The Hemipelvic Muscle Mass in Small to Medium Size Dogs and its Association with Canine Hip Dysplasia: A Cadaveric Study

Shipov, A. and Milgram, J.

Laboratory of Biomechanics and Applied Anatomy, Koret School of Veterinary Medicine, The Hebrew University of Jerusalem

* **Corresponding:** author – Dr. Joshua Milgram; Laboratory of Biomechanics and Applied Anatomy, Koret School of Veterinary Medicine Hebrew University of Jerusalem. Email- josh.milgram@mail.huji.ac.il; PO Box 12, Rehovot, 761001, Israel; Tel – 972-3-9688558; Fax – 972-3-9604079

ABSTRACT

Canine hip dysplasia (CHD) is among the most common orthopedic diseases in dogs. In large breed dogs, increase in pelvic muscle mass is associated with decreased incidence of CHD. The aim of this study was to characterize individual hip joint muscle contributions to the pelvic muscle mass, and evaluate the suitability of the parameters previously shown to identify large-breed dogs predisposed to CHD to small to medium sized dogs. Fifteen cadavers of mixed-breed dog (age range, 1-4 years; body weight range 6-28 kg) were used. Twenty-four muscles, directly acting on the hip joint were identified during dissection. Each muscle was dissected from surrounding tissues and weighed. These muscles were divided into five groups based on their innervation. The hip joint and surrounding bone were visually examined for cartilage damage and articular osterophyte formation. The hemipelvic muscle weight, as a percentage of body weight, was 4.85%±0.81. The contributions of the muscles innervated by the sciatic, femoral, obturator and the gluteal nerves were 35.73%±1.21, 28.45%±1.49, 21.28%±0.73 and 14.54%±1.17 of the entire pelvic muscle mass, respectively. Based on the previously published criteria for large breed dogs, as many as 67% of dogs in this study would have been expected to be predisposed to CHD; however, all the hip joints of these dogs were free of pathology. It was concluded that hemipelvic muscle mass relationship parameters applied to large breed dogs predisposed to CHD might be unsuitable for small to medium size dogs.

Keywords: Hip joint; Musculature; Degenerative Joint Disease.

INTRODUCTION

The primary functions of skeletal muscles are stabilizing and moving diarthrodial joints. Periarticular muscles contribute significantly to the forces acting on the joint, which compress the joint during weight bearing (1). The contribution of an individual muscle to joint stability depends on its force and stiffness (2). The latter is positively associated with muscle mass (3). Muscle force and stiffness are produced by the cross-bridges cycling as muscles contract and relax (4). Greater muscle mass indicates greater total number of cross-bridges available for spontaneous re-attachment, as well as a larger volume of passive connective tissue components, which contribute to passive stiffness, both of which have significant roles in joint stabilization. Muscles with higher stiffness store more energy when stretched, thereby creating higher system stability level (4). Furthermore, muscles with greater cross-sectional area produce greater active force (5).

Canine hip dysplasia (CHD) is characterized by progressive hip joint remodeling and degeneration, with incompletely defined etiology and pathogenesis (6). Estimates of the heritability of CHD range from 0.2 to 0.6, although non-genetic factors also play roles in its development (6). Healthy hip joint development depends on the congruency between the femoral head and the acetabulum being maintained as the

hip joint begins to be loaded (6). It is proposed that in CHD-susceptible individuals, periarticular muscle mass growth and maturation are disproportionate to that of the skeleton (6). Individuals susceptible to CHD have normal hip joints at birth; however, as the puppy begins to ambulate, the forces generated in the hip joint exceed the supporting soft tissue strength. Stretching of the soft tissues results in joint laxity, subluxation and abnormal loading of the articular cartilage, which damages the cartilage, and initiates inflammation that causes degenerative joint disease. This theory is based primarily on the findings that puppies which subsequently develop CHD are heavier and have smaller peri-articular muscle mass than puppies which do not develop CHD (7, 8).

Previous studies examining the pelvic muscle mass in dogs sought to demonstrate associations between decreased pelvic muscle mass and increased prevalence of CHD (8-11). To determine association between pelvic muscle mass and joint pathology, a previous study examined the relationship between the severity of bony changes characteristic of CHD and the weight of muscles surrounding the hip joint (9). Such bony changes were detected and characterized either visually or radiographically, while pelvic muscle weight was determined by the sum of weights of all the individual muscles acting on the hip joint. That study showed that the frequency of CHD increased with decreasing pelvic muscle mass. Nevertheless, the presence of disuse atrophy, which possibly resulted from pain experienced by dogs with CHD, and its potential impact on the results was not addressed (9).

