

Dog	Breed	Age (Years)	Sex	Weight (kg)	History
#1	Boxer	4	MN	30	Two-month history of progressively worsening right hind lameness, difficulty rising and reluctance to jump into the back of a truck for 5 months. Resting the leg when standing.
#2	Labrador	10	FS	25	Twelve-month history of lower back pain and reluctance to jump into a car. Gradual hind limb stiffening and decreased exercise tolerance in the last 6 months. Previous marginal response to NSAIDs has been treated for possible disco spondylitis with extended antibiotic course.
#2	Beagle	7	FS	14.5	Two-month history of back pain after a traumatic incident. Intermittent exercise induced left hind lameness. Hunched back and abnormal tail carriage.
#4	GSD x Labrador	6	M	31	Two-month history of back pain, intermittent right hind lameness and difficulty jumping, reluctance to exercise. Eighteen-month history of incontinence.
#5 PDS	GSD	8	M	39	Three-year history of mild lameness after heavy work, lordosis and lumbosacral epaxial muscle pain. CT showed congenital stenosis and transitional vertebra. Recent poor work performance, reluctance to jump, apparent pain and bunny hopping gait. Mild urinary incontinence for several weeks.
#6	Rhodesian Ridgeback	8	MN	65.5	Seven-year history of progressive hind limb weakness. Mildly faecal and urinary incontinence. Transitional vertebral anomaly found on radiographs and MRI recommended but not performed
#7	Rottweiler	3	M	39	Growls with any manipulation, tense and aggressive. Unable to work, severe difficulty jumping into vehicles, scaling walls/fences/lame after jumping.

***** PDS = New Zealand Police Dog Section, M=male, MN=male neutered, F=female, FS=female spayed

Table 25. Clinical signs, CT findings, treatment performed and outcome of seven dogs with degenerative lumbosacral stenosis that underwent lateral foraminotomy. XR = plain radiography, MRI = Magnetic Resonance Imaging, CT = Computed Tomography, R = right, L = left, DLSS = degenerative lumbosacral stenosis, LS = lumbosacral, H=hind limb.

Dog Number	Clinical Signs	CT findings	Treatment	Outcome
#1	RH lame 8/10 at walk, 10/10 when trotting, RH muscle atrophy, no neurological signs, pain on right hind limb extension and lordosis testing, resentment of right L/S epaxial muscle pressure.	R L7-S1 foramen compression Soft tissue opacity effacing fat. Mild nerve enlargement at entrance, bony stenosis from S1 osteophytes on exit of foramen.	R L7-S1 lateral foraminotomy, nerve sheath biopsy (nerve was swollen at surgery) histopathology revealed inflammatory peri-neuritis	One week after surgery required additional analgesia (tramadol 50mg bid for 7 days) but the pain resolved after 2 days. At two weeks one episode of holding the right hind limb up which resolved without treatment. A 35-month post-op the dog was active and not lame, with no apparent pain. Mildly stiff in the morning, but warms out of it. Muscle atrophy no longer present.
#2	Stiff hind limb gait, reluctant to walk, pain on hind limb extension and lordosis, pain on L/S epaxial muscle pressure. No abnormalities on neurological examination.	Circumferential *spondylosis deformans* L7-S1. <50% protrusion of disc dorsally, foraminal compression and loss of perineural fat on extension. , bilateral L>R, middle and exit zone predominantly due to new bone formation, inactive discospondylitis	Dorsal S1 laminectomy and bilateral lateral foraminotomies	3-months post-op not improved at all, difficulty rising, has to be lifted, only walked very short distances, Weak but not painful, Strains to defecate and urinate and falls whilst doing so. Neurological examination revealed decreased patellar reflex and marked flexor reflex loss. Marked muscle atrophy of hind limbs. Repeat CT showed evidence of new bone formation associated with the foraminotomy sites but it did not appear to causing compression on the nerve roots, although minimal epidural fat was seen in the foramina. Suspected myelopathy or peripheral neuropathy. The dog was euthanased a month later. A post-mortem examination was not performed.
#3	2/10 left hind lameness, pain on lordosis and L/S epaxial muscle palpation. Neurological examination normal.	Calcified and protruding L7-S1 disc, left foraminal impingement	Dorsal L7-S1 laminectomy, disc fenestration and curettage, left L7-S1 lateral foramenotomy	31-month follow-up: very active, 1 hour walks 3 x a week, chases rabbits, jumps into vehicles and on beds fine. No further problems associated with the back any more. No evidence of pain, no medication required very successful outcome.

Table 25. continued

Dog Number	Clinical Signs	CT findings	Treatment	Outcome
#4	Pain on LS epaxial muscle pressure, lordosis and hip extension. Mild iliopsoas pain bilaterally. Neurological exam normal.	Ventral and lateral spondylosis and sacral wing osteophytes present causing foraminal narrowing. Loss of peri-neural L7 fat R>L. DLSS	Bilateral lateral foraminotomies	33-month follow-up, back to full health without back pain. Can jump into vehicles and not longer limited in his level of activity, no medication required. Took 8 months to fully recover, needed ongoing analgesia to start with, then swimming as physiotherapy. Owners consider him to be cured, very successful outcome.
#5	Hind limb gait crouched, more plantigrade than normal, LS epaxial pain, lordosis test painful, dribbling urine standing, normal conscious proprioceptive responses. Scuffs nails, patellar reflexes brisk to exaggerated, flexor reflexes decreased,[i.e. psuedohyperreflexia of patellar reflexes.	Transitional vertebral anomaly. Excessive dorsal sacral laminar overhang, sacral canal stenosis mild ventral displacement of the sacrum, disc degeneration with annular prolapse. DLSS	Dorsal S1 decompression, bilateral lateral foraminotomies	26-month follow-up. Returned to work 6 months after surgery. Another 2 months before considered fully recovered. Able to perform limited duties, part-time, limited number of jobs, but same type of work. Rated 9/10 for extent of recovery and work performance as the result of surgery cf. pre-op.
#6	Paraparesis, flaccid tail. Only mild pain on dorsal LS pressure and LS extension, decrease LS ROM. Severe muscle wasting of the hind limbs.	Transitional vertebral anomaly. L7 sacralised. Severe L6-7 disc prolapse. L6 nerve root compression in extension. DLSS	Dorsal laminectomy and foramenotomy of the entrance zones L6-7. Bilateral lateral foraminotomies for decompression of the middle and exit zones of L6-7.	23-month follow-up. Developed worsening paresis, unable to walk without assistance, intermittent faecal incontinence, no urinary incontinence, tail tone and movement. Conscious proprioceptive reflex deficits, crosses over then collapses. Euthanased. Post mortem examination inconclusive.
#7	Lower back pain, root signature right hind. Pain on tail jack/lordosis/hip extension. Slow conscious proprioceptive response right hind.	L7-S1 IVD degeneration with protrusion. Bilateral L7-S1 foraminal compression. Nerve root enlargement. Bony stenosis due to osteophytes within the exit zones of the foramina. DLSS	Dorsal L7-S1 laminectomy, disc fenestration and bilateral lateral foraminotomies	12-month follow-up. Excellent recovery, free of pain and returned to full function. At 15 months developed immune mediated disease and subsequently discospondylitis at L7-S1 diagnosed. Treated with antibiotics. At 26-months there was a mild recurrence of signs, a further CT was performed and a diagnosis of discospondylitis was made and antibiotics prescribed

Table 26. Volume of the L7-S1 lateral intervertebral foramen in seven dogs, prior to lateral foramenotomy, immediately post-foramenotomy and at follow-up (median 23 months), measured using 3-dimensional CT tissue management protocols. The lumbosacral junction was positioned in extension. The volumes are measured in mm^3 and have a measurement error of +/- 10%. Dog #6 had a transitional vertebral anomaly with sacralisation of L7. Disc protrusion was present at L6-7 whereas L7-S1 was unaffected.*

Dog # L left R right foramen	Pre-op vol. (mm^3) L7S1 foramen	Post-op vol. (mm^3) L7S1 foramen	Percentage increase in vol. (mm^3) L7S1 foramen Post-op	Length of 1st follow-up (months from initial CT)	Follow-up vol. (mm^3) L7S1 foramen	Percentage increase in vol. (mm^3) L7S1 foramen at follow-up	% of Post-op L7S1 foramen	Length of 2nd follow-up (months from initial CT)	Post-op vol. (mm^3) L7S1 foramen	% of Post-op L7S1 foramen
#1 R	106.6			35	133.6	125				
L	111.2*				102.2	92*				
#2 R	87.96			3	136.72	155				
L	48.04				240.62	501				
#3 R	38.9			33	64.26	165*				
L	24.88				88.42	355				
#4 R	47.36			36	125.84	266				
L	52.16				160.9	308				
#5 R	34.72	266.8	768	15	183.44	528	69			
L	33.52	218.8	653		203.16	606	93			
#6 R	308.8	1243.6	403	23	670.8	217	54			
L	220	839.2	381		578.2	263	69			
#7 R	66.8	531.2	795	14	310.8	465	59	26	244	46
L	81.4	544.8	669		412.2	506	76	26	395	73

** only a unilateral lateral foramentomy of the contralateral foramen was performed, not this foramen*

Immediate post-operative CT volumetric analysis of the L7-S1 lateral intervertebral neurovascular foramina indicated a median percentage increase in volume of 660% in extension achieved by foramenotomy (combined left and right foramen data). The increase in volume achieved by surgery ranged from 380 to 1200%.

Follow-up CT examination performed at a median of 23 months post-operatively indicated a median percentage increase in volume of 330% in extension (combined left and right foramenotomy data) compared to pre-operative foraminal volume. The persistent increase in volume compared to pre-operative volume ranged from 125 to 606%.

In the three dogs with both immediate post-operative and follow-up CT data the foraminal volume had reduced to a median of 69% of that achieved by surgery (range 54 to 93%). Stated another way the lateral intervertebral neurovascular foramen had remodelled by 7 to 47% (median 31% reduction in volume from that achieved immediately after surgery). In the one dog which had a second follow-up CT there was a further reduction of 13% of the neuroforaminal volume on the left and a 2% reduction on the right, from 14 to 26 months. The reduction in the neuroforaminal volume was due to bone regrowth at the edges of the foraminotomy, not fibrous scar tissue or synovial hypertrophy, as these tissues have opacities outside the acquisition window.

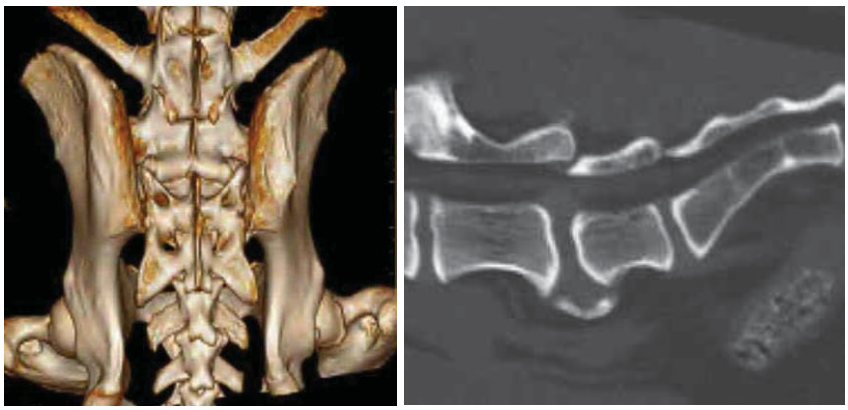
In all dogs the foraminal volume at follow-up was still larger than pre-operatively.

Clinical evaluation

Five out of seven dogs (71%) had successful outcomes (range 13 to 33 months follow-up). These dogs returned to either full, or near full, pre-disease degrees of activity. The Police GSD returned to work. The owners of the four pet dogs with successful outcomes commented on how active their dogs were, returning to activities such as jumping and running for extended periods, free of apparent pain or lameness. Two owners commented that the recovery time after surgery was long (6 and 8 months) before their dog fully returned to normal. Dog #3 recovered well initially then represented with pelvic limb stiffness and an apparent return of clinical signs. However physical examination revealed no signs referable to the LS junction and bilateral cranial cruciate rupture was diagnosed. The owners declined stifle stabilisation surgery in favour of weight loss and analgesia. The dog subsequently improved and despite stifle arthrosis became very active once more, without any signs that the owners perceived to be referable to the lumbosacral region. It was thus included in the successful outcome category. There were two dogs (29%) with poor outcomes despite seemingly effective decompression of the LS nerve roots. Dog #2 underwent uncomplicated surgery and was discharged from hospital at the same neurological grade as prior to

surgery. However the dog had deteriorated by three months post-operatively. The dog was less mobile although no pain was elicited when the LS junction was extended. There was marked atrophy of the pelvic limb muscles including both the quadriceps (femoral nerve) and stifle flexor muscle groups (sciatic nerve). A neurological examination revealed hyporeflexic patellar and poor flexor reflexes bilaterally. A repeat CT scan revealed some modelling of the foramenotomy site but the LS lateral intervertebral foraminal volume was still wider than pre-surgery and a fat signal was evident surrounding the *L7* nerve roots as they traversed the neurovascular foramen. The *L7* nerve root on one side was enlarged. The lower motor neuron signs localised to the femoral nerve indicated a cord lesion cranial to the LS junction. A second neurological condition was therefore likely however a lesion involving the *L4-6* cord segments was not seen on CT. Degenerative myelopathy was considered the prime differential diagnosis. The owners requested euthanasia of the dog without further investigation and a post-mortem examination was declined.

Dog #6 had progression of neurological signs following uncomplicated surgery. The LS junction was transitional with the transverse processes of L7 fused to the ilial wings, Figure 40. Prior to surgery the dog could walk but was paretic with marked conscious proprioceptive deficits in both pelvic limbs. The patellar myotactic reflexes were mildly exaggerated whereas the flexor myotactic reflexes were mildly reduced, suggesting pseudo-hyperreflexia of the patellar reflex rather than an upper motor neuron lesion to the femoral segment. Following surgery the dog required some assistance to stand and the owner reported he never regained the mobility he had before surgery. This dog had the longest duration of clinical signs prior to surgery (seven years since signs were first noticed at the age of 2 years) and the most severe neurological dysfunction of any dog in this study. Further imaging investigation was offered to determine if surgery had caused collapse of the intervertebral space or L7-S1 lateral foramen and to rule out vertebral neoplasia or discospondylitis, but the owners declined. *Figure 40.*



Reconstructed 3-D CT image (left) and sagittal CT (right) of the lumbosacral junction of dog #6. The transverse processes of L7 are attached to the ilial wings effectively making the L6-7 intervertebral disc space the LS junction.

At 23-months follow-up the dog was unable to walk without assistance and had intermittent faecal incontinence. Urinary incontinence was not present and tail tone and movement were considered normal. He was profoundly ataxic with severe proprioceptive deficits of both pelvic limbs. There was severe muscle atrophy and decreased patellar and flexor reflexes. On post-mortem CT there was no evidence of *cauda equina* compression and the lateral neuroforamina of the transitional LS junction were still effectively decompressed by the lateral foraminotomy performed two years earlier. Gross inspection ruled out other sites of compression, as well as spinal cord or vertebral neoplasia, and epidural empyema. Histopathology of the spinal cord at the lumbosacral intumescence showed no convincing evidence of myelin or axonal loss. Skeletal muscle sections exhibited marked and chronic atrophy with myofibre loss and extensive replacement by fat. These changes were considered non-specific but compatible with both disuse and denervation atrophy. A conclusive diagnosis was not made.

Discussion

Lateral foraminotomy, as performed for DLSS, is intended to decompress the nerve roots passing through the LS lateral intervertebral neurovascular foramen. Measuring foraminal volume using CT allowed the effect of lateral foraminotomy of the LS junction to be quantified. Immediate post-operative CT volumetric measurement in three dog (six foramina) confirmed that a lateral foraminotomy procedure increased the volume of the L7-S1 lateral intervertebral neurovascular foramen to greater than six times its initial volume. Regrowth of bone occurred at the foramenotomy site in all three dogs. Two of the three dogs had an excellent outcome and no recurrence of clinical signs was noted, indicating that bone regrowth at the site of the foramenotomy did not result in compression of *L7* nerve roots. A separate disease process was suspected of causing progressive neurological decline in the other dog.

In five of seven dogs foramenotomy was successful at relieving clinical signs and had a sustained effect. Re-evaluation of all seven dogs at a median of 23 months indicated persistence of the effects of decompression. The foraminal volume was on average three times greater than prior to surgery. Increased foraminal volume was present even in the three dogs with greater than 30-month follow-up. In the one dog that had a CT at both 14 and 26 months post-operatively there was a further reduction in foraminal volume between these scans but it represented only a quarter of the initial reduction from 0 to 12 months follow-up, suggesting that over time modelling of the foramenotomy site abated.

Lateral foramenotomy was an effective technique for decompression of the nerve roots at the LS junction. The majority of dogs had a successful outcome as subjectively assessed by the owner. Pain was resolved in all cases, as physically assessed by the author at six-month follow-up, and again at the time of final CT assessment.

The two dogs with a poor outcome were both suspected to have another cause of progressive neurological disease with signs that could not be attributed to DLSS alone. In previous studies in dogs undergoing dorsal laminectomy, more advanced neurological dysfunction prior to surgery, and increased age at onset of clinical signs were associated with a poorer post-operative outcome, while younger dogs and those with LS pain only tending to have a better prognosis for return to normal activity (De Riseo *et al.* 2001, Linn *et al.* 2003, Suwankong *et al*. 2008).

Traditional decompression of the LS junction by a dorsal laminectomy alone does not provide good access to the L7-S1 lateral intervertebral neurovascular foramen, which may result in the continuation of clinical signs in dogs with static or dynamic narrowing of the foramen. A facetectomy (*sic*), performed via a dorsal approach provides decompression of the L7-S1 lateral intervertebral neurovascular foramen (Tarvin and Prata 1980), but this technique is no longer recommended due to the risk of inducing instability of the LS junction (Smith *et al.* 2004, Godde and Steffen 2007). Compared with an intact cadaveric LS specimen, dorsal laminectomy combined with bilateral facetectomy decreased stiffness of the LS junction by 44% in extension, and 31% in flexion. The combination of dorsal laminectomy, facetectomy and discectomy decreased stiffness of the LS junction by 48% in extension and 57% in flexion (Smith *et al.* 2004). Enlargement on the entrance/middle zones of the foramen can be attempted by careful extension of the dorsal laminectomy beneath the caudal articular process of L7 (Chambers 1989). However access is limited and therefore a dorsal approach runs the risk of incomplete decompression of the middle and exit zones of the L7 foramen (Godde and Steffen 2007). In an experimental study a rigid arthroscope was used to aid entrance and middle zone enlargement via curettage after limited dorsal laminectomy (Wood et al. 2004). This technique does not provide access to the exit zone and to date has not been reported as effective in dogs with clinical signs of DLSS.

In order to access the exit zone, Godde and Steffen (2007) developed the lateral foramenotomy technique, which involves removing bone from the pedicle, directly over the lateral recess thus allowing decompression of the entrance, exit and middle zones of the neuroforamen. The technique of Godde and Steffen (2007) offers a means to decompress the foramen with less destabilisation of the L7-S1 articular process joints.

As stated in chapter seven, this novel technique of volumetric analysis of CT images has some limitations. Volumetric analysis using the chosen windowing pre-sets will

only delineate the bony perimeter of the foramen without the inclusion of any soft tissue structures. Hypertrophic soft tissues can also be responsible for nerve root compression therefore this method potentially over-estimates the *in-vivo* volume of each neuroforamen. Volumetric increase has a cubic relationship to changes in foraminal length and height achieved by surgery. The decompressive effect of foraminotomy is dependant on enlarging the narrowest point of the foramen rather than the overall increase in volume *per se.* For example a large amount of the lateral pedicle of L7 could be removed without breaking though into the lateral recess. The post-operative CT volume of the foramen would be measurably increased but the nerve pathway would remain compressed due to the remaining inner cortex of the pedicle.

The use of immediate post-operative CT provided feedback on the degree of decompression achieved, and improved the surgeons understanding of the relevant anatomy. The dog with the smallest increase in foraminal volume was dog #1, the author's first foramentomy. Performing an immediate post-operative CT scan is recommended for surgeons gaining experience with foramenotomy.

This study is the first to quantify the extent and effectiveness of the lateral foraminotomy technique at increasing the volume of the L7-S1 lateral intervertebral neurovascular foramen. In this small prospective case series, lateral LS junction foramenotomy achieved a successful outcome in 5/7 dogs and the remaining two dogs were suspected to have a second neurological disease. Lateral foramenotomy is a complex procedure that requires an understanding of the appearance of the exit zone. Bone regrowth did occur but the volume of the foramen 23 months post-operatively was still substantially greater than the pre-surgical volume. The author recommends that the foramenotomy be subjectively enlarged to a size that mitigates the effects of the regrowth of bone and still provide sufficient decompression for at least the medium term. Due to the immediate feedback it provides, the author recommends that surgeons performing lateral foraminotomy for the first time perform post-operative CT assessment to gain insight into their technique.

Biomechanical assessment of dorsal stabilisation of the lumbosacral junction in dogs and the development of a custom designed implant for dorsal stabilisation of the lumbosacral junction

Introduction

In chapter one, degenerative disease of the lumbosacral (LS) junction was a significant cause of early retirement in NZ Police GSDs. Based on the author's experience, the majority of NZ Police GSD's presented to the MUVTH with pain and/or neurological dysfunction localised to the LS junction are affected by degenerative lumbosacral stenosis (DLSS). Ideally, accurate diagnosis and effective therapy would allow a GSD with DLSS to return to active duty as a Police dog indefinitely. However, despite improvements in diagnostic imaging and a variety of surgical techniques having been proposed to manage DLSS, a recent review by an eminent veterinary neurologist commented that there has been little advance in our understanding or treatment of DLSS in the last three decades (Jeffery *et al.* 2014).

As outlined in previous chapters, there are two contrasting paradigms for surgical management of DLSS, these being decompression of the LS junction and distraction of the LS junction with stabilisation. Although dorsal decompressive surgery achieves a successful outcome in the majority of pet dogs, there is a relatively high recurrence rate and the result of this surgery in working dogs is less predictable (Danielsson and Sjöström 1999, DeRisio *et al* 2001). The high rate of recurrence after decompressive surgery of the LS junction has led to distraction/stabilisation being the preferred technique for surgical management of working dogs with DLSS by some surgeons. However, in distraction/stabilisation procedures, implant failure is a common complication (Hankin *et al.* 2012, Golini *et* al 2014). Loss of vertebral stability can result in recurrence of compression of the *cauda equina*. To date there have been no well-designed studies on which to base recommendations for the treatment of dogs with DLSS (Jeffery *et al.* 2014), and the outcomes of these two differing surgical approaches have not been critically compared in a cohort of working dogs with defined entry criteria.

The purpose of the final chapter of this thesis is to critically appraise the surgical techniques currently used to treat DLSS and to introduce a concept of therapy that will be part of the author's future ongoing studies; computer designed custom implants for dorsal stabilisation.

In their review, Jeffery *et al.* (2014) called for randomized comparative trials to evaluate therapies for DLSS, with rigorous case definition and objective outcome evaluation. Whilst chapters six to nine of this thesis may not fulfil these desirable criteria, they do provide a retrospective comparison of methods of surgical management of DLSS in working dogs and specifically evaluate the utility of annulectomy and lateral foramenotomy by undertaking volumetric analyses.

Dynamic imaging of the LS junction at the extreme ranges of motion has shown that the L7-S1 lateral intervertebral neurovascular foramen is narrowed in extension (chapter seven), an event that would not be prevented by dorsal decompression of the LS junction alone. In addition, dorsal decompression combined with a dorsal annulectomy of the L7-S1 intervertebral disc, as commonly recommended, may exacerbate dynamic narrowing of the lateral intervertebral neurovascular foramen (chapter eight). Although decompression of the L7-S1 lateral intervertebral foramen by lateral foraminotomy can be achieved with lasting effect (chapter nine) there are limited data on the effectiveness of lateral foraminotomy for DLSS in working dogs. Lateral foraminotomy is a difficult and time-consuming surgical procedure with a risk of injury to the *L7* nerve roots during burring of the L7 pedicle. In contrast, a dorsal stabilisation procedure is simpler and involves less muscle dissection reducing the duration of surgery and post-operative morbidity. In the authors experience NZ Police GSDs are examined early in the degenerative process and have dynamic foraminal narrowing in extension rather than static narrowing due to osteophyte development around the L7-S1 lateral intervertebral neurovascular foramina. Stabilisation of the LS junction should therefore be suitable to counter dynamic lateral intervertebral foraminal narrowing. However, in working dogs the existing methods of stabilisation of the LS junction have proven to result in weak vertebral stabilisation and are subject to early failure (see chapter six). Of the seven Police GSDs with DLSS treated by trans-articular screw fixation, six returned to full work but there were complications in five of those dogs and further surgical intervention was required in four. Screws broke in three dogs and in an additional dog the screws bent, suggesting that two stainless steel screws (3.5mm thread diameter (shaft diameter 2.5mm, cortical pitch) inserted across the L7-S1 articular process joints are not sufficiently durable in a working GSD.

Composite dorsal stabilisation of the LS junction in distraction has been recently reported (Beam *et al.* 2014). Multiple bone screws and pins were inserted in both L7 and S1 and bonded into one composite implant with polymethylmethylacrylate. By distracting the L7-S1 IVD space and stabilising the LS junction in a neutral position those authors sought to maintain the volume of the lateral intervertebral foramina. The published abstract reported a 2% major and 13% minor complication rate and a good

to excellent outcome in 81% of dogs with DLSS (n=51) according to responses to questionnaires sent to the owners of the dogs. This technique has the advantage of using multiple implants, which should increase the construct strength and decrease the risk of implant failure. However, a lasting bond between the metallic implants and the bone cement may not be consistently achieved. Composite dorsal stabilisation is non-standardised, time consuming and introduces a significant amount of foreign material on which a biofilm will form, increasing the risk of infection. Clinical studies with longer follow-up intervals are needed before this composite method of dorsal stabilisation of the LS junction can be recommended.

Iatrogenic nerve injury is a concern for all dorsal stabilisation procedures, as implants must be inserted without the ability to directly view the *cauda equina* (Smolders *et al.* 2012). In human medicine, posterior stabilisation of the vertebral column is mostly performed using pedicle fixation devices. The pedicle joins the dorsal arch of the lamina to its vertebral body and forms the lateral border of the vertebral canal. Dorsal fixation of the LS junction using the L7 pedicles and the body of the sacrum as the points of fixation has been investigated in dogs (Meheust *et al.* 2000). The width, orientation and length of the L7 pedicles were measured radiographically in twenty lumbar vertebrae from dogs weighing more than 20 kg to determine the ideal location of pedicle screws for dorsal stabilisation procedures. The results obtained indicated that the ideal entry point for a single implant in the L7 pedicle remained constant. The entry point of the screw into the L7 pedicle was determined to be the base of the cranial articular process. The entry point of the screw into the sacrum was the sloped plane of bone several millimetres caudal to the cranial articular process. Those authors advised that screws should be inserted perpendicular to the dorsal plane of the vertebral body. The main restriction was the width of the pedicle, which varies with the size of the dog. Meij *et al.* (2007) described the intra-operative landmarks for the insertion of an L7 pedicle screw as a vertical line tangential to the lateral border of the cranial articular process of L7 and a horizontal line bisecting the transverse process of L7. The entry point for an S1 screw was located at the intersection of a vertical line tangential to the caudal border of the cranial articular process of the sacrum, and a horizontal line midway between the caudal border of the cranial articular process of S1 and the cranial border of the intermediate sacral ridge, [Figure 41]. However, the latter description relies on consistent anatomy and dog breeds vary in their vertebral morphology. The length and width of the pedicle has not been described in a reference group of GSDs. Standard anatomical descriptions are also not helpful where an individual has a transitional LS vertebral segment.

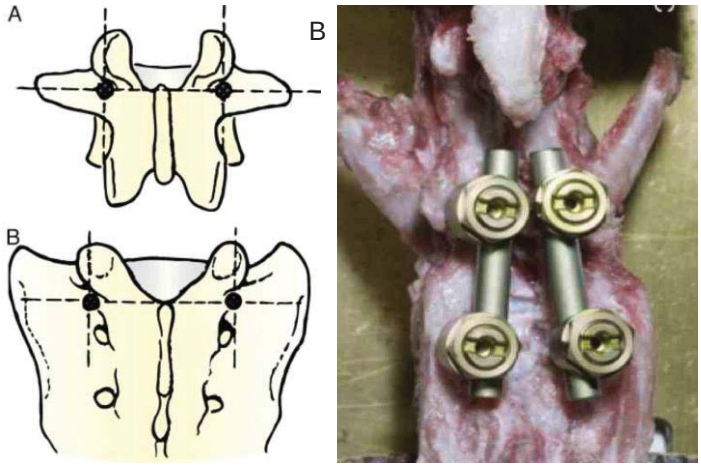


Figure 41 (Left). Schematic drawing of dorsal view of L7 (A) and the sacrum (B) with entry points (•) for pedicle screws. Figure 41 (right) Canine lumbosacral cadaver following 4.0mm titanium pedicle screw-rod fixation (USS Small Stature, Synthes, Zeist, the Netherlands).

Reproduced with permission from Meij, et al. (2007). Veterinary Surgery 36: 742-751.

Meheust (2000) was the first author to report the clinical use of pedicle screw fixation in dogs (n=5) with 'lumbosacral instability' using specialised medical devices. The technique consisted of four pedicle screws: two monoaxial screws located vertically in the L7 pedicles and two polyaxial screws located in the sacral pedicles parallel to the vertebral end-plate of the sacrum. Each L7 pedicle screw was connected to the ipsilateral sacral screw by a separate rod. At a minimum follow-up of six months, the dogs were pain-free with a normal gait and there were no complications resulting from the devices. Radiographs did not reveal any evidence of loosening of the implants. The use of specialised medical devices for dorsal stabilisation of the LS junction has since been investigated by others (LG Carpenter pers comm., Meij *et al.* 2007, Smolders *et al.* 2012).

Dr Larry Carpenter [Military Working Dog Veterinary Services] and Brett Taylor [Wilford Hall Medical Centre, San Antonio, TX] collaborated to perform decompression and spinal fusion on a working GSD using the Xia Spinal System [Stryker Howmedica] (pers comm.). This system (available in stainless steel and titanium alloy) uses polyaxial screws for treatment of degenerative and traumatic spinal collapse in humans. Titanium bars are placed between ipsilateral L7 and sacral screws that have been optimally placed through the pedicles without requiring them to be in the same vertical and horizontal plane (as is the case with monoaxial screws). The patented buttress thread closure mechanism then locks the screw to the 4.5mm rod, providing stability to the LS junction. The screws are available in 4.5 or 5.5 mm thread diameter, which is too large for all but the largest GSDs.

Meij *et al.* (2007) investigated the utility of 4.0mm titanium pedicle screw-rod fixation (USS Small Stature, Synthes, Zeist, the Netherlands) of the LS junction in 12 cadavers (Figure 2). They concluded that dorsal pedicle-screw fixation could be used to stabilise an unstable LS junction in dogs with DLSS. The same researchers have more recently

reported an 18-month evaluation of pedicle screw-rod fixation in three dogs with DLSS (Smolders *et al.* 2012). Prior to dorsal fixation, a dorsal laminectomy was performed and the *nucleus pulposus* on the L7-S1 intervertebral disc was evacuated using a motorised burr through a small incision in the dorsal *annulus fibrosus*. The disc space was packed with a mixture of autogenous cancellous bone graft and tricalcium phosphate. Post-mortem examination at 18 months post-operatively indicated that despite grafting, intervertebral fusion did not occur in any dog. Force plate data 18 months after surgery showed improvement in the pelvic to thoracic limb use ratio (increased propulsive force) in the three dogs treated.

Specialised medical devices are expensive NZD $2-3000 per dog for the implants, preventing their widespread use in canine patients. A more cost effective alternative to medical devices would be to use currently available veterinary implants such as the Synthes locking (LCP) or Orthomed UK String of Pearls (SOP) plates, provided suitable fixation and orientation of the implant can be obtained. Alternatively, a custom designed implant for each patient would solve many of the limitations of the devices in use.

