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Skeletal morphology and morphometry of the lumbosacral junction in German shepherd dogs and an evaluation of the possible genetic basis for radiographic findings

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ABSTRACT

The aim of this study was to identify skeletal variations in the lumbosacral junction (LSJ) of the German shepherd dog (GSD) compared with other large breeds. The radiographic traits of the LSJ were investigated in a group of 733 GSDs and a control group of 334 dogs of other breeds that were matched in terms of size. Nine morphological and 17 morphometric traits were recorded and analysed. Furthermore, the possibility of a genetic basis for these radiographic features was evaluated by calculation of genetic variance components.

Five of the morphological and 14 of the morphometric traits varied significantly between the GSD group and the control group ($P < 0.05$). Osteochondrosis of the sacral endplate (SOC) had a higher prevalence in the GSDs (10.1%) compared with controls (5.7%). The majority of LSJ degenerative changes recorded from the radiographs occurred in the GSDs. The extent and relative proportion of lumbosacral step formations were greater in the GSD group compared with controls ($P < 0.001$). The lumbosacral vertebral canal height was reduced in the GSD compared with the control dogs ($P < 0.001$) suggesting a primary stenosis. This was accentuated by an abrupt tapering of the vertebral canal at the level of the LSJ indicated by a lumbosacral ratio of 1.51 in the GSD.

The skeletal morphology and morphometry of the LSJ in the GSD seem to be different from that found in other large dogs. For multiple traits frequently observed in GSD such as SOC, step formations, and LSJ stenosis, moderate to high non-zero heritabilities were noted. As these features are also assumed to promote lumbosacral disease, selection against these traits is suggested.

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Introduction

Cauda equina syndrome (CES) refers to a complex of clinical signs caused by lumbosacral vertebral canal stenosis and subsequent compression of the cauda equina nerve roots. The breed most commonly affected by clinical signs related to lumbosacral stenosis is the German shepherd dog (GSD) (Indrieri, 1988; Watt, 1991; Ness, 1994; Danielsson and Sjöström, 1999; DeRiso et al., 2000; Suwankong et al., 2008). The aetiopathogenesis of the disease is considered to be complex and congenital, developmental and acquired abnormalities may contribute to narrowing of the lumbosacral vertebral canal, nerve root compression, and progressive clinical signs (Lang et al., 1992; Morgan et al., 1993; DeRiso et al., 2000; Seiler et al., 2002; Flückiger et al., 2006; Meij and Bergknut, 2010).

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Individual characteristics of lumbosacral morphology predisposing the GSD to CES have been identified in previous studies. Lumbosacral transitional vertebrae (LTV) and sacral osteochondrosis (SOC) are believed to contribute to the breed-predisposition for CES because of their pathological potential and frequent occurrence in the breed (Lang et al., 1992; Morgan et al., 1993; Hanna, 2001; Damur Djuric et al., 2006; Flückiger et al., 2006). There may be a genetic background for conditions promoting CES (Lang et al., 1992; Morgan et al., 1993; Damur Djuric et al., 2006; Flückiger et al., 2006), and at least partial genetic determination is indispensable in the justification and efficacy of breeding selection against traits predisposing the GSD to lumbosacral stenosis. However, to date, the only trait known to increase the risk of CES in which the genetic background has been investigated is LTV (Wigger et al., 2009) and these authors give a heritability range of 20–30% in the GSD. Information on the genetic involvement of many other skeletal characteristics of the lumbosacral junction (LSJ) is lacking.

A number of studies have examined the diagnostic value of several radiographic and tomographic features in the quest to differentiate between dogs with and those without clinical signs of CES (Mattoon and Koblik, 1993; Morgan et al., 1993; Schmid and Lang, 1993; Rossi et al., 2004; Scharf et al., 2004; Flückiger et al., 2006; Suwankong et al., 2006; Steffen et al., 2007). However, extensive interbreed comparisons regarding the general skeletal conformation of the LSJ have not been undertaken using normal dogs. In light of the high incidence of CES in GSDs, there is a need for comprehensive data on the general LSJ differences between GSDs and other dogs of similar size. Knowledge of the morphological and morphometric variations of the LSJ of GSDs compared to other large dogs would help to understand why GSDs are prone to lumbosacral disease.

The purpose of this study was to analyse the variation in congenital, developmental and acquired features of the LSJ between clinically normal GSD and dogs from other large breeds. Morphological and morphometric radiographic traits of the LSJ in a large population of GSDs were investigated and compared with a control group. We hypothesized that there is breed-specific variation in the radiographic morphology and morphometry of the lumbosacral region in the GSD. Furthermore, our aim was to determine potential genetic involvement in the development of phenotypic characteristics of the LSJ based upon the pedigree data of the GSD.

Materials and methods

Study design and patient material

The observational study was based on a retrospective analysis of radiographic material from the Small Animal Clinic, Department of Veterinary Clinical Sciences, University of Giessen. The study comprises a group of GSDs and a control group containing other canine breeds matched to the GSD in terms of size. All dogs were presented for the purpose of screening for canine hip dysplasia (CHD) according to the Fédération Cynologique Internationale (FCI). To meet the inclusion criteria of this study, the clinical histories of the dogs had to be devoid of back pain or gait abnormalities, and the medical records had to be negative for lumbosacral pain, hind limb ataxia, and neurological deficits at the time of presentation. To be included in the control group, the height of the withers of a specific breed had to overlap within the range 60–65 cm for males and 55–60 cm for females according to the breed standard of the FCI. For all dogs, a lateral radiograph of the pelvis in neutral position centered on the LSJ (Morgan, 1993) was available and had been obtained at the request of the owner.

Evaluation of radiographs

Image analysis was performed using a dedicated computer software program (DicomWorks 1.3.5 imaging software¹). All images were reviewed by a radiology resident (NO) who was unaware of the breed of the dog. In case of equivocal findings images were re-evaluated by the resident and a board-certified radiologist (BT), and consensus was reached.