The aims of the present study were to characterize the contribution of individual muscles acting on the hip joint to the total hemi-pelvic muscle mass in small to medium sized, mixed breed dogs, and evaluate the suitability of previously suggested parameters for identifying individual large breed dogs expected to develop CHD.

MATERIAL & METHODS

Dogs

The dogs included in this study were euthanized at their owners' request, and donated to science with the owners' signed consent. This study was approved by the institutional animal care and use committee (reference number KSVM-VTH/32_17).

Dogs were examined prior to their inclusion in the study, and were excluded if the hip joints muscles were asymmetrical or atrophied, and if a decrease in range of motion of the hip joints and crepitation were detected during hip joint manipulation. Dogs grossly identified as representing particular dog breeds and those showing conformational deviations (e.g., chondrodystrophic breeds) were excluded. Post mortem, the cadavers were refrigerated at -4°C pending dissection, which was performed within 24 hours from euthanasia.

Determination of muscle mass

Immediately after euthanasia, the coxo-femoral joints of each cadaver were examined for presence of joint laxity, crepitation and decreased motion range. The skin and subcutaneous tissues were then carefully removed from the entire hindquarter. All the muscles surrounding the hip joint were identified and meticulously excised, while preserving the entire muscle tissue, and discarding all tendinous and fat tissue. The muscles evaluated included the following (Table 1): superficial gluteal, middle gluteal, deep gluteal, piriformis, tensor fascia lata, lateral sartorius, medial sartorius, vastus lateralis and intermedius, vastus medialis, rectus femoris, biceps femoris, abductor cruris caudalis, semimembranosus, semitendinosus, gracillis, adductor longus, adductor magnus et brevis, pectineus, obturator internus, obturator externus, gemelli, quadratus femoris, articularis coxae and iliopsoas. Each muscle was weighed using a digital scale (YEB-B Electronic Weighing Balance, 1000/0.01g. Shanghai Yong Cheng Scale Co., Shanghai, China).

After completely removing all muscles, the remaining periarticular soft tissues, bone and cartilage were assessed visually for presence of abnormalities. The joint capsule thickness was assessed visually, and its insertion on the femoral neck and acetabulum was inspected for evidence of osteophyte formation. In addition, the capital ligament, labrum and articular cartilage were inspected visually for presence of tears and erosion.

In the first part of the study, muscles of both the right and left hemipelves of four dogs were dissected and weighed. The left and right limb individual muscle weights of each dog were compared. Since differences were insignificant, only unilateral dissections were performed on the remaining cadavers.

Muscle groups and indices

Individual muscles were grouped based on their innervation (12) as following: Group 1, cranial and caudal gluteal nerves;

Table 1: List of innervation-based hemiplevic muscle groups and their individual muscles and the individual muscle mass coefficient of variation of 15 small to medium sized dogs

Group number	Innervating nerve	Muscle	Coefficient of variation
1	Cranial and caudal gluteal	Superficial gluteal	0.12
		Middle gluteal	0.05
		Deep gluteal	0.21
		Piriformis	0.16
		Tensor fascia lata	0.11
2	Sciatic	Biceps femoris	0.04
		Semimembranosus	0.08
		Semitendinosus	0.09
3	Sciatic (outward hip rotators)	Internal obturator	0.09
		Gemelli	0.36
		Quadratus femoris	0.14
4	Femoral	Vastus lateralis and intermedius	0.06
		Vastus medius	0.18
		Rectus femoris	0.08
		Iliopsoas	0.15
		Cranial sartorius	0.15
		Caudal sartorius	0.10
5	Obturator	Gracillis	0.10
		Adductor longus	0.20
		Pectineus	0.13
		External obturator	0.17
		Adductor magnus et brevis	0.05

Group 2 (hip joint extensors) and 3 (hip joint rotators), sciatic nerve; Group 4, femoral nerve; Group 5, obturator nerve (Table 1).

The hemipelvic muscle mass index (HMI) was defined as total hemipelvic muscle weight (T) divided by whole body weight (B) (9) as following: HMI = [T (g)/B (g)] x100. The group muscle mass index (GMI) was defined for each of the five muscle groups as group weight (G) divided by total hemipelvic muscle weight (T) as following: GMI = [G (g)/T (g)] x100. Individual muscle mass index (IMMI) was defined as individual muscle mass (IMM) divided by its total muscle group weight (G) as follows: IMI = [IMM (g)/G (g)] x100.