Locking plates were developed to overcome the shortcomings of conventional plating systems used for fracture repair. Conventional dynamic compression plates (DCP, Sythnes, Switzerland) require that the plate is held compressed against the bone by the screw head due to the tightened pressure of the screw. The screw head is not locked to the plate and the screws are therefore said to be “non-angle stable”. Any loosening of the screw leads to loss of compression of the plate against the bone surface, which results in instability of the entire construct. Conventional plating requires the plate to be contoured to the surface of the bone to achieve the necessary contact with underlying bone. The complex anatomy of the dorsal LS junction precludes using conventional plates, due to the extensive contouring required. The Synthes locking plate (LCP$^{(TM)}$, Sythnes, Switzerland) was developed utilising the concepts of external skeletal fixation (ESF). The plate is placed adjacent to the bone but does not require compression against the bone surface since the screws are rigidly locked to the plate. By locking the screws to the plate, the screws become angle-stable and the construct then acts like an internal, ESF device. The LCP system allows the surgeon to perform less invasive procedures since accurate contouring of the plate is no longer required to ensure stability. The LCP was designed with a B-shaped screw hole that allows insertion of conventional or locking screws. Locking screws have a fine machine thread around a conical screw head that locate into a corresponding thread at the locking end of the B hole. The LCP screws are unique to the system and are approximately four times the cost of a conventional screw limiting the use the LCP system in veterinary

surgery. An additional limitation of the LCP is its conventional design, being ribbon shaped so that it can only be contoured in four degrees of freedom. It can be bent across its thickness or twisted around its long axis but it cannot be bent across its width.

The SOP locking plating system was specially designed for veterinarians and uses conventional screws. It comprises a series of non-deformable screw holes (pearls) connected by short internodes that have a circular cross-section. The cross-section of the internodes gives the implant a consistent stress profile along its length without concentrating stress at the screw holes, a weakness of conventionally designed plates. The SOP plate is stiffer and capable of sustaining greater static and cyclical loading than the equivalent LCP (Malenfant *et al*. 2014). Additionally, the circular profile of an SOP plate provides six degrees of freedom, allowing it to be bent in all planes and twisted along its long axis. The conventional screws provide angular stability by locking through a threaded section at the base of the pearl and by an interference fit between the screw head and the convex interior of the pearl. Conventional screws of high quality are required to meet the manufacturers' tolerances for the inner dimensions of the pearl but conventional screws are substantially less expensive than Synthes LCP locking screws.

The SOP system was introduced for fracture repair of long bones in cats and dogs but it has also been used to stabilise vertebral fractures (McKee *et al*. 2008). The use of the SOP system for fixation of a fracture involving the LS junction is now taught as part of advanced neurosurgical courses for veterinarians. Due to the proximity of the ilial wing it is difficult to apply plates to the lateral aspect of the body of L7, as is recommended for the thoraco-lumbar region. Insertion of screws through the pedicle of L7 dorsally is more practical but the surgeon must avoid penetrating the lateral recess. The surgeon must use precise vertebral landmarks to determine the entry point and angle of the drill, as there is only a narrow corridor within the pedicle for safe placement of the implant. In a study of six cadavers and three clinical cases there was a 33% incidence of unacceptable screw placement including penetration into the vertebral canal (Smolders *et al.* 2012).

The standard recommendation for stabilising a vertebral fracture using an SOP plate is for bilateral plates with two screws placed in the vertebrae on either side of the fracture (Karl Kraus pers comm.). Constructs using a single screw per plate per vertebra are less desirable due to the concentration of stress causing disruption of the bone supporting the screw. However, the spacing (12 mm) between adjacent pearls of the 3.5 mm SOP plate may exceed the usable length of the pedicle in GSDs, which prevents insertion of two screws per pedicle. The stiffness of dorsal fixation of the LS

junction using dual SOP plates with a single screw per pedicle has not been reported in the veterinary literature.

Following the high rate of failure of standard trans-articular screw fixation of the LS junction, the author used dual SOP locking plates with single screws per vertebra inserted into the pedicles of L7 and the sacral body (chapter six). Three working Police dogs with DLSS were treated by the application of dual SOP locking plates bridging the LS junction dorsally. The first dog had the articular processes of L7 removed to access the *L7* nerve roots, which precluded the use of trans-articular screws. In the second dog the SOP locking plate was applied for dorsal stabilisation with the articular processes intact and with the third dog the plates were augmented by the addition of trans-articular fixation screws through the articular processes. Despite the theoretical improvement in the strength of the fixation with the combination of the trans-articular screws and dual SOPs bridging the LS junction, the latter fixation failed due to breakage of multiple screws within six weeks of surgery.

Thus, as a finale to this study of DLSS and due to the variable results with use of SOP implants, the following four pilot studies were initiated, with the ultimate aim of fixation of the LS junction with a stable implant.

Pilot study 1: To document the dimensions of the L7 pedicle in GSDs to determine if the insertion of two 3.5mm screws, at the spacing of the pearls of a 3.5mm SOP plate, can be achieved without entering the vertebral canal or lateral recess of L7.

Pilot study 2: To investigate the mechanical properties (stiffness and yield strength) of dorsal fixation of the LS junction, using trans-articular screws, dual SOP constructs, and their combination.

Pilot study 3: To develop a custom-designed dorsal implant unique to each dog to overcome the known deficiencies of the implant systems currently available.

Pilot study 4: Application of a custom-designed dorsal LS junction implant in a dog with DLSS.

Pilot study 1:

Aim. To document the dimensions of the L7 pedicle in GSDs to determine if the insertion of two 3.5mm screws, at the spacing of the pearls of a 3.5mm SOP plate, can be achieved without entering the vertebral canal/lateral recess of L7 with part of the screw.

Materials and methods.

Data for this study was gained from CT scans previously obtained from GSDs with DLSS. These dogs were the affected cohort of NZ PDS GSDs described in chapter six. For each dog the length and width of the L7 pedicle was measured from transverse images of L7 using a digital measurement line tool [E-film] (Figure 42). Prior to measurements being taken a specimen of bone from an L7 vertebra of a GSD was used as a reference for selection of appropriate measurement points.

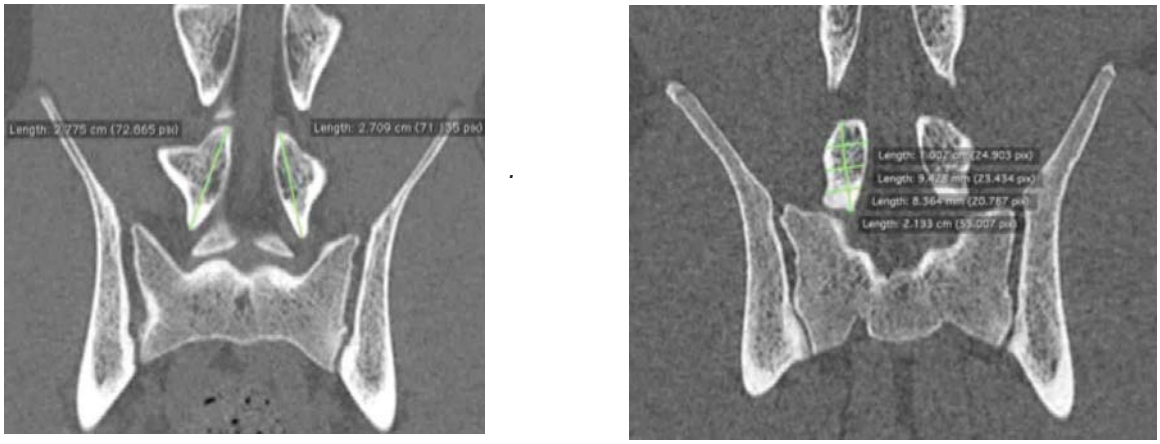


Figure 42. Transverse plane CT images of the lumbosacral junction of a German shepherd dog. The slice with the smallest pedicle length was subjectively chosen for measurement. The length (left) and width (right) of the pedicle were measured using E-film digital software. Each pedicle was measured three times using the line tool, by manually selecting the dorsal plane image with the smallest pedicle length.

The length of the pedicle was measured on the transverse CT slice with the shortest visible pedicle cross-section. The width of each pedicle was measured at three sites on a transverse slice of the pedicle (at its smallest subjective length) at the cranial, middle and caudal third. The length and width measurements were performed three times then averaged, Table 27.

Results

The mean lengths of the right and left L7 pedicles were 23.1mm, and 22.4mm respectively, with a pooled mean length of 22.7mm. The mean widths of the cranial and caudal thirds of the L7 pedicles were 7.7mm and 7.25mm respectively. The pooled pedicle width (including cranial, middle and caudal width measurements) was 8.3mm.

Table 27. Radiological measurements of the L7 pedicle performed on transverse plane CT images of the vertebrae of nine German shepherd Police dogs with degenerative lumbosacral stenosis.

	Right L7 pedicle length (mm)			**Left L7 pedicle length (mm)**			**Right L7 pedicle width (mm)**			**Left L7 pedicle width (mm)**		
Dog	Dorsal	Middle	Ventral	Dorsal	Middle	Ventral	Cranial	Middle	Caudal	Cranial	Middle	Caudal
1	21.4	21.8	21.4	20.0	20.5	20.1	7.8	11.0	7.1	7.3	12.3	6.8
2	21.9	20.8	21.4	23.5	22.9	21.8	8.8	9.6	7.9	7.6	10.5	7.1
3	22.7	23.4	22.1	19.3	19.6	19.3	7.2	9.5	7.6	7.3	9.4	7.2
4	23.6	23.7	22.8	22.0	22.6	22.5	6.1	8.4	5.9	6.2	8.9	5.0
5	25.1	26.3	27.9	23.7	24.8	24.2	7.8	10.2	6.3	7.2	9.8	5.5
6	23.2	23.9	23.1	23.4	24.2	22.5	7.2	10.8	7.8	7.3	12.7	7.4
7	21.2	21.7	22.0	23.3	23.3	23.4	8.6	11.2	6.3	8.5	8.8	7.9
8	26.0	26.2	25.7	25.1	24.8	25.8	7.7	10.2	7.3	8.5	10.7	6.9
9	21.7	22.1	21.8	19.9	20.9	20.4	8.6	9.6	6.9	8.6	10.7	7.9
Mean	**23.0**	**23.3**	**23.1**	**22.2**	**22.6**	**22.2**	**7.8**	**10.1**	**7.0**	**7.6**	**10.4**	**6.9**

Discussion

The 3.5mm SOP plate has a spacing of 12mm between each pearl centre. In a two-screw SOP pedicle construct the cranial screw would be located approximately at the site of the cranial width measurement and the caudal screw would be located approximately at the location of the caudal width measurement. As each screw is 3.5mm in diameter and the maximum width of the pedicle is approximately 7.5mm at these sites, and assuming there is no directional error during placement, the surgeon would have a margin of 2 mm of bone in either side of the ideal location. In addition, the pedicle is obliquely positioned in relation to the vertebral body. Therefore the SOP plate would have to be aligned along the cranio-caudal pedicle axis not the longitudinal axis of L7. The SOP plate would need to be bent to bring the caudal screw holes (that need to be located on the sacrum) into alignment. The narrow width and sloping angulation of the L7 pedicle increases the risk of the surgeon making an error and the drill bit entering the lateral recess of L7, or entering the L7-S1 lateral intervertebral neurovascular foramen, potentially injuring the *L7* nerve roots.

Pilot study 2:

The evaluation of medical devices for vertebral fixation involves testing their strength, fatigue characteristics, and stability when implanted. The strength test evaluates the failure load of the device, determines its weak points, and is helpful in the initial development of the device. Fatigue testing provides a measure of longevity of the device by testing the device to failure using cyclically varying loads. In contrast, the stability test measures the capability of the device to provide multi-directional stability to the injured vertebral column (Panjabi 1988). Force-displacement curves are used to compare material properties. Metal devices have an initial elastic phase, temporarily deforming without injury to the material (bounce back without permanent deformation). Once the elastic limit is reached the material deforms without application of significantly more force (plastic deformation) and is permanently deformed – the material has failed as a construct.

Aim. To investigate the stiffness and yield strength of dorsal fixation of the LS junction using either trans-articular screws, dual SOP constructs, and the combination of trans-articular screws, and dual SOP constructs.

Materials and Methods

The vertebral column of a 32kg, adult GSD bitch was dissected. The soft tissues were removed from the LS junction and the L7 and sacral vertebrae were disarticulated. The L7 and sacral bone specimens were imaged separately using transverse 1mm CT slices (bone algorithm with scan width: 1500 Hounsfield units, scan length: 500 Hounsfield units) [a]. The CT data was converted from DICOM to STL formats by Axia 3D Design (Napier NZ) using ScanIP[b] software. The STL data could then be manipulated with CopyCAD Pro design software[c]. Three-dimensional computer models were generated from the data in SolidWorks. The pedicle length of the bone specimens was measured and compared to the mean pedicle length obtained in the first pilot study. The computer model was scaled by 108% to equal the mean pedicle length of the dogs in the dataset in the first pilot study. Cylindrical extensions were added to the cranial endplate of the computer model of L7 and the caudal aspect of the computer model of the sacrum to act as locating pegs in a testing jig once the design was rendered in plastic.

[a] Phillips Brilliance, Philips Healthcare, Andover, MA, USA. [b] Simpleware Ltd, Exeter, UK

[c] Delcam PLC, Birmingham, UK

The dorsal surface of each peg was designed with a flat surface to allow application of a locking clamp. Nine identical stereo-lithographic models of the two vertebrae were then produced in acryl-nitrile butadiene styrene (ABS) plastic using a Fuse Deposition Modelling 3-Dimensional printer[d], Figure 43.

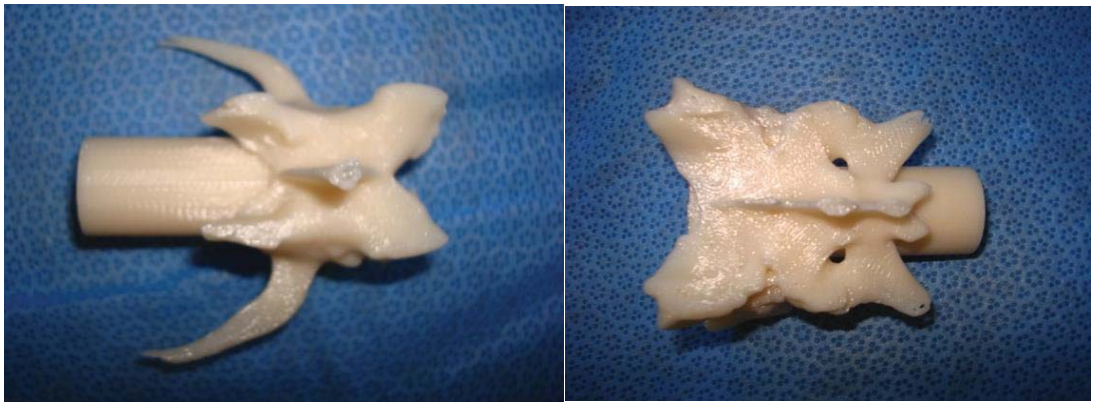


Figure 43. Acryl-nitrile butadiene styrene plastic renditions of L7 (left) and the sacrum (right) of a German shepherd dog created from CT data using a Fuse Deposition Modelling 3-Dimensional printer.

Each LS junction unit was prepared for mechanical testing by clamping the two separate rendered plastic L7 and sacral vertebrae into a custom-made wooden jig to maintain a neutral LS angle and replicate normal intervertebral spacing. Silicone sealant was injected between the vertebral bodies and articular processes to mimic the intervertebral discs and the joint capsule respectively. Each LS junction motion unit was left locked in the jig until the silicon had cured. Nine 'elastically-articulated LS junction units' were thus created.

To ensure consistency of application of the dorsal stabilisation, each model was stabilised whilst locked into the wooden jig. The nine ABS vertebral LS junction units were stabilised in three groups of three according to the method of dorsal stabilisation used for each. Group 1 was stabilised with bilateral trans-articular screws (2 x 3.5mm 316L stainless steel cortical screws, 38mm long, Orthomed UK). Group 2 was stabilised with dual SOP constructs (bilateral 3-hole sections of 3.5mm SOP plate with one L7 pedicle screw and one sacral screw per plate, Orthomed UK) with the open hole located over the LS junction. Group 3 was stabilised with a combination of trans-articular screws and dual SOP plates (Group 1 plus Group 2 fixation). The landmarks for pedicle and trans-articular screw placement were consistent between models. Once the fixation was complete the model was removed from the wooden jig.

[d] Dimension Elite, Dimension, Inc, Eden Prairie, MN, USA.

The models were then tested in a materials testing machine (Instron) at the School of Engineering, Massey University. A steel jig was fabricated to hold the LS junction units in the testing rig beneath the actuator of the Instron to which a load cell had been fitted. Load cells measure the force experienced by the specimen under test that give an electrical signal output used to precisely monitor, report and control the applied force. The sacral peg of the model was clamped to an upright support on the steel jig. The jig was adjusted so the peg of L7 lay beneath the load sensor and then the jig's base plate was clamped to the Instron to prevent motion. The load sensor was fitted with a rod and bracket system such that upward motion of the testing rig translated into vertical tension on the peg. The ventral surface of the L7 peg was modified by filing a dome shaped channel in which to locate the actuator of the load-testing device. The channel in the L7 peg prevented the testing jig pulling off the peg as the LS extension angle increased. With the sacral peg clamped to the test unit, tension on the L7 peg was translated into cantilever bending of the LS junction mimicking an extension force applied to the LS junction, Figure 44. Extension was chosen as the test condition because reduction of foraminal volume during extension is most relevant to DLSS in dogs as shown in chapters six and seven. The displacement of the test rig was plotted on a force-displacement curve against the tensile force generated in the load cell. Each construct model was tested to the point of plastic deformation (as determined by a plateau in the force-displacement curve) by application of force in tension at 10mm per minute. Stiffness is the ratio of the force required to create a specified deflection or movement of a part. Stiffness is the ratio of force to displacement, and is expressed in N/m. During testing the models were observed for any indication of structural failure and one model under test from each group was continuously recorded using a digital camera. After testing, the plates and screws were removed from the models for inspection and recording of the nature of any observable failure of the implants or plastic models.

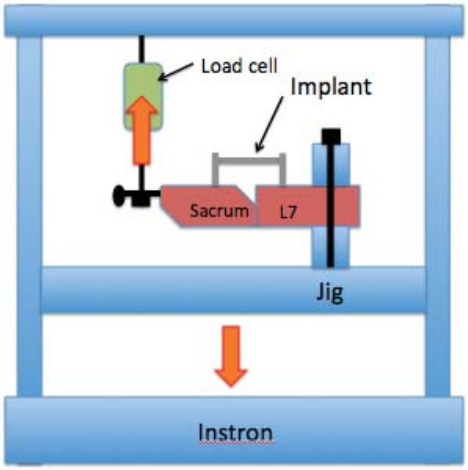


Figure 44. Diagram of the testing jig for lumbosacral junction units printed in plastic and mounted in an Instron materials testing machine. The sacral segment of the unit was clamped to a stainless steel jig, which was clamped to the actuating arm of the Intron beneath the load cell. The load cell was connected to L7 via an arm that could pivot on the L7 unit peg.

Results

Mechanical data

The data from the three models within each group were averaged. The force-displacement data is shown in Table 28. Trans-articular screw fixation (Group 1) was marginally stiffer than dual SOP (Group 2) [mean 159N versus 132N peak force before plastic deformation respectively]. Group 2 constructs allowed a much larger maximal elastic deformation of the LSJ model compared to Group 1 [38mm cf. 21mm]. As this displacement was measured from the Intron crossbeam, it is non-linearly related to the angulation of the LS junction. The combination in Group 3 of trans-articular screws and dual SOP pedicle plates was markedly stiffer than either method alone (289 N peak load before plastic deformation). A representative force:displacement curve from each group is depicted in Figures 45, 46 and 47.

Table 28. Mechanical test data on a single bending-to-deformation trial for dorsal stabilisation of acryl-nitrile butadiene styrene plastic lumbosacral junction constructs. Group 1 had trans-articular screws alone. Group 2 had dual SOP plates alone. Group 3 had combined dual SOP and trans-articular screw fixation. Three models were tested in each Group.

	Peak force (N)	Max displacement (mm)	Force at 3cm displacement (N)
Group 1	140.6	23.6	69.4
	152.2	19.9	83.5
	184.6	21.5	47.9
Group 2	129.5	35.6	56.9
	137.1	44.0	45.3
	130.5	35.0	43.9
Group 3	322.5	43.9	262.6
	235.9	30.1	230.0
	308.5	37.0	216.8

Mode of failure

Group 1 constructs failed by bending of the trans-articular screws. Group 2 constructs failed by bending of the two sacral screws. The screws bent in the open section between the plate and the dorsal surface of the sacrum. Groups 3 constructs failed by bending of the sacral screws in the section of screw unsupported by bone. In addition one model in Group 3 failed when the articular process fractured. The SOP plates were not visibly deformed in any test (Groups 2 and 3), which indicates that the screws are

weak when unsupported by bone but the plate is sufficiently rigid to avoid plastic deformation. Due to the dorsal projection of the L7 and S1 articular processes, it is not possible to place the SOP plates adjacent to the sacrum, a position that leads to several millimetres of the screw being unsupported by bone or the plate. Thus the screws' elastic limit is exceeded and stabilisation of the LS junction fails due to plastic deformation of the screws without deformation of the plates. Whilst the plates themselves may be sufficiently rigid to stabilise the LS junction this pilot study suggests that the section of unsupported screw is the weak point that causes failure of SOP LS stabilisation in dogs with DLSS. In this pilot study a single static force-to-failure was tested and the test load may have exceeded physiological forces. To the authors knowledge the natural forces applied through the LS junction have not been measured *in-vivo*.

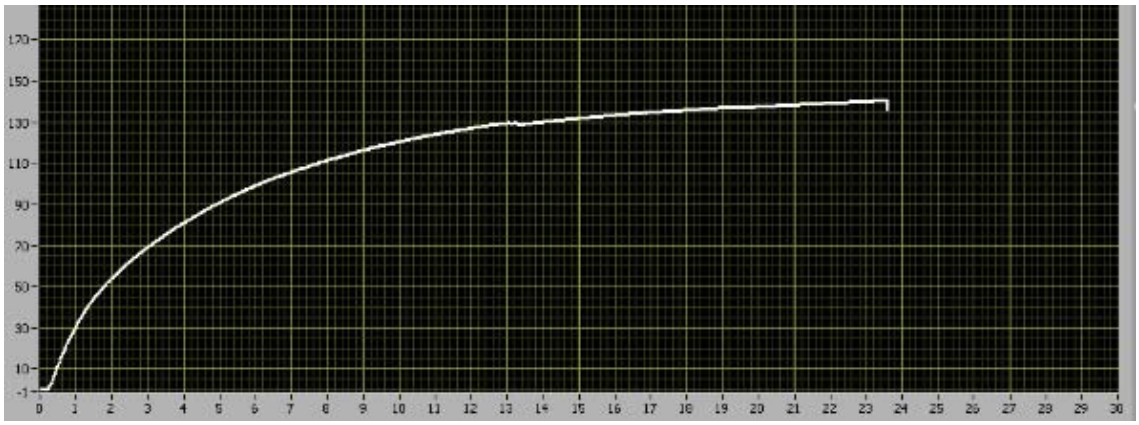


Figure 45. Force-displacement curve of a model from group 1. (Trans-articular screw fixation only). The x-axis is graduated in millimetres of displacement. The y-axis is graduated in Newtons of force.

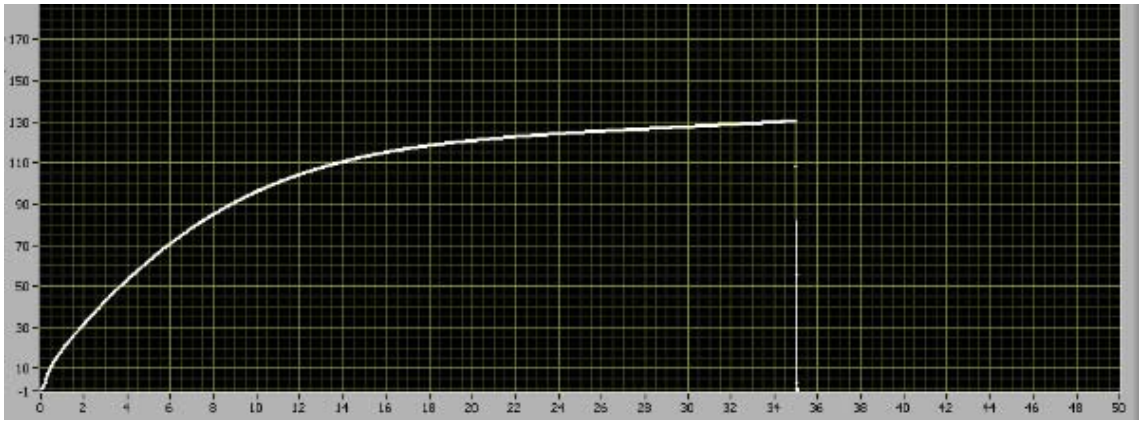


Figure 46. Force-displacement curve of a model from group 2. (Bilateral SOP stabilisation only). The x-axis is graduated in millimetres of displacement. The y-axis is graduated in Newtons of force.

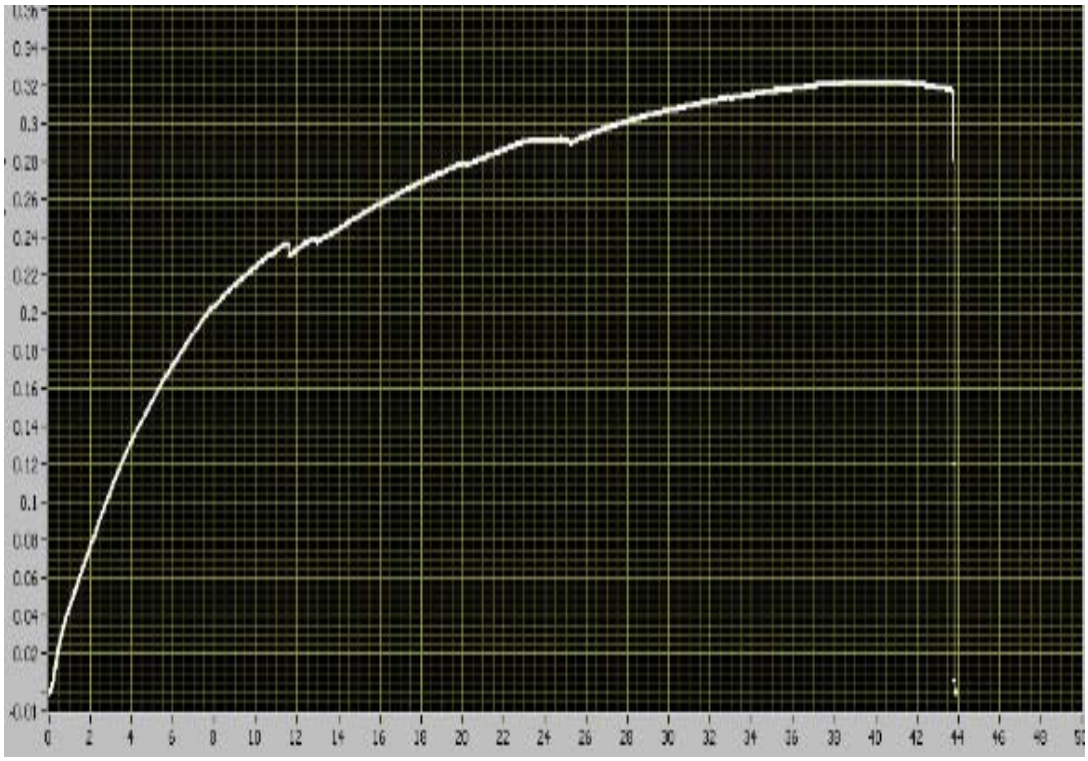


Figure 47. Force-displacement curve of a model from group 3. (Trans-articular screw fixation combined with bilateral SOP stabilisation). The x-axis is graduated in millimetres of displacement. The y-axis is graduated in kilo Newtons of force.

Pilot study 3:

Aim: To develop a custom-designed dorsal implant unique to each dog to overcome the known deficiencies of the implant systems currently available.

The mechanical test in the second pilot study indicated that a combination of trans-articular screws and dual SOP plates is twice as stiff compared to trans-articular screw fixation or SOP plate fixation alone as a method of dorsal stabilisation of the LS junction. However, in the single use of this technique in a clinical case the trans-articular screws and the sacral screws were all broken six weeks post-operatively and stabilisation of the LS junction was not achieved, (chapter six.) In clinical cases the implants are subjected to cyclic loading during activity, which over thousands of cycles, would be expected to result in failure if those forces are close to the elastic limit of the implant material. The length of unprotected sacral screw spanning the gap between the plate and the sacrum likely contributes to failure.

The following were seen as potential improvements to the design of an implant for dorsal stabilisation of the LS junction.

i) the design should eliminate the length of screw not surrounded by bone or implant by extending in a turret to the bone surface
ii) the implant must be applied with minimum disruption to the epaxial muscles
iii) the implant should provide a mechanism for directing the drill position to avoid the drill or screws entering the vertebral canal or lateral recess and injuring the nerve roots.

In human surgery there are sophisticated computer assisted surgical systems that use fiducial markers. A fiducial marker is an object placed in the field of view of an imaging system that appears in the image produced, for use as a point of reference during surgery. These systems employ 3-dimensional optical arrays that allow the surgeon to map the location of a surgical tool to the patient's anatomy based on previously obtained CT or MRI data and "registering" the tool to known anatomical landmarks. The expense of such a system currently precludes their use at the MUVTH. An alternative approach to fiduciary systems would be to custom design an implant that acts as its own drill jig, thus allowing safe insertion of screws at pre-determined sites based on knowledge of the dog's individual anatomy.

The previously described 3-dimensional LS computer model (Pilot study 2) was used as the subject for a custom designed LS dorsal stabilisation implant.

The computer model of the LS junction was set at a neutral angle. Using SolidWorks, an implant was designed which consisted of eight screw “turrets” fused into one implant that was fitted dorsally to the pedicle of L7, the L7-S1 articulation and the sacrum (Axia 3-D Design, Napier). Screw positions were determined to optimise the depth of bone purchase without penetrating the lateral recess of L7, L7-S1 intervertebral foramina or sacral canal. The implant was designed for two screws in the L7 pedicle per side, one trans-articular screw per side, and one screw in the body of the sacrum per side. The construct was designed to hug the contours of L7 and S1 with the ‘turrets’ for each screw position having different length and orientation protecting the entire length of screw external to the bone. After the first prototype plate was generated in ABS plastic using a rapid prototyping process, detailed changes were made to the contour of the implant and a second reinforcing rib was added, connecting both sides at the caudal end of the implant. Once design changes had been finalised the CAD file was sent to TIDA (the Titanium Industry Development Association, Tauranga, NZ) for manufacture. Using laser sintering, the custom implant was formed from titanium powder in a rapid prototype printer. The custom implant was mounted on the ABS plastic LSJ model as described in Pilot study 2. The implant was fixed in position with eight stainless steel standard 3.5mm cortical screws, Figure 48. Each turret acted as a drill guide for the correct angle of screw insertion after insertion of a Synthes 2.5/3.5mm drill guide. The guide extended the working length of the turret to ensure accurate concentric alignment of each drill hole. Once the custom implant was screwed to the ABS plastic LS junction model, the custom construct was stressed using the same testing conditions as described in the second pilot study, namely a single test to failure in extension.