Nine qualitative traits were evaluated and graded binary according to their absence (grade 0) or presence (grade 1). These included SOC, LTV identified by the presence of a radiolucent disc space between the first and second sacral segment, spondylosis deformans of the opposing lumbosacral vertebral endplates (SPON^{7/1}) and the opposing endplates of the lumbar vertebrae 6 and 7 (SPON^{6/7}), lumbosacral facet joint arthropathy (ARTH), sclerosis of the cranial sacral endplate (SCLER), opacification associated with the intervertebral disc space (O-DISC) or vertebral canal (O-CAN), and reduced delineation of the ventral contour of the sacral roof (ROOF). The morphological traits and their abbreviations are summarized in Table 1.

The following 17 quantitative morphometric traits were obtained from the radiographs and scaled as continuous measurements based on previously published methods (Wright, 1980; Feeney and Wise, 1981; Walla, 1986; Morgan and Bailey, 1990; Mattoon and Koblik, 1993; Schmid and Lang, 1993; Scharf et al., 2004). The metric measurements are quoted in mm, and the angles in degrees (°). Lumbosacral step formations (STEP) were measured at the level of the caudal endplate of L7 as the distance between two lines joining the dorsal contour of the vertebral bodies of L7 and S1. Width of the lumbosacral intervertebral space (IVS) was defined as the distance between the centers of the opposing lumbosacral endplates along a line dividing the vertebral body of S1 into equal halves. The lengths of the vertebral bodies of L6

(L_L6), L7 (L_L7), and S1 (L_S1) were measured as a line intersecting these vertebral bodies. The heights of the bodies of L6 (H_L6) and L7 (H_L7) were measured in the mid-portion of the vertebral bodies. The height of S1 (H_S1) was measured at the level of the cranial sacral endplate. The height of the vertebral canal was measured at six locations—the cranial and caudal endplate of L6 (CAN_L6_{CR}, CAN_L6_{CD}), L7 (CAN_L7_{CR}, CAN_L7_{CD}), and the sacrum (CAN_S1, CAN_S3). Table 2 summarizes the morphometric traits including their abbreviations and units of measurements.

To compensate for possible bias from individual body size, the values obtained from metric measurements were normalized using anatomical reference points within the same radiograph as stated below. The unitless normalized values are indicated by the superscript REL.

$$REL_STEP = STEP/CAN_L7_{CD},$$

$$REL_IVS = IVS/CAN_L7_{CD},$$

and

$$REL_LX = LX/[(L_L6 + L_L7 + L_S1)/3],$$

$$REL_HX = HX/[(H_L6 + H_L7 + H_S1)/3],$$

$$REL_CAN_X_{CR} = CAN_X_{CR}/H_X,$$

$$REL_CAN_Y_{CD} = CAN_Y_{CD}/H_X,$$

with

$$X = L6, L7, S1 \text{ and } Y = L6, L7, S3 \text{ respectively.}$$

The lumbosacral ratio (LS_R) was calculated by dividing the vertebral canal height $REL_CAN_L7_{CD}$ by the vertebral canal height REL_CAN_S1 as in a previous study (Mattoon and Koblik, 1993). The lumbosacral angle was recorded in two different ways. The dorsal lumbosacral angle (LS_A_D) was derived from a line aligned with the dorsal contour of the vertebral body of the lumbar vertebrae 6 and 7 and a line that joined the dorsal contour of the sacral vertebral bodies. The central lumbosacral angle (LS_A_C) was measured between lines intersecting the vertebral bodies of the last lumbar vertebrae and the sacrum centrally. In addition, the intervertebral angle (EP_A) was obtained from the lines being tangential to the opposing lumbosacral vertebral endplates.

Statistical analysis of radiographic traits

The prevalence of the morphological traits among the two groups was calculated. Descriptive statistics were computed for the morphometric traits. In the analysis of variance, both morphologic and morphometric data were analysed for significant differences regarding group, sex, and age. Group (GSD, control group) and sex (male, female) were regarded fixed effects; age at the time of presentation was included as linear covariate. To allow for eventual differences between the groups caused by the varying age at presentation, age within the group was taken into account. Preliminary trials revealed that the effect of sex between the groups was negligible. Thus, the interaction between group and sex was not integrated in the statistical model applied (model 1):

$$y_{ijkm} = \mu + bA_i(Gr_j) + RGr_j + S_k + e_{ijkm}$$

where y_{ijkm} is the morphologic or morphometric trait obtained from the radiographs, μ is the model constant, $A_i(Gr_j)$ is the age within the group in months with the linear regression factor b , RGr_j is the fixed effect of the group (GSD group vs. control group), S_k is the fixed effect of sex, and e_{ijkm} is the random residual error.

When prior testing revealed an influence of the presence of an LTV on a trait, the presence of an LTV was integrated in the model as a fixed effect and the respective model was used for that specific trait (model 2):

$$y_{ijkm} = \mu + bA_i(RGr_j) + RGr_j + G_k + LTV_1 + e_{ijkm}$$

where LTV_1 is the fixed effect of the presence of an LTV.

For the morphologic traits, a GENMOD procedure (Generalized Linear Model) was employed using a binomial distribution function and probit link function. The analysis of variance of the morphometric traits was exhibited using the SAS-procedure General Linear Model (GLM). Results were considered significant at $P < 0.05$. All statistical analyses were performed using a commercial statistical software package (SAS version 9.2, SAS Institute).

Genetic analysis

In a subset of the GSD group, pedigree data archived by the national GSD Kennel Club was available, and for these dogs, analyses of genetic variance components were performed. The morphological and morphometric criteria of these dogs were analysed for a possible partial genetic background. The genetic analysis of the morphological traits was restricted to traits with a proportion >5%. Hence, two of the morphological traits (SPON^{7/1} and SPON^{6/7}) were excluded.

¹ Puech, P., and Bousset, L. See: <<http://www.dicomworks.com/>> – accessed January 2008.

Table 1

Survey of the nine morphological traits evaluated from the radiographs with their abbreviations.

Morphological traits	
Graded in a binary mode as absent or present	
Variable	Abbreviation
Osteochondrosis of the cranial sacral endplate	SOC
Lumbosacral transitional vertebra	LTV
Spondylosis deformans at lumbosacral junction	SPON ^{7/1}
Spondylosis deformans at L6/L7 intervertebral disc space	SPON ^{6/7}
Lumbosacral facet joint arthropathy	ARTH
Sclerosis of the cranial sacral endplate	SCLER
Opacification associated with lumbosacral intervertebral disc space	O-DISC
Opacification associated with vertebral canal at lumbosacral junction	O-CAN
Reduced delineation of the sacral roof	ROOF

Table 2

Survey of the 17 morphometric traits obtained from the radiographs with their abbreviations and units of measurements.