Evaluation of the hip joint

After all soft tissue removal, the acetabulum and femoral head and neck were carefully examined visually for signs of degenerative joint disease associated with CHD, including capital ligament integrity, presence of synovitis, joint capsule thickness, bony proliferation evidence in the origin and insertion of the joint capsule on the acetabulum and the femoral neck and changes consistent with CHD in the coxo-femoral joint labrum and cartilage, particularly in the femoral head and acetabular rim dorsal aspects.

Statistical analyses

The Shapiro-Wilk's test was used to assess normality. For the first part of the study a Mann-Whitney test was used to compare muscle weights between the right and left hemipelves. The mean and standard deviation were calculated for HMI and GMI using the data from all dogs. The IMI coefficient of variation was calculated for each muscle to evaluate its variation among dogs. Pearson's correlation coefficient was calculated for all possible pairs of muscles within each group. Only significant correlations are reported. A $P<0.05$ was considered statistically significant.

RESULTS

Fifteen well-muscled small to medium sized mixed breed dogs, ranging in age from 1 – 4 years, were included in this study.

The mean HMI of all joints was 4.85%±0.81. HMI ranged from 3.03% in a 6-kg dog to 6.13% in a 26-kg dog. The HMI was less than 5% in 66.7% of the dogs (Figure 1). None of the joints showed abnormalities associated with hip joint degenerative joint disease.

The GMI data of each muscle group are illustrated in Figure 2. Muscle groups 2 and 3, innervated by the sciatic nerve, were the most massive muscles, contributing 35.7%±1.2% to the hemi-pelvic muscle mass. The muscles innervated by the femoral, obturator and gluteal nerves contributed 28.5%±1.5%, 21.28%±0.7% and 14.54%±1.2% to the hemipelvic muscle mass, respectively.

The coefficients of variation (CV) of the IMMI data are listed in Table 1. The gemelli muscle IMMI CV was highest (0.36), while for the remaining muscles, CVs were ≤ 0.21.

The correlations ($r > |0.40|$) of muscle mass between muscle pairs within each muscle group are listed in Table 2.