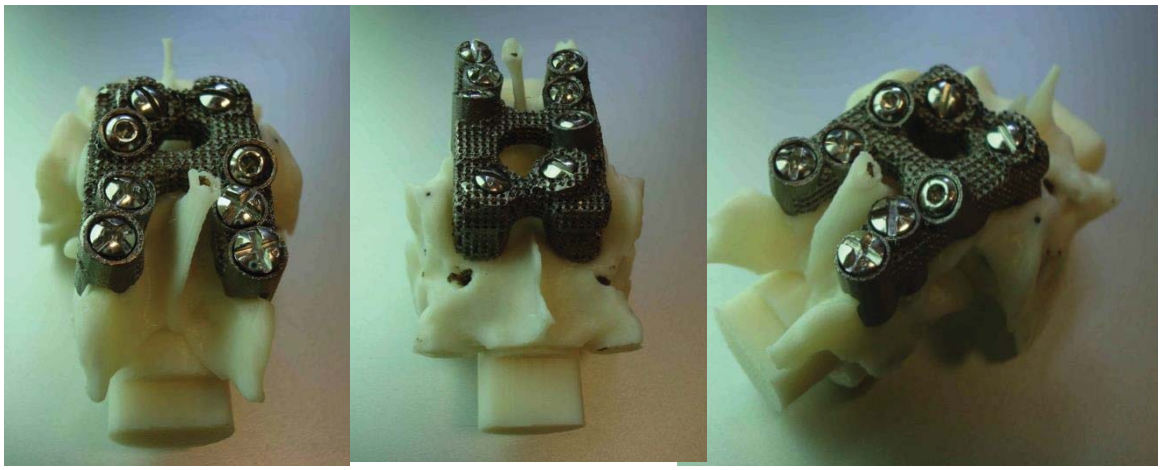
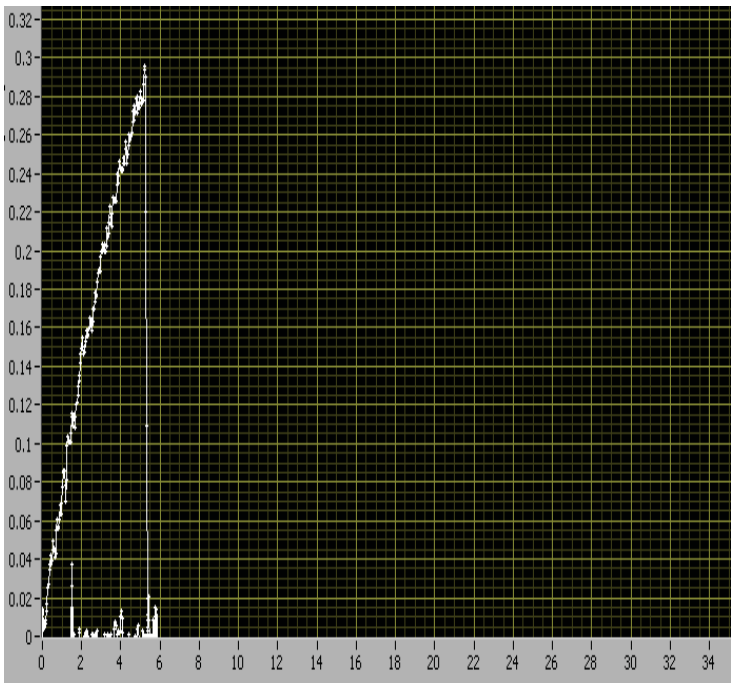


Figure 48. Computer designed titanium implant mounted on acryl-nitrile butadiene styrene plastic renditions of L7 and the sacrum of a GSD.

The angle of the load displacement curve, Figure 49, indicated greater implant stiffness compared to the other three groups previously tested. At 294N the plastic LS junction model failed with complete fracture of the sacral peg at the point where the clamp constrained the model. The plastic model failed before the titanium implant reached its elastic limit as shown by the slope angle of the force-displacement curve.

In Groups 1 to 3, the constructs failed by deformation of the screws. Screw failure did not occur with the titanium implant. The video recording showed no appreciable bending of the titanium implant itself (visual assessment), whereas the plastic pegs of the L7 and S1 models bent, which was likely responsible for the majority of the recorded displacement of the titanium implant/LS model (5.2mm). For the titanium implant the force required to reach a 3cm vertical displacement was 3-4 times higher than the equivalent force recorded for Groups 1 and 2 in Pilot study 2. For the titanium implant the force required to reach a 3cm vertical displacement was 50% higher than the combination of trans-articular screw fixation and dual SOP plates dorsally.

Figure 49.
Force-displacement curve of a stabilised LS junction model with a custom made titanium implant. The x-axis is graduated in millimetres of displacement. The y-axis is graduated in Newtons of force



Pilot Study 4:

Clinical application of a custom-designed titanium LS junction implant in a dog

A 41.5 kg 18-month-old male Akita was admitted to the MUVTH with a mild pain response upon pressure applied over the LS junction, and resentment to lordosis testing. Dynamic CT was undertaken using the protocol described in chapter seven. Significant narrowing of the L7-S1 lateral intervertebral neurovascular foramina was present on extension with effacement of peri-neural fat and prolapse of the L7-S1 dorsal disk annulus. A diagnosis of degenerative lumbosacral stenosis was made. A conservative approach with rest and anti-inflammatory medication was unsuccessful and surgical management was recommended. A titanium implant was designed based on the prototype tested in Pilot study 3. Due to the dog's narrow pedicles the implant was designed for 2.7mm screws, Figure 50. Stainless steel screws were utilised since titanium screws of suitable length were unavailable.

Figure 50. Computer generated 3-dimensional model of the LS junction of a dog with DLSS. The custom designed implant for dorsal stabilisation is in purple, the sacrum is in brown and L7 is in grey. On the right the implant has been rendered into a porous titanium scaffold with screw turrets that guide screw application in pedicles of L7 and the sacrum.

The dog was positioned in sternal recumbency with the hind limbs flexed and supported beside the trunk. A midline skin incision was made from the spinous process of L6 to the S3-Cd vertebral junction. The spinous ligaments and the epaxial muscles were separated in the midline and the LS junction was exposed. The interarcuate ligament was removed and the LS vertebral canal was inspected for possible disc protrusion. The dorsal annulus was left intact. The custom implant was fitted to the vertebral body of L7 and the degree of extension of the LS junction was adjusted to fit

the angle of the design. A drill guide was placed in the first screw hole, which corresponded to the cranial screw that was to pass into the right pedicle of L7. A 2mm bit was drilled through the pedicle. The hole was measured for depth and the length of screw required. A 2.7mm screw was inserted and tightened, ensuring that the implant was drawn tightly onto the surface of the bone. The corresponding screw position on the left pedicle was drilled and measured and similarly fixed with a 2.7mm screw. The trans-articular screws were then inserted, followed by the sacral body screws and finally the proximal L7 pedicle screws, Figure 51. The lumbodorsal fascia was reopposed with 2/0 USP Polydioxanone (PDS, Johnson and Johnson) and the subcutaneous tissues and skin were sutured. Post-operative radiographs showed good alignment of the LS junction. The patient recovered uneventfully and was discharged from the hospital 48 hours of the operation.

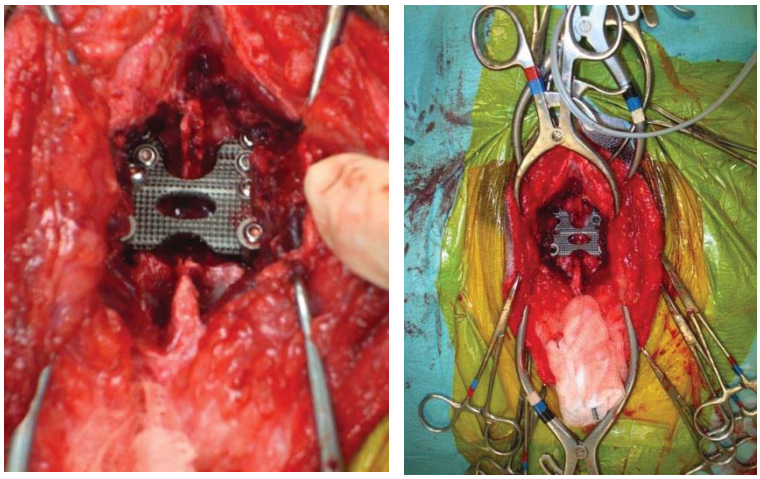


Figure 51. Intra-operative images of the application of a novel computer-designed titanium implant to the LS junction of an eighteen month old Akita with DLSS.

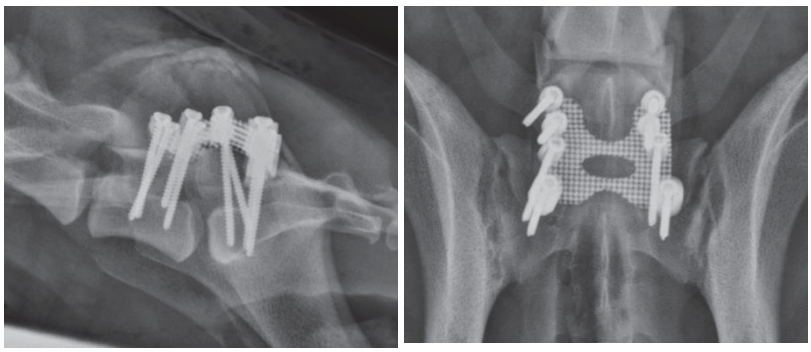


Figure 52. Post-operative radiographs following application of a novel computer-designed titanium implant to the LS junction of an eighteen month old Akita with DLSS.

A small seroma developed at the incision site but did not require drainage. At the 6-week follow-up visit, the owner reported that the dog was very keen to exercise, but was still lame intermittently. Radiographs showed failure of one of the sacral screws at 8 weeks but the titanium implant was stable and in position. There was no pain on manipulation of the LS junction and neurological deficits were not apparent. At 12-week follow-up the owner reported the dog was sound and without any signs of pain from the LS junction.

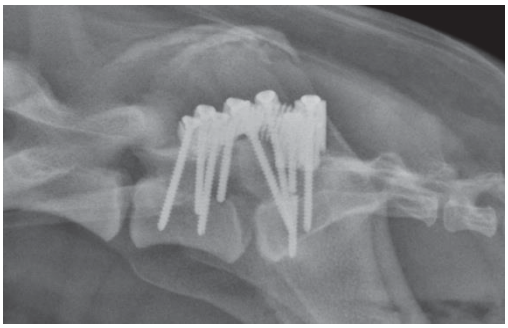


Figure 53. Six week post-operative radiographs following application of a novel computer-designed titanium implant to the LS junction of an eighteen month old Akita with DLSS. There is fracture of one of the screws inserted into the sacrum. The plate is unchanged in position and the dog was improving despite this complication.

The surgical procedure itself was straightforward. Accurate positioning of the implant is critical in order that the turrets correctly determine the intended position of the screw holes. Intra-operative reference to 3D images of the implant in position is useful to determine important landmarks. The implant should be correctly designed to accept a drill sleeve to extend the length of the turret. A long sleeve is more accurate at aligning the drill hole with the turret. The screw that failed was drilled free hand as inaccuracy in the manufacturing tolerance of the turret internal diameter prevented the use of a drill sleeve at that location. Similarly the right cranial L7 pedicle screw was drilled free hand and was slightly mal-aligned. The screw that failed was likely subjected to eccentric loading due to the disparity between the axis of the sacral drill hole and the turret itself, brought about by the free-hand drilling.

This method of dorsal stabilisation of the LS junction appears to have merit and warrants further development. A prospective clinical trial has been initiated using implants modified to accept a drill guide and accept titanium 2.7mm and 3.5mm screws. Further improvements may include custom designed screws of greater size and tapering girth to lessen the risk of failure at the bone-screw interface.

Summary

Working German shepherd dogs (GSDs) play a vital role in law enforcement in New Zealand (NZ). This thesis investigated the predominant causes of death or early retirement of GSDs working for the NZ Police Dog Section (PDS). Both osteoarthritis of the coxo-femoral joints and degeneration of the L7-S1 intervertebral disc with resultant lumbosacral stenosis were major causes of early retirement because of the dogs' inability to meet the physical demands required by the PDS.

Dysplasia of the coxo-femoral joints in dogs (CHD) is a multi-factorial trait with genetic and environmental influences. Radiographic screening of the coxo-femoral joints in order to select dogs for breeding that have a more favourable hip phenotype, has traditionally been based on an extended ventro-dorsal hip radiograph. In chapter three analysis of the hip score data held by the NZ Veterinary Association revealed minimal improvement in the total hip score of German shepherd dogs when analysed by year of birth. The median total hip score improved from a total NZVA score of 8 to 7 over 17 years, an improvement of 12%, or 0.73% per year. The NZVA scoring scheme, based on that of the BVA, uses a scale from 0 to a maximum of 106, therefore a change of one total score unit in 17 years as stated, represents only a 0.05% change (as a proportion of score range) per year.

Date of birth explained just 1.5% of the variation in total score according to the statistical model. By implication, the improvement in the median NZVA total score was predominantly due to effects other than selection pressure. However, the chosen method of analysis (logistic regression) may have underestimated improvements in the NZVA CHD phenotype for an individual dog. Genetic trend (the effectiveness of the selection differential) can be better determined by estimated breeding value analysis. Estimated breeding value analysis of the NZVA CHD database in a subsequent study published outside this thesis revealed a statistically significant trend towards a lowering of the NZVA total CHD score in the German shepherd breed, but the magnitude of the improvement was small (0.13 total hip score units per year) despite the trait demonstrating moderate heritability (Soo and Worth 2015).

Breeders lack of compulsion to radiograph and score all dogs used for breeding, a closed database, and pre-screening are all potential reasons for the lower than expected rate of improvement in the NZVA CHD score data. During the same period, more substantial improvement had been achieved in reducing the prevalence of Elbow dysplasia (Worth *et al.* 2010). Given that the aforementioned deficiencies also apply to the NZVA Elbow Dysplasia (ED) scheme, the radiographic screening for ED would

appear to be based on a more accurate assessment of the presence of dysplasia than the NZVA CHD scheme. Positioning a dog for an extended ventro-dorsal radiograph of the pelvis results in a tightening of the passive constraints of the coxo-femoral joint, reducing coxo-femoral joint subluxation in dogs affected by CHD. Under-estimation of coxo-femoral joint subluxation will therefore reduce the potential genetic gain from selection and may explain the slow improvement documented for CHD in NZ GSDs using the NZVA scheme.

Distraction radiography using the PennHIP method was introduced to the NZ Police Dog Section with the intent of improving the effect of selection against CHD. Distraction radiography objectively measures passive hip laxity, which is strongly predictive of the development of hip dysplasia in dogs. Correlation between the distraction index (DI) and the NZVA total and subtotal scores in NZ Police GSDs found only low agreement between total NZVA score and DI (Kappa value 0.31) but moderate agreement between the subtotal NZVA score and DI (Kappa value 0.55). The poor correlation between the NZVA total scores is of concern as ranking dogs by each method gave disparate results. There was evidence from this study that using a subtotal NZVA score moderately improved agreement between the NZVA scheme scores and the PennHIP DI, but only at a higher threshold of DI.

The poor correlation between an NZVA and a PennHIP score in the same dog precludes these two different scoring systems being used interchangeably to guide breeding decisions regarding hip dysplasia. As a result of this study, (Worth *et al.* 2009), the NZ Police Dog section abandoned the NZVA HD scoring system and now exclusively utilises the PennHIP system for providing phenotypic data related to hip dysplasia in potential breeding animals. Future work will involve determining the heritability and genetic trend for the PennHIP distraction index in GSDs of the NZPDS using estimated breeding value analysis.

Degenerative lumbosacral stenosis (DLSS) was examined in the second part of this thesis as a leading cause of loss or retirement of NZ Police dogs. A cohort of working GSDs with DLSS treated surgically at the MUVTH underwent retrospective assessment. Fifteen of sixteen dogs returned to service as working dogs, twelve of which were capable of full duties. In the eight dogs that completed follow-up the median length of service after surgery for DLSS was 2.6 years. The use of multiple techniques [dorsal or lateral decompression, stabilisation, or both] resulted in small numbers of dogs in each group, preventing statistical analysis. A number of deficiencies were noted in the existing surgical techniques, which were then investigated with a series of experiments.

The extent of dynamic narrowing of the L7-S1 lateral intervertebral neurovascular foramen during LS junction extension is an important factor in the pathogenesis of DLSS and was investigated using a volumetric analysis of CT images. Positioning a dog with the LS junction in extension mimics the position of the LS junction during activity and significantly decreases the volume of the L7-S1 lateral intervertebral neurovascular foramen. Greyhounds unaffected by DLSS had a smaller reduction in foraminal volume at full LS extension than GSDs. GSDs with DLSS had greater dynamic narrowing of the L7-S1 lateral intervertebral neurovascular foramen than GSDs without evidence of DLSS. When making treatment decisions about DLSS in dogs a veterinarian should consider the degree and nature (static or dynamic) of foraminal narrowing as indicated by dynamic CT studies of the LS junction. Stabilisation of the LS junction in a neutral to flexed position should be an effective form of therapy for DLSS associated with dynamic foraminal narrowing. A threshold value of <90m3 was proposed for predicting dogs with DLSS from foraminal data. Though it demonstrated a positive predictive value of 75% and a negative predictive value of 86%, a larger, blinded, prospective study is required to validate this finding.

In chapter eight a cadaveric model revealed that a dorsal annulectomy with partial discectomy could induce further dynamic reduction of the L7-S1 lateral intervertebral neurovascular foraminal volume. Post-operatively the resultant narrowing of the L7-S1 lateral intervertebral neurovascular foramen may lead to persistence or recurrence of clinical signs of nerve root compression. These findings suggest that annulectomy may contribute to the recurrence of nerve root pain and neurological dysfunction following surgical management for DLSS unless concurrent stabilisation of the LS junction is performed.

Lateral foraminotomy is an alternative technique that has been advocated for surgical treatment of DLSS. A prospective evaluation in a small cohort of dogs confirmed effective enlargement of the L7-S1 lateral intervertebral neurovascular foraminal volume, but showed that by 12-months post-operatively there was bone regrowth partially attenuating the effect (chapter nine). Further evaluation of the efficacy of this procedure in a larger number of working Police dogs is required before recommending lateral foraminotomy as the definitive treatment of DLSS, however it does hold promise as an effective treatment for static foraminal narrowing.

Finally, the reportedly high recurrence rate of clinical signs of DLSS following dorsal decompressive techniques and the high failure rate of implants used for dorsal stabilisation techniques led to the development of a novel method of dorsal stabilisation through a series of pilot studies. By utilising CT reconstruction a custom implant was

designed by computer using engineering software and printed in titanium using rapid prototyping technology. The implant utilised dual pedicle screw fixation in L7, a trans-articular screw and a sacral body screw on each side of midline. This is the first time that CT, CAD design and rapid prototyping technologies using titanium have been linked together to manufacture a custom implant for spinal surgery in dogs. According to the author's knowledge, this approach to spinal surgery is unknown in the medical literature

Cantilever bending data showed the custom implant to be stiffer than the other methods of dorsal stabilisation tested. Initial use in one dog with DLSS demonstrated that the implant could be successfully placed and was effective at negating the signs of DLSS in the dog at 12-week follow-up. Further clinical trials are now needed to refine the shape and structure of the implant and test the efficacy of custom designed implants in multiple dogs. It is possible that this novel implant represents a significant step forward in surgical management of DLSS. This approach could provide greater safety during implant positioning and reliable maintenance of the lateral intervertebral neurovascular foraminal volume in working dogs with DLSS. Clinical trials will be required to test the long-term durability of the implant and the potential risk of a domino lesion, i.e. degeneration of the L6-7 intervertebral disc that could lead to recurrent back pain.

The collective publications emanating from this body of work have provided a contemporary scientific approach to two of the most serious diseases of GSDs in the NZ Police Dog Section colony. In so doing the outlook for preventing CHD and diagnosing and treating DLSS in these dogs and the population at large should be improved.

Appendix 1

Handler Survey of the Causes of Loss of Working Police Dogs

The purpose of this survey is to identify the major causes of loss of working police dogs. Police dogs are retired/rehomed, die on the job or are euthanased for many reasons. If we can identify these causes then effective strategies to mitigate losses may be possible.

The Centre for Service and Working Dog Health and Research at Massey University and the Police Dog Training Centre would appreciate your time to fill out this survey as the information is vital to developing future strategies to mitigate these losses.

In order for an accurate analysis it is important that we also collect data on dogs that are still in service, especially if they are known to be suffering from an ailment that could lead to retirement. For fit and healthy dogs we are simply recording numbers in service.

For **each dog** that you are currently handling, or have worked in the past (i.e. was once your **operational dog**) can you please give as many of the following details as possible. If a date is not known completely accurately, please estimate the month and/or year.

1. Date of survey completion

2. Your Name

3. Name of dog

4. Date of birth of dog

5. Tattoo number if known

6. Microchip number if known

7. Police District to whom you were assigned at the time his dog was in service.

8. Date of this dog entering service with you as handler

9. Is this dog still in active service with no known health issues for which retirement may be indicated, if **yes** go to survey completion, if **no** (health issues or not in service/died) continue to 10.

10. Date of loss or retirement from active service or still in service [ ] tick

11. Reason for loss or retirement from active service

Retired due to inability to continue to work [ ] tick go to 12

Euthanased (put to sleep) whilst in active service [ ] tick go to 13

Died of a disease / illness whilst in service [ ] tick go to 14

Killed whilst in active service [ ] tick go to 15

12. What was the MAJOR reason your dog was unable to continue to work and which led to retirement?
Please indicate by placing a numeral 1 in the appropriate box

(if there was more than one contributing factor please indicate a (1) for the most important reason and a (2, 3 etc) for other minor but contributing causes).

Behavioural problem [ ]

Type of behavioural problem aggression [ ]

not following commands [ ]

other [ ]

_____

Loss of tracking [ ]

Inability to meet the physical demands of the job [ ]

Other, please describe [ ]

13. What was the MAJOR reason your dog was euthanased whilst still in active duty

Please place a numeral 1 in the box for the most important contributing factor and a (2, 3 etc) for other minor but contributing causes).

Continued overleaf

Behavioural problem [ ]

Type of behavioural problem aggression [ ]

not following commands [ ]

other [ ]

_____

Medical problem [ ]
Heart problem [ ]
Musculoskeletal [ ]
Neurological [ ]
Breathing issues [ ]
Urinary Issues [ ]
Back/Spinal problems [ ]
Arthritis [ ]
Stomach problems [ ] but not bloat/GDV see below
Intestinal [ ]
Gastic Bloat/GDV [ ]

Other [ ] Please give the name of the medical condition (if known)

_____

_____

Inability to meet the physical demands of the job
(please describe the issues)

_____

14. What was the medical condition / illness which led to the death of your dog whilst on active duty
(if there was more than one contributing factor please indicate a (1) for the most important reason and a (2, 3 etc) for other minor but contributing causes).

Medical problem
Heart problem [ ]
Poisoning [ ]
Seizuring [ ]
] Breathing problem [ ]
Infectious disease [ ]
Spinal problems [ ]
Bloat/GDV [ ]
Perforated bowel [ ]
Other (please describe_____)

15. How was your dog killed in active service.

Injured by offender (stabbed / gun shot / other please describe) _____

Motor vehicle accident

Poisoning

Other (please describe) _____

16. If you indicated arthritis as a cause of retirement or euthanasia in 12 or 13 above, please state the joint/s affected

Hip	[ ]	Stifle (knee)	[ ]
Hock (ankle)	[ ]	Hind foot	[ ]
Shoulder	[ ]	Elbow	[ ]
Carpus (wrist)	[ ]	Fore foot	[ ]

17. If you indicated back/spinal problem as a cause of retirement or euthanasia in 12 or 13 above, please state the area/s affected

Neck [ ]
Thoracic spine (between the neck and the last rib) [ ]
Lumbar spine (the rest of the spine except for the lumbosacral junction [ ]
Lumbo-sacral junction (junction between the lower back and the pelvis/tail bone) [ ]

Yours sincerely

Andrew Worth
Senior Lecturer and Registered Specialist in Small Animal Surgery
Massey University Veterinary Teaching Hospital

Sergeant Mark Sandford
Police Dog Training Centre
Trentham

New Zealand Veterinary Journal
Publication details, including instructions for authors and subscription information:
http://www.tandfonline.com/loi/tnzv20

Canine hip dysplasia: phenotypic scoring and the role of estimated breeding value analysis

M Soo[ab] & AJ Worth[a]

[a] Massey University Veterinary Teaching Hospital, Institute of Veterinary, Animal and Biomedical Sciences, Massey University, Private Bag 11222, Palmerston North 4442, New Zealand

[b] Cahill Animal Hospital, Palmerston North, New Zealand

Accepted author version posted online: 29 Jul 2014.Published online: 01 Dec 2014.

To cite this article: M Soo & AJ Worth (2015) Canine hip dysplasia: phenotypic scoring and the role of estimated breeding value analysis, New Zealand Veterinary Journal, 63:2, 69-78, DOI: 10.1080/00480169.2014.949893

To link to this article: http://dx.doi.org/10.1080/00480169.2014.949893

Review Article

Canine hip dysplasia: phenotypic scoring and the role of estimated breeding value analysis

M Soo*† and AJ Worth*§

Abstract

Canine hip dysplasia (CHD) is a developmental orthopaedic disease of the coxofemoral joints with a multifactorial mode of inheritance. Multiple gene effects are influenced by environmental factors; therefore, it is unlikely that a simple genetic screening test with which to identify susceptible individuals will be developed in the near future. In the absence of feasible methods for objectively quantifying clinical CHD, radiographic techniques have been developed and widely used to identify dogs for breeding which are less affected by the disease. A hip-extended ventrodorsal view of the pelvis has been traditionally used to identify dogs with subluxation and/or osteoarthritis of the coxofemoral joints. More recently, there has been emphasis on the role of coxofemoral joint laxity as a determinant of CHD and methods have been developed to measure passive hip laxity. Though well-established worldwide, the effectiveness of traditional phenotypic scoring schemes in reducing the prevalence of CHD has been variable. The most successful implementation of traditional CHD scoring has occurred in countries or breeding colonies with mandatory scoring and open registries with access to pedigree records. Several commentators have recommended that for quantitative traits like CHD, selection of breeding stock should be based on estimated breeding values (EBV) rather than individual hip score/grade. The EBV is a reflection of the genetic superiority of an animal compared to its counterparts and is calculated from the phenotype of an individual and its relatives and their pedigree relationship. Selecting breeding stock on the basis of a dog's genetic merit, ideally based on a highly predictive phenotype, will confer the breeder with greater selection power, accelerate genetic improvement towards better hip conformation and thus more likely decrease the prevalence of CHD.

KEY WORDS: *Canine hip dysplasia, phenotypic scoring, estimated breeding values, genetic improvement, hip scores, hip laxity*

Introduction

Canine hip dysplasia (CHD) is a developmental condition primarily affecting medium-sized and large-breed dogs, which is characterised by instability of the hip joint, leading to degenerative arthritis (Todhunter and Lust 2003).

Canine hip dysplasia is a heritable and multifactorial disorder, meaning that its expression is influenced by the effect of several genes and many, often unidentified, environmental factors (Cook *et al.* 1996; Bliss *et al.* 2002; Smith *et al.* 2006). The disease is characterised by coxofemoral joint pain leading to lameness, stiffness and a progressive decline in function of the joint. CHD is one of the most common orthopaedic diseases affecting companion animals. Recognition of the heritable nature of the disease and use of effective breeding selection methods is critical to achieve a reduction in the prevalence of CHD. This paper will summarise the current understanding of the pathogenesis and radiographic diagnosis of CHD, and outline methods for selective breeding guided by genetic analyses.

Pathogenesis of CHD

Canine hip dysplasia was first described in 1937 as a congenital subluxation of the coxofemoral joint (Schnelle 1937). It is more correctly described as a developmental condition because the coxofemoral joints of dogs that later become dysplastic initially appear normal and congruent at birth (Riser 1975b). Whilst the original anatomical conformation of the coxofemoral joint and its surrounding structures are genetically pre-determined, continued growth and development are synchronised and dependent on mechanical function, joint congruency and the balance of forces applied across the joint. An alteration in any or all of these factors may affect or interfere with development of the hip joint (Riser 1975a; Frost 1989).

The hip joint is stabilised by its joint capsule and surrounding pelvic musculature. In 1966, Henricson, Norberg and Olson

* Massey University Veterinary Teaching Hospital, Institute of Veterinary, Animal and Biomedical Sciences, Massey University, Private Bag 11222, Palmerston North 4442, New Zealand
† Cahill Animal Hospital, Palmerston North, New Zealand
§ Author for correspondence. Email: a.j.worth@massey.ac.nz

http://dx.doi.org/10.1080/00480169.2014.949893

BVA	British Veterinary Association
CHD	Canine hip dysplasia
EBV	Estimated breeding value
FCI	Federation Cynologique Internationale
NZVA	New Zealand Veterinary Association
OFA	Orthopaedic Foundation for Animals
SNP	Single nucleotide polymorphisms

described a link between early joint laxity and the later development of CHD (Henricson *et al.* 1966). When laxity of the femoral head is present, the stabilising structures are assumed to fail to restrain the head of the femur within the acetabulum. The femoral head shifts laterally during weight-bearing, and force is concentrated on the dorsal acetabular rim. Prior to 6 months of age, the dorsal acetabular rim in dogs is largely cartilaginous and very plastic. Concentration of weight-bearing forces on the dorsolateral rim leads to microfractures and modelling of the acetabulum (Riser 1963, 1975b). The joint capsule is stretched and its attachments to the labrum can tear. After 6 months of age, changes in joint shape are only possible through the production or resorption of bone. Therefore, the phenotypic expression of CHD in genetically susceptible dogs may be preventable if coxofemoral joint congruency can be maintained until ossification of the acetabulum is complete (Riser 1975b).

The first 60 days of a puppy's life is thought to be the most critical period in terms of development of the coxofemoral joint, a period during which the joint is susceptible to modelling under abnormal stress loading (Riser 1975c). During this period, a puppy may be presented to a veterinarian because of pain from acetabular microfractures and traumatic synovitis. Clinical signs often improve with conservative management (weight reduction, confinement, analgesics) and many dogs become free of clinical signs until osteoarthritis develops and progresses to the stage of full-thickness cartilage loss later in life. In mild cases, the degree of laxity and change in loading is insufficient to induce osteochondral lesions that would result in lameness. In more severe cases, the trauma to the dorsal acetabulum caused by excessive loading leads to abrasion of the articular cartilage, inducing synovitis and effusion. Changes to the composition of the synovial fluid within the dysplastic hip joint reduce its ability to lubricate, leading to increased cartilage wear (Riser 1975c). As osteoarthritis progresses, joint effusion worsens the stability of the joint by loss of hydrostatic pressure (Smith *et al.* 1990). These changes further destabilise the joint, sometimes to the extent of coxofemoral subluxation or complete luxation (Riser 1975c).

Whilst the concept of laxity leading to subluxation is well accepted, the inciting cause of laxity is still uncertain. Factors that have been implicated in predisposing or causing a genetically susceptible individual to develop CHD include, but are not limited to: the disparity in the rate of maturity of the pelvic muscle mass and skeletal structures that support the coxofemoral joint in genetically susceptible dogs (Cardinet *et al.* 1997), spasms or contracture of the pectineus muscle (Ihemelandu *et al.* 1983), hormonal influence of relaxin and/or oestrogen (Steinetz *et al.* 2008), increased synovial fluid volume and osmolality (Lust *et al.* 1980; Kealy *et al.* 1993), caloric intake and rate of weight gain (Kealy *et al.* 1992; Comhaire and Snaps 2008) and level of exercise in the skeletally immature animal (Krontveit *et al.* 2012).

The genetic basis of CHD

Canine hip dysplasia has been accepted to be a heritable condition since the 1950s. Original hypotheses that a single gene with a recessive or dominant Mendelian pattern of transmission was responsible (Hutt 1967) were later doubted and the concept of incomplete manifestation and variable penetrance were promoted (Janutta and Distl 2006). By the 1970s, a multifactorial mode of inheritance was considered to be the most probable genetic basis for CHD (Leighton *et al.* 1977; Lust and Farrell 1977; Hedhammar *et al.* 1979).

In order to understand the concept of a multifactorial mode of inheritance, we must define the concepts of phenotype and genotype. The expression of a trait is known as the phenotype, which is the sum of the genetic and environmental effects, expressed as $P=G+E$, where P is the phenotypic value, G is the genotypic value and E is non-genetic, environmental deviation (Nicholas 2010a). An animal's genotypic value is the combined effect of the animal's genes at all loci affecting the phenotype of interest. For any measured trait, G is fixed at conception and E is the effect of all environmental factors influencing trait expression between conception and measurement of P. CHD may be expressed in a variety of phenotypes, each determined and measured by one or a combination of methods. For a quantitative trait, it is possible for two dogs of identical genotypes to express very different phenotypes under differing environmental influences. It is also possible that a genetically susceptible individual may have a hip phenotype that passes for normal, if environmental conditions are favourable. Multifactorial inheritance refers to inheritance of a phenotypic characteristic (trait) that is attributable to two or more genes with an unknown number of non-genetic (environmental) factors. In such multifactorial diseases, the actual number of genes determining the manifestation of the condition is commonly unknown. CHD is considered by many to be a quantitative trait due to its continuous phenotypic expression from normal to abnormal. Quantitative traits are determined by the action of different genes at many quantitative trait loci and the effects of each of the individual alleles are often not immediately distinguishable (Lust and Farrell 1977; Nicholas 2010a).