Morphometric traits		
Graded as continuous measurements		
Variable	Abbreviation	Unit
Lumbosacral step formation	STEP	mm
Width of lumbosacral intervertebral disc space	IVS	mm
Vertebral body length of L6	L_L6	mm
Vertebral body length of L7	L_L7	mm
Vertebral body length of S1	L_S1	mm
Vertebral body height of L6	H_L6	mm
Vertebral body height of L7	H_L7	mm
Vertebral body height of S1	H_S1	mm
Height of vertebral canal cranial at L6	CAN_L6 _{CR}	mm
Height of vertebral canal caudal at L6	CAN_L6 _{CD}	mm
Height of vertebral canal cranial at L7	CAN_L7 _{CR}	mm
Height of vertebral canal caudal at L7	CAN_L7 _{CD}	mm
Height of vertebral canal at S1	CAN_S1	mm
Height of vertebral canal at S3	CAN_S3	mm
Dorsal lumbosacral angle	LS_A _D	°
Central lumbosacral angle	LS_A _C	°
Lumbosacral endplate angle	EP_A	°

For seven morphological and 17 morphometric traits, a statistical model was evolved via SAS-procedure GLM, entering age as linear variable and considering sex plus month and quarter of birth as fixed effects. A comparison of models adjusted for the inclusion of the fixed effects using a likelihood ratio test yielded no significant advance for factoring in the month and quarter of birth as compared to the model involving sex as the only fixed effect. This applied for both the morphological and the morphometric traits equally.

The following linear model was therefore adopted for all 24 traits:

$$y_{ijkl} = \mu + bA_i + G_j + a_k + e_{ijkl}$$

where y_{ijkl} is the morphologic or morphometric trait obtained from the radiographs, μ is the model constant, A_i is the age in months with the linear regression factor b , G_j is the sex as fixed effect, a_k is the random additive genetic effect of the animal, and e_{ijkl} the random residual error.

Univariate estimation of genetic parameters was conducted. Accordingly, multivariate estimates were obtained to account for possible correlation existing between the traits.

Restricted maximum likelihood procedures were applied to obtain variance component estimates using dedicated computer software (VCE-5, version 5.1.2 FAL; Mariensee-Neustadt; Kovac et al., 2003). Heritabilities (h^2) and genetic (r_g) and residual correlations (r_r) of two traits each (traits 1 and 2) were calculated based on their estimated additive genetic (σ_a^2 , cov_a) and residual (co)variance (σ_e^2 , cov_e) as follows:

$$h_1^2 = \sigma_a^2 / (\sigma_a^2 + \sigma_e^2) \text{ or } h_2^2 = \sigma_a^2 / (\sigma_a^2 + \sigma_e^2),$$

$$r_{g12} = \text{cov}_{a12} / (\sigma_{a1} * \sigma_{a2}), \text{ and}$$

$$r_{e12} = \text{cov}_{e12} / (\sigma_{e1} * \sigma_{e2}).$$

The implementation of linear models in the analysis of parameters graded in a binary mode (morphologic traits) leads to underestimation of heritabilities and

Table 3

Prevalence of morphological traits reported as number of dogs (%) for the German shepherd dog (GSD) group ($n = 733$) and the control group comprising 334 dogs of 46 breeds.

Variable	GSD group	Control group	P
SOC	74 (10.1)	19 (5.7)	0.013
LTV	50 (6.8)	14 (4.2)	0.270
SPON ^{7/1}	79 (10.8)	74 (22.2)	0.915
SPON ^{6/7}	31 (4.2)	26 (7.8)	0.963
ARTH	168 (22.9)	28 (8.4)	<0.001
SCLER	319 (43.5)	96 (28.7)	<0.001
O-DISC	97 (13.2)	21 (6.3)	<0.001
O-CAN	166 (22.7)	12 (3.6)	<0.001
ROOF	295 (40.3)	145 (43.4)	0.600

A legend to the abbreviations of the variables is provided in Table 1. $P < 0.05$ considered statistically significant (95% confidence interval).

Table 4

Distribution of the results for the morphometric traits in the German shepherd dog (GSD) group ($n = 733$) and the control group ($n = 334$) quoted as unitless normalized values (indicated by the superscript REL). Data are presented as means (standard deviation). Absolute values are given for the lumbosacral angles with the unit°.

Variable	GSD group	Control group	P
REL ^{STEP}	0.26 (0.18)	0.09 (0.15)	<0.001
REL ^{IVS}	0.54 (0.14)	0.48 (0.12)	<0.001
REL ^{L_L6}	1.19 (0.04)	1.21 (0.04)	<0.001
REL ^{L_L7}	1.04 (0.03)	1.03 (0.03)	<0.001
REL ^{L_S1}	0.76 (0.04)	0.76 (0.04)	0.374
REL ^{H_L6}	0.88 (0.04)	0.92 (0.04)	<0.001
REL ^{H_L7}	0.96 (0.03)	0.96 (0.03)	0.092
REL ^{H_S1}	1.16 (0.05)	1.12 (0.05)	<0.001
REL ^{CAN_L6_{CR}}	0.66 (0.08)	0.71 (0.10)	<0.001
REL ^{CAN_L6_{CD}}	0.62 (0.09)	0.67 (0.10)	<0.001
REL ^{CAN_L7_{CR}}	0.51 (0.07)	0.60 (0.08)	<0.001
REL ^{CAN_L7_{CD}}	0.50 (0.07)	0.57 (0.08)	<0.001
REL ^{CAN_S1}	0.34 (0.05)	0.42 (0.06)	<0.001
REL ^{CAN_S3}	0.25 (0.05)	0.31 (0.05)	<0.001
LS_A _D	160.66 (6.10)	157.73 (7.67)	<0.001
LS_A _C	152.99 (7.26)	148.51 (7.22)	<0.001
EP_A	17.10 (5.24)	17.50 (4.44)	0.283

A legend to the abbreviations of the variables is provided in Table 2. The superscript REL is assigned for the variables analyzed as unitless normalized values.