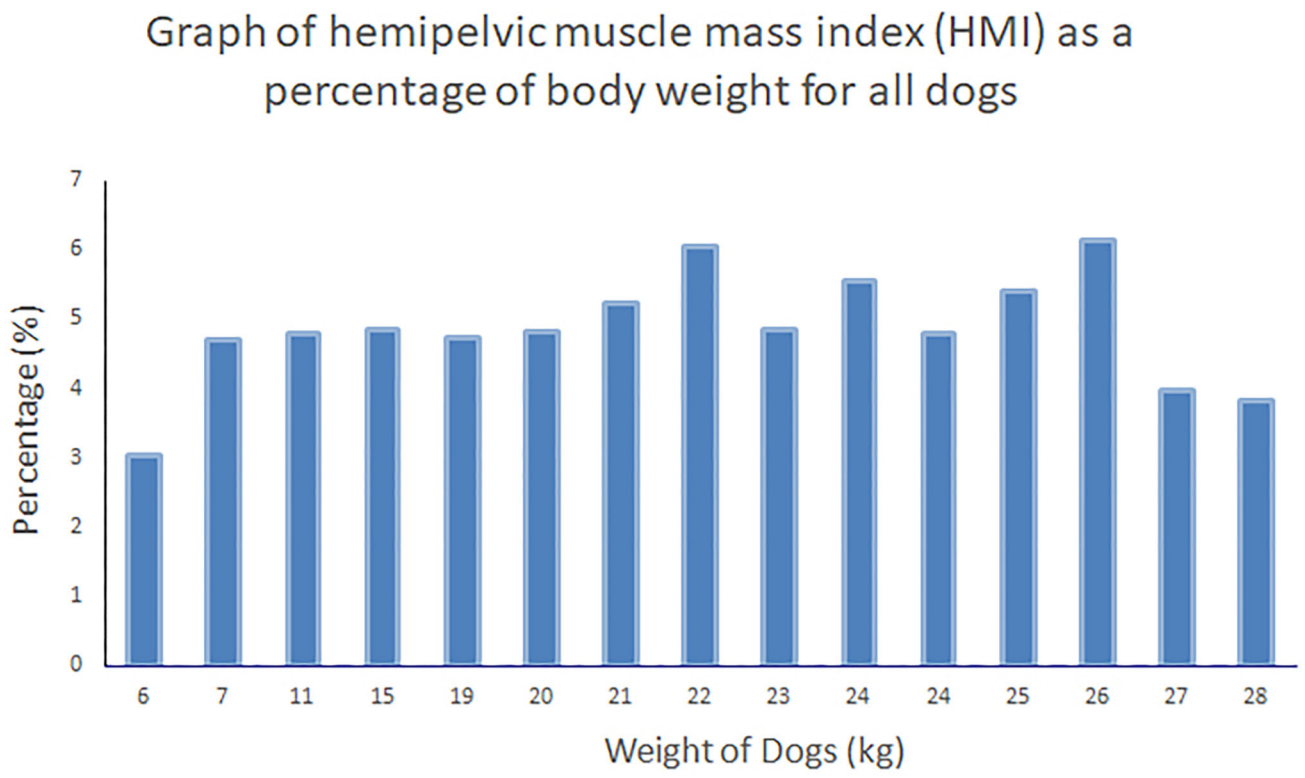

Figure 1. Hemipelvic muscle mass index (HMI) which represents the total muscle mass around a single hip joint as a percentage of body weight, for all subjects.

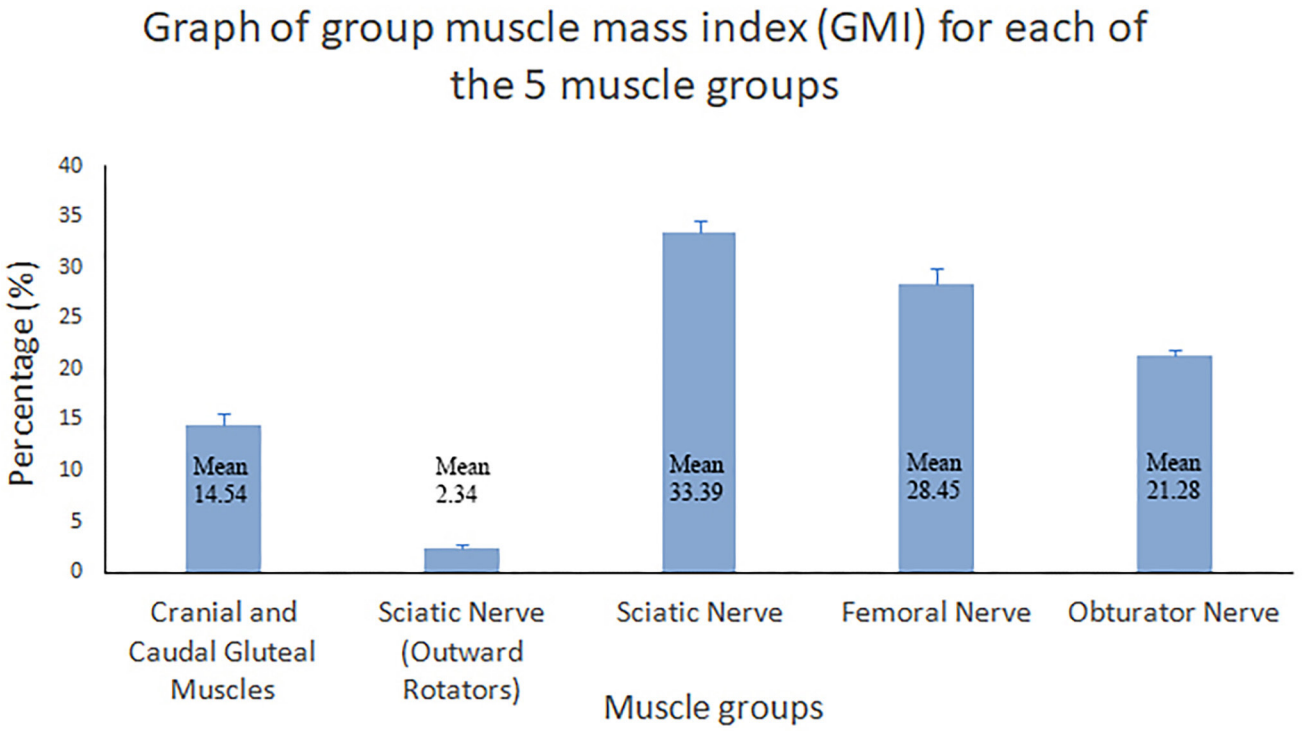

Figure 2. Group muscle mass index (GMI