Several key steps have been taken to develop a genetic test for CHD (Zhu 2009). The canine genome has been mapped, opening the possibility that genes responsible for the expression of CHD may be identifiable (Guo *et al.* 2011). Mutations related to hip laxity and Norberg angles have been identified by mapping the chromosomes of cohorts of dysplastic Labrador Retrievers and disease-free Greyhound crossbred dogs (Todhunter *et al.* 2005). Twelve candidate (approximate chromosomal) locations for CHD were found. Further work on the German Shepherd dog genome using quantitative trait loci analysis revealed 19 candidate loci associated with CHD, located on nine different chromosomes, of which chromosome CFA9 was the strongest possible candidate (Marschall and Distl 2007). In another genome-wide association study, Pfahler and Distl (2012) identified three quantitative trait loci for CHD in Bernese Mountain dogs harbouring significantly associated single-nucleotide polymorphisms (SNP). Three SNP were found to be significantly associated with CHD on dog chromosomes CFA14 and 37, with candidate genes of interest being paraoxonase-2 (*PON2*) on CFA14 and fibronectin-1 (*FN1*) on CFA37. The *PON2* and *FN1* genes were hypothesised to be part of the pathogenesis of CHD due to their involvement with bone mineral density and extracellular matrix in cartilage respectively (Pfahler and Distl 2012). Four SNP associated with CHD and two SNP loci associated with hip osteoarthritis have also been identified in a separate study (Zhou *et al.* 2010). Friedenberg *et al.* (2011) identified an association between a mutation-deletion haplotype in the candidate gene, fibrillin-2 gene (*FBN2*) and CHD. The *FBN2* gene is the first gene reported to be associated with four phenotypic markers for the presence of CHD (the Norberg angle, distraction index, dorsolateral subulaxation score and the Orthopaedic Foundation for Animals (OFA) hip grade,

see later). Dogs homozygous for the deletion *FBN2* haplotype were found to have worse hip joint conformation (i.e. more severely affected by CHD) as characterised by having a lower Norberg angles value and dorsolateral subulaxation score, higher distraction index and poorer OFA hip grade. It was also found that dogs with incipient osteoarthritis at necropsy had an approximately 50% greater *FBN2* mRNA in their hip joint capsule in contrast to non-osteoarthritic dogs. However, the authors stressed that the *FBN2* locus did not explain all the genetic trait variation observed in CHD, indicating that other genes must contribute to the expression of the disease (Friedenberg *et al.* 2011).

Whilst current DNA marker technology is not yet sufficiently refined to be used in the selection of breeding animals, DNA marker information acquired via such means may be available to breeders in the future (Marschall and Distl 2007; Zhu *et al.* 2008; Zhou *et al.* 2010). It has been suggested that employing DNA markers as part of a genomic selection scheme would be an alternative means of reducing the prevalence of CHD (Sánchez-Molano *et al.* 2014). A genomic selection strategy involves a genomic (DNA) test that provides information on a large number of genotype markers in a population of phenotypically scored dogs. A subset of markers is then produced via linkage disequilibrium with the genes associated with the disease. These markers are later used to calculate genomic estimates of the true breeding values (genomic estimated breeding values), which are in turn used for subsequent breeding selections within the same breed. In the simulated study population, Sánchez-Molano *et al.* (2014) showed that genomic selection could have achieved greater genetic progress as compared to selection based on the phenotype alone (British Veterinary Association (BVA) hip scoring scheme). Compared to phenotypic selection, genomic selection accelerates the rate of genetic progress due to higher selection accuracy (Sánchez-Molano *et al.* 2014). Genomic selection also allows breeding selection to be carried out earlier because the DNA tests can be performed on animals at a younger age. More importantly, genomic estimated breeding values distinguish between littermates, thereby minimising the rate of inbreeding. Unlike phenotypic selection schemes, genomic selection removes environmental biases (such as age at scoring) which may artefactually influence breeding selection. Sánchez-Molano *et al.* (2014) emphasised that one potential drawback of this selection strategy is that due to the decay of the linkage disequilibrium between the genotype markers and causative loci, the accuracy of the genomic selection will likely decrease over time. Therefore, re-estimates (via phenotypic scoring) of the marker effects will still be necessary every few generations in order to maintain the level of accuracy. Until DNA marker technology has been further refined for routine use in screening for CHD, clinicians and breeders will have to rely on other modalities of breeding selection, as discussed in the following sections.

Phenotypic selection of dogs for breeding based on radiology: worldwide CHD scoring systems

Extended hip view

Due to the difficulty in objectively quantifying clinical CHD, radiological scoring/grading methods have been developed in an attempt to allow dog breeders to select less affected stock for breeding. There are several systems of radiographic screening currently in use around the world. The majority are based on images of the hips and pelvis taken under deep sedation or general anaesthesia, with the dog in ventral recumbency and with the hindlimbs extended (Flückiger 2007). An extended-hip view places the femoral head and neck consistently in a position which allows observation of the typical sites of coxofemoral joint osteophyte development. In addition, this view can reveal subluxation of the femoral head. However, the extent of subluxation can be underestimated due to the inherent positioning of the limbs for radiography and the level of sedation. If the patient is incompletely anaesthetised or sedated, it will respond to hindlimb extension by contracting its pelvic muscles, thereby improving the seating of the femoral head within the acetabulum and falsely lowering the extent of subluxation. Additionally, when the coxofemoral joints are positioned in the extended-hip view, the joint capsule and ligament of the head of the femur are both twisted, tightening the tensile elements of the joint capsule (Smith *et al.* 1990). As the tensile elements tighten, the femoral head is forced deeper into the acetabulum in a phenomenon known as "screw-home" tightening, a term borrowed from human medicine. In the canine hip, screw-home tightening may lead to false negative grades assigned to dogs screened at an early age, as osteoarthritis may not be evident on radiographs and the assessor is relying on the evidence for subluxation as an indicator of the severity of the disease. Furthermore, the extent of laxity seen on the extended-hip view is affected by the accuracy of patient positioning and the method of holding the limbs in extension. An oblique pelvic position increases the extent of femoral head coverage of one hip joint whilst lowering it on the other. If the dog's limbs are hand-held rather than being strapped in position for radiography, then the holder can also influence the degree of subluxation and falsely tighten the joints (Rendano and Ryan 1985).

Features of CHD on the extended-hip view vary depending on the age of the animal when it is radiographed and these features have been extensively documented by Riser (1975c). There is continual osteophyte development over the life of a dysplastic dog, indicating that osteoarthritis associated with CHD is progressive (Smith *et al.* 2006, 2012). Similarly, increased age at the time of assessment is significantly correlated with dysplastic radiological changes. These radiological signs are indirect markers of cartilage degeneration secondary to abnormal biomechanics of the hip joint. When CHD-affected patients were examined at the end of life, osteoarthritis was observed using histopathology in 96% (43/45) of the dogs compared to only 67% (32/48) of dogs seen radiographically (Smith *et al.* 2012). Therefore, osteophyte development observed radiographically is insensitive when compared to a gold standard of joint histopathology.

In the early stages of CHD, the radiological signs lag behind cartilage injury, but as a dog ages, radiography becomes more accurate at predicting the status of the hip joint morphology (Smith *et al.* 2012). When a dog is younger than 5 years of age, the radiological diagnosis of CHD is reliant on evidence of femoral head subluxation and/or osteoarthritis (Smith *et al.* 2012). In older dogs, radiological identification of CHD may encompass the presence of a shallow acetabulum, coxofemoral incongruity and osteoarthritis (Riser 1975c). After 6 years of age, newly diagnosed cases of CHD appear to be entirely dependent on radiographic evidence of coxofemoral osteoarthritis, with no new diagnoses based on subluxation (Smith *et al.* 2012).

The three most well-utilised CHD radiographic schemes are operated by the OFA in the United States of America, the BVA (United Kingdom and Australasia) and the Fédération Cynologique Internationale (FCI) in Europe (Flückiger 2007). A comparison of these grading schemes for CHD is shown in Table 1, but as each of these independent schemes is based on subjective assessments, direct comparisons between scheme grades are largely speculative. The OFA has been grading hip radiographs of dogs in North America and Canada for CHD since 1966, using subjective criteria (Flückiger 2007; Anonymous 2010). Dogs must be >24 months of age to be eligible for the scheme and an extended-hip view is taken under anaesthesia.

In Europe, the FCI is a cooperative of national kennel clubs which has been screening dogs for CHD for over 40 years (Flückiger 2007). Dogs must be >12 months of age or >18 months of age for the large and giant breeds. The FCI scoring system combines subjective assessment of the degree of subluxation, congruity of the femoral head and acetabulum and osteophyte development on the extended-hip view with objective measurement of subluxation using the Norberg angle (Morgan and Stephens 1985; Henry 1992). A normal Norberg angle is considered to be 105° and greater; however, this upper limit of normal value varies between breeds and should be interpreted with caution (Tomlinson and Johnson 2000; Culp *et al.* 2006). Additionally, the Norberg angle is affected by screw-home tightening, as described above. Analyses of inter-observer agreement of the FCI system showed that assessment of the morphological characteristics of the hip joints and the final score are highly variable between observers (Verhoeven *et al.* 2007, 2009).

In the United Kingdom, the BVA and the Kennel Club implemented a CHD screening programme in 1965. This technique of hip scoring was also adopted in Ireland, Australia and New Zealand (Flückiger 2007). Up to 53 demerit points are awarded *per hip* in nine categories to yield a total score of 0 (best) to 106 (worst) (Dennis 2012). Dogs are eligible for this programme from 12 months of age.

The New Zealand Veterinary Association (NVZA) introduced the Canine Hip Dysplasia Scheme in the mid-1980s (Hunter 1986) in co-operation with the New Zealand Kennel Club. A national computerised database was maintained from 1989. Dogs need to be >12 months of age to be eligible to be scored under this scheme and it is recommended that giant breeds are scored at 18 months of age or older. Readers are referred elsewhere for further information on the NZVA CHD scheme (Worth *et al.* 2009; Anonymous 2011).

Table 1. An attempted comparison of the different grading systems for canine hip dysplasia, modified from Verhoeven *et al.* (2012). Each system is based on subjective criteria such that direct comparisons are largely speculative, therefore, this table is only a guide.

	Grading System and Grade				
	Fédération Cynologique Internationale (Europe)		British Veterinary Association (UK)		Orthopedic Foundation for Animals (USA)
Descriptor					
No signs of hip dysplasia	A	NA[a] >105°	A1	0–4 (not >3/hip)	Excellent
			A2	5–10 (not >6/hip)	Good
Near normal hip joints	B	NA ≤105°	B1	11–18	Fair
			B2	19–25	Borderline
Mild hip dysplasia	C	NA 100°	C1	26–35	Mild
			C2		
Moderate hip dysplasia	D	NA 90–100°	D1	36–50	Moderate
			D2		
Severe hip dysplasia	E	NA <90°	E1	51–106	Severe
			E2		

[a] The Norberg angle (NA) measured under the Fédération Cynologique Internationale scheme allows for the objective assessment of femoral head subluxation. A normal NA is considered to be ≥105°.

Distraction view

Building on the link between early joint laxity and later development of CHD described by Henricson *et al.* (1966), the concept of passive versus functional hip laxity was introduced to distinguish between subluxation evident radiologically compared to subluxation induced during weight-bearing (Smith *et al.* 1990). Those authors proposed that the joint capsule, round ligament of the femoral head and the hydrostatic stability factor are passive constraints to hip laxity and each play a role in maintaining congruency between the femoral head and acetabulum. The active constraints are the muscles surrounding the hip joint capable of imparting a force that reduces the femoral head into the acetabulum. Passive constraints restrict the amount of hip extension possible as well as creating a force that acts to drive the femoral head into the acetabulum, minimising laxity. If the constraints maintained by the soft tissue surrounding the hip joint do not function properly, the coxofemoral joint will not receive and effectively transmit load during weight-bearing. The inability or failure of these soft tissue passive constraints to limit the amount of lateral displacement of the femoral head in the relaxed dog is defined as passive laxity. Functional laxity is defined as the lateral translation of the femoral head out of the acetabulum during weight-bearing. Passive laxity is a pre-requisite for, but not a cause of, functional laxity and may not always correlate with an equal extent of functional laxity (Smith *et al.* 1990). Passive laxity has been reported to be highly correlated with the development of osteoarthritis and the clinical signs of hip dysplasia (Smith *et al.* 1990, 1995). Smith *et al.* (1990) developed a stress-radiographic method (distraction radiography) using a radiolucent fulcrum placed between the femurs to quantify the degree of passive laxity in an anaesthetised or heavily sedated dog. The adjustable radiolucent fulcrum is placed between the thighs of the dog and the femurs are pulled together by gripping the distal limb, inducing femoral head subluxation at which point a radiograph (the distraction view) is taken (Figure 1). This distraction radiography method formed the foundation of PennHIP (formerly the University of Pennsylvania Hip Improvement Program), which became commercially available in 1993 (Anonymous 2013). The PennHIP distraction index is a measure of passive hip laxity and is calculated by determining the degree to which the femoral head subluxates relative to the acetabulum. The linear distance between the centre of the acetabulum and the centre of the femoral head on the distracted radiographic view is divided by the radius of the femoral head to achieve a unit-less measure of laxity; the distraction index (Figure 2). The PennHIP evaluation comprises three ventrodorsal radiographic views; the distraction, compression and extended-hip views. The extended-hip view is used to assess the presence or absence of osteoarthritis and the compression view is used to assess the overall coxofemoral congruency. The presence of osteophyte

Figure 1. Photograph showing positioning of a dog for measurement of the PennHIP distraction index. A radiograph is taken with the dog heavily sedated or anaesthetised and positioned in ventrodorsal recumbency with the distraction device placed between the thighs. The operator uses the device as a fulcrum to achieve distraction of the coxofemoral joints (passive hip laxity).

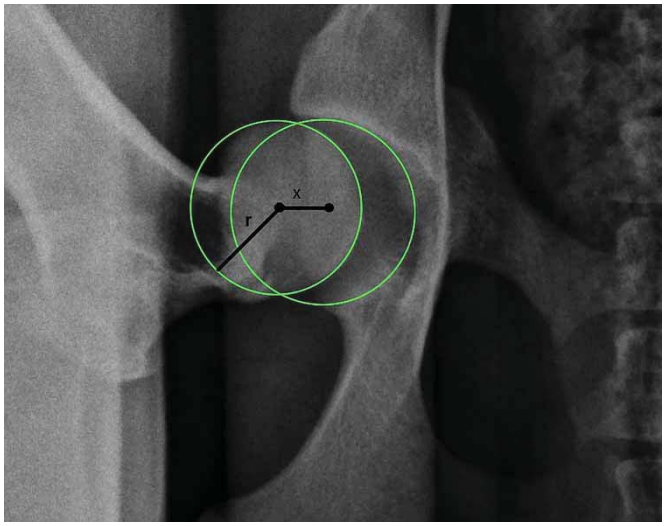


Figure 2. Radiograph showing measurements used for evaluation of the distraction index, calculated as the ratio of the distraction between the centres of the acetabulum and femoral head (x) to the radius of the femoral head (r), when a distraction force is applied to the sedated/anaesthetised patient as shown in Figure 1.

development on the extended-hip view results in the dog being classified as having CHD (Anonymous 2013). Of the three PennHIP radiographic views, the distraction view is most frequently utilised in genetic studies because it quantifies passive joint laxity, which has been widely recognised for its important role in the pathophysiology of CHD.

Heritability and breeding selection for a superior hip phenotype

Genetic gain through selective breeding is a product of the heritability of a trait and the selection pressure applied over generations. The selection pressure is related to the extent of phenotypic variation and the intensity of selection set by dog breeders. CHD scoring systems were introduced to assist dog breeders with the goal of reducing the prevalence and severity of hip dysplasia in future generations. The key assumption in any phenotypic scoring scheme is that successive generations will benefit from the selection pressure exerted against the trait in their parents and therefore, a better phenotype will be produced over time.

Heritability measures the extent to which offspring resemble their parents for a certain trait. Estimation of heritability uses statistical techniques to determine the extent to which relatives resemble each other for a particular trait (Nicholas 2010a). Heritability ranges from 0–1. If the heritability is 0, it means that relatives do not resemble each other at all for the trait of interest. Conversely, a trait with heritability of 1 is completely genetically determined. Traits with heritability less than 0.1 are considered lowly heritable; 0.2–0.3 moderately heritable and 0.4–1.0 to be highly heritable (Mackenzie 1985). The practical importance is that if the heritability is more than 0, then it should be possible to decrease the prevalence of a particular trait via selective breeding. In general, the higher the heritability, the greater the influence of genetic effects on the trait and thus the greater the potential response to breeding selection pressures (Mackenzie 1985; Nicholas 2010a). Heritability for the same trait may vary from one estimate to another. Estimates may differ due to sampling variation, the phenotype (method of hip scoring) being examined, the breed or population involved, the degree of inbreeding, environmental factors, and the method of calculation (Mackenzie 1985; Nicholas 2010a).

Historically, depending on the method of evaluation, the estimated heritability of CHD has been found to be >0.22–0.25, meaning that no less than approximately 22% of the disease is genetically controlled (Leighton *et al.* 1977; Fox *et al.* 1987). Tables 2–4 provide a non-exhaustive overview of the reported estimated breed heritabilities based on OFA, FCI and BVA scoring systems. The heritability estimates in these studies were performed using a variety of methods such as regression, Bayesian and/or Restricted Maximal Likelihood analyses. As a general rule, traits with heritability estimates >0.15 are considered to be under adequate genetic influence such that sufficient response will be seen with selective breeding (Wilson *et al.* 2011), thus decreasing the prevalence of the disease. Heritability estimates of previous studies performed on the OFA, FCI and BVA scoring systems have generally met or exceeded this level. To the author's knowledge, there have been no estimates of heritability performed specifically for dogs scored under the NZVA CHD Scheme, but, because the NZVA CHD Scheme is based on the BVA

Table 2. Reported heritabilities (h^2) for the Orthopaedic Foundation for Animals hip grading system for canine hip dysplasia, using the hip-extended radiographic view (subjective scoring system).

Breed	h^2 (SE)	No. dogs	Study
Bernese Mountain Dog	0.30 (0.04)	4,151	Reed *et al.* (2000)
Chinese Shar-pei	0.31 (0.05)	3,360	Reed *et al.* (2000)
English Setter	0.17 (0.05)	3,876	Reed *et al.* (2000)
German Shepherd Dog	0.22 (0.06)	1,186	Leighton *et al.* (1977)
German Shepherd Dog	0.43	575	Mackenzie *et al.* (1985)
Labrador Retriever	0.21 (0.006)	154,352	Hou *et al.* (2010)
Portuguese Water Dog	0.30 (0.06)	1,337	Reed *et al.* (2000)
Pooled h^2 for 17 breeds	0.76	2,716	Zhang *et al.* (2009)
Pooled h^2 for 74 breeds	0.22 (0.002)	760,455	Hou *et al.* (2013)

Table 3. Reported heritabilities (h^2) for the Fédération Cynologique Internationale hip grading system for canine hip dysplasia, using the hip-extended radiographic view (subjective scoring system).

Breed	h^2 (SE)	No. dogs	Study
Bernese Mountain Dog	0.42 (0.03)	8,221	Malm *et al.* (2008)
Bernese Mountain Dog	0.31 (0.06)	1,479	Lavrijsen *et al.* (2014)
Estrela Mountain Dog	0.3–0.43[a]	313	Silvestre *et al.* (2007)
German Shepherd Dog	0.31–0.35[a]	10,335	Leppänen *et al.* (2000b)
German Shepherd Dog	0.24–0.26[a]	21,371	Hamann *et al.* (2003)
German Shepherd Dog	0.25 (0.01)	47,730	Stock *et al.* (2011)
Golden Retriever	0.17 (0.03)	22,934	Lingaas and Klemetsdal (1990)
Golden Retriever	0.18 (0.04)	2,412	Lavrijsen *et al.* (2014)
Labrador Retriever	0.44	664	Ohlerth *et al.* (2001)
Labrador Retriever	0.24–0.29[a]	3,151	Vostrý *et al.* (2012)
Labrador Retriever	0.10 (0.03)	3,746	Lavrijsen *et al.* (2014)
Rottweiler	0.58 (0.04)	2,764	Mäki *et al.* (2000)
Rottweiler	0.38 (0.02)	14,693	Malm *et al.* (2008)
Newfoundland	0.26–0.28[a]	1,372	Dietschi *et al.* (2003)
Newfoundland	0.23 (0.08)	788	Lavrijsen *et al.* (2014)

[a] Heritability value varies with model and method of estimation.

Table 4. Reported heritabilities (h^2) for the British Veterinary Association hip scoring system for canine hip dysplasia, using the hip-extended radiographic view (semi-subjective scoring system).

Breed	h^2 (SE)	No. dogs	Study
Akita	0.39 (0.053)[a]	152	Lewis *et al.* (2013)
Bearded Collie	0.46 (0.048)[a]	350	Lewis *et al.* (2013)
Bernese Mountain Dog	0.36 (0.040)[a]	450	Lewis *et al.* (2013)
Border Collie	0.44 (0.033)[a]	1,008	Lewis *et al.* (2013)
English Setter	0.35 (0.049)[a]	198	Lewis *et al.* (2013)
Flat-coated Retriever	0.74 (0.25)	1,258	Wood *et al.* (2000a)
Flat-coated Retriever	0.28 (0.032)[a]	1,121	Lewis *et al.* (2013)
German Shepherd Dog	0.30 (0.02)[b]	13,124	Wilson *et al.* (2012)
German Shepherd Dog	0.35 (0.015)[a]	3,680	Lewis *et al.* (2013)
Golden Retriever	0.40 (0.017)[a]	5,374	Lewis *et al.* (2013)
Gordon Setter	0.20–0.38[c]	1,152	Wood *et al.* (2000b)
Gordon Setter	0.43 (0.062)[a]	175	Lewis *et al.* (2013)
Labrador Retriever	0.34 (0.02)	13,382	Wood *et al.* (2002)
Labrador Retriever	0.35 (0.016)[a] 0.50 (0.018)[d]	25,243	Lewis *et al.* (2010)
Labrador Retriever	0.35 (0.02)	25,243	Woolliams *et al.* (2011)
Labrador Retriever	0.33 (0.012)[a]	17,164	Lewis *et al.* (2013)
Newfoundland	0.49 (0.08)	1,566	Wood *et al.* (2000a)
Newfoundland	0.46 (0.041)[a]	478	Lewis *et al.* (2013)
Rhodesian Ridgeback	0.33 (0.048)[a]	541	Lewis *et al.* (2013)
Rottweiler	0.39 (0.028)[a]	616	Lewis *et al.* (2013)
Siberian Husky	0.48 (0.038)[a]	300	Lewis *et al.* (2013)
Tibetan Terrier	0.34 (0.048)[a]	757	Lewis *et al.* (2013)

[a] Heritability estimate performed on hip scores transformed onto a logarithmic scale.
[b] Heritability estimate study based on the Australian Veterinary Association hip scoring system, which is similar to the BVA system.
[c] Heritability value varies with model and method of estimate.
[d] Heritability estimate performed on original, untransformed hip score.

hip scoring system, it is possible to infer the heritability of the NZVA hip phenotype from the BVA hip phenotype.

While a thorough quantitative genetic analysis of the entire PennHIP database has not been published, a small number of studies using the distraction index have been performed. Todhunter *et al.* (2003) found the heritability of the distraction index in 147 Labrador Retrievers across four generations to be 0.5. A larger study by Zhang *et al.* (2009) comprising 2,716 dogs across 17 breeds reported a heritability of 0.61 for the distraction index. A recent genetic analysis of the distraction index in the Estrela Mountain dog, using a linear animal model, reported a very high heritability of 0.83 (Ginja *et al.* 2008). This estimate was based only on 215 observations and has an SE of 0.11. As heritability is unique to the phenotype and population in which it is estimated (Mackenzie 1985), this high heritability estimate from an atypical breed cannot be directly applied to other breeds.

Effectiveness of the radiological scoring systems in lowering the prevalence of CHD

Several authors have commented that despite >40 years of radiographic screening, the prevalence of CHD remains high and have questioned the efficacy of the schemes or programmes adopted by the OFA, FCI and BVA (e.g. Leppanen *et al.* 2000a; Verhoeven *et al.* 2012). There are somewhat conflicting reports as to the efficacy of the OFA scheme. One evaluation of the OFA database showed a steady, albeit slow, increase in the proportions of dogs graded as excellent and good, whereas proportions of fair and mild/moderate/severe dysplastic grades significantly decreased over the period 1989–2003 (Kaneene *et al.* 2009). More recently, Hou *et al.* (2013) performed an estimated breeding value (EBV) analysis on 760,455 hip scores across 74 breeds listed in the OFA database to evaluate genetic trends between the period 1970–2009 inclusive. The study found a genetic improvement of 0.1 units of hip score during the study period, which was equivalent to 16.4% of the phenotypic standard deviation. These values corresponded to a 0.52% decrease in incidence of CHD in the study population. Additionally, the study emphasised that while some genetic improvement was evident on the basis of the EBV analysis of the OFA database, there was a variation in the amount of genetic improvement amongst breeds (Hou *et al.* 2013). These studies suggest that whilst the OFA system has been effective at reducing the number of severely affected dogs, there has been only limited progress towards reducing the overall impact of CHD.

A genetic evaluation of the effectiveness of the BVA scoring system in reducing the prevalence of CHD in UK Labrador Retrievers reported a genetic progress of 0.376 untransformed (or 0.155 log-transformed) hip score units per annum (Lewis *et al.* 2010). This translates to a 1.4% decline year on year or a 13% improvement in hip scores over the 10-year study period. The authors concluded that this was very minimal progress against CHD, and was equivalent to only avoiding 15% of the

worst animals for breeding. More recently, an EBV analysis on 142,287 hip scores across fifteen breeds listed on the BVA CHD database evaluated the genetic improvement made when breeding selection was carried out on the basis of phenotypic scores (Lewis *et al.* 2013). Regression of the EBV on the date of birth revealed that 14/15 breeds exhibited some genetic improvement during the study period. The authors discussed that despite the significant genetic progress, the extent of improvement was only small with a 0.13% to 1.98% decline of hip scores per year, indicating a low selection intensity was employed throughout the study period (Lewis *et al.* 2013). To the authors' knowledge, no genetic studies have been conducted on the efficacy of the NZVA CHD scheme at reducing the prevalence of CHD. However, an evaluation of the phenotypic trend of the NZVA hip scores in four populous breeds of dogs revealed a small but significant phenotypic trend towards an improved radiographic hip conformation in German Shepherd dogs, but not in the Labrador Retrievers, Golden Retrievers or Rottweilers (Worth *et al.* 2011). However, the chosen method of regression analysis based on individual animal hip scores may have overlooked the genetic gain made in some lines within breeds, by not evaluating the genetic trend.

Significant selection bias has been reported in the OFA system with radiographs of normal-appearing hips being 8.2 times more likely to be submitted by veterinarians than radiographs that showed the dogs were clearly dysplastic (Paster *et al.* 2005); a process termed pre-screening. Pre-screening is a criticism of most CHD scoring schemes and skews the population of scored dogs by removing the worst-affected individuals (Paster *et al.* 2005). Submission of radiographs is voluntary in all but a few countries. In New Zealand, there is no compulsion to submit all radiographs performed for the purpose of scoring and, anecdotally, pre-screening does occur. There is also no legal requirement to hip score dogs that are to be used for breeding. Additionally, when a lame dog is diagnosed with clinical CHD, a radiologic score is not captured by any database. Thus, the published average hip scores are not a true reflection of the disease prevalence or severity within a given breed. The quality of radiographs also has a significant effect on the ability of any assessor to accurately determine a dog's CHD radiological phenotype. Thus the credibility of screening methods for CHD using the extended-hip view is questionable (Verhoeven *et al.* 2009).

The use of radiological scoring methods in countries or breeding colonies with mandatory scoring programmes has been reported to be more successful at lowering the prevalence of CHD (Hedhammar *et al.* 1979; Swenson *et al.* 1997; Genevois *et al.* 2008). In Sweden (which uses the FCI method of hip scoring), it has been mandatory since 1984 for the hip joint status of both parents to be published before the Swedish Kennel Club will register the puppies of that mating. This open registration and mandatory scoring may have contributed to the observed improvement in the median score for CHD of several breeds in Sweden (Swenson *et al.* 1997).

As previously discussed, the higher the heritability of a trait, the larger the contribution of genetic factors to the resultant phenotype and thus the greater the response to selection (Mackenzie 1985; Nicholas 2010b). While a thorough genetic evaluation of the PennHIP database has yet to be conducted, preliminary studies suggest that the PennHIP distraction index phenotype potentially has a higher heritability than the hip-extended radiographic phenotype and could therefore yield a better response to selection than the OFA, FCI or BVA phenotypes. This is because higher heritability indicates that a larger proportion of the phenotype is genetically determined and therefore more susceptible to manipulation by selective breeding to decrease the prevalence of disease, given adequate selection intensity. At present, no national veterinary association has adopted the PennHIP method as the basis for recommendations regarding the use of breeding stock.

Discussion

The small amount of genetic progress attained using selection of breeding stock based on individual extended-hip view phenotype has led several commentators to recommend the use of EBV in CHD breeding schemes (Hou *et al.* 2013; Lewis *et al.* 2013). Selection of breeding stock solely on the basis of their individual phenotypic hip status is not the most accurate method of identifying dogs with superior genes, because for a quantitative trait like CHD, it is possible for two dogs of identical genotypes to express very different phenotypes under differing environmental influences (Lust and Farrell 1977). A dog with phenotypically better hips may not have better genes for transmission to its offspring as the favourable hip score may have been a result of a better environment. Instead of the phenotypic hip score or grade, the identification of dogs with superior genes is best determined using EBV. The EBV is a measure of the genetic superiority of an animal as compared to its counterparts and is calculated from the phenotypes of the individual, their relatives and pedigree data (Nicholas 2010a). The EBV is a more accurate predictor of an individual's genetic merit because it takes into account the genetic contribution of superior genes from all relatives (such as offspring or siblings) as well as any other available information about the individual in question (Wilson *et al.* 2011; Woolliams *et al.* 2011). The accuracy of an individual's EBV increases as information becomes available from its relatives. EBV confers greater selection power allowing accelerated genetic gain over time, compared to using individual phenotypic scores (Lewis *et al.* 2010; Keller *et al.* 2011).

In a population of German Shepherd dogs, selection procedures based on breeding values were more efficient than selection schemes based on phenotypic records of parents (FCI hip scoring) (Janutta *et al.* 2008). In another study, it was estimated that it would take approximately 44 years to decrease the median hip score from 10 to 5 if identification of dogs for breeding were to continue on the basis of the phenotypic selection (BVA hip scores). In contrast, by utilising an EBV-based selection scheme to identify dogs with superior genes, it would take approximately 37 years (19% faster) to achieve the same amount of improvement in the BVA hip scores (Lewis *et al.* 2010). Similarly, in a range of simulated scenarios (e.g. large *vs.* small population; low *vs.* high prevalence of CHD), the average genetic gain attained from selection based on Best Linear Unbiased Prediction (estimated) breeding value was 1.03–1.25 times higher than selection based on phenotype (FCI hip scores) alone (Malm *et al.* 2013). Additionally, because the accuracy of breeding selection is directly correlated with the extent of genetic progress, higher selection accuracy will result in greater genetic improvement towards better hip conformation. This was demonstrated in a study Lewis *et al.* (2013), whereby the mean accuracy of selection based on individual or parental phenotype (BVA hip score) was found to be between 1.16 and 1.30, as compared to the higher mean accuracy of 1.44 when

selection was made on the basis of EBV (Lewis *et al.* 2013). In a simulated study population of German Shepherd dogs, breeding selection based on EBV was up to three times more efficient in achieving genetic progress than selection on phenotype alone; however, the study also revealed that an even higher response to selection was obtained via genomic selection (Stock and Distl 2010). Genomic selection has been shown to be the most superior method breeding selection, as compared to EBV or phenotypic selection, in a number of simulated populations (Stock and Distl 2010; Sánchez-Molano *et al.* 2014). However, there is only limited use of DNA marker technology at present (Woolliams *et al.* 2011) whilst it is still undergoing further development and refinement for routine clinical use (see above discussion). Therefore, in the short term at least, it is the authors' view that breeding selection on the basis of EBV remains the next best alternative.

Estimated breeding values can be generated for dogs that have not been hip scored, as long as they have relatives within their pedigree that have been previously scored. This allows an EBV-based selection of breeding stock to be carried out even if not all animals within the breeding population have been scored. This also means that utilising the available information from relatives will allow the EBV of a puppy to be calculated the moment it is born (Lewis *et al.* 2010); this is beneficial as it allows breeders to plan ahead as well as potentially assisting with the process and progress of selection. Another major advantage for the utilisation of EBV for breeding selection is that EBV are corrected for identifiable non-genetic (environmental) effects which may cause bias on the hip score (Lewis *et al.* 2010; Wilson *et al.* 2011; Woolliams *et al.* 2011).