$P < 0.05$ considered statistically significant (95% confidence interval).

residual correlations relative to the prevalence of the trait in question. This underestimation can be compensated for via transformation of the estimates based upon the threshold value scheme, which was applied according to Dempster and Lerner (1950) and Vinson et al. (1976). Heritability was transformed according to the following equation:

$$h_{i\text{iab}}^2 = h_{i\text{obs}}^2 [p_i(1 - p_i)] / z_i^2$$

where $h_{i\text{iab}}^2$ is the heritability of the trait i on the underlying continuous scale, $h_{i\text{obs}}^2$ is the heritability of the trait i as recorded from the observed (binary) scale, p_i is the prevalence of the trait 1, and z_i the ordinate of the standard-normal distribution at the threshold that corresponds to p_i .

The transformation of residual correlation between two traits (traits 1 and 2) was obtained as follows:

$$r_{e\text{iab}} = r_{e\text{obs}} \{ [p_{11}(1 - p_{11})] / z_{11}^2 \}^{1/2} \{ [p_{22}(1 - p_{22})] / z_{22}^2 \}^{1/2}$$

We report below the transformed heritability and residual correlation for all binary (morphologic) traits. As additive genetic correlation estimates are not affected by underestimation, a transformation was unnecessary. The heritability estimates of all traits investigated demonstrated a high level of agreement according to the various multivariate analytic procedures. Therefore, the results presented are confined to the means and standard error.

Results

Descriptive statistics

Seven hundred and thirty-three GSDs met the inclusion criteria (392 males, 341 females). The mean age was 15 months (range, 9–

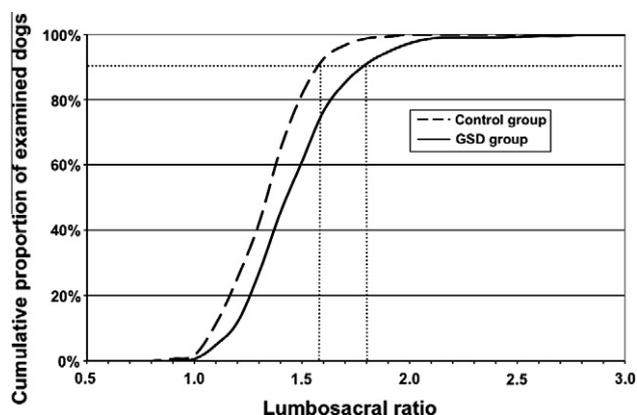


Fig. 1. Proportion of dogs in the German shepherd (GSD) and Control group as a function of their lumbo-sacral ratio. Dotted lines define the 90%-quantiles, which = 1.80 in the GSD group and 1.60 in the control group. There is a shift in distribution to higher lumbo-sacral ratios in the GSD group, indicating an acute drop in vertebral canal height between the last lumbar vertebra and the sacrum in the GSDs compared to the control dogs.

145 months) with 90% being <18 months. The control group comprised 334 dogs of 46 breeds. These included: Labrador Retrievers ($n = 49$), Golden Retrievers ($n = 33$), Bernese Mountain dogs

($n = 29$), Boxers ($n = 26$), Rottweilers ($n = 24$), Doberman Pinschers ($n = 23$), Giant Schnauzers ($n = 21$). There were also 129 dogs of 39 other breeds. The control group comprised 190 males and 144 females. The mean age was 29 months (range, 10–94 months). Less than 50% of the control dogs were younger than 18 months.

Results of radiographic evaluation

The prevalence of the morphological traits in GSDs and control dogs is shown in Table 3. SOC lesions were seen in 10.1% of the GSDs and in 5.7% of the controls ($P = 0.013$). We also found a greater proportion of LTV in the GSD group (6.8%) compared to the control group (4.2%) but there was no statistical significance. Differences with a high level of significance ($P < 0.001$) were evident for four of the morphological traits, namely, ARTH, SCLER, O-DISC and O-CAN which each occurred at a higher frequency in the GSD group than in the control group. Only two of the morphological traits were significantly influenced by the sex of the animals ($P < 0.05$): males were more frequently affected by SCLER than females, whereas females had a higher proportion of SPON^{6/7} ($P < 0.05$).

The descriptive statistics of the morphometric lumbo-sacral traits are listed in Table 4. REL^{STEP} was significantly more pronounced in the GSD group with a value of 0.26 ± 0.18 vs.

Table 5

Multivariate estimates of heritability (h^2), genetic and residual correlation for the morphological traits of the lumbo-sacral junction in a subset of 572 German shepherd dogs (GSDs). h^2 estimates are arranged diagonally (bold type), the genetic correlation is quoted above the diagonal, and residual correlation below the diagonal. Standard errors are presented in parentheses below the estimates.

Variable	SOC	LTV	SCLER	ARTH	ROOF	O-DISC	O-CAN
SOC	0.505 (0.128)	-0.215 (0.189)	-0.247 (0.131)	0.084 (0.122)	0.871 (0.090)	-0.173 (0.155)	-0.243 (0.172)
LTV	-0.016 (0.142)	0.639 (0.159)	0.544 (0.148)	0.095 (0.145)	0.053 (0.168)	0.112 (0.177)	0.190 (0.191)
SCLER	0.554 (0.104)	-0.573 (0.133)	0.597 (0.077)	0.839 (0.057)	-0.110 (0.107)	0.800 (0.119)	0.881 (0.086)
ARTH	0.004 (0.107)	0.206 (0.132)	0.213 (0.091)	0.620 (0.114)	0.001 (0.110)	0.919 (0.062)	0.928 (0.061)
ROOF	-0.178 (0.116)	0.143 (0.148)	0.152 (0.088)	0.202 (0.086)	0.619 (0.099)	-0.347 (0.166)	-0.332 (0.149)
O-DISC	0.190 (0.109)	-0.148 (0.133)	-0.065 (0.102)	-0.073 (0.120)	0.296 (0.114)	0.436 (0.104)	0.984 (0.041)
O-CAN	0.392 (0.083)	0.019 (0.105)	0.467 (0.061)	0.715 (0.073)	0.147 (0.079)	-0.044 (0.086)	0.219 (0.054)

A legend to the abbreviations of the variables is provided in Table 1.

Table 6

Multivariate estimates of heritability (h^2), genetic and residual correlation for the morphometric traits of the lumbo-sacral junction in a subset of 572 German shepherd dogs (GSDs). h^2 estimates are arranged diagonally (bold type), the genetic correlation is quoted above the diagonal and residual correlation below the diagonal. Standard errors are presented in parentheses below the estimates.

Variable	LS _{Ad}	LS _{Ac}	EP _A	STEP	IVS
LS _{Ad}	0.768 (0.079)	0.865 (0.021)	-0.840 (0.036)	-0.045 (0.115)	-0.572 (0.070)
LS _{Ac}	0.672 (0.085)	0.759 (0.058)	-0.821 (0.034)	0.299 (0.110)	-0.165 (0.085)
EP _A	0.168 (0.221)	0.084 (0.179)	0.754 (0.052)	0.070 (0.100)	0.442 (0.097)
STEP	-0.332 (0.138)	-0.385 (0.119)	0.257 (0.103)	0.338 (0.062)	0.473 (0.149)
IVS	0.305 (0.087)	0.278 (0.068)	-0.292 (0.094)	0.197 (0.070)	0.250 (0.064)

	L _{L6}	L _{L7}	L _{S1}	H _{L6}	H _{L7}	H _{S1}
L _{L6}	0.431 (0.087)	-0.501 (0.163)	-0.849 (0.047)	-0.139 (0.238)	0.269 (0.360)	0.043 (0.272)
L _{L7}	-0.042 (0.093)	0.266 (0.074)	-0.031 (0.192)	-0.146 (0.216)	-1.000 (0.362)	0.250 (n.a.)
L _{S1}	-0.1787 (0.029)	-0.3583 (0.060)	0.270 (0.081)	0.258 (0.240)	0.243 (0.540)	-0.355 (0.352)
H _{L6}	0.300 (0.086)	-0.320 (0.063)	-0.053 (0.073)	0.225 (0.081)	-0.141 (0.419)	0.924 (0.074)
H _{L7}	0.231 (0.073)	-0.029 (n.a.)	-0.173 (0.066)	0.014 (0.063)	0.052 (0.048)	-0.249 (0.430)
H _{S1}	-0.374 (0.072)	0.281 (0.060)	0.148 (0.072)	-0.751 (0.028)	-0.670 (0.034)	0.136 (0.067)

	CAN _{L6_{CR}}	CAN _{L6_{CD}}	CAN _{L7_{CR}}	CAN _{L7_{CD}}	CAN _{S1}	CAN _{S3}
CAN _{L6_{CR}}	0.665 (0.056)	0.661 (0.037)	0.798 (0.052)	0.455 (0.074)	0.345 (0.098)	0.295 (0.072)
CAN _{L6_{CD}}	0.641 (0.067)	0.730 (0.047)	0.516 (0.050)	0.467 (0.066)	-0.121 (0.080)	-0.217 (0.059)
CAN _{L7_{CR}}	0.307 (0.101)	0.476 (0.073)	0.574 (0.048)	0.675 (0.038)	0.744 (0.052)	0.666 (0.048)
CAN _{L7_{CD}}	0.406 (0.086)	0.475 (0.080)	0.535 (0.043)	0.494 (0.045)	0.266 (0.086)	0.265 (0.071)
CAN _{S1}	0.129 (0.078)	0.431 (0.071)	0.070 (0.055)	0.470 (0.051)	0.308 (0.049)	0.954 (0.026)
CAN _{S3}	-0.047 (0.134)	0.453 (0.098)	-0.258 (0.100)	0.282 (0.089)	0.236 (0.062)	0.722 (0.037)

A legend to the abbreviations of the variables is provided in Table 2.

n.a., not applicable.

0.09 ± 0.15 in the control group ($P < 0.001$). Moreover, an absolute measurement for STEP > 0 was noted at a ratio of 2:1 in the GSDs compared with the control dogs. REL_{IVS} was significantly larger in the GSD group (0.54 ± 0.14) than in the control dogs (0.48 ± 0.12). Regarding the dimensions of the vertebral bodies, the significant differences ($P < 0.001$) included a shorter REL_{L_L6} , a longer REL_{L_L7} , a lower REL_{H_L6} , and a higher REL_{H_S1} . Furthermore, differences with a high level of significance ($P < 0.001$) were observed for the dorsoventral dimension of the vertebral canal in general.

At any of the six points measured, the height of the vertebral canal was reduced in the GSDs. The LS_R was significantly higher for the GSDs (1.51 ± 0.31) compared to the control dogs (1.38 ± 0.19, $P < 0.001$), indicating a greater degree of reduction of spinal canal height at the level of the LSJ. Fig. 1 demonstrates the distribution of the lumbosacral ratio (LS_R) between the GSD and the control groups. Regardless of which method was used to obtain the measurements, the lumbosacral angles (LS_A_D and LS_A_C) were significantly higher for the GSDs ($P < 0.001$).

Results of genetic analysis

Pedigree data were available in a subset of 572 GSDs. The records represented 298 males and 274 females. The mean litter size was 1.93 for the sires and 1.35 for the dams. Table 5 contains multivariate estimates of heritability (h^2), genetic correlation (r_g), and residual correlation (r_r) of the morphologic traits of the LSJ. The heritability estimates for the morphologic traits ranged from 0.22 to 0.64. Negative genetic correlations < -0.2 were estimated for SOC with LTV; SCLER; and O-CAN, ROOF with O-DISC, and ROOF with O-CAN. Moderate to high positive genetic correlations (>0.5) were calculated for SOC with ROOF, LTV with SCLER, SCLER with ARTH; O-DISC; and O-CAN, ARTH with O-DISC and O-CAN, and O-DISC with O-CAN.

The variance component estimates of the morphometric traits of the LSJ of the GSDs are illustrated in Table 6. Low-to-moderate heritabilities were estimated for IVS ($h^2 = 0.25$) and STEP ($h^2 = 0.34$). In the domain of the normalized vertebral body measurements, multivariate analysis revealed low-to-moderately high heritabilities ($h^2 = 0.05$ –0.43). Among the six reference points for the lumbosacral vertebral canal height, moderate-to-high heritabilities ranging from 0.31 to 0.72 were detected. The vertebral canal measurements proved to be predominantly positively correlated with $r_g \geq 0.3$. In particular, for CAN_{S1} and CAN_{S3} a close positive genetic correlation ($r_g = 0.95$) was estimated.