The use of a phenotypic screening test with a higher heritability than the current extended-hip view phenotype would aid an EBV scheme for CHD. Although published studies suggest the heritability of the distraction index is higher than the heritability of the extended-hip view, only a limited number of genetic analyses have been performed on the distraction index to date. The authors are hopeful that a heritability estimate of the distraction index based on a larger dataset will be available in the near future, because using a hip phenotype with a high heritability in an EBV-based selection scheme will likely accelerate the rate of genetic progress, compared to hip phenotypes with lower heritability.

Conclusion

Canine hip dysplasia is a multifactorial trait with a moderate to high heritability. By applying selection pressure appropriately, a reduction in the prevalence of CHD should be achievable. Despite many decades of use, selection using traditional radiological phenotypic scoring schemes has had only modest success. Selection of dogs for breeding on the basis of EBV may prove more effective at reducing the prevalence of CHD than selection based on individual hip scores/grades. The incorporation of EBV into selective breeding programmes will also enable genetic trends to be monitored prospectively and as dynamically as possible in all populations under selection.

Declaration of interest

Andrew Worth is the current Convenor of the NZVA Dysplasia Schemes and receives an honorarium for this position. He is also a PennHIP-certified veterinarian, whose training was supported by the New Zealand Police.

References

*Anonymous. *Orthopedic Foundation for Animals. An Examination of Hip Grading.* http://www.offa.org/hd_grades.html (accessed 24 February 2014). Orthopedic Foundation for Animals, Columbia, MO, USA, 2010

*Anonymous. *Interpretation of the NZVA Hip Dysplasia Scheme Scoring System.* http://www.nzva.org.nz/sites/default/files/domain-0/Interpretation%20of%20Hip%20Dysplasia%20Grade%20-%202011.pdf (accessed 24 February 2014). New Zealand Veterinary Association, Wellington, NZ, 2011

*Anonymous. *PennHIP Hip Improvement Program.* http://info.antechimagingservices.com/pennhip/navigation/general/what-is-PennHIP.html (accessed 25 February 2014). ANTECH Imaging Services, Irvine, CA, USA, 2013

Bliss S, Todhunter RJ, Quaas R, Casella G, Wu R, Lust G, Williams AJ, Hamilton S, Dykes NL, Yeager A, *et al.* Quantitative genetics of traits associated with hip dysplasia in a canine pedigree constructed by mating dysplastic Labrador Retrievers with unaffected greyhounds. *American Journal of Veterinary Research* 63, 1029–35, 2002

Cardinet GH 3rd, Kass PH, Wallace LJ, Guffy MM. Association between pelvic muscle mass and canine hip dysplasia. *Journal of the American Veterinary Medical Association* 210, 1466–73, 1997

Comhaire FH, Snaps F. Comparison of two canine registry databases on the prevalence of hip dysplasia by breed and the relationship of dysplasia with body weight and height. *American Journal of Veterinary Research* 69, 330–3, 2008

*Cook JL, Tomlinson JL, Constantinescu GM. Pathophysiology, diagnosis, and treatment of canine hip dysplasia. *Compendium on Continuing Education for the Practicing Veterinarian* 18, 853–66, 1996

Culp WTN, Kapatkin AS, Gregor TP, Powers MY, McKelvie PJ, Smith GK. Evaluation of the Norberg angle threshold: a comparison of Norberg angle and distraction index as measures of coxofemoral degenerative joint disease susceptibility in seven breeds of dogs. *Veterinary Surgery* 35, 453–9, 2006

Dennis R. Interpretation and use of BVA/KC hip scores in dogs. *In Practice* 34, 178–94, 2012

Dietschi E, Schawalder P, Gaillard C. Estimation of genetic parameters for canine hip dysplasia in the Swiss Newfoundland population. *Journal of Animal Breeding and Genetics* 120, 150–61, 2003

Flückiger MA. Scoring radiographs for canine hip dysplasia – the big three organizations of the world. *European Journal of Companion Animal Practice* 17, 135–40, 2007

Fox SM, Burns J, Burt J. The dysplastic hip: a crippling problem in dogs. *Veterinary Medicine* 82, 684–93, 1987

Friedenberg SG, Zhu L, Zhang Z, Foels WvdB, Schweitzer PA, Wang W, Fisher PJ, Dykes NL, Corey E, Vernier-Singer M, *et al.* Evaluation of a fibrillin 2 gene haplotype associated with hip dysplasia and incipient osteoarthritis in dogs. *American Journal of Veterinary Research* 72, 530–40, 2011

Frost HM. Pathogenesis of congenital hip dysplasia (CHD). A proposal. *Veterinary and Comparative Orthopaedics and Traumatology* 1, 1–10, 1989

Genevois JP, Remy D, Viguier E, Carozzo C, Collard F, Cachon T, Maitre P, Fau D. Prevalence of hip dysplasia according to official radiographic screening, among 31 breeds of dogs in France. *Veterinary and Comparative Orthopaedics and Traumatology* 21, 21–4, 2008

Ginja MMD, Silvestre AM, Ferreira AJA, Gonzalo-Orden JM, Orden MA, Melo-Pinto P, Llorens-Pena MP, Colaco J. Passive hip laxity in Estrela Mountain dog – distraction index, heritability and breeding values. *Acta Veterinaria Hungarica* 56, 303–12, 2008

Guo G, Zhou Z, Wang Y, Zhao K, Zhu L, Lust G, Hunter L, Friedenberg S, Li J, Zhang Y, *et al.* Canine hip dysplasia is predictable by genotyping. *Osteoarthritis and Cartilage* 19, 420–9, 2011

Hamann H, Kirchhoff T, Distl O. Bayesian analysis of heritability of canine hip dysplasia in German Shepherd dogs. *Journal of Animal Breeding and Genetics* 120, 258–68, 2003

Hedhammar A, Olsson SE, Andersson SA, Persson L, Pettersson L, Olausson A, Sundgren PE. Canine hip dysplasia: study of heritability in 401 litters of German Shepherd dogs. *Journal of the American Veterinary Medical Association* 174, 1012–6, 1979

Henricson B, Norberg I, Olssons SE. On the etiology and pathogenesis of hip dysplasia: a comparative review. *Journal of Small Animal Practice* 7, 673–88, 1966

Henry GA. Radiographic development of canine hip dysplasia. *Veterinary Clinics of North America: Small Animal Practice* 22, 559–78, 1992

Hou Y, Wang Y, Lust G, Zhu L, Zhang Z, Todhunter RJ. Retrospective analysis for genetic improvement of hip joints of cohort labrador retrievers in the United States: 1970–2007. *PLoS One* 5, e9410, 2010

Hou Y, Wang Y, Lu X, Zhang X, Zhao Q, Todhunter RJ, Zhang Z. Monitoring hip and elbow dysplasia achieved modest genetic improvement of 74 dog breeds over 40 years in USA. *PLoS One* 8, e76390, 2013

Hunter AC. Radiography for hip dysplasia evaluation. *New Zealand Veterinary Journal* 34, 43–5, 1986

Hutt FB. Genetic selection to reduce the incidence of hip dysplasia in dogs. *Journal of the American Veterinary Medical Association* 151, 1041–8, 1967

Ihemelandu EC, Cardinet GHr, Guffy MM, Wallace LJ. Canine hip dysplasia: differences in pectineal muscles of healthy and dysplastic German Shepherd dogs when two months old. *American Journal of Veterinary Research* 44, 411–6, 1983

Janutta V, Distl O. Inheritance of canine hip dysplasia: Review of estimation methods and of heritability estimates and prospects on further developments. *Deutsche tierarztliche Wochenschrift* 113, 6–12, 2006

Janutta V, Hamann H, Distl O. Genetic and phenotypic trends in canine hip dysplasia in the German population of German shepherd dogs. *Berliner Und Munchener Tierarztliche Wochenschrift* 121, 102–9, 2008

Kaneene JB, Mostosky UV, Miller R. Update of a retrospective cohort study of changes in hip joint phenotype of dogs evaluated by the OFA in the United States, 1989–2003. *Veterinary Surgery* 38, 398–405, 2009

Kealy RD, Olsson SE, Monti KL, Lawler DF, Biery DN, Helms RW, Lust G, Smith GK. Effects of limited food consumption on the incidence of hip dysplasia in growing dogs. *Journal of the American Veterinary Medical Association* 201, 857–63, 1992

Kealy RD, Lawler DF, Monti KL, Biery D, Helms RW, Lust G, Olsson SE, Smith GK. Effects of dietary electrolyte balance on subluxation of the femoral head in growing dogs. *American Journal of Veterinary Research* 54, 555–62, 1993

Keller GG, Dziuk E, Bell JS. How the orthopedic foundation for animals (OFA) is tackling inherited disorders in the USA: using hip and elbow dysplasia as examples. *Veterinary Journal* 189, 197–202, 2011

Krontveit RI, Trangerud C, Sævik BK, Skogmo HK, Nødtvedt A. Risk factors for hip-related clinical signs in a prospective cohort study of four large dog breeds in Norway. *Preventive Veterinary Medicine* 103, 219–27, 2012

Lavrijsen ICM, Heuven HCM, Meij BP, Theyse LFH, Nap RC, Leegwater PAJ, Hazewinkel HAW. Prevalence and co-occurrence of hip dysplasia and elbow dysplasia in Dutch pure-bred dogs. *Preventive Veterinary Medicine* 114, 114–22, 2014

Leighton EA, Linn JM, Willham RL, Castleberry MW. A genetic study of canine hip dysplasia. *American Journal of Veterinary Research* 38, 241–4, 1977

Leppänen M, Maki K, Juga J, Saloniemi H. Factors affecting hip dysplasia in German Shepherd dogs in Finland: efficacy of the current improvement programme. *Journal of Small Animal Practice* 41, 19–23, 2000a

Leppänen M, Mäki K, Juga J, Saloniemi H. Estimation of heritability for hip dysplasia in German Shepherd dogs in Finland. *Journal of Animal Breeding and Genetics* 117, 97–103, 2000b

Lewis TW, Blott SC, Woolliams JA. Genetic evaluation of hip score in UK Labrador Retrievers. *PLoS One* 5, e12797, 2010

Lewis TW, Blott SC, Woolliams JA. Comparative analyses of genetic trends and prospects for selection against hip and elbow dysplasia in 15 UK dog breeds. *BMC Genetics* 14, 16, 2013

Lingaas F, Klemetsdal G. Breeding values and genetic trend for hip dysplasia in the Norwegian golden retriever population. *Journal of Animal Breeding and Genetics* 107, 437–43, 1990

Lust G, Farrell PW. Hip dysplasia in dogs: the interplay of genotype and environment. *Cornell Veterinarian* 67, 447–67, 1977

Lust G, Beilman WT, Rendano VT. A relationship between degree of laxity and synovial fluid volume in coxofemoral joints of dogs predisposed for hip dysplasia. *American Journal of Veterinary Research* 41, 55–60, 1980

Mackenzie SA. Why heritability estimates differ: canine hip dysplasia. *Canine Practice* 12, 19–22, 1985

Mackenzie SA, Oltenacu EAB, Leighton EA. Heritability estimate for temperament scores in German shepherd dogs and its genetic correlation with hip dysplasia. *Behavior Genetics* 15, 475–82, 1985

Mäki K, Liinamo AE, Ojala M. Estimates of genetic parameters for hip and elbow dysplasia in finnish rottweilers. *Journal of Animal Science* 78, 1141–8, 2000

Malm S, Fikse WF, Danell B, Strandberg E. Genetic variation and genetic trends in hip and elbow dysplasia in Swedish Rottweiler and Bernese Mountain dog. *Journal of Animal Breeding and Genetics* 125, 403–12, 2008

Malm S, Sørensen AC, Fikse WF, Strandberg E. Efficient selection against categorically scored hip dysplasia in dogs is possible using best linear unbiased prediction and optimum contribution selection: a simulation study. *Journal of Animal Breeding and Genetics* 130, 154–64, 2013

Marschall Y, Distl O. Mapping quantitative trait loci for canine hip dysplasia in German Shepherd dogs. *Mammalian Genome* 18, 861–70, 2007

*__Morgan JP, Stephens M__. *Radiographic Diagnosis and Control of Canine Hip Dysplasia*. Iowa State University Press, Ames, USA, 1985

*__Nicholas FW__. Quantitative variation. In: *Introduction to Veterinary Genetics*. 3rd Edtn. Pp 216–29. Wiley-Blackwell, Chichester, UK, 2010a

*__Nicholas FW__. Familial disorders not due to a single gene. In: *Introduction to Veterinary Genetics*. 3rd Edtn. Pp 139–150. Wiley-Blackwell, Chichester, UK, 2010b

Ohlerth S, Lang J, Busato A, Gaillard C. Estimation of genetic population variables for six radiographic criteria of hip dysplasia in a colony of Labrador Retrievers. *American Journal of Veterinary Research* 62, 846–52, 2001

Paster ER, LaFond E, Biery DN, Iriye A, Gregor TP, Shofer FS, Smith GK. Estimates of prevalence of hip dysplasia in golden retrievers and Rottweilers and the influence of bias on published prevalence figures. *Journal of the American Veterinary Medical Association* 226, 387–92, 2005

Pfahler S, Distl O. Identification of quantitative trait loci (QTL) for canine hip dysplasia and canine elbow dysplasia in Bernese Mountain dogs. *PLoS One* 7, e49782, 2012

Reed AL, Keller GG, Vogt DW, Ellersieck M, Corley EA. Effect of dam and sire qualitative hip conformation scores on progeny hip conformation. *Journal of the American Veterinary Medical Association* 217, 675–80, 2000

Rendano VT, Ryan G. Canine hip dysplasia evaluation. *Veterinary Radiology* 26, 170–86, 1985

Riser WH. A new look at developmental subluxation and dislocation: hip dysplasia in the dog. *Journal of Small Animal Practice* 4, 421–34, 1963

Riser WH. Hip dysplasia as a disease. *Veterinary Pathology* 12, 316–23, 1975a

Riser WH. Observations and research on hip dysplasia. *Veterinary Pathology* 12, 239–63, 1975b

Riser WH. The dysplastic hip joint: radiologic and histologic development. *Veterinary Pathology* 12, 279–305, 1975c

Sánchez-Molano E, Woolliams JA, Blott SC, Wiener P. Assessing the impact of genomic selection against hip dysplasia in the labrador retriever dog. *Journal of Animal Breeding and Genetics* 131, 134–45, 2014

*__Schnelle GB__. Congenital subluxation of the coxofemoral joint in a dog. *University of Pennsylvania Bulletin* 65, 15–6, 1937

Silvestre AM, Ginja MMD, Ferreira AJA, Colaço J. Comparison of estimates of hip dysplasia genetic parameters in Estrela Mountain dog using linear and threshold models. *Journal of Animal Science* 85, 1880–4, 2007

Smith GK, Biery DN, Gregor TP. New concepts of coxofemoral joint stability and the development of a clinical stress-radiographic method for quantitating hip joint laxity in the dog. *Journal of the American Veterinary Medical Association* 196, 59–70, 1990

Smith GK, Popovich CA, Gregor TP. Evaluation of factors contributing to the risk of developing degernative joint disease associated with hip dysplasia in dogs. *Journal of the American Veterinary Medical Association* 206, 642–7, 1995

Smith GK, Paster ER, Powers MY, Lawler DF, Biery DN, Shofer FS, McKelvie PJ, Kealy RD. Lifelong diet restriction and radiographic evidence of osteoarthritis of the hip joints in dogs. *Journal of the American Veterinary Medical Association* 229, 690–3, 2006

Smith GK, Lawler DF, Biery DN, Powers MY, Shofer FS, Gregor TP, Karbe GT, McDonald-Lynch MB, Evans RH, Kealy RD. Chronology of hip dysplasia development in a cohort of 48 labrador retrievers followed for life. *Veterinary Surgery* 41, 20–33, 2012

Steinetz BG, Williams AJ, Lust G, Schwabe C, Büllesbach EE, Goldsmith LT. Transmission of relaxin and estrogens to suckling canine pups via milk and possible association with hip joint laxity. *American Journal of Veterinary Research* 69, 59–67, 2008

Stock KF, Distl O. Simulation study on the effects of excluding offspring information for genetic evaluation versus using genomic markers for selection in dog breeding. *Journal of Animal Breeding and Genetics* 127, 42–52, 2010

Stock KF, Klein S, Tellhelm B, Distl O. Genetic analyses of elbow and hip dysplasia in the German shepherd dog. *Journal of Animal Breeding and Genetics* 128, 219–29, 2011

Swenson L, Audell L, Hedhammar A. Prevalence and inheritance of and selection for hip dysplasia in seven breeds of dogs in Sweden and benefit: cost analysis of a screening and control program. *Journal of the American Veterinary Medical Association* 210, 207–14, 1997

*Todhunter RJ, Lust G. Hip dysplasia: pathogenesis. In: Slatter DH. (ed). *Textbook of Small Animal Surgery*. 3rd Edtn. Pp 2009–19. Saunders, Philadelphia PA, USA, 2003

Todhunter RJ, Bliss SP, Casella G, Wu R, Lust G, Burton-Wurster NI, Williams AJ, Gilbert RO, Acland GM. Genetic structure of susceptibility traits for hip dysplasia and microsatellite informativeness of an outcrossed canine pedigree. *Journal of Heredity* 94, 39–48, 2003

Todhunter RJ, Mateescu R, Lust G, Burton-Wurster NI, Dykes NL, Bliss SP, Williams AJ, Vernier-Singer M, Corey E, Harjes C, *et al*. Quantitative trait loci for hip dysplasia in a cross-breed canine pedigree. *Mammalian genome* 16, 720–30, 2005

Tomlinson JL, Johnson JC. Quantification of measurement of femoral head coverage and Norberg angle within and among four breeds of dogs. *American Journal of Veterinary Research* 61, 1492–500, 2000

Verhoeven G, Coopman F, Duchateau L, Saunders JH, Van Rijssen B, Van Bree H. Interobserver agreement in the diagnosis of canine hip dysplasia using the standard ventrodorsal hip-extended radiographic method. *Journal of Small Animal Practice* 48, 387–93, 2007

Verhoeven GEC, Coopman F, Duchateau LUC, Bosmans TIM, Van Ryssen B, Van Bree H. Interobserver agreement on the assessability of standard ventrodorsal hip-extended radiographs and its effect on agreement in the diagnosis of canine hip dysplasia and on routine FCI scoring. *Veterinary Radiology and Ultrasound* 50, 259–63, 2009

Verhoeven G, Fortrie R, Van Ryssen B, Coopman F. Worldwide screening for canine hip dysplasia: where are we now? *Veterinary Surgery* 41, 10–9, 2012

Vostrý L, Čapková Z, Šebková N, Přibyl J. Estimation of genetic parameters for hip dysplasia in Czech Labrador Retrievers. *Journal of Animal Breeding and Genetics* 129, 60–9, 2012

Wilson B, Nicholas FW, Thomson PC. Selection against canine hip dysplasia: success or failure? *Veterinary Journal* 189, 160–8, 2011

Wilson BJ, Nicholas FW, James JW, Wade CM, Tammen I, Raadsma HW, Castle K, Thomson PC. Heritability and phenotypic variation of canine hip dysplasia radiographic traits in a cohort of Australian German shepherd dogs. *PLoS One* 7, e39620, 2012

Wood JLN, Lakhani KH, Dennis R. Heritability and epidemiology of canine hip-dysplasia score in flat-coated retrievers and Newfoundlands in the United Kingdom. *Preventive Veterinary Medicine* 46, 75–86, 2000a

Wood JLN, Lakhani KH, Dennis R. Heritability of canine hip dysplasia score and its components in Gordon Setters. *Preventive Veterinary Medicine* 46, 87–97, 2000b

Wood JLN, Lakhani KH, Rogers K. Heritability and epidemiology of canine hip dysplasia score and its components in Labrador Retrievers in the United Kingdom. *Preventive Veterinary Medicine* 55, 95–108, 2002

Woolliams JA, Lewis TW, Blott SC. Canine hip and elbow dysplasia in UK Labrador retrievers. *Veterinary Journal* 189, 169–76, 2011

Worth AJ, Laven RA, Erceg VH. An assessment of the agreement between the New Zealand veterinary association hip dysplasia scoring system and the PennHIP distraction index in German Shepherd dogs. *New Zealand Veterinary Journal* 57, 338–45, 2009

Worth AJ, Bridges JP, Jones G. Trends in the phenotypic hip status of selected breeds of dog as measured by the New Zealand veterinary association hip dysplasia scheme (1990–2008). *New Zealand Veterinary Journal* 59, 67–74, 2011

Zhang Z, Zhu L, Sandler J, Friedenberg SS, Egelhoff J, Williams AJ, Dykes NL, Hornbuckle W, Krotscheck U, Moise NS, *et al*. Estimation of heritabilities, genetic correlations, and breeding values of four traits that collectively define hip dysplasia in dogs. *American Journal of Veterinary Research* 70, 483–92, 2009

Zhou Z, Sheng X, Zhang Z, Zhao K, Zhu L, Guo G, Friedenberg SG, Hunter LS, Vandenberg-Foels WS, Hornbuckle WE, *et al*. Differential genetic regulation of canine hip dysplasia and osteoarthritis. *PLoS One* 5, e13219, 2010

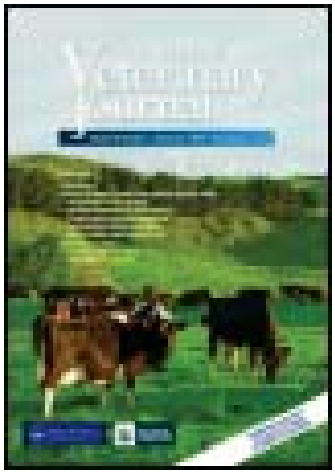
Zhu L, Zhang Z, Feng F, Schweitzer P, Phavaphutanon J, Vernier-Singer M, Corey E, Friedenberg S, Mateescu R, Williams A, *et al*. Single nucleotide polymorphisms refine QTL intervals for hip joint laxity in dogs. *Animal Genetics* 39, 141–6, 2008

Zhu L, Zhang Z, Friedenberg S, Jung S-W, Phavaphutanon J, Vernier-Singer M, Corey E, Mateescu R, Dykes N, Sandler J, *et al*. The long (and winding) road to gene discovery for canine hip dysplasia. *Veterinary Journal* 181, 97–110, 2009

Submitted 22 November 2013

Accepted for publication 10 June 2014

First published online 29 July 2014



New Zealand Veterinary Journal
Publication details, including instructions for authors and subscription information:
http://www.tandfonline.com/loi/tnzv20

Genetic evaluation of the total hip score of four populous breeds of dog, as recorded by the New Zealand Veterinary Association Hip Dysplasia Scheme (1991–2011)

M Soo[ab], NW Sneddon[c], N Lopez-Villalobos[c] & AJ Worth[a]

[a] Massey University Veterinary Teaching Hospital, Institute of Veterinary, Animal and Biomedical Sciences, Massey University, Private Bag 11222, Palmerston North 4442, New Zealand

[b] Cahill Animal Hospital, Palmerston North, New Zealand

[c] Institute of Fundamental Sciences, Massey University, Private Bag 11222, Palmerston North 4442, New Zealand

Accepted author version posted online: 11 Sep 2014.Published online: 27 Jan 2015.

To cite this article: M Soo, NW Sneddon, N Lopez-Villalobos & AJ Worth (2015): Genetic evaluation of the total hip score of four populous breeds of dog, as recorded by the New Zealand Veterinary Association Hip Dysplasia Scheme (1991–2011), New Zealand Veterinary Journal, DOI: 10.1080/00480169.2014.961581

To link to this article: http://dx.doi.org/10.1080/00480169.2014.961581

Scientific Article

Genetic evaluation of the total hip score of four populous breeds of dog, as recorded by the New Zealand Veterinary Association Hip Dysplasia Scheme (1991–2011)

M Soo*†, NW Sneddon‡, N Lopez-Villalobos‡ and AJ Worth*§

Abstract

AIM: To use estimated breeding value (EBV) analysis to investigate the genetic trend of the total hip score (to assess canine hip dysplasia) in four populous breeds of dogs using the records from the New Zealand Veterinary Association (NZVA) Canine Hip Dysplasia Scheme database (1991 to 2011).

METHODS: Estimates of heritability and EBV for the NZVA total hip score of individual dogs from the German Shepherd, Labrador Retriever, Golden Retriever and Rottweiler breeds were obtained using restricted maximum likelihood procedures with a within-breed linear animal model. The model included the fixed effects of gender, birth year, birth season, age at scoring and the random effect of animal. The pedigree file included animals recorded between 1990 and 2011. A total of 2,983 NZVA hip score records, from a pedigree of 3,172 animals, were available for genetic evaluation. Genetic trends of the NZVA total hip score were calculated as the regression coefficient of the EBV (weighted by reliabilities) on year of birth.

RESULTS: The estimates of heritability for hip score were 0.32 (SE 0.08) in German Shepherd, 0.37 (SE 0.08) in Labrador Retriever, 0.29 (SE 0.08) in Golden Retriever and 0.52 (SE 0.18) in Rottweiler breeds. Genetic trend analysis revealed that only the German Shepherd breed exhibited a genetic trend towards better hip conformation over time, with a decline of 0.13 (SE 0.04) NZVA total hip score units per year ($p<0.001$). The genetic trends of total hip score for the remaining three breeds were not significantly different from zero ($p>0.1$).

CONCLUSIONS: Despite moderate heritability of the NZVA total hip score, there has not been substantial improvement of this trait for the four breeds analysed in the study period.

CLINICAL RELEVANCE: Greater improvement in reducing the prevalence of canine hip dysplasia may be possible if screening were to be compulsory as a requirement for registration of pedigree breeding stock, greater selection pressure were to be applied and selection of breeding stock made on the basis on an individual's EBV rather than the NZVA total hip score alone.

KEY WORDS: *Canine hip dysplasia, estimated breeding values, genetic trend, New Zealand Veterinary Association, hip scores, hip laxity*

Introduction

Recognised as one of the most common orthopaedic diseases in dogs, canine hip dysplasia (CHD) is a developmental condition primarily affecting medium- and large-breed dogs. Canine hip dysplasia is characterised by coxofemoral joint instability, leading to osteoarthritis and progressive decline in joint function. This is manifested clinically as coxofemoral joint pain and hind-limb lameness/stiffness. Canine hip dysplasia is first detected radiologically as subluxation of the femoral head in affected puppies. In older patients, it is distinguished by osteophyte development and remodelling of the acetabulum and femoral head (Todhunter and Lust 2003). The inheritance of CHD is considered to be multifactorial, meaning that its expression is influenced by the effect of multiple genes and environmental factors (Bliss *et al.* 2002; Smith *et al.* 2006). Due to the complexity of the inheritance of CHD, there is a current lack of readily available genomic tests for routine clinical use. In the absence of feasible methods for direct clinical assessment of CHD, radiological scoring/grading methods have been developed and are widely used to identify dogs less phenotypically affected by the disease.

The New Zealand Veterinary Association (NVZA) introduced a "Hip Scheme" in the mid 1980s (Hunter 1986) as a method for identifying dogs with CHD. In co-operation with the New Zealand Kennel Club (NZKC), the NZVA hip scoring records were maintained in a national computerised database from 1989. The NZVA CHD Scheme is based on the British Veterinary Association (BVA)/Kennel Club CHD scoring method

* Massey University Veterinary Teaching Hospital, Institute of Veterinary, Animal and Biomedical Sciences, Massey University, Private Bag 11222, Palmerston North 4442, New Zealand
† Cahill Animal Hospital, Palmerston North, New Zealand
‡ Institute of Fundamental Sciences, Massey University, Private Bag 11222, Palmerston North 4442, New Zealand
§ Author for correspondence: Email: a.j.worth@massey.ac.nz

http://dx.doi.org/10.1080/00480169.2014.961581

BVA	British Veterinary Association
CHD	Canine hip dysplasia
EBV	Estimated breeding value
EHV	Extended hip view
h^2	Estimated heritability
NZKC	New Zealand Kennel Club
NZVA	New Zealand Veterinary Association

(Burbidge 2003; Worth *et al.* 2009). In order to reduce the prevalence of CHD via selective breeding, the trait being selected for/against must be genetically influenced, i.e. possess heritability, and be directly measurable on live animals. The higher the heritability, the greater the influence of genetic effects on the trait and thus the greater the response to selection (Mackenzie 1985). Recognition of the heritable nature of CHD and the use of effective breeding selection methods is critical to achieve a reduction in the prevalence of the disease. To date, a number of studies have shown that the BVA phenotype possesses sufficient heritability to manipulate the prevalence of CHD through selective breeding (Lewis *et al.* 2010, 2013; Wilson *et al.* 2012). However a report by Willis (1997), which analysed the BVA hip score trends in six different breeds, concluded that the expected progress towards lower hip scores had not been achieved at that time. In New Zealand, work by Worth *et al.* (2011) showed a small but significant trend towards an improved phenotypic NZVA hip conformation in German Shepherd dogs, but not in the remaining three breeds analysed (Labrador Retriever, Golden Retriever and Rottweiler) in the period 1990–2008.

The phenotypic trend is the sum of the environmental and genetic trends. However, the chosen method of regression analysis in the Worth *et al.* (2011) study was based on individual animal scores and may have overlooked the genetic improvement made in some lines within breeds, by not evaluating the genetic trend. The genetic trend can be determined by the regression coefficient of estimated breeding values (EBV) on year of birth of the animals. The EBV is a measure of the genetic superiority of an animal as compared to its contemporaries and is calculated from the phenotypes of the individual and its relatives, and pedigree data (Nicholas 2010). Therefore, an analysis of the genetic trend calculated using EBV may provide a more accurate indication of the amount of genetic progress attained by the respective breeds towards hips with less laxity, as compared to using the phenotypic trend. Genetic trend analysis of the hip scoring data held by the NZVA has not been previously reported.

The aims of this study were to develop a within-breed animal evaluation system to estimate the heritability of the NZVA hip phenotype, to obtain breeding values for total hip score and to estimate the genetic trends for this trait in four breeds of dogs scored under the NZVA CHD Scheme. The breeds considered in this study were the German Shepherd dog, Labrador Retriever, Golden Retriever and Rottweiler.

Materials and methods

The database of the NZVA CHD Scheme was made available for this study. Records of the four most populous breeds in the NZVA database (German Shepherd dog, Labrador Retriever, Golden Retriever and Rottweiler) were selected for analysis. Data consisting of each dog's unique NZKC registration number, date of birth, gender, date of radiography, age at radiographic scoring, subtotal and total hip score, were collated by breed. If a dog's gender was missing, the data were corrected from NZKC records. Under the rules of the NZVA CHD Scheme, a dog must be at least 12 months of age to be eligible for scoring, hence the year of birth precedes the year of hip scoring (radiography). Though many breeders present dogs for scoring soon after they are 12 months of age, some dogs are not scored until they are older, so dogs scored in the same year can have different birth years. Age at scoring (in days) was determined by subtracting the date of birth from date of radiography. Age at scoring was then divided by 30.4 to give an age at scoring in months. The season during which the animals were born was determined from the month of birth. Summer was designated to cover December to February, autumn was assigned to March to May, winter was allocated to June to August and spring was September to November.

In order to generate breeding values, pedigree information on all animals scored under the NZVA CHD Scheme was obtained from the NZKC and sorted by breed. The pedigree file was created by extracting the names of animal, sire and dam and tracked back until the first animal was recorded. The unique NZKC identification number for each named individual allowed the generation of a pedigree database. Missing parents or grandparents were denoted as 0 to indicate unknown individuals. The NZKC pedigree information was sorted and cleaned using the Structured Query Language procedure of SAS 9.3 (SAS Institute Inc., Cary NC, USA, 2011). All animals included in the study were hip scored between 1991 and 2011. A total of 3,172 dogs born between 1988 and 2010 were included in the pedigree dataset, which was sub-categorised for each breed. After the removal of dogs with inconsistent NZVA hip scoring data (such as incorrectly entered radiography dates or the same animal having two different hip scores), a total of 2,983 NZVA hip scoring records from a pedigree of 3,172 animals were included in the study.

The German Shepherd pedigree contained a total of 848 animals over four generations. There were 110 sires, 18 sires of sires, 37 dams of sires, 249 dams, 41 sires of dams, and 72 dams of dams, with 322 dogs in the most recent generation. There were 547 females and 256 males in this pedigree; no hip scoring data were available for 45 dogs. Fifty-eight German Shepherd dogs were identified as having been bred by the New Zealand Police Dog Breeding Centre (Trentham, New Zealand). The EBV analysis was performed on this subset of German Shepherd dogs and the remaining pedigree file without the Police dogs.

The Labrador Retriever pedigree contained a total of 1,277 animals over six generations. There were 163 sires, 41 sires of sires, 48 dams of sires, 370 dams, 72 sires of dams and 123 dams of dams, with 461 dogs in the most recent generation. There were 843 females and 363 males in this pedigree; no hip scoring data were available for 71 dogs.