Discussion

Our findings indicate that there is variation in skeletal morphology and morphometry of the LSJ in GSDs compared with dogs of similar size. The percentage of SOC in the GSD group was approximately twice the percentage of the control group (10.1% and 5.7%, respectively). As SOC has been reported to disrupt the integrity of the lumbosacral intervertebral disc, it is regarded as a promoter of lumbosacral stenosis and CES (Lang et al., 1992; Hanna, 2001; Glyde et al., 2004; Michal et al., 2004; Mathis et al., 2009). Considering the frequent occurrence of this finding in GSDs, SOC may be a factor exposing particularly this breed to lumbosacral disease.

The prevalence of an LTV separated from the fused sacral segments by a rudimentary intervertebral space was greater in the GSD group (6.8%) than in the controls (4.2%), although this difference was not statistically significant. A significantly higher prevalence of LTV in GSDs than in other breeds has been reported by others (Winkler and Loeffler, 1986; Morgan et al., 1993; Breit and Künzel, 1998; Morgan, 1999; Breit et al., 2003; Damur Djuric

et al., 2006; Flückiger et al., 2006), but was not apparent in our study. Our results would suggest that this disorder is not a major contributor to the frequent occurrence of lumbosacral stenosis and CES in the GSDs.

The analysis of the morphometric data highlights the differences in skeletal morphology between GSD and other large breeds of dogs. The reduced vertebral canal height throughout the entire length of the lumbosacral transition, when compared with other breeds, suggests the presence of lumbosacral stenosis existing presumably as a primary malformation in the GSD. The confined LSJ noted in the GSD is further supplemented by the significantly increased LS_R , which indicates an abrupt drop in the dorsoventral lumbosacral vertebral canal height between the last lumbar vertebra and the sacrum.

In our study sample, significantly larger STEPs were found in the GSDs. Furthermore, and regardless of their metric extent, STEPs were twice as frequent in the GSD group as in the control group. Despite disagreement on the metric extent of lumbosacral step formations necessary to cause clinical disease, i.e., >4 mm (Schmid and Lang, 1993) or as small as 2 mm (Suwankong et al., 2006), this finding is generally considered to be important in promoting lumbosacral disease (Schmid and Lang, 1993; Suwankong et al., 2006).

The significant differences in lumbosacral vertebral canal height, LS_R and STEP characterize the aberrant morphology of the LSJ in the GSD. Given that we focused on young dogs without signs of lumbosacral disease, these findings suggest that the changes are of primary origin. It is known from human medicine that most of the congenital conditions compromising the spinal canal do not manifest until traumatic or degenerative processes ensue (Kirkaldy-Willis et al., 1974; Verbiest, 1975; Munday et al., 1994).

In veterinary medicine, it has been proposed that the combination of congenital lumbosacral stenosis and degenerative changes may cause cauda equina compression in large breed dogs (Oliver et al., 1978; Tarvin and Prata, 1980; Palmer and Chambers, 1991; Bailey and Morgan, 1992; Jones et al., 1996). Due to the reduced spatial tolerance towards secondary degenerative or hypertrophic changes in the LSJ, we assume that the risk of developing clinically relevant stenosis is increased in animals with a narrow lumbosacral vertebral canal or other anomalies such as REL_{STEP} (as we have specifically identified in GSDs). With regard to the confined LSJ in GSDs, the morphological disorders SOC and premature degeneration are of special interest.

Previous work has documented the prevalence of LTV to be as high as 29% in some populations of GSDs (Larsen, 1977; Winkler and Loeffler, 1986; Morgan et al., 1993; Damur Djuric et al., 2006; Flückiger et al., 2006; Wigger et al., 2009). The discrepancy between the prevalence of LTV recorded in our study and in previous work may reflect natural fluctuations through unequal study populations and age groups and, in particular, the difference in inclusion criteria. Studies reporting higher percentages of LTV provide for different types of LTV. Isolation of the 'spinous process type' vertebrae account for approximately 80% of LTV (Wigger et al., 2009), but were disregarded in our study because based on our experience and that of others (Flückiger et al., 2009) they are inconsistently identified radiographically, and also as these forms of LTV can be considered insignificant in the individual dog (Flückiger et al., 2009).

It has been postulated that abnormal biomechanics associated with a translational motion type and/or altered range of motion is the most significant aspect of LTV with regard to early disc degeneration and CES (Bürger and Lang, 1992; Morgan et al., 2000; Luoma et al., 2004; Flückiger et al., 2006). To date, no study has documented the actual change in kinetics with a transitional vertebra, and an alteration in lumbosacral mobility seems to apply only to complete isolation of the abnormal vertebral segment from the fused sacral vertebrae.

Heritability conveys the proportion of the total phenotypic variance that is attributed to genetic factors and heritability estimates give a measure of the relative importance of genetic variance in determining the phenotypic variance of a specific trait (Conner and Hartl, 2004). If the heritability of a trait is between 0.4 and 1.0, it is generally considered high and the phenotype is a good predictor of the genetic disposition of an animal. With high heritability estimates, beneficial results might be expected by phenotypic selection for or against any trait.

Genetic correlations between traits are of substantial interest, because depending on their value (positive or negative) and the desired breeding objective regarding the individual trait, they can either facilitate or hamper phenotypic selection of the traits. Genetic correlations may produce beneficial or undesirable changes in selection programs. Undesirable correlations have to be known and must not be ignored in meeting breeding objectives as they may influence the rate of progress.

With regard to the morphological and morphometric traits that presumably advance the susceptibility of GSD to lumbosacral disease, the results of our genetic analysis indicate that a relevant proportion of the phenotypic variance of these traits is determined by genetic factors. Heritability estimates for SOC and LTV were high (>0.5) although a low negative correlation was calculated between SOC and LTV. Fortunately, within that range, minimal (if any) effect of the traits on each other would be expected and the selection of one of these traits should not impede selection of the other significantly.