The Golden Retriever pedigree contained a total of 770 animals over seven generations. There were 103 sires, 25 sires of sires, 34 dams of sires, 228 dams, 45 sires of dams and 78 dams of dams, with 258 dogs in the most recent generation. There were 500 females and 228 males in this pedigree; no hip scoring data were available for 42 dogs.

The Rottweiler pedigree contained a total of 277 animals over four generations. There were 39 sires, 4 sires of sires, 5 dams of sires, 78 dams, 9 sires of dams, and 14 dams of dams, with 128 dogs in the most recent generation. There were 155 females and 88 males in this pedigree; no hip scoring data were available for 34 dogs.

Genetic analysis

The NZVA total hip score data distribution was not significantly different from a Normal distribution on the basis of the Sharpiro-Wilk, Kolmogorov-Smirnov, Cramer-von Mises or Anderson-Darling tests. Therefore, each breed's hip data were treated as normally distributed for further analysis.

Estimates of variance components and EBV for the NZVA total hip score were obtained using ASReml software program release 3.0 (VSN International Ltd, Hemel Hempstead, UK) with a single trait linear animal model within-breed. The model included the fixed effects of gender, year of birth, season of birth, age at scoring, the random animal effect and residual error for each observation. In matrix form, the model can be written as follows:

$$[\mathbf{y}] = [\mathbf{X}][\mathbf{b}] + [\mathbf{Z}][\mathbf{u}] + [\mathbf{e}]$$

where,

y	is the vector of observations for hip score.
b	is the vector of fixed effects of year of birth, season of birth, gender and as a covariable age at scoring.
u	is a vector of random animal effects.
e	is the vector of residual errors.
X and **Z**	are design matrices for fixed and random animal effects, respectively.

The expectation of **y**, **u** and **e** are assumed to be:

$$\mathrm{E}\begin{bmatrix}\mathbf{y}\\ \mathbf{u}\\ \mathbf{e}\end{bmatrix} = \begin{bmatrix}\mathbf{Xb}\\ \mathbf{0}\\ \mathbf{0}\end{bmatrix}$$

with variance-covariances matrices

$$\mathrm{var}\begin{bmatrix}\mathbf{u}\\ \mathbf{e}\end{bmatrix} = \begin{bmatrix}\mathbf{G} & \mathbf{0}\\ \mathbf{0} & \mathbf{R}\end{bmatrix}$$

and

$$\mathrm{var}(\mathbf{y}) = \mathbf{V} = \mathbf{ZGZ}' + \mathbf{R}$$

The structure of **G** and **R** were assumed to be

$$\mathbf{G} = \sigma_a^2\mathbf{A},$$

and

$$\mathbf{R} = \sigma_e^2\mathbf{I},$$

therefore

$$\mathrm{var}\begin{bmatrix}\mathbf{u}\\ \mathbf{e}\end{bmatrix} = \begin{bmatrix}\alpha^{-1}\mathbf{A} & \mathbf{0}\\ \mathbf{0} & \mathbf{I}\end{bmatrix}\sigma_e^2$$

with

$$\alpha^{-1} = \frac{\sigma_a^2}{\sigma_e^2}$$

where, **A** is the numerator relationship matrix between individuals in **u**, σ_a^2 is the animal variance, **I** is an identity matrix and σ_e^2 is the residual variance.

Estimates of the animal and residual variances and the solution of the random animals effects (vector **u**), were obtained using restricted maximum likelihood analysis. The estimated heritability (h^2) for total hip score was calculated as follows:

$$h^2 = \frac{\sigma_a^2}{\sigma_a^2 + \sigma_\theta^2}$$

The estimate of animal variance is equal to the additive genetic variance, and therefore phenotypic variance (σ_p^2) is estimated as $\sigma_p^2 = \sigma_a^2 + \sigma_\theta^2$. Estimated breeding value for animal (i) was obtained as $EBV_i = u_i$ where u_i is the estimate of animal effect from the mixed model. The reliability of EBV was estimated as

$$\text{Reliability} = \left(1 - \left(\frac{\mathrm{SE}}{\sigma_a}\right)^2\right) \times 100$$

where SE is the standard error of the estimate of animal effect and σ_a is the square root of the animal variance for the NZVA total hip score.

Even though some of the dogs in the pedigree records did not have a NZVA total hip score, their offspring were still able to be identified through the NZKC pedigree dataset and an EBV was generated for each animal based on the mean performance of their offspring. A negative EBV is interpreted as the animal having favourable (lower) total hip score compared to the mean of the breed population. A positive EBV is interpreted as the animal having unfavourable (higher) total hip score compared to the mean of the breed population. Therefore, a negative EBV is the goal for selection.

Statistical analysis

Genetic trend of the NZVA total hip score for each of the breeds were estimated as the weighted regression line of EBV on year of birth using SAS 9.3 (SAS Institute Inc., Cary NC, USA 2011). The EBV were weighted by their reliabilities. A favourable genetic trend (improvement in the hip phenotype within a breed over time) would be represented by a negative regression line of EBV on birth year. This regression analysis can be considered as an indirect measure of the genetic trend over time to evaluate the efficiency of a breeding programme. However, this regression analysis was not an attempt to use birth year as a predictor of animal EBV.

Within the German Shepherd dogs, 58 were identified as having been bred by the New Zealand Police Dog Breeding Centre (Trentham, New Zealand) and genetic trends were obtained for police and non-police dogs.

Results

Descriptive statistics

Table 1 presents the descriptive statistics for total hip score for all data and by breed. The mean score for the Labrador Retriever breed was higher than the other breeds but the German Shepherd dog had the largest SD and range. The coefficients of skewness for the whole dog population and for each of the breeds indicate that the distributions of NZVA total hip score were skewed to the right, but still followed a normal distribution based on the normality tests.

Estimates of heritability

Table 2 presents the estimates of phenotypic, additive genetic and residual variances, and the h^2 of the NZVA total hip score for the four breeds of dogs examined in this study. The Rottweiler had the highest h^2 and the Golden Retriever had the lowest h^2 amongst the four breeds analysed. The German Shepherd dog had the largest phenotypic and residual variances in the study.

Genetic trends

Figures 1–4 show the genetic trends of total hip score for each of the breeds. Amongst the four breeds investigated, the largest amount of genetic change was seen in the German Shepherd breed at −0.13 (SE 0.04) NZVA total hip score units per year;

Table 1. Descriptive statistics for total hip score in the German Shepherd, Labrador Retriever, Golden Retriever, and Rottweiler dog breeds recorded in the New Zealand Veterinary Association Canine Hip Dysplasia database from 1991–2011.

Breed	n	Mean	SD	Min, max	Skewness
German Shepherd	804	10.5	11.7	0, 96	4.5
Labrador Retriever	1,207	8.9	6.8	0, 82	3.9
Golden Retriever	729	12.2	6.4	1, 59	2.6
Rottweiler	243	8.2	6.7	0, 65	3.7
All	2,983	10.9	8.5	0, 96	4.5

Table 2. Estimates of heritabilities (h^2±SE), phenotypic, additive genetic and residual variances for the total hip score in the German Shepherd, Labrador Retriever, Golden Retriever, and Rottweiler dog breeds recorded in the New Zealand Veterinary Association Canine Hip Dysplasia database from 1991–2011.

Breed	h^2	Phenotypic variance[a]	Additive genetic variance[b]	Residual variance[c]
German Shepherd	0.33 ± 0.08	140.1	45.7	94.4
Labrador Retriever	0.37 ± 0.08	47.0	17.3	29.7
Golden Retriever	0.30 ± 0.08	40.7	12.2	28.5
Rottweiler	0.56 ± 0.20	46.4	25.9	20.5

[a] Phenotypic variance is defined as the extent to which individuals differ in their observed trait values.
[b] Additive genetic variance is defined as the extent to which animals differ in their breeding values.
[c] Residual variance represents the portion of the phenotypic variance unexplained by the model.

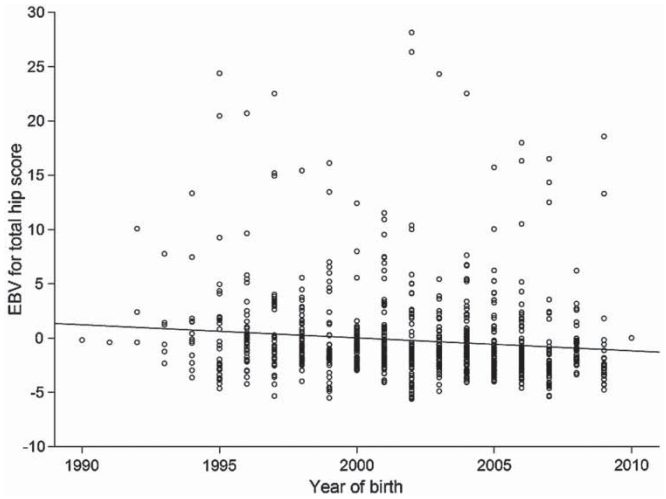


Figure 1. Genetic trend of total hip score estimated breeding value (EBV) for German Shepherd dogs recorded in the New Zealand Veterinary Association Canine Hip Dysplasia database from 1990–2010.

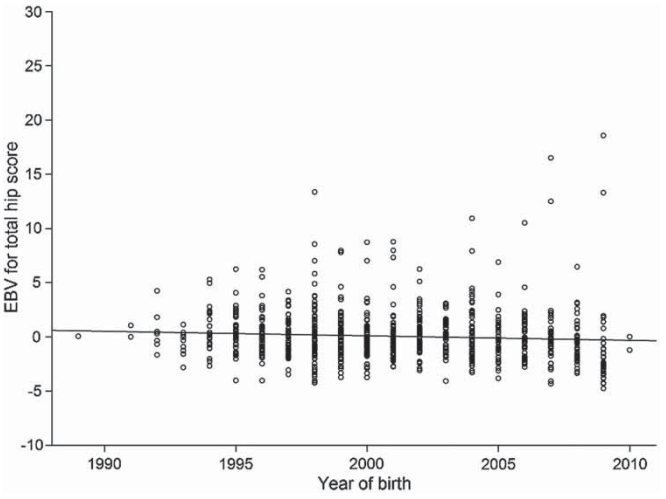


Figure 2. Genetic trend of total hip score estimated breeding value (EBV) for Labrador Retrievers recorded in the New Zealand Veterinary Association Canine Hip Dysplasia database from 1989–2010.

the slope of the regression line was significantly different from zero (p=0.001). The R^2 for this regression model was 0.01. The genetic trend for total hip score for the 58 German Shepherd dogs that were bred by the New Zealand Police dog breeding programme was −0.22 (SE 0.17) total hip score units per year and the genetic trend for the remaining 746 non-Police dogs was −0.11 (SE 0.04). The difference between the genetic trends in these two groups was significant (p=0.009). The genetic trends of total hip score for the Labrador Retriever, Golden Retriever, and Rottweiler breeds were −0.02 (SE 0.02), −0.02 (SE 0.02) and −0.08 (SE 0.06), respectively, and were not significantly different from zero (p>0.1).

Discussion

Genetic analysis of the NZVA total hip score data demonstrated moderate heritability in three of the four breeds analysed and high heritability in the Rottweiler. Traits with h^2 of 0.20–0.39 are considered to be moderately heritable, and possess sufficient genetic influence such that selective breeding should decrease the prevalence of the disease (Mackenzie 1985). Heritability estimates are unique to the population and method of analysis, but as the NZVA CHD Scheme is based on the BVA scoring method it is reasonable to compare the h^2 values in this study to those of

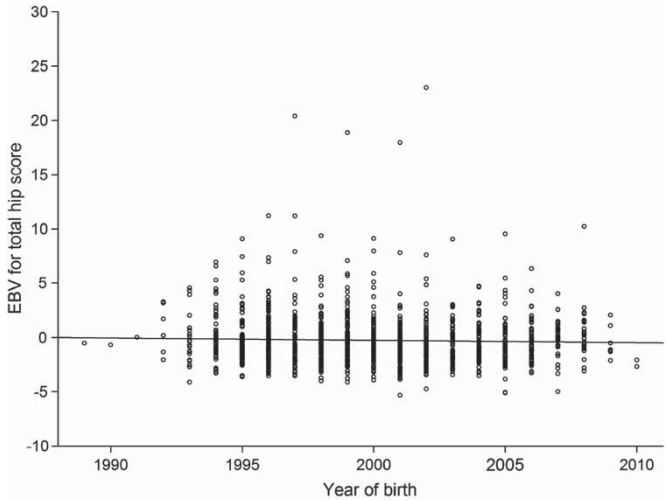


Figure 3. Genetic trend of total hip score estimated breeding value (EBV) for Golden Retrievers recorded in the New Zealand Veterinary Association Canine Hip Dysplasia database from 1989–2010.

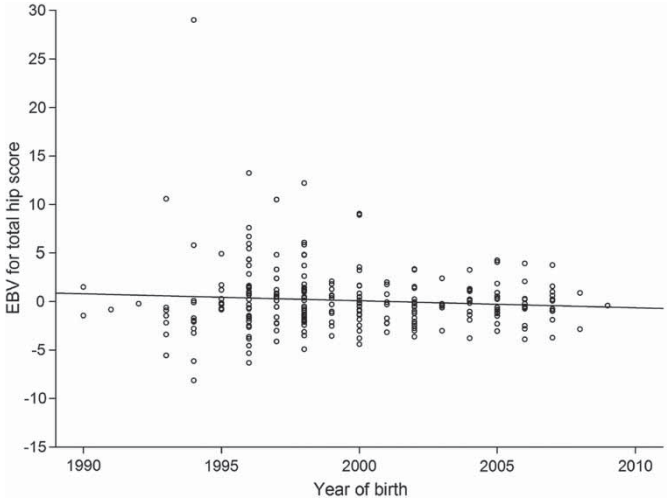


Figure 4. Genetic trend of total hip score estimated breeding value (EBV) for Rottweilers recorded in the New Zealand Veterinary Association Canine Hip Dysplasia database from 1990–2009.

the BVA phenotype. Wilson *et al.* (2012) found that the h^2 for the BVA hip phenotype was 0.30 (SE 0.02) for German Shepherd dogs in Australia, which is similar to our results for the German Shepherd breed. Other recent studies found the h^2 for the BVA hip phenotype to be 0.34–0.35 for the Labrador Retriever breed, obtained via a variety of estimation methods (Lewis *et al.* 2010, 2013; Woolliams *et al.* 2011). These values are similar to the h^2 obtained for the Labrador Retrievers in our study, using the NZVA data. Lewis *et al.* (2013) found that the h^2 of the BVA phenotype was 0.40 (SE 0.017) for the Golden Retriever and 0.39 (SE 0.028) for the Rottweiler, which are also similar to the h^2 of these two breeds in our study. It is important to note that the German Shepherd breed in the current study had the largest phenotypic variance for total hip score arising from both greater additive genetic and greater residual variances than the other breeds. A large additive genetic variance indicates a wider range of animals for breeders to select from, with the potential of obtaining higher genetic gains if a breeding programme is implemented for the improvement of CHD. This is of relevance because the residual variance is a component of phenotypic variance. As a result of the closely intertwined relationships between response to selection, h^2 and phenotypic variation, a large residual variance will ultimately also impact the response to selection. The larger the additive genetic variance, the larger will be the genetic response seen if a breeding programme was implemented to reduce the NZVA total hip score.

The coefficient of regression of EBV weighted by reliabilities, on year of birth was negative and significantly different from zero in the German Shepherd dog population, indicating a significant genetic reduction in the NZVA total hip score from 1991–2011. This suggests that sufficient selection pressure has been applied to this breed to improve NZVA hip phenotype over time, consistent with the previous findings of Worth *et al.* (2011). For many of the years of this current study, the New Zealand Police dog breeding programme utilised the NZVA CHD Scheme for phenotypic scoring and 58 New Zealand Police German Shepherd dogs were included in the pedigree dataset used in this study. German Shepherd dogs bred by the New Zealand Police had a genetic trend that was twice that of the non-Police bred German Shepherd dogs, indicating greater active selection for superior hip conformation. Thus the inclusion of the New Zealand Police dogs favourably influenced the overall genetic trend of the breed. While the genetic trend for the NZVA total hip score in the German Shepherd breed was statistically significant and in a favourable direction towards better hip conformation, in practical terms, the magnitude of the change was low, equivalent to lowering of the total hip score by only approximately two hip score units over 20 years. Moreover, the R^2 value of the regression model was very low indicating that total hip score EBV were widely spread around the regression line indicating that the genetic change for total hip score in the population has been very variable, as shown in Figure 1. Other authors have performed log-transformation to improve the normality of the hip score data prior to genetic analysis (Lewis *et al.* 2010, 2013; Woolliams *et al.* 2011). Our genetic analysis was performed without log-transformation on the basis that the dataset satisfied tests for normality. In order to confirm that the genetic trend was not influenced by the data distribution, a log-transformation of the German Shepherd data was performed post-analysis. The correlation coefficient between the untransformed and log-transformed EBV was 0.87, indicating that both methods would yield similar results.

The genetic change observed in the Golden Retriever, Labrador Retriever and Rottweiler breeds were not significantly different from zero, indicating that less selection intensity had been applied to reduce the NZVA total hip score in these breeds compared to the German Shepherd dog.

In New Zealand, the Labrador Retriever is commonly utilised by the Blind Foundation Guide Dog Service (formerly Royal New Zealand Foundation of the Blind). Similar to the police dogs, Labrador Retriever guide dogs are actively selected for superior hip status. However, as previously noted by Worth *et al.* (2011), their hip data are not included in the NZVA CHD database because hip conformation of Blind Foundation guide dogs is assessed by a private CHD film-review service.

Comparing genetic trends between studies and especially between different hip scoring systems is problematic as EBV analyses are very specific to the phenotype (hip scoring system), breed and population on which the analysis is based (Mackenzie 1985; Wilson *et al.* 2011). Nonetheless, a comparison between the BVA and NZVA hip scoring systems may be valid, because

they utilise the same radiographic hip phenotype. The reported genetic change for the BVA phenotype in the Labrador Retriever was 0.376 untransformed hip score units per annum (Lewis *et al.* 2010) which, is almost three times greater than the genetic change observed for the German Shepherd dogs in this study. This implies that using the same phenotype for selection, greater genetic change has been achieved in the United Kingdom compared to New Zealand. Given the comparable estimates of h^2, our analysis would indicate that, as compared to the United Kingdom, relatively lower selection intensity has been applied to these four breeds within New Zealand.

A number of plausible reasons may be conjectured to explain the limited genetic improvement seen in the NZVA total hip score found in this study. Worth *et al.* (2011) showed that less than a quarter of whelped German Shepherd dogs, Labrador Retrievers, Golden Retrievers and Rottweilers registered with the NZKC between the years 1990–2008 inclusive, were scored under the NZVA CHD scheme. There was also a notable decline in percentage of registered dogs that were scored between 2005 and 2008, though younger dogs may not have been scored at the time of the study (Worth *et al.* 2011). The authors also acknowledged the possibility that dogs not scored under the NZVA CHD Scheme may have been scored under other hip scoring systems such as the Australian Veterinary Association CHD Scheme, PennHIP (formerly the University of Pennsylvania Hip Improvement Program) or private CHD film-review services (Worth *et al.* 2011). The NZVA CHD scheme is not a genetic disease control programme but a voluntary scheme which is intended to assist breeders to select dogs with a favourable hip phenotype. Hip dysplasia scoring is not a requirement of pedigree registration with the NZKC, and the NZVA scheme is only selectively used by breeders (Burbidge 2003). Whilst the NZVA provides recommendations for selection of phenotypically superior breeding stock, there is no regulation or means of compulsion to follow them. Therefore, breeding selection is not tightly regulated, and any possible genetic improvement can be diluted by the proportion of random selection occurring within the population. Pre-screening also lowers submission rates and affects selection intensity. Pre-screening occurs when veterinarians or breeders fail to submit the radiographs of obviously radiologically abnormal dogs for evaluation under the NZVA CHD Scheme (Worth *et al.* 2011). Additionally, the lack of active selection pressure against CHD can also occur when breeders are more focused on selection for or against other genetic diseases. Hip conformation is one of the many characteristics a dog breeder has to consider when a breeding assessment is made. These factors may have contributed to the limited genetic improvement in the NZVA total hip score found in this study.

Alternatively, if breeding selection is based on a phenotypic measure with poor reliability, the rate of genetic improvement towards better hip conformation may be negatively affected. The accuracy and reliability of the ventrodorsal extended hip view (EHV) in identifying the presence of hip dysplasia in dogs has been questioned (Kapatkin *et al.* 2004; Powers *et al.* 2010). The EHV was found to have a relatively higher rate of false negative diagnoses and severely underestimated the number of dogs with coxofemoral joint laxity, as compared to the distraction (stress) radiographic view employed under the PennHIP scoring method (Kapatkin *et al.* 2004; Powers *et al.* 2010). This is because the EHV positioning of the hindlimbs (as utilised for the NZVA hip radiographs) places the coxofemoral joint in its tightest (least lax) position due to increased tension in the joint capsule and capital ligament (Smith *et al.* 1990). This masks the true extent of laxity of the coxofemoral joint and falsely lowers the NZVA hip score as a result. In contrast, the distraction index, as determined by the PennHIP method of hip scoring maximises passive hip laxity (Smith *et al.* 1990). While there is good agreement between the EHV and distraction radiographic views in identifying the normal or tight hip, investigators have found that distraction radiography is superior to the EHV in the detection of the dysplastic hip (Kapatkin *et al.* 2004; Powers *et al.* 2010). Hence, to maximise the response to selection for good hip conformation, breeders should consider the move towards using a more reliable hip scoring method for CHD.

Breeding selection strategies also have a role in the rate of genetic improvement. It is critical to realise that throughout the period examined by this study, NZKC-registered breeders did not have EBV data available to them on which to base breeding selections. Therefore, calculation and provision of EBV information to New Zealand dog breeders would be advantageous. An EBV-based selection strategy is a more accurate indicator of an animal's genetic superiority because it provides valuable information of the genetic risk of CHD being transmitted to the offspring, as compared to using its radiographic hip phenotype (Soo and Worth 2014). Therefore, calculation and provision of EBV information to New Zealand dog breeders would be advantageous. As the extent of genetic progress is directly correlated with the accuracy of breeding selection, greater genetic improvement could have been achieved with EBV-based selection as compared to hip phenotype-based selection (Lewis *et al.* 2013; Malm *et al.* 2013; Wilson *et al.* 2013). In simulated populations, while an EBV-based selection scheme is shown to be more efficient in achieving genetic progress than phenotypic selection, an even greater response to selection can be obtained with genomic (DNA marker-assisted) selection (Stock and Distl 2010; Sánchez-Molano *et al.* 2014). However, there is only limited use of DNA marker technology at present (Woolliams *et al.* 2011) which is still undergoing development and refinement for routine clinical use. Therefore, in the short term at least, it is in the authors' view that breeding selection on the basis of EBV of the phenotypic hip score remains the next best alternative to genomic selection. While the use of EBV to select against CHD is still in its infancy stage, it carries a lot of potential if utilised appropriately as part of selective breeding schemes to effectively enhance the rate of genetic gain towards better hip conformation in dogs.

In conclusion, this study demonstrates that the NZVA total hip score phenotype in the four studied breeds has sufficient heritability to allow for genetic improvement through the use of selective breeding, provided that there is adequate selection intensity. Based on the EBV analysis of the NZVA total hip scores, only the German Shepherd dog exhibited significant genetic change towards better hip conformation, but even then, the magnitude of genetic change was not substantial. There was no genetic change in the remaining three breeds over the study period, suggesting random selection or very low selection intensity towards a better hip phenotype. Greater genetic improvement could be achievable if EBV were to be used for selection against CHD instead of individual phenotypic records.

Declaration of interest

Andrew Worth is the current Convenor of the NZVA Hip and Elbow Dysplasia Scheme and receives an honorarium for this position. He is also a PennHIP-certified veterinarian, whose training was supported by the New Zealand Police.

Acknowledgements

The authors would like to thank the New Zealand Companion Animal Health Fund, Massey University, The Golden Retriever Club Inc., the Tasman District Gundog Society, the Wanganui & Districts Gundog Society and Keridale Golden Retrievers for their generosity in funding this study. The authors would also like to thank Trish Thorpe of NZVA, Aaron Fulton of VetLearn and Pam Douglas of NZKC for the extraction, provision and spread-sheeting of the data.

References

Bliss S, Todhunter RJ, Quaas R, Casella G, Wu R, Lust G, Williams AJ, Hamilton S, Dykes NL, Yeager A, et al. Quantitative genetics of traits associated with hip dysplasia in a canine pedigree constructed by mating dysplastic Labrador Retrievers with unaffected Greyhounds. *American Journal of Veterinary Research* 63, 1029–35, 2002

*__Burbidge HM__. Changes proposed for inherited disorder control schemes. *VetScript* 16 (7), 28–9, 2003

Hunter AC. Radiography for hip dysplasia evaluation. *New Zealand Veterinary Journal* 34, 43–5, 1986

Kapatkin AS, Gregor TP, Hearon K, Richardson RW, McKelvie PJ, Fordyce HH, Smith GK. Comparison of two radiographic techniques for evaluation of hip joint laxity in 10 breeds of dogs. *Journal of the American Veterinary Medical Association* 224, 542–6, 2004

Lewis TW, Blott SC, Woolliams JA. Genetic evaluation of hip score in UK Labrador Retrievers. *PLoS One* 5, e12797, 2010

Lewis TW, Blott SC, Woolliams JA. Comparative analyses of genetic trends and prospects for selection against hip and elbow dysplasia in 15 UK dog breeds. *BMC genetics* 14, 16, 2013

Mackenzie SA. Why heritability estimates differ: Canine hip dysplasia. *Canine Practice* 12, 19–22, 1985

Malm S, Sørensen AC, Fikse WF, Strandberg E. Efficient selection against categorically scored hip dysplasia in dogs is possible using best linear unbiased prediction and optimum contribution selection: a simulation study. *Journal of Animal Breeding and Genetics* 130, 154–64, 2013

*__Nicholas FW__. Quantitative variation. In: *Introduction to Veterinary Genetics*. 3rd Edtn. Pp 216–9. Wiley-Blackwell, Chichester, United Kingdom, 2010

Powers MY, Karbe GT, Gregor TP, McKelvie P, Culp WT, Fordyce HH, Smith GK. Evaluation of the relationship between Orthopedic Foundation for Animals' hip joint scores and PennHIP distraction index values in dogs. *Journal of the American Veterinary Medical Association* 237, 532–41, 2010

Sánchez-Molano E, Woolliams JA, Blott SC, Wiener P. Assessing the impact of genomic selection against hip dysplasia in the Labrador Retriever dog. *Journal of Animal Breeding and Genetics* 131, 134–45, 2014

Smith GK, Biery DN, Gregor TP. New concepts of coxofemoral joint stability and the development of a clinical stress-radiographic method for quantitating hip joint laxity in the dog. *Journal of the American Veterinary Medical Association* 196, 59–70, 1990

Smith GK, Paster ER, Powers MY, Lawler DF, Biery DN, Shofer FS, McKelvie PJ, Kealy RD. Lifelong diet restriction and radiographic evidence of osteoarthritis of the hip joint in dogs. *Journal of the American Veterinary Medical Association* 229, 690–3, 2006

Soo M, Worth AJ. Canine hip dysplasia: Phenotypic scoring and the role of estimated breeding value analysis. *New Zealand Veterinary Journal* doi:10.1080/00480169.2014.949893, 2014

Stock KF, Distl O. Simulation study on the effects of excluding offspring information for genetic evaluation versus using genomic markers for selection in dog breeding. *Journal of Animal Breeding and Genetics* 127, 42–52, 2010

*__Todhunter RJ, Lust G__. Hip dysplasia: Pathogenesis. In: Slatter D (ed). *Textbook of Small Animal Surgery*. Pp 2009–19. Saunders, Philadelphia, PA, USA, 2003

Willis MB. A review of the progress in canine hip dysplasia control in Britain. *Journal of the American Veterinary Medical Association* 210, 1480–2, 1997

Wilson B, Nicholas FW, Thomson PC. Selection against canine hip dysplasia: Success or failure? *Veterinary Journal* 189, 160–8, 2011

Wilson BJ, Nicholas FW, James JW, Wade CM, Tammen I, Raadsma HW, Castle K, Thomson PC. Heritability and phenotypic variation of canine hip dysplasia radiographic traits in a cohort of Australian German Shepherd dogs. *PLoS One* 7, e39620, 2012

Wilson B, Nicholas FW, James JW, Wade CM, Thomson PC. Estimated breeding values for canine hip dysplasia radiographic traits in a cohort of Australian German Shepherd dogs. *PLoS One* 8, e77470, 2013

Woolliams JA, Lewis TW, Blott SC. Canine hip and elbow dysplasia in UK Labrador Retrievers. *Veterinary Journal* 189, 169–76, 2011

Worth AJ, Laven RA, Erceg VH. An assessment of the agreement between the New Zealand Veterinary Association hip dysplasia scoring system and the PennHIP distraction index in German Shepherd dogs. *New Zealand Veterinary Journal* 57, 338–45, 2009

Worth AJ, Bridges JP, Jones G. Trends in the phenotypic hip status of selected breeds of dog as measured by the New Zealand Veterinary Association Hip Dysplasia scheme (1990–2008). *New Zealand Veterinary Journal* 59, 67–74, 2011

Submitted 25 November 2013

Accepted for publication 26 August 14

First published online 11 September 2014

*Non-peer-reviewed

Telephone questionnaire used to assess the outcome of surgical management of degenerative lumbosacral stenosis in GSDs of the NZ PDS.

Hi this is Lauren from the Massey Vet Clinic, where *name* had surgery *when*. We are doing a study, looking at the outcome of these types of surgeries on working dogs. Would it be possible to ask you a few questions about your dog?

1. Date of survey completion 2. Your Name

3. Your assigned Police District at the time this dog had surgery

4. Dog's Name 5. Dog's birth date

6. Date this dog began service with you as handler

7. What signs was your dog showing prior to the diagnosis of lumbosacral disease (select as many as apply) 0 normal, 1 mild, 2 moderate, 3 severe problem

	Mild	Mod	Sev
Poor bite work / man work	[ ]	[ ]	[ ]
Poor tracking	[ ]	[ ]	[ ]
Decreased endurance	[ ]	[ ]	[ ]
Loss of interest, less keen to work	[ ]	[ ]	[ ]
Difficulty jumping into vehicle	[ ]	[ ]	[ ]
Difficulty scaling walls/fences	[ ]	[ ]	[ ]
Displaying pain/yelping	[ ]	[ ]	[ ]
Displaying pain when handled	[ ]	[ ]	[ ]
Abnormally aggressive when handled	[ ]	[ ]	[ ]
A limp when walking / trotting	[ ]	[ ]	[ ]
A limp when running	[ ]	[ ]	[ ]
Lifting/carrying leg after jumping	[ ]	[ ]	[ ]

Other (please specify) _____

8. Did your dog return to work as a Police Dog after lumbosacral surgery?

YES How many months after surgery before he was back at work?

_____

NO Describe the reasons your dog was not returned to work:

_____

➔ skip to Q16

9. If your dog returned to work, how long do you estimate it took for him to either fully recover or perform to his maximum ability?

10. If your dog did return to work, how would you rate its ability?

☐ Able to perform full duties including bite work, tracking, scaling fences

☐ Able to perform most duties but some allowances had to be made
Specify what the dog cannot do
_____

☐ Able to perform limited duties
Specify what the dog is allowed to do
_____

☐ The dog required transferral to another role _____

☐ Semi-retired but still kept in training and occasionally used
Specify what the dog is used for and allowed to do
_____

11. On scale of 1 to 10, how would you rate the success of the surgery at returning your dog to active duty? 1 being much worse, 5 no change, 10 an exceptional result

12. Using the same signs we discussed as being present prior to surgery, which signs are/were still present even after recovery from surgery. 0 normal, 1 mild, 2 moderate, 3 severe problem

		Mild	Mod	Sev
Poor bite work / man work	[ ]			
Poor tracking	[ ]			
Decreased endurance	[ ]			
Loss of interest, less keen to work	[ ]			
Difficulty jumping into vehicle	[ ]			
Difficulty scaling walls/fences	[ ]			
Displaying pain/yelping	[ ]			
Displaying pain when handled	[ ]			
Abnormally aggressive when handled	[ ]			
A limp when walking / trotting	[ ]			
A limp when running	[ ]			
Lifting/carrying leg after jumping	[ ]			

Other (please specify) _____

13. Where there any complications as a result of the surgery? Please specify:

14. Is this dog still in active service as a Police dog?

NO Date the dog left service - _____

YES ➔ Skip to question 17

15. If your dog returned to work, but the effect of surgery was temporary and he had to be subsequently retired/euthanased, how long was he able to remain in work?

16. If the dog is no longer in active duty as a Police dog was it

- ☐ Retired due to inability to continue to work ➔Complete Section 16A
- ☐ Euthanased (put to sleep) whilst in active service ➔Complete Section 16A
- ☐ Died of a disease / illness whilst in service ➔Complete Section 16B
- ☐ Killed whilst in active service ➔Complete Section 16C

SECTION 16A

What was the MAJOR reason your dog was retired/euthanised
(1) JUST ASK: _____
Where there any secondary considerations in the decision to euthanase
(2) : _____

☐ Behavioural problem
- Aggression [ ]
- Not following commands [ ]
- Other: _____ [ ]

☐ Loss of tracking [ ]

☐ Inability to meet the physical demands of the job [ ]
Please describe:
_____

☐ Medical problem
- Heart problem [ ]
- Musculoskeletal [ ]
- Neurological [ ]
- Breathing issues [ ]
- Urinary Issues [ ]
- Intestinal [ ]
- Gastric Bloat/GDV [ ]
- Stomach problem (not Gastric Bloat/GDV) [ ]
- Other Please describe: [ ]

Back/Spinal problems (please specify which region/s)
Neck [ ]Thoracic spine (between the neck and the last rib) [ ]
Lumbar spine (after the last rib, excluding the lumbosacral junction) [ ]
Lumbo-sacral junction (where the lower back meets the pelvis/tail bone) [ ]

Arthritis (please specify which joint/s)

Shoulder	[ ]	Hip	[ ]
Elbow	[ ]	Stifle (knee)	[ ]
Carpus (wrist)	[ ]	Hock (ankle)	[ ]
Fore foot	[ ]	Hind foot	[ ]

Other medical problem (please describe): _____

SECTION 16B

What was the medical condition / illness which led to the death of your dog whilst on active duty? If more than one, please rank in order of relevance (1 for the most important, 2, 3, etc)

Heart problem	[ ]
Poisoning	[ ]
Seizuring	[ ]
Breathing problem	[ ]
Infectious disease	[ ]
Spinal problems	[ ]
Bloat/GDV	[ ]
Perforated bowel	[ ]
Other: _____	

SECTION 16C

How was your dog killed in active service?

Stabbed	[ ]
Gun shot	[ ]
Poisoning	[ ]
Motor vehicle accident	[ ]
Other: _____	

17. If you had another dog with lumbosacral disease with similar signs, would you be in favour of surgery on that dog?

18. Are there any comments you would like to add on the benefits/problems associated with the surgery your dog had?

_____

Adams WH, Daniel GB, Pardo AD, Selcer RS. Magnetic-resonance-imaging of the caudal lumbar and lumbosacral spine in 13 dogs (1990–1993). *Veterinary Radiology and Ultrasound* 36, 3–13, 1995

Adams WM, Dueland RT, Meinen J, O'Brien RT, Giuliano E, Nordheim. Early detection of canine hip dysplasia: Comparison of two palpation and five radiographic methods. *Journal of the American Animal Hospital Association.* 34, 339-47, 1998

Adams WM. Adams WM. Radiographic diagnosis of hip dysplasia in the young dog. *Veterinary Clinics of North America: Small animal practice* 30, 267-80, 2000

Axlund TW, Hudson JA. Computed tomography of the normal lumbosacral intervertebral disc in 22 dogs. *Veterinary Radiology & Ultrasound* 44, 630–4, 2003

***Bagley RS.** Surgical stabilisation of the lumbosacral joint. In: Slatter DH (ed). *Textbook of Small Animal Surgery*. 3rd Edtn. Pp 1238–43. Saunders, Philadelphia, USA, 2003

Bardens JW. Palpation for the detection of joint laxity. In: *Proceedings of the Canine Hip Dysplasia Symposium and Workshop*. St. Louis, Missouri. Pp 105-9. Orthopedic Foundation for Animals, 1972

Bardens JW, Hardwick H. New observations on the diagnosis and cause of hip dysplasia. *Veterinary Medicine, Small Animal Clinician* 63, 238-45, 1968

Barlow TG. Early diagnosis and treatment of congenital dislocation of the hip. *Journal of Bone & Joint Surgery - British Volume* 44B, 292-301, 1962

Barthez PY, Morgan JP, Lipsitz D. Discography and epidurography for evaluation of the lumbosacral junction in dogs with *cauda equina* syndrome. *Veterinary Radiology & Ultrasound* 35, 152–7, 1994

***Beam R, Powers D, Hauptman J, Sanders S.** (Abstract). Dynamic lumbosacral instability; clinical evaluation and owner perceived outcome of a novel approach for surgical distraction-stabilisation of L7-S1: 51 cases (2008-2013). *Journal of Veterinary Internal Medicine* 28, 1016, 2014

Benninger MI, Seiler GS, Robinson LE, Ferguson SJ, Bonél HM, Busati AR, Lang J. Three-dimensional motion pattern of the caudal lumbar and lumbosacral portions of the vertebral column of dogs. *American Journal of Veterinary Research* 65, 544-551, 2004

Benninger MI, Seiler GS et al. Effects of anatomic conformation on three-dimensional motion of the caudal lumbar and lumbosacral portions of the vertebral column of dogs. *American Journal of Veterinary Research* 67, 43-50, 2006

Bergknut N. Intervertebral Disc Degeneration in Dogs. *Doctoral Thesis No. 2010:91 Faculty of Veterinary Medicine and Animal Science*, *Uppsala, Sweden and Faculty of Veterinary Medicine, Utrecht University, The Netherlands.* 2011

Bergknut N, Egenvall A, Hagman R, Gustås P, Hazewinkel HAW, Meij BP, Lagerstedt AS. Incidence of intervertebral disk degeneration-related diseases and associated mortality rates in dogs. *Journal of the American Veterinary Medical Association* 240, 1300-1309, 2012

Bliss S, Todhunter RJ, Quaas R, Casella G, Wu R, Lust G, Williams AJ, Hamilton S, Dykes NL, Yeager A, Gilbert RO, Burton-Wurster NI, Acland GM. Quantitative genetics of traits associated with hip dysplasia in a canine pedigree constructed by mating dysplastic Labrador retrievers with unaffected Greyhounds. *American Journal of Veterinary Research* 63, 1029–35, 2002

Blumensaat C, Clasing C. Anatomie and Klinik der Übergangswirbel (Sakralisation und Lumbalisation). *Ergebnisse der Chirurgie und Orthopädie* 25, 1-59, 1932

Bodner DR, Delamarter RB, Bohlman HH, *et al.* Urologic changes after *cauda equina* compression in dogs. *Journal of Urology* 143, 186–190, 1990

Bowen JM, Lewis RE, Kneller SK, Wilson RC, Arnold RA. Progression of hip dysplasia in German shepherd dogs after unilateral pectineal myotomy. *Journal of the American Veterinary Medical Association* 161, 899-904, 1972

Bray JP, Burbidge HM. The canine intervertebral disk - Part one: Structure and function. *Journal of the American Animal Hospital Association* 34, 55-63, 1998a

Bray JP, Burbidge HM. The canine intervertebral disk - Part two: Degenerative changes - Nonchondrodystrophoid versus chondrodystrophoid disks. *Journal of the American Animal Hospital Association* 34, 135-44, 1998b

Breen M, Hitte C, Lorentzen TD, Thomas R, Cadieu E, Sabacan L, Scott A, Evanno G, Parker HG, Kirkness EF, Hudson R, Guyon R, Mahairas GG, Gelfenbeyn B, Fraser CM, Andre C, Galibert F, Ostrander EA. An integrated 4249 marker FISH/RH map of the canine genome. *BMC Genomics* 5, 65, 2004

Breur GJ, Lust G, Todhunter RJ. Genetics of canine hip dysplasia and other orthopaedic traits. In: Ruvinsky A, Sampson J (eds). *The Genetics of the Dog*. Pp 267-98. CAB International, New York, USA, 2001

[*]**Burbidge HM.** Changes proposed for inherited disorder control schemes. *VetScript* XVI (Vol 7), 28–9, August 2003

Cardinet GH, 3rd, Wallace LJ, Fedde MR, Guffy MM, Bardens JW. Developmental myopathy in the canine with type II muscle fiber hypotrophy. *Archives of Neurology* 21, 620-30, 1969

Cardinet GH, 3rd, Guffy MM, Wallace LJ. Canine hip dysplasia: Effects of pectineal tenotomy on the coxo-femoral joints of German shepherd dogs. *Journal of the American Veterinary Medical Association* 164, 591-8, 1974

Cardinet GH, 3rd, Leong CL, Means PS. Myofiber differentiation in normal and hypotrophied canine pectineal muscles. *Muscle & Nerve* 5, 665-73, 1982

Cardinet GH, 3rd, Kass PH, Wallace LJ, Guffy MM. Association between pelvic muscle mass and canine hip dysplasia. *Journal of the American Veterinary Medical Association* 210, 1466-73, 1997

*__Carozzo C, Cachon T, Genevois JP, Fau D, Remy D, Daniaux L, Collard F, Viguier E.__ Transiliac approach for exposure of lumbosacral intervertebral disk and foramen: Technique description. *Annual Scientific Meeting of the ECVS*. Pp 27–31, 2006

Chalman JA, Butler HC. Coxo-femoral joint laxity and the Ortolani sign. *Journal of the American Animal Hospital Association* 21, 671-6, 1985

Chambers J. Degenerative lumbosacral stenosis in dogs. *Veterinary Medical Report* 1, 166–80, 1989

Chen Q, Baba H, Kamitani K ET AL. Post-operative bone regrowth in lumbar spinal stenosis; a multivariate analysis of 48 patients. Spine 18,2144-9,1994

Clements DN, Carter SD, Innes JF, Ollier WER. Genetic basis of secondary osteoarthritis in dogs with joint dysplasia. *American Journal of Veterinary Research* 67, 909–17, 2006

Comhaire FH, Snaps F. Comparison of two canine registry databases on the prevalence of hip dysplasia by breed and the relationship of dysplasia with body weight and height. *American Journal of Veterinary Research* 69, 330-3, 2008

Cook JL, Tomlinson JL, Constantinescu GM. Pathophysiology, diagnosis, and treatment of canine hip dysplasia. *Compendium of Continuing Education for the Practicing Veterinarian* 18, 853–66, 1996

Coopman F, Verhoeven G, Paepe D, van Bree H, Duchateau L, Saunders JH. Inter-observer agreement on the radiographic assessment of canine hip dysplasia. *Vlaams Diergeneeskundig Tijdschrift* 76, 417-422, 2007

Corley EA, Hogan PM. Trends in hip dysplasia control: Analysis of radiographs submitted to the Orthopaedic Foundation for Animals, 1974–1984. *Journal of the American Veterinary Medical Association* 187, 805–9, 1985

Corley EA. Role of the Orthopedic Foundation for Animals in the control of canine hip dysplasia. *Veterinary Clinics of North America: Small animal practice* 22, 579-93, 1992

Corley EA, Keller GG, Lattimer JC, Ellersieck MR. Reliability of early radiographic evaluation for canine hip dysplasia obtained from the standard ventrodorsal radiographic projection. *Journal of the American Veterinary Medical Association* 211, 1142-6, 1997

Culp WTN, Kapatkin AS, Gregor TP, Powers MY, McKelvie PJ, Smith GK. Evaluation of the Norberg Angle and Distraction Index as measures of coxofemoral degenerative joint disease susceptibility in seven breeds of dogs. *Veterinary Surgery* 35, 453-9, 2006

Damur-Djuric N, Steffen F, Hassig M, Morgan JP, and Fluckiger MA. Lumbo-sacral transitional vertebrae in dogs: classification, prevalence, and association with sacroiliac morphology. *Veterinary Radiology & Ultrasound* 47**,** 32-38, 2006

Danielsson F, Sjöström L. Surgical treatment of degenerative lumbosacral stenosis in dogs. *Veterinary Surgery* 28, 91–8, 1999

De Decker S, Wawrzenski LA, Volk HA. Clinical presentation and outcome for dogs treated medically for degenerative lumbosacral stenosis. *Journal of Veterinary Internal Medicine* 28, 944–975, 2014

Delamarter RB, Bohlman HH, Dodge LD, Biro C. Experimental lumbar spinal stenosis. Analysis of the cortical evoked potentials, microvasculature, and histopathology. *Journal of Bone and Joint Surgery America* 72, 110–120, 1990

Demoulin C, Distree V, Tomasella M, Crielaard JM, Vanderthommen M. Lumbar functional instability: a critical appraisal of the literature. *Annales de Réadaptation et de Médecine Physique* 50, 10–16, 2007

Dennis R. Interpretation and use of BVA/KC hipscores in dogs. *In Practice* 34, 178-92, 2012

Denny HR, Gibbs C, Holt PE. The diagnosis and treatment of *cauda equina* lesions in the dog. *Journal of Small Animal Practice* 23, 435–43, 1982

[*]**DeRisio L, Thomas W, Sharp N.** Degenerative lumbosacral stenosis. *Veterinary Clinics of North America* 30, 130–3, 2000

DeRisio L, Sharp N, Olby N, Munana K, Thomas W. Predictors of outcome after dorsal decompressive laminectomy for degenerative lumbosacral stenosis in dogs: 69 cases (1987–97). *Journal of the American Veterinary Medical Association* 219, 624–8, 2001

Dietschi E, Schawalder P, Gaillard C. Estimation of genetic parameters for canine hip dysplasia in the Swiss Newfoundland population. *Journal of Animal Breeding and Genetics* 120, 150-61, 2003

[*]**Dupuis P.** *The Natural History of Degenerative Changes in the Lumbar Spine*. Aspen Publishers, Rockville MD, USA, 1987

Dutton R, and Moore GE. Clinical review of death or euthanasia in 123 military working dog necropsies. *Military Medicine* 152**,** 489-493, 1987

Early P, Mente P, Dillard S, Roe S. In vitro biomechanical comparison of the flexion/extension mobility of the canine lumbosacral junction before and after dorsal laminectomy and partial discectomy. *The Veterinary Journal* 196, 533–535, 2013

Engler J, Hamann H, Distl O. (abstract) Estimation of genetic parameters for radiographic signs of hip dysplasia in Labrador retrievers. *Berliner Und Munchener Tierarztliche Wochenschrift* 121, 359-64, 2008

Evans HE. *Miller's Anatomy of the Dog. 3rd Edition.* W.B.Saunders Co. Elsivier, Philadelphia, Pennsylvania, USA, 1993

Evans R, Herbold JR, Bradshaw BS, and Moore GE. Causes for discharge of military working dogs from service: 268 cases (2000-2004). *Journal of the American Veterinary Medical Association* 231**,** 1215-1220, 2007

Farese JP, Todhunter RJ, Lust G, Williams AJ, Dykes NL. Dorsolateral subluxation of hip joints in dogs measured in a weight-bearing position with radiography and computed tomography. *Veterinary Surgery* 27, 393-405, 1998

Farrell M, Clements DN, Mellor D, Gemmill T, Clarke SP, Arnott JL, Bennett D, Carmichael S. Retrospective evaluation of the long-term outcome of non-surgical management of 74 dogs with clinical hip dysplasia. *Veterinary Record* 160, 506-11, 2007

Flückiger MA. Scoring radiographs for canine hip dysplasia - The big three organizations of the world. *European Journal of Companion Animal Practice* 17, 135-40, 2007

Flückiger MA, Damur-Djuric N, Hassig M, Morgan JP, Steffen F. A lumbosacral transitional vertebra in the dog predisposes to *cauda equina* syndrome. *Veterinary Radiology & Ultrasound* 47, 39–44, 2006

Flückiger MA, Friedrich GA, Binder H. Correlation between hip joint laxity and subsequent coxarthrosis in dogs. *Journal of Veterinary Medicine Series A* 45, 199-207, 1998

Flückiger MA, Friedrich GA, Binder H. A radiographic stress technique for evaluation of coxofemoral joint laxity in dogs. *Veterinary Surgery* 28, 1–9, 1999

Fox SM, Burns J, Burt J. The dysplastic hip: A crippling problem in dogs." *Veterinary Medicine* 82, 684-693, 1987

Friedenberg SG, Zhu L, Zhang Z, Foels WvdB, Schweitzer PA, Wang W, Fisher PJ, Dykes NL, Corey E, Vernier-Singer M, Jung SW, Sheng X, Hunter LS, McDonough SP, G. Lust, Bliss SP, Krotscheck U, Gunn TM, Todhunter RJ. Evaluation of a fibrillin 2 gene haplotype associated with hip dysplasia and incipient osteoarthritis in dogs. *American Journal of Veterinary Research* 72, 530-540, 2011

Fries CL, Remedios AM. The pathogenesis and diagnosis of canine hip dysplasia: A review. *Canadian Veterinary Journal-Revue Veterinaire Canadienne* 36, 494-502, 1995

Fritsh E, Heisel J, Rupp S. The failed back surgery syndrome: reasons, intra-operative findings and long term results (a report of 182 operative treatments). *Spine* 21, 626-33, 1996

Fry TR and Clark DM. Canine hip dysplasia: clinical signs and physical diagnosis. *Veterinary Clinics of North America: Small Animal Practice* 22, 551-558, 1992

Frost HM. Pathogenesis of congenital hip dysplasia (CHD). A proposal. *Veterinary and Comparative Orthopaedics and Traumatology* 1, 1-10, 1989

Fox SM, Burns J, Burt J. The dysplastic hip: A crippling problem in dogs. Veterinary Medicine 82, 684-93, 1987

Friedenberg SG, Zhu L, Zhang Z, Foels WvdB, Schweitzer PA, Wang W, Fisher PJ, Dykes NL, Corey E, Vernier-Singer M, Jung S-W, Sheng X, Hunter LS, McDonough SP, Lust G, Bliss SP, Krotscheck U, Gunn TM, Todhunter RJ. Evaluation of a fibrillin 2 gene haplotype associated with hip dysplasia and incipient osteoarthritis in dogs. *American Journal of Veterinary Research* 72, 530-40, 2011

Fry TR, Clark DM. Canine hip dysplasia: clinical signs and physical diagnosis. *Veterinary Clinics of North America: Small Animal Practice* 22, 551-8, 1992

Gal JM. Mammalian spinal biomechanics I. Static and dynamic mechanical properties of intact intervertebral joints. *Journal of Experimental Biology* 174, 247-80, 1993

Genevois JP, Remy D, Viguier E, Carozzo C, Collard F, Cachon T, Maitre P, Fau D. Prevalence of hip dysplasia according to official radiographic screening, among 31 breeds of dogs in France. *Veterinary and Comparative Orthopaedics and Traumatology* 21, 21-4, 2008

Gibbs C. The BVA/KC scoring scheme for control of hip dysplasia: interpretation of criteria. *Veterinary Record* 141, 275–84, 1997.

Ginja MMD, Ginzalo-Orden JM, Melo-Pinto P, Bulas-Cruz J, Orden MA, San Roman F, Llorens-Pena MP, Ferreira AJA. Early hip laxity examination in predicting moderate and severe hip dysplasia in Estrela mountain dogs. *Journal of Small Animal Practice* 49, 641-6, 2008a

Ginja MMD, Silvestre AM, Ferreira AJA, Ginzalo-Orden JM, Orden MA, Melo-Pinto P, Llorens-Pena MP, Colaco J. Passive hip laxity in Estrela mountain dog – Distraction index, heritability and breeding values. *Acta Veterinaria Hungarica* 56, 303–12, 2008b

Ginja MMD, Ferreira AJ, Jesus SS, Melo-Pinto P, Bulas-Cruz J, Orden MA, San-Roman F, Llorens-Pena MP, Gonzalo-Orden JM. Comparison of clinical, radiographic, computed tomographic, and magnetic resonance imaging methods for early prediction of canine hip laxity and dysplasia. *Veterinary Radiology & Ultrasound* 50, 135-43, 2009a

Ginja MMD, Silvestre AM, Colaço J, Gonzalo-Orden JM, Melo-Pinto P, Orden MA, Llorens-Pena MP, Ferreira AJ. Hip dysplasia in Estrela mountain dogs: Prevalence and genetic trends 1991–2005. *Veterinary Journal* 182, 275-82, 2009b

Ginja MMD, Silvestre AM, Gonzalo-Orden JM, Ferreira AJA. Diagnosis, genetic control and preventive management of canine hip dysplasia: A review. *Veterinary Journal* 184, 269-76, 2010

Glickman LT, Glickman NW, Schellenberg DB, and Lantz GC. Multiple risk factors for gastric dilatation and dilatation-volvulus in dogs: a practitioner case/control study. *Journal of the American Animal Hospital Association* 33, 197-204, 1997.

Godde T, Steffen F. Surgical treatment of lumbosacral foraminal stenosis using a lateral approach in twenty dogs with degenerative lumbosacral stenosis. *Veterinary Surgery* 36, 705–13, 2007

Goldsmith LT, Lust G, Steinetz BG. Transmission of relaxin from lactating bitches to their offspring via suckling. *Biology of Reproduction* 50, 258-65, 1994

Golini L, Kircher PR, Lewis FI, Steffen F. Transarticular fixation with cortical screws combined with dorsal laminectomy and partial discectomy as surgical treatment of degenerative lumbosacral stenosis in 17 dogs: clinical and computed tomography follow-up. *Veterinary Surgery* 43, 405–413, 2014

Grumme Th, Bittl M. Imaging and therapy of degenerative spine diseases—A neurosurgeon's view. *European Journal of Radiology* 27, 235–240, 1998

Gustafsson P-O, Olsson S-E, Kasström H. Skeletal development of Greyhounds, German shepherd dogs and their crossbreed offspring. An investigation with special reference to hip dysplasia. *Acta Radiologica Supplementum* 344, 81-107, 1975

Hamann H, Kirchhoff T, Distl O. Bayesian analysis of heritability of canine hip dysplasia in German shepherd dogs. *Journal of Animal Breeding and Genetics* 120, 258-68, 2003

Hankin EJ, Jerram RM, Walker AM, King MD, Warman CGA. Transarticular facet screw stabilization and dorsal laminectomy in 26 dogs with degenerative lumbosacral stenosis with instability. Veterinary Surgery 41, 611–619, 2012

Hanna FY. Lumbo-sacral osteochondrosis: radiological features and surgical management in 34 dogs. *Journal of Small Animal Practice* 42, 272-278, 2001

Hassinger KA, Smith GK, Conzemius MG, Saunders HM, Hill CM, Gregor TP. Effect of the oestrus cycle on coxofemoral joint laxity. *Veterinary Comparative Orthopaedics and Traumatology* 10, 69–74, 1997

Hathcock JT. Vacuum phenomenon of the canine spine – CT findings in 3 patients. *Veterinary Radiology & Ultrasound* 35, 285–9, 1994

Hathcock JT, Pechman RD, Dillon AR, Knecht CD, Braund KG. Comparison of three radiographic contrast procedures in the evaluation of the canine lumbosacral spinal canal. *Veterinary Radiology* 29, 4–15, 1988

Haverbeke A, De Smet A, Depiereux E, Giffroy J, and Deiderich C. Assessing undesired aggression in military working dogs. *Applied Animal Behaviour Science* 117, 55-62, 2009

Hazewinkel HAW, Goedegebuuure SA, Poulos PW, Wolvekamp WThC. Influences of chronic **c**alcium excess on the skeletal development of growing Great Danes. *Journal of the American Animal Hospital Association* 21, 377-91, 1985

Hazewinkel HA, Van den Brom WE, Van TKAT, Voorhout G, Van Wees A. Calcium metabolism in Great Dane dogs fed diets with various calcium and phosphorus levels. *Journal of Nutrition* 121, S99-S106, 1991

Hedhammar A, Wu FM, Krook L, Schryver HF, De Lahunta A, Whalen JP, Kallfelz FA, Nunez EA, Hintz HF, Sheffy BE, Ryan GD. Overnutrition and skeletal disease. An experimental study in growing Great Dane dogs. *Cornell Veterinarian* 64, 5-160, 1974

Hedhammer A, Olsson SE, Andersson SA, Persson L, Pettersson L, Olausson A, Sundgren PE. Canine hip dysplasia: a study of heritability in 401 litters of German shepherd Dogs. *Journal of the American Veterinary Medical Association* 174, 1012–6, 1979

Hediger KU, Ferguson SJ, Gedet P, Busato A, Forterre F, Isler S, Barmettler R, Lang J. Biomechanical Analysis of Torsion and Shear Forces in Lumbar and Lumbosacral Spine Segments of Nonchondrodystrophic Dogs *Veterinary Surgery* 38, 874–880, 2009

Henninger W, Werner G. CT examination of the canine lumbosacral spine in extension and flexion. Part 1: Bone window. *European Journal of Companion Animal Practice* 13, 215–26, 2003 a

Henninger W, Werner G. CT examination of the canine lumbosacral spine in extension and flexion. Part 2: Soft-tissue window. *European Journal of Companion Animal Practice* 13, 227–33, 2003 b

Henricson B, Norberg I, Olsson SE. On the etiology and pathogenesis of hip dysplasia: A comparative review. *Journal of Small Animal Practice* 7, 673–87, 1966

Henry GA. Radiographic development of canine hip dysplasia. *Veterinary Clinics of North America: Small Animal Practice* 22, 559-78, 1992

Heyman SJ, Smith GK, Cofone MA. Biomechanical study of the effect of coxofemoral positioning on passive hip joint laxity in dogs. *American Journal of Veterinary Research* 54, 210–5, 1993

Higgins BM, Cripps PJ, Baker M, Moore L, Penrose FE, McConnell JF. Effects of body position, imaging plane, and observer on computed tomographic measurements of the lumbosacral intervertebral foraminal area in dogs. American Journal of Veterinary Research 72, 905-17, 2011

Hou Y, Wang Y, Lust G, Zhu L, Zhang Z, Todhunter RJ. Retrospective analysis for genetic improvement of hip joints of cohort Labrador retrievers in the United States: 1970–2007. *PLoS ONE* 5 (2), e9410. doi:10.1371/journal.pone.0009410, 2010

Hou Y, Wang Y, Lu X, Zhang X, Zhao Q, Todhunter RJ, Zhang Z. Monitoring hip and elbow dysplasia achieved modest genetic improvement of 74 dog breeds over 40 years in USA. *PLoS One* 8, e76390, 2013

Hunter AC. Radiography for hip dysplasia evaluation. *New Zealand Veterinary Journal* 34, 43-5, 1986

Hutt FB. Genetic selection to reduce the incidence of hip dysplasia in dogs. *Journal of the American Veterinary Medical Association* 151, 1041-8, 1967

Ihemelandu EC, Cardinet GHr, Guffy MM, Wallace LJ. Canine hip dysplasia: differences in pectineal muscles of healthy and dysplastic German shepherd dogs when two months old. *American Journal of Veterinary Research* 44, 411-6, 1983

Janssens LAA, Moens Y, Coppens P, Peremans K, Vinck H. Lumbosacral degenerative stenosis in the dog: the results of dorsal decompression with dorsal annulectomy and nuclectomy. *Veterinary Comparative Orthopaedics and Traumatology* 13, 97–103, 2000

[*]**Janssens LA, Beosier YM, Daems R.** Lumbosacral degenerative stenosis in the dog: The results of epidural infiltration with methylprednisolone acetate: a retrospective study. *36th Annual Conference of the Veterinary Orthopaedic Society*. P 9, 2009

Janutta V, Distl O. Inheritance of canine hip dysplasia: review of estimation methods and of heritability estimates and prospects on further developments. *Deutsche Tierärztliche Wochenschrift* 113, 6–12, 2006.

Janutta V, Hamann H, Distl O. Complex segregation analysis of canine hip dysplasia in German shepherd dogs. *Journal of Heredity* 97, 13-20, 2006

Jeffery ND, Barker A, Harcourt-Brown T. What progress has been made in the understanding and treatment of degenerative lumbosacral stenosis in dogs during the past 30 years? *The Veterinary Journal 201, 9–14, 2014*

Jones JC, Inzana KD. Subclinical CT abnormalities in the lumbosacral spine of older large-breed dogs. *Veterinary Radiology & Ultrasound* 41, 19–26, 2000

Jones JC, Cartee RE, Bartels JE. Computed tomographic anatomy of the canine lumbosacral spine. *Veterinary Radiology & Ultrasound* 36, 91–9, 1995

Jones JC, Banfield CM, Ward DL. Association between postoperative outcome and results of magnetic resonance imaging and computed tomography in working dogs with degenerative lumbosacral stenosis. *Journal of the American Veterinary Medical Association* 216, 1769–74, 2000

Jones JC, Davies SE, Werre SR, Shackelford KL. Effects of body position and clinical signs on L7–S1 intervertebral foraminal area and lumbosacral angle in dogs with lumbosacral disease as measured via computed tomography. *American Journal of Veterinary Research* 69, 1446–54, 2008

Kaneene JB, Mostosky UV, Miller R. Retrospective cohort study of changes in hip joint phenotype of dogs in the United States. *Journal of the American Veterinary Medical Association* 211, 1542–4, 2009

Kapatkin AS, Fordyce HH, Mayhew PD, Smith GK. Canine hip dysplasia: The disease and its diagnosis. *Compendium on Continuing Education for the Practicing Veterinarian* 24, 526-35, 2002

Kapatkin AS, Gregor TP, Heaton K, Richardson RW, McKelvie PJ, Fordyce HH, Smith GK. Comparison of two radiographic techniques for evaluation of hip joint laxity in 10 breeds of dogs. *Journal of the American Veterinary Medical Association* 224:542-6, 2004

Kapatkin AS, Mayhew PD, Smith GK. Genetic control of canine hip dysplasia. *Compendium on Continuing Education for the Practicing Veterinarian* 24, 681–7, 2002

Karkkainen M, Punto LU, Tulamo RM. Magnetic-resonance-imaging of canine degenerative spinal diseases. *Veterinary Radiology & Ultrasound* 34, 399–404, 1993

Kasström H. Nutrition, weight gain and development of hip dysplasia. An experimental investigation in growing dogs with special reference to the effect of feeding intensity. *Acta Radiologica Supplementum* 344, 135-79, 1975

Kealy RD, Olsson SE, Monti KL, Lawler DF, Biery DN, Helms RW, Lust G, Smith GK. Effects of limited food consumption on the incidence of hip dysplasia in growing dogs. *Journal of the American Veterinary Medical Association* 201, 857-63, 1992

Kealy RD, Lawler DF, Monti KL, Biery D, Helms RW, Lust G, Olsson SE, Smith GK. Effects of dietary electrolyte balance on subluxation of the femoral head in growing dogs. *American Journal of Veterinary Research* 54, 555-62, 1993

Kealy RD, Lawler DF, Ballam JM, Lust G, Smith GK, Biery DN, Olsson SE. Five-year longitudinal study of limited food consumption and development of osteoarthritis in coxofemoral joints of dogs. *Journal of the American Veterinary Medical Association* 210, 222-5, 1997

Kealy RD, Lawler DF, Ballam JM, Lust G, Biery DN, Smith GK, Mantz SL. Evaluation of the effect of limited food consumption on radiographic evidence of osteoarthritis in dogs. *Journal of the American Veterinary Medical Association* 217, 1678–80, 2000

Kim NH, Yang IH. A study of motor and sensory evoked potentials in chronic *cauda equina* compression of the dog. *European Spine Journal* 5, 338–344, 1996

Kinsel S, Koch J, Stopinski. *Cauda equina* compression syndrome: retrospective study of surgical treatment with patial laminectomy in 86 dogs with lumbosacral stenosis. *Berl Munch Tierartzl Wuchenshr* 117, 334-40, 2004

Kippenes H, Gondalen J. Health status of police patrol dogs and selected German shepherds. Helsetilstanden hos politiets patruljehunder og et utvalg schaferhunder. *Norsk Veterinartidsskrift* 111, 145-50, 1999.

Klaveren NJ, Suwankong N, De Boar S, Van Den Brom WE, Voorhout G, Hazewinkel HAW, Meij BP. Force plate analysis before and after dorsal decompression for treatment of degenerative lumbosacral stenosis in dogs. *Veterinary Surgery* 34, 450–6, 2005

Krontveit RI, Nødtvedt A, Sævik BK, Ropstad E, Trangerud C. Housing- and exercise-related risk factors associated with the development of hip dysplasia as determined by radiographic evaluation in a prospective cohort of Newfoundlands, Labrador retrievers, Leonbergers, and Irish Wolfhounds in Norway. *American Journal of Veterinary Research* 73, 838-46, 2012a

Krontveit RI, Trangerud C, Sævik BK, Skogmo HK, Nødtvedt A. Risk factors for hip-related clinical signs in a prospective cohort study of four large dog breeds in Norway. *Preventive Veterinary Medicine* 103, 219-27, 2012b

Lang J. Flexion-extension myelography of the canine cauda-equina. *Veterinary Radiology* 29, 242–57, 1988

Lang J, Hani, H. Schwalder, P. A sacral lesion resembling osteochondrosis in the german shepherd dog. *Veterinary Radiology* 33**,** 69-72, 1992.