The heritability estimates of the relevant morphometric traits, including STEP and lumbosacral vertebral canal height, were moderate to high. Furthermore, a distinctive positive correlation ($r_g = 0.95$) was noted at the crucial site of spinal canal narrowing between the caudal endplate of L7 and the cranial endplate of the sacrum creating favourable conditions for breeding selection. Due to the relatively limited sample size of animals studied in the genetic analysis, the definite values estimated have to be interpreted with some caution, and a re-evaluation with adjustment of the population size may be beneficial (Klein, 1974). Nevertheless, there are non-zero heritabilities of relevant morphological and morphometric lumbosacral features in the GSD. Our results not only demonstrate that genetic effects contribute to the phenotypic variance of the LSJ in GSDs, but also provide efficacy for selective breeding against factors potentially associated with lumbosacral disease.

The limitations of the study are mainly attributable to its retrospective nature which may have introduced confounding factors regarding the selection of the study population and the retrospective matching process of the control dogs to the GSDs. The even distribution of physical characteristics between the GSD and the control groups may have been limited by this procedure so a generalization of the results may not be wholly applicable. The analysis of the genetic variance components makes use of a subpopulation of GSDs from the GSD group. In addition, the relatively small sample size and deviating population structure (owing to different ancestry) represent a potential source of bias, particularly in comparison with other populations.

The appearance of acquired lumbosacral features may have been influenced by the disparity in age between the GSDs and the dogs in the control group. However, despite the distinctively lower age of the GSDs compared with the controls, the prevalence of acquired traits such as ARTH, SCLER, and both O-DISC and O-CAN, was greater in the GSD. Thus, although a general distortion of the results by age differences may exist, it should not have led to overestimation of findings in the GSD group. This finding rather suggests premature onset of and/or general disposition towards degeneration of lumbosacral segments specifically in this breed.

Conclusions

An aberrant skeletal conformation of the LSJ was found in GSDs with morphological and morphometric findings when compared with other large breed dogs. The findings suggest the presence of a primary lumbosacral stenosis in GSDs that has not been documented before. Various features that predispose to lumbosacral disease are greater in frequency or magnitude in GSDs, such as SOC, early onset of degenerative changes, and STEP of the LSJ. In addition, relevant proportions of the phenotypic variance of most of the promoting phenotypic features, including SOC, LTV, STEP and lumbosacral stenosis, are determined by genotypic variance. Selection against these factors appears to be feasible and is suggested.

Conflict of interest statement

None of the authors of this paper has a financial or personal relationship with other people or organisations that could inappropriately influence or bias the content of the paper.

References

- Bailey, C.S., Morgan, J.P., 1992. Congenital spinal malformations. *Veterinary Clinics of North America* 22, 985–1015.
- Breit, S., Knaus, I., Künzel, W., 2003. Differentiation between lumbosacral transitional vertebrae, pseudolumbarisation, and lumbosacral osteophyte formation in ventrodorsal radiographs of the canine pelvis. *The Veterinary Journal* 165, 36–42.
- Breit, S., Künzel, W., 1998. Osteologische Besonderheiten an Wirbelsäulen von Rassehunden: Eine röntgenologische und morphologische Studie. *Wiener Tierärztliche Monatsschrift* 85, 340–350.
- Bürger, R., Lang, J., 1992. Kinetische Studie über die Lendenwirbelsäule und den lumbosakralen Übergang beim Deutschen Schäferhund. *Schweizer Archiv für Tierheilkunde* 134, 411–416.
- Conner, J.K., Hartl, D.L., 2004. *A Primer of Ecological Genetics*. Sinauer Associates Inc., Massachusetts, USA.
- Damur Djuric, N., Steffen, F., Hässig, M., Morgan, J.P., Flückiger, M.A., 2006. Lumbosacral transitional vertebrae in dogs: Classification, prevalence, and association with sacroiliac morphology. *Veterinary Radiology and Ultrasound* 47, 32–38.
- Danielsson, F., Sjöström, L., 1999. Surgical treatment of degenerative lumbosacral stenosis in dogs. *Veterinary Surgery* 28, 91–98.
- Dempster, E.R., Lerner, I.M., 1950. Heritability of threshold characters. *Genetics* 35, 212–235.
- DeRísio, L., Thomas, W.B., Sharp, N.J.H., 2000. Degenerative lumbosacral stenosis. *Veterinary Clinics of North America Small Animal Practice* 30, 111–132.
- Feeney, D.A., Wise, M., 1981. Epidurography in the normal dog: Technique and radiographic findings. *Veterinary Radiology* 22, 35–39.
- Flückiger, M.A., Damur-Druric, N., Hässig, M., Morgan, J.P., Steffen, F., 2006. A lumbosacral transitional vertebra in the dog predisposes to cauda equina syndrome. *Veterinary Radiology and Ultrasound* 47, 39–44.
- Flückiger, M.A., Geissbühler, U., Lang, J., 2009. Lumbosakrale Übergangswirbel: Welche Bedeutung haben sie für die Gesundheit von betroffenen Hunden? *Schweizer Archiv für Tierheilkunde* 3, 133–135.
- Glyde, M., Doyle, H., McAllister, H., Campoy, L., Callanan, J.J., 2004. Magnetic resonance imaging in the diagnosis and surgical management of sacral osteochondrosis in a mastiff dog. *Veterinary Record* 155, 83–86.
- Hanna, F.Y., 2001. Lumbosacral osteochondrosis: Radiological features and surgical management in 34 dogs. *Journal of Small Animal Practice* 42, 272–278.
- Indrieri, R.J., 1988. Lumbosacral stenosis and injury of the cauda equina. *Veterinary Clinics of North America Small Animal Practice* 18, 697–710.
- Jones, J.C., Sorjonen, D.C., Simpson, S.T., Coates, J.R., Lenz, S.D., Hathcock, J.T., Agee, M.W., Bartels, J.E., 1996. Comparison between computed tomography and surgical findings in nine large-breed dogs with lumbosacral stenosis. *Veterinary Radiology and Ultrasound* 37, 247–256.
- Kirkaldy-Willis, W.H., Paine, K.W.L., Cauchoix, J., McIvor, G., 1974. Lumbar spinal stenosis. *Clinical Orthopaedics* 99, 30–49.
- Klein, T.W., 1974. Heritability and genetic correlation: Statistical power, population comparisons, and sample size. *Behavioral Genetics* 3, 355–364.
- Kovac, M., Groenenveld, E., Garcia-Cortez, A., 2003. *VCE-5 Users Guide and Reference Manual Version 5.1.2*. Institut für Tierzucht und Tierverhalten, Bundesforschungsanstalt für Landwirtschaft, Mariensee/Neustadt.
- Lang, J., Häni, H., Schawwalder, P., 1992. A sacral lesion resembling osteochondrosis in the German shepherd dog. *Veterinary Radiology and Ultrasound* 33, 69–76.
- Larsen, J.S., 1977. Lumbosacral transitional vertebrae in the dog. *Journal of the American Veterinary Radiology Society* 18, 76–79.

- Luoma, K., Vehmas, T., Raininko, R., Luukkonen, R., Riihimäki, H., 2004. Lumbosacral transitional vertebra: Relation to disc degeneration and low back pain. *Spine* 29, 200–205.
- Mathis, K., Havlicek, M., Beck, J., Eaton-Wells, R.D., Park, F.M., 2009. Sacral osteochondrosis in two German shepherd dogs. *Australian Veterinary Journal* 87, 249–252.
- Mattoon, J.S., Koblik, P.D., 1993. Quantitative survey radiographic evaluation of the lumbosacral spine of normal dogs and dogs with degenerative lumbosacral stenosis. *Veterinary Radiology and Ultrasound* 34, 194–206.
- Meij, B.P., Bergknut, N., 2010. Degenerative lumbosacral stenosis in dogs. *Veterinary Clinics of North America Small Animal Practice* 40, 983–1009.
- Michal, U., Steffen, F., Hauser, B., Damur, D., Philipp, M., 2004. Sacral osteochondrosis dissecans in a Bernese Mountain dog: Diagnosis and treatment. *Schweizer Archiv für Tierheilkunde* 146, 233–238.
- Morgan, J.P., 1993. *Techniques of Veterinary Radiography*, Fifth Ed. Iowa State University Press.
- Morgan, J.P., 1999. Transitional lumbosacral vertebral anomaly in the dog: A radiographic study. *Journal of Small Animal Practice* 40, 167–172.
- Morgan, J.P., Bahr, A., Franti, C.E., Bailey, C.S., 1993. 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, 1877–1882.
- Morgan, J.P., Wind, A., Davidson, A.P., 2000. Transitional lumbosacral vertebrae. In: Morgan, J.P., Wind, A., Davidson, A.P. (Eds.), *Hereditary Bone and Joint Diseases in the Dog*. Schlütersche, Hannover, Germany, pp. 223–229.
- Morgan, J.P., Bailey, C.S., 1990. Cauda equina syndrome in the dog: Radiographic evaluation. *Journal of Small Animal Practice* 31, 69–77.
- Munday, T.L., Johnson, M.H., Hayes, C.W., Thompson, E.O., Smoker, W.R.K., 1994. Musculoskeletal causes of spinal axis compromise: Beyond the usual suspects. *RadioGraphics* 14, 1225–1245.
- Ness, M.G., 1994. Degenerative lumbosacral stenosis in the dog: A review of 30 cases. *Journal of Small Animal Practice* 35, 185–190.
- Oliver, J.R., Selcer, R.R., Simpson, S., 1978. Cauda equina compression from lumbosacral malarticulation malformation in the dog. *Journal of the American Veterinary Medical Association* 17, 207–214.
- Palmer, P.H., Chambers, J.N., 1991. Canine lumbosacral diseases. Part I. Anatomy, pathophysiology, and clinical presentation. *Compendium on Continuing Education* 13, 61–69.
- Rossi, F., Seiler, G., Busato, A., Wacker, C., Lang, J., 2004. 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 and Ultrasound* 45, 381–387.
- Scharf, G., Steffen, F., Grünenfelder, F., Morgan, J.P., Flückiger, M., 2004. The lumbosacral junction in working German Shepherd Dogs: Neurological and radiological evaluation. *Journal of Veterinary Medicine Series A* 51, 27–32.
- Schmid, V., Lang, J., 1993. Measurements on the lumbosacral junction in normal dogs and those with cauda-equina compression. *Journal of Small Animal Practice* 34, 437–442.
- Seiler, G.S., Häni, H., Busato, A.R., Lang, J., 2002. 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.
- Steffen, F., Hunold, K., Scharf, G., Roos, M., Flückiger, M., 2007. 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, 1259–1533.
- Suwankong, N., Voorhout, G., Hazewinkel, H.A.W., Meij, B.P., 2006. Agreement between computed tomography, magnetic resonance imaging, and surgical findings in dogs with degenerative lumbosacral stenosis. *Journal of the American Veterinary Medical Association* 229, 1924–1929.
- Suwankong, N., Meij, B.P., Voorhout, G., 2008. Review and retrospective analysis of degenerative lumbosacral stenosis in 156 dogs treated by dorsal laminectomy. *Veterinary and Comparative Orthopaedics and Traumatology* 21, 285–293.
- Tarvin, G., Prata, R.G., 1980. Lumbosacral stenosis in dogs. *Journal of the American Veterinary Medical Association* 177, 154–159.
- Verbiest, H., 1975. Pathomorphologic aspects of developmental lumbar stenosis. *Orthopedic Clinics of North America* 6, 177–196.
- Vinson, W.E., White, J.M., Kliewer, R.H., 1976. Overall classification as a selection criterion for improving categorically scored components of type in Holsteins. *Journal of Dairy Science* 59, 2104–2114.
- Walla, L., 1986. Die Kompression der Cauda equina beim Hund. *Kleintierpraxis* 31, 315–322.
- Watt, P.R., 1991. Degenerative lumbosacral stenosis in 18 dogs. *Journal of Small Animal Practice* 32, 125–134.
- Wigger, A., Julier-Franz, C., Tellhelm, B., Kramer, M., 2009. 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.
- Winkler, W., Loeffler, K., 1986. Lumbosakrale Übergangswirbel beim Hund. *Berliner und Münchner Tierärztliche Wochenschrift* 99, 343–346.
- Wright, J.A., 1980. Spondylosis deformans of the lumbo-sacral joint in dogs. *Journal of Small Animal Practice* 21, 45–58.