Lauten SD. Nutritional risks to large-breed dogs: From weaning to the geriatric years. Veterinary Clinics of North America-Small Animal Practice 36, 1345–59, 2006

Lavrijsen ICM, Heuven HCM, Meij BP, Theyse LFH, Nap RC, Leegwater PAJ, Hazewinkel HAW. Prevalence and co-occurrence of hip dysplasia and elbow dysplasia in Dutch pure-bred dogs. *Preventive Veterinary Medicine* in press (e-publication ahead of print), *in press*

Lawson DD. The radiographic diagnosis of hip dysplasia in the dog. *Veterinary Record* 75, 445–56, 1963

Lee C, Rauschning W, Glenn W. Lateral lumbar spinal canal stenosis: classification, pathologic anatomy and surgical decompression. *Spine* 13, 313-20, 1988

Leighton EA. Genetics of canine hip dysplasia. *Journal of the American Veterinary Medical Association* 210, 1474–9, 1997

Leighton EA, Linn JM, William RL, Castleberry MW. A genetic study of canine hip dysplasia. *American Journal of Veterinary Research* 38, 241–4, 1977

Leppänen M, Mäki K, Juga J, Saloniemi H. Estimation of heritability for hip dysplasia in German shepherd dogs in Finland. *Journal of Animal Breeding and Genetics* 117, 97-103, 2000

Leppanen M, Saloniemi H. Controlling canine hip dysplasia in Finland. *Prev. Vet. Med.* 42, 121-31, 1999

Lewis TW, Blott SC, Woolliams JA. Genetic evaluation of hip score in UK Labrador retrievers. *PLoS One* 5, e12797, 2010

Lewis TW, Blott SC, Woolliams JA. Comparative analyses of genetic trends and prospects for selection against hip and elbow dysplasia in 15 UK dog breeds. *BMC genetics* 14, 16, 2013

Lingaas F, Klemetsdal G. Breeding values and genetic trend for hip dysplasia in the Norwegian Golden retriever population. *Journal of Animal Breeding and Genetics* 107, 437-43, 1990

Linn LL, Bartels KE, Rochat MC, Payton ME, Moore GE. Lumbosacral stenosis in 29 military working dogs: epidemiologic findings and outcome after surgical intervention (1990–1999). *Veterinary Surgery* 32, 21–9, 2003

Lust G, Geary JC, Sheffy BE. Development of hip dysplasia in dogs. *American Journal of Veterinary Research* 34, 87-91, 1973

Lust G, Farrell PW. Hip dysplasia in dogs: The interplay of genotype and environment. *Cornell Veterinarian* 67, 447-67, 1977

Lust G, Beilman WT, Rendano VT. A relationship between degree of laxity and synovial fluid volume in coxo-femoral joints of dogs predisposed for hip dysplasia. *American Journal of Veterinary Research* 41, 55-60, 1980

Lust G, Todhunter RJ, Erb HN, Lust G, Dykes NL, Williams AJ, Burton-Wurster NI, Farese JP. Repeatability of dorsolateral subluxation scores in dogs and correlation with macroscopic appearance of hip osteoarthritis. *American Journal of Veterinary Research* 62, 1711–1715, 2001

Lust G, Todhunter RJ, Erb HN, Dykes NL, Williams AJ, Burton-Wurster NI, Farese JP. Comparison of three radiographic methods for diagnosis of hip dysplasia in eight-month-old dogs. *Journal of the Veterinary Medical Association* 219, 1242–6, 2001

McKee WM, Downes CJ. Vertebral stabilisation and selective decompression for the management of triple thoracolumbar disc protrusions. *Journal of Small Animal Practice* 49, 536-539, 2008

Mackenzie SA. Why heritability estimates differ: Canine hip dysplasia. *Canine Practice* 12, 19-22, 1985

Mackenzie SA, Oltenacu EAB and Leighton EA. Heritability estimate for temperament scores in German shepherd dogs and its genetic correlation with hip dysplasia. *Behavior Genetics* 15, 475-482, 1985

Mäki K, Liinamo AE, Ojala M. Estimates of genetic parameters for hip and elbow dysplasia in Finnish Rottweilers. *Journal of Animal Science* 78, 1141-8, 2000

Mäki K, Groen AF, Liinamo AE, Ojala M. Genetic variances, trends and mode of inheritance for hip and elbow dysplasia in Finnish dog populations. *Animal Science* 75, 197-207, 2002

Mäki K, Janss LLG, Groen AF, Liinamo AE, Ojala M. An indication of major genes affecting hip dysplasia and elbow dysplasia in foor Finnish dog populations. *Journal of Heredity* 92, 402-8, 2004

Malenfant, Renee-Claire; Sod, Gary A. In vitro biomechanical comparison of 3.5 string of pearl plate fixation to 3.5 locking compression plate fixation in a canine fracture gap model. *Veterinary Surgery* 43, 465-70, 2014

Malm S, Fikse WF, Danell B, Strandberg E. Genetic variation and genetic trends in hip and elbow dysplasia in Swedish Rottweiler and Bernese Mountain Dog. *Journal of Animal Breeding and Genetics* 125, 403-12, 2008

Marschall Y, Distl O. Mapping quantitative trait loci for canine hip dysplasia in German shepherd dogs. *Mammalian Genome* 18(12): 861-870, 2007.

Mathews KA. Neuropathic pain in dogs and cats: if only they could tell us if they hurt. *Veterinary Clinics of North America-Small Animal Practice* 38, 1365-1414, 2008

Mathis KR, Havlicek M, Beck JB, Eaton-Wells RD, and Park FM. Sacral osteochondrosis in two German shepherd dogs. *Australian Veterinary Journal* 87, 249–252, 2009.

Mattoon JS, Koblik PD. Quantitative survey radiographic evaluation of the lumbosacral spine of normal dogs and dogs with degenerative lumbosacral stenosis. *Veterinary Radiology & Ultrasound* 34, 194–206,1993

Mayhew PD, Kapatkin AS, Wortman JA, Vite CH. Association of *cauda equina* compression on magnetic resonance images and clinical signs in dogs with degenerative lumbosacral stenosis. *Journal of the American Animal Hospital Association* 38, 555–62, 2002

Meheust P, Mallet C, Marouze C. A new surgical technique for lumbosacral stabilisation: arthrodesis using pedicle screw fixation. Anatomical aspects. *Pratique Médicale et Chirurgicale de L'Animal de Compaignie* 35, 193–9, 2000

McKee M, Dennis R. Radiology corner: lumbosacral radiography. *Veterinary Radiology & Ultrasound* 44, 655–6, 2003

Meheust P, Mallet C, Marouze C. A new surgical technique for lumbosacral stabilisation: arthrodesis using pedicle screw fixation. Anatomical aspects. *Pratique Médicale et Chirurgicale de L'Animal de Compaignie* 35, 193–9, 2000

Meij BP and Bergknut N. Degenerative lumbosacral stenosis in dogs. *Veterinary Clinics Small Animal* 20, 983-1009, 2010

Meij BP, Suwankong N, Van Der Veen AJ, Hazewinkel HAW. Biomechanical flexion-extension forces in normal canine lumbosacral cadaver specimens before and after dorsal laminectomy-discectomy and pedicle screw-rod fixation. *Veterinary Surgery* 36, 742–51, 2007

Moens N, Runyon C. Fracture of L7 vertebral articular facets and pedicles following dorsal laminectomy in a dog. *Journal of the American Veterinary Medical Association* 221, 807–10, 2002

Moore GE, Burkman KD, Carter MN, and Peterson MR. Causes of death or reasons for euthanasia in military working dogs: 927 cases (1993-1996). *Journal of the American Veterinary Medical Association* 219, 209-214, 2001

Morgan JP, Bailey CS. *Cauda equina* syndrome in the dog: Radiographic evaluation. *Journal of Small Animal Practice* 31, 69–77, 1990

Morgan JP, Stephens M. Radiographic diagnosis and control of canine hip dysplasia. *Iowa State University Press*, 1985

Morgan JP, Bahr A, Franti CE, Bailey CS. Lumbosacral transitional vertebrae as a predisposing cause of *cauda-equina* syndrome in German shepherd dogs – 161 cases (1987–1990). *Journal of the American Veterinary Medical Association* 202, 1877–82, 1993

Ness MG. Degenerative lumbosacral stenosis in the dog: A review of 30 cases. Journal of Small Animal Practice 35, 185–90, 1994

***Nicholas FW.** Hip Dysplasia in dogs. In: Nicholas FW (ed) *Veterinary Genetics*, 1st Edtn. Pp 331-3, Clarendon Press, Oxford, England, 1987

***Nicholas FW.** Is it inherited? In: *Introduction to Veterinary Genetics*. 3rd Edtn. Pp 150-7. Wiley-Blackwell, Chichester, United Kingdom, 2010a

***Nicholas FW.** Molecular Biology. In: *Introduction to Veterinary Genetics*. 3rd Edtn. Pp 46-84. Wiley-Blackwell, Chichester, United Kingdom, 2010b

***Nicholas FW.** Familial disorders not due to a single gene. In: *Introduction to Veterinary Genetics*. 3rd Edtn. Wiley-Blackwell, Chichester, United Kingdom, 2010c

***Nicholas FW.** Selection within populations. In: *Introduction to Veterinary Genetics*. 3rd Edtn. Pp 235-48. Wiley-Blackwell, Chichester, United Kingdom, 2010d

***Nicholas FW.** Quantitative variation. In: *Introduction to Veterinary Genetics*. 3rd Edtn. Pp 216-29. Wiley-Blackwell, Chichester, United Kingdom, 2010e

Nunamaker DM, Biery DN, Newton CD. Femoral neck anteversion in the dog: Its radiographic measurement. *Veterinary Radiology* 14, 45-8, 1973

Ogden DM, Scrivani PV, Dykes N, Lust G, Friedenberg SG, Todhunter RJ. The S-Measurement in the diagnosis of canine hip dysplasia. *Veterinary Surgery* 41, 78-85, 2012

Ohlerth S, Busato A, Rauch M, Weber U, Lang J. Comparison of three distraction methods and conventional radiography for early diagnosis of canine hip dysplasia. *Journal of Small Animal Practice* 44, 524-9, 2003

Ohlerth S, Lang J, Busato A, Gaillard C. Estimation of genetic population variables for six radiographic criteria of hip dysplasia in a colony of Labrador retrievers. *American Journal of Veterinary Research* 62, 846-52, 2001

Olson RC. Physical evaluation and selection of military dogs. *Journal of the American Veterinary Medical Association* 159**,** 1444-1446, 1971

Olsewski JM, Lust GL, Rendano VT et al. Degenerative joint disease: multiple joint involvement in young and mature dogs. *American Journal of Veterinary Research* 44, 1300-1308, 1983

Ondeka N, Amort KH, Stock KF, Tellhelm B, Klumpp SW, Kramer M, Schmidt MJ. Skeletal morphology of the lumbosacral junction in German shepherd dogs and an evaluation of the possible genetic basis for radiographic findings. The Veterinary Journal 196, 64-70, 2013

Ortolani M. Congenital hip dysplasia in the light of early and very early diagnosis. *Clinical orthopaedics and related research*, 6-10, 1976

[*]**Paatsama S.** Hip dysplasia in dogs: a controlled restricted breeding programme in Finland. *Twenty-first World Veterinary Congress Summaries, Moscow* 4, 42–3, 1979

Paatsama S, Rissanen P, Rokkanen P. Changes in the hip joint induced with certain hormones: An experimental study on young dogs. *Journal of Small Animal Practice* 9, 433-40, 1968

Palmer RH, Chambers JN. Canine lumbosacral diseases. Part 1. Anatomy, pathophysiology and clinical presentation. *Compendium on Continuing Education for the Practicing Veterinarian* 13, 61–8, 1991 a

Palmer RH, Chambers J. Canine lumbosacral diseases. Part II. Definitive diagnosis, treatment, and prognosis. *Compendium on Continuing Education for the Practicing Veterinarian* 13, 213–22, 1991 b

Panjabi, Manohar M. Biomechanical Evaluation of Spinal Fixation Devices: I. A Conceptual Framework. *Spine 13, 1988*

Parr JR, Otto CM. Emergency visits and occupational hazards in German shepherd police dogs (2008–2010). *Journal of Veterinary Emergency and Critical Care* 23, 591–597, 2013

Paster ER, LaFond E, Biery DN, Iriye A, Gregor TP, Shofer FS, Smith GK. Estimates of prevalence of hip dysplasia in Golden retrievers and Rottweilers and the influence of bias on published prevalence figures. *Journal of the American Veterinary Medical Association* 226, 387–92, 2005

Patterson DF. Companion animal medicine in the age of medical genetics. *Journal of Veterinary Internal Medicine* 14**,** 1-9, 2000

PennHIP AIS. What is PennHIP? http://info.antechimagingservices.com/pennhip/navigation/general/what-is-PennHIP.html (accessed 25 February, 2014). 2013

Pfahler S, Distl O. Identification of Quantitative Trait Loci (QTL) for canine hip dysplasia and canine elbow dysplasia in Bernese Mountain dogs. *PLoS One* 7, e49782, 2012

Pierce KR, Bridges GH, Banks WC. Hormone induced hip dysplasia in dogs. *Journal of Small Animal Practice* 6, 121-6, 1965

Pierce KR, Bridges CH. The role of estrogens in the pathogenesis of canine hip dysplasia. Metabolism of exogenous estrogens. *Journal of Small Animal Practice* 8, 383-9, 1967

Popovitch CA, Smith GK, Gregor TP, Shofer FS. Comparison of susceptibility for hip dysplasia between Rottweilers and German shepherd Dogs. *Journal of the Veterinary Medical Association* 206, 648-50, 1995

Prieur WD. Coxarthrosis in the dog Part I: Normal and abnormal biomechanics of the hip joint. *Veterinary Surgery* 9, 145-9, 1980

Ramirez O, Thrall DE. A review of imaging techniques for canine *cauda equina* syndrome. *Veterinary Radiology & Ultrasound* 39, 283–96, 1998

Reed AL, Keller GG, Vogt DW, Ellersieck M, Corley EA. Effect of dam and sire qualitative hip conformation scores on progeny hip conformation. *Journal of the American Veterinary Medical Association* 217, 675-80, 2000

Rendano VT, Ryan G. Canine hip dysplasia evaluation. *Veterinary Radiology* 26, 170-86, 1985

Rettenmaier JL, Constantinescu GM. Canine hip dysplasia. *Compendium on Continuing Education for the Practicing Veterinarian* 13, 643-54, 1991

Richardson DC. The role of nutrition in canine hip dysplasia. *Veterinary Clinics of North America: Small animal practice* 22, 529-40, 1992

Riser WH. A new look at developmental subluxation and dislocation: Hip dysplasia in the dog. Journal of Small Animal Practice 4, 421-34, 1963

Riser WH. The dysplastic hip joint: Radiologic and histologic development. *Veterinary Pathology* 12, 279-305, 1975a

Riser WH. Hip dysplasia as a disease. *Veterinary Pathology* 12, 316-23, 1975b

Riser WH. Growth and development of the normal canine pelvis, hip joints, and femur from birth to maturity. *Veterinary Pathology* 12, 264-78, 1975c

Riser WH. Observations and research on hip dysplasia. *Veterinary Pathology* 12, 239-63, 1975d

Riser WH. Canine hip dysplasia. In: Bojrab MJ (ed). *Disease Mechanisms in Small Animal Surgery*. 2nd Edtn. Pp 797-803. Lea & Febiger, Philadelphia, 1993

Riser WH, Cohen D, Lindqvist S, Mansson J, Chen S. Influence of early rapid growth and weight gain on hip dysplasia in the German shepherd Dog. *Journal of the American Veterinary Medical Association* 145, 661-8, 1964

Riser WH, Shirer JF. Correlation between canine hip dysplasia and pelvic muscle mass: A study of 95 dogs. A*merican Journal of Veterinary Research* 28, 769-77, 1967

Rossi F, Seiler G, Busato A, Wacker C, Lang J. Magnetic resonance imaging of articular process joint geometry and intervertebral disk degeneration in the caudal lumbar spine (L5–S1) of dogs with clinical signs of *cauda equina* compression. *Veterinary Radiology & Ultrasound* 45, 381–7, 2004

Sallander MH, Hedhammar Å, Trogen MEH. Diet, exercise, and weight as risk factors in hip dysplasia and elbow arthrosis in Labrador retrievers. *Journal of Nutrition* 136, 2050S-2S, 2006

Sánchez-Molano E, Woolliams JA, Blott SC, Wiener P. Assessing the impact of genomic selection against hip dysplasia in the Labrador retriever dog. *Journal of Animal Breeding and Genetics* in press (e-publication ahead of print), 2013

Scharf G, Steffen F, Grunenfelder F, Morgan JP, and Fluckiger M. The lumbo-sacral junction in working German shepherd dogs - neurological and radiological evaluation. *Journal of Veterinary Medicine. Series A* 51**,** 27-32, 2004

Schmid V, Lang J. Measurements on the lumbosacral junction in normal dogs and those with cauda-equina compression. *Journal of Small Animal Practice* 34, 437–42, 1993

Schnelle GB. Congenital subluxation of the coxo-femoral joint in a dog. *Univ Pa Bull* 65: 15-16, 1937

Schoenmakers I, Hazewinkel HAW, Voorhout G, Carlson CS, Richardson D. Effect of diets with different calcium and phosphorus contents on the skeletal development and blood chemistry of growing Great Danes. *Veterinary Record* 147, 652-60, 2000

Schwarz T, Owen MR, Long S, Sullivan M. Vacuum disk and facet phenomenon in a dog with *cauda equina* syndrome. *Journal of the American Veterinary Medical Association* 217, 862–4, 2000

Seiler GS, Hani H, Busato AR, Lang J. Facet joint geometry and intervertebral disk degeneration in the L5–S1 region of the vertebral column in German shepherd Dogs. American Journal of Veterinary Research 63, 86–90, 2002

Silvestre AM, Ginja MMD, Ferreira AJA, Colaço J. Comparison of estimates of hip dysplasia genetic parameters in Estrela Mountain Dog using linear and threshold models. *Journal of Animal Science* 85, 1880-4, 2007

***Sharp NJH, Wheeler SJ.** Lumbosacral disease. In: Wheeler SJ (ed). *Small Animal Spinal Disorders.* Pp 181-209. Elsevier Mosby, Philadelphia. USA, 2005

Sisson AF, LeCouteur RA, Ingram JT, Park RD, Child G. Diagnosis of *cauda equina* abnormalities by using electromyography, discography, and epidurography in dogs. Journal of Veterinary Internal Medicine 6, 253–63, 1992

Slocum B, Devine T. L7–S1 fixation-fusion for treatment of *cauda equina* compression in the dog. *Journal of the American Veterinary Medical Association* 188, 31–5, 1986

Smith GA, Aspden RM, Porter RW. Measurement of vertebral foraminal dimensions using three-dimensional computerized tomography. *Spine* 18, 629-36, 1993

Smith GK. Advances in diagnosing canine hip dysplasia. *Journal of the American Veterinary Medical Association* 211, 1451–7, 1997

Smith GK, Biery DN, Gregor TP. New concepts of coxofemoral joint stability and the development of a clinical stress-radiographic method for quantitating hip joint laxity in the dog. *Journal of the American Veterinary Medical Association* 196, 59-70, 1990

Smith GK, Gregor TP, Rhodes WH, Biery DN. Coxofemoral joint laxity from distraction radiography and its contemporaneous and prospective correlation with laxity, subjective score and evidence of degenerative joint disease from conventional hip-extended radiography. *Journal of the American Veterinary Medical Association* 54, 1021–42, 1993

Smith GK, Lawler DF, Biery DN, Powers MY, Shofer FS, Gregor TP, Karbe GT, McDonald-Lynch MB, Evans RH, Kealy RD. Chronology of hip dysplasia development in a cohort of 48 labrador retrievers followed for life. *Veterinary Surgery* 41, 20-33, 2012

Smith GK, Mayhew PD, Kapatkin AS, McKelvie PJ, Shofer FS. Evaluation of risk factors for degenerative joint disease associated with hip dysplasia in German shepherd dogs, Golden retrievers, Labrador retrievers, and Rottweilers. *Journal of the American Veterinary Medical Association* 219, 1719-24, 2001

Smith GK, Paster ER, Powers MY, Lawler DF, Biery DN, Shofer FS, McKelvie PJ, Kealy RD. Lifelong diet restriction and radiographic evidence of osteoarthritis of the hip joints in dogs. *Journal of the American Veterinary Medical Association* 229, 690–3, 2006

Smith GK, Popovitch CA, Gregor TP. Evaluation of factors contributing to the risk of developing degenerative joint disease associated with hip dysplasia in dogs. *Journal of the American Veterinary Medical Association* 206, 642–7, 1995.

Smith MEH, Bebchuk TN, Shmon CL, Watson LG, Steinmetz H. An in vitro biomechanical study
of the effects of surgical modification upon the canine lumbosacral spine. *Veterinary Comparative Orthopaedics and Traumatology* 17, 17–24, 2004

Smolders LA, Voorhout G, van de Ven R, Bergknut N, Grinwis GCM, Hazewinkel HAW, BP Meij. Pedicle screw-rod fixation of the canine lumbosacral junction. *Veterinary Surgery* 41, 720-32, 2012

Soo M. Estimated breeding value analysis of the New Zealand Veterinary Association Canine Hip Dysplasia score in four large breeds of dogs (1991 – 2011). A dissertation presented in partial fulfilment of the requirements for the degree of Master of Veterinary Medicine in Companion Animal Surgery at Massey University, Palmerston North, New Zealand, 2013

Soo M, Worth AJ. Canine hip dysplasia: Phenotypic scoring and the role of estimated breeding value analysis. *New Zealand Veterinary Journal* 63, 69-78, 2015

Soo M, Sneddon NW, Lopez-Villalobos N, Worth AJ. Genetic evaluation of the total hip score of four populous breeds of dog, as recorded by the New Zealand Veterinary Association Hip Dysplasia Scheme (1991–2011). *New Zealand Veterinary Journal* 63, 79-85, 2015

Stambough J. Failed back: etiologies. *Seminars in Spine Surgery* 11, 162-75, 1999

Steffen F, Berger M, Morgan J. Asymmetrical, transitional, lumbosacral stenosis in six dogs: a characteristic clinical syndrome. *Journal of the American Animal Hospital Association* 40, 338-44, 2004

Steffen F, Hunold K, Scharf G, Roos M, Flückiger M. A follow-up study of neurologic and radiographic findings in working German shepherd dogs with and without degenerative lumbosacral stenosis. *Journal of the American Veterinary Medical Association* 231, 1529–33 2007

Steinetz BG, Williams AJ, Lust G, Schwabe C, Büllesbach EE, Goldsmith LT. Transmission of relaxin and estrogens to suckling canine pups via milk and possible association with hip joint laxity. *American Journal of Veterinary Research* 69, 59-67, 2008

Stock KF, Distl O. Simulation study on the effects of excluding offspring information for genetic evaluation versus using genomic markers for selection in dog breeding. *Journal of Animal Breeding and Genetics* 127, 42-52, 2010

Stock KF, Klein S, Tellhelm B, Distl O. Genetic analyses of elbow and hip dysplasia in the German shepherd dog. *Journal of Animal Breeding and Genetics* 128, 219-29, 2011

Strickland, Winifred and Moses, James. The German shepherd today: Complete reference for the German shepherd owner; *Canada: Macmillan Publishing Company,* 1988

Suwankong N, Voorhout G, Hazewinkel HAW, Meij BP. Agreement between computed tomography, magnetic resonance imaging, and surgical findings in dogs with degenerative lumbosacral stenosis. *Journal of the American Veterinary Medical Association* 229, 1924–9, 2006

Suwankong N, Meij BP, Klaveren NJ, Van Wees AMTC, Meijer E, Van Den Brom WE, Hazewinkel HAW. Assessment of decompressive surgery in dogs with degenerative lumbosacral stenosis using force plate analysis and questionnaires. *Veterinary Surgery* 36, 423–31, 2007

Suwankong N, Meij BP, Voorhout G, de Boer AH, Hazewinkel HAW. Review and retrospective analysis of degenerative lumbosacral stenosis in 156 dogs treated by dorsal laminectomy. *Veterinary and Comparative Orthopaedics and Traumatology* 21, 285–93, 2008

Swenson L, Audell L, Hedhammar A. Prevalence and inheritance of and selection for hip dysplasia in seven breeds of dogs in Sweden and benefit:cost analysis of a screening and control program. *Journal of the American Veterinary Medical Association* 210, 207–14, 1997

Tarvin G, Prata RG. Lumbosacral stenosis in dogs. *Journal of the American Veterinary Medical Association* 177, 154–9, 1980

Theyse LFH, van de Brom WE, van Sluijs FJ. Small size of food particles and age as risk factors for gastric dilatation volvulus in Great Danes. Veterinary Record 143, 48–50, 1998

[*]**Todhunter RJ, Lust G.** Hip dysplasia: Pathogenesis. In: Slatter D (ed). *Textbook of Small Animal Surgery.* 3rd Edtn. Pp 2009–19. Saunders, Philadelphia PA, USA, 2003

Todhunter RJ, Acland GM, Olivier M, Williams AJ, Vernier-Singer M, Burton-Wurster N, Farese JP, GrÖhn YT, Gilbert RO, Dykes NL, Lust G. An outcrossed canine pedigree for linkage analysis of hip dysplasia. *Journal of Heredity* 90, 83-92, 1999

Todhunter RJ, Bliss SP, Casella G, Wu R, Lust G, Burton-Wurster NI, Williams AJ, Gilbert RO, Acland GM. Genetic structure of susceptibility traits for hip dysplasia and microsatellite informativeness of an outcrossed canine pedigree. *Journal of Heredity* 94, 39-48, 2003

Todhunter RJ, Mateescu G, Lust RG, Burton-Wurster NI, Dykes NL, Bliss SP, Williams AJ, Vernier-Singer M, Corey E, Harjes C, Quaas RL, Zhang Z, Gilbert RO, Volkman D, Casella G, Wu R, Acland GM. Quantitative trait loci for hip dysplasia in a cross-breed canine pedigree. *Mammalian Genome* 16, 720-730, 2005

Tomlinson JL, Johnson JC. Quantification of measurement of femoral head coverage and Norberg angle within and among four breeds of dogs. *American Journal of Veterinary Research* 61, 1492-500, 2000

Vaughan L, Jones D, Lane J. Pectineus muscle resection as a treatment for hip dysplasia in dogs. *Veterinary Record* 96, 145-8, 1975

Verhoeven GEC, Coopman F, Duchateau LUC, Bosmans TIM, Van Ryssen B, Van Bree H. Interobserver agreement on the assessability of standard ventrodorsal hip-extended radiographs and its effect on agreement in the diagnosis of canine hip dysplasia and on routine FCI scoring. *Veterinary Radiology & Ultrasound* 50, 259-63, 2009

Verhoeven G, Fortrie R, Van Ryssen B, Coopman F. Worldwide screening for canine hip dysplasia: where are we now? *Veterinary Surgery* 41, 10-19, 2012

Vostrý L, Čapková Z, Šebková N, Přibyl J. Estimation of genetic parameters for hip dysplasia in Czech Labrador retrievers. *Journal of Animal Breeding and Genetics* 129, 60-9, doi:10.1111/j.1439-0388.2011.00946.x, 2012

Ward MP, Patronek GJ, and Glickman LT. Benefits of prophylactic gastropexy for dogs at risk of gastric dilatation-volvulus. *Preventative Veterinary Medicine* 60**,** 319-329, 2003

Wigger A, Julier-Franz C, Tellhelm B, Kramer M. Lumbosacral transitional vertebrae in the German shepherd dog: prevalence, classification, genetics, and association with canine hip dysplasia. *Tierärztliche Praxis Ausgabe Kleintiere Heimtiere* 37, 7–13, 2009

Wilkinson JA. Prime factors in the etiology of congenital dislocation of the hip. *Journal of Bone and Joint Surgery* 45B, 268-83, 1963

Willis MB. A review of the progress in canine hip dysplasia control in Britain. *Journal of the American Veterinary Medical Association* 210, 1480–2, 1997

Wilson B, Nicholas FW, Thomson PC. Selection against canine hip dysplasia: Success or failure? *Veterinary Journal* 189, 160-8, 2011

Wilson BJ, Nicholas FW, James JW, Wade CM, Tammen I, Raadsma HW, Castle K, Thomson PC. Heritability and phenotypic variation of canine hip dysplasia radiographic traits in a cohort of Australian German shepherd dogs. *PLoS One* 7, e39620, 2012

Witte P, Scott H. Investigation of lameness in dogs. *In Practice* 33, 58-66, 2011

Wood JLN, Lakhani KH, Dennis R. Heritability and epidemiology of canine hip-dysplasia score in flat-coated retrievers and Newfoundlands in the United Kingdom. *Preventive Veterinary Medicine* 46, 75-86, 2000a

Wood JLN, Lakhani KH, Dennis R. Heritability of canine hip dysplasia score and its components in Gordon Setters. *Preventive Veterinary Medicine* 46, 87-97, 2000b

Wood JLN, Lakhani KH, Rogers K. Heritability and epidemiology of canine hip dysplasia score and its components in Labrador retrievers in the United Kingdom. *Preventive Veterinary Medicine* 55, 95-108, 2002

Wood JLN, Lakhani KH, Henley WE. An epidemiological approach to prevention and control of three common heritable diseases in canine pedigree breeds in the United Kingdom. *Veterinary Journal* 168, 14–27, 2004a

Wood BC, Lanz OI, Jones JC, Shires PK. Endoscopic-assisted lumbosacral foraminotomy in the dog. *Veterinary Surgery* 33, 221–31, 2004b

Woolliams JA, Lewis TW, Blott SC. Canine hip and elbow dysplasia in UK Labrador retrievers. *Veterinary Journal* 189, 169-76, 2011

Worth AJ, Laven RA, Erceg VH. An assessment of the agreement between the New Zealand Veterinary Association Hip Dysplasia Scoring System and the PennHIP Distraction Index in German shepherd dogs. *New Zealand Veterinary Journal* 57, 338–45, 2009

Worth AJ, Bridges JP, Jones G. Reduction in the incidence of elbow dysplasia in four breeds of large dog as measured by the New Zealand Veterinary Association scoring scheme. *New Zealand Veterinary Journal* 58, 190–5, 2010

Wright PJ, Mason TA. The usefulness of palpation of joint laxity in puppies as a predictor of hip dysplasia in a guide dog breeding programme. *Journal of Small Animal Practice* 18, 513-22, 1977

Zhang Z, Zhu L, Sandler J, Friedenberg SS, Egelhoff J, Williams AJ, Dykes NL, Hornbuckle W, Krotscheck U, Moise NS, Lust G, Todhunter RJ. Estimation of heritabilities, genetic correlations, and breeding values of four traits that collectively define hip dysplasia in dogs." *American Journal of Veterinary Research* 70, 483-492, 2009

Zhou Z, Sheng X, Zhang Z, Zhao K, Zhu L, Guo G, Friedenberg G, Hunter, LS, Vandenberg-Foels WS, Hornbuckle WE, Krotscheck U, Corey E, Moise NS, Dykes NL, Li J, Xu S, Du L, Wang Y, Sandler J, Acland GM, Lust G, Todhunter RJ. Differential genetic regulation of canine hip dysplasia and osteoarthritis. *PLoS One* 5, e13219, 2010

Zhu L, Zhang Z, Feng F, Schweitzer P, Phavaphutanon J, Vernier-Singer M, Corey E, Friedenberg S, Mateescu R, Williams A, Lust G, Acland G, Todhunter RJ. Single nucleotide polymorphisms refine QTL intervals for hip joint laxity in dogs. *Animal Genetics* 39, 141-146, 2008

Zhu L, Zhang Z, Friedenberg S, Jung SW, Phavaphutanon J, Vernier-Singer M, Corey E, Mateescu R. Dykes N, Sandler J, Acland G, Lust G, Todhunter R. The long (and winding) road to gene discovery for canine hip dysplasia. *Veterinary Journal* 181, 97–110, 2009

Zorko B, Ivanusa T, Pelc R. Progression of Hip Dysplasia in 40 Police working dogs. *European Journal of Companion Animal Practice* 17, 145-148, 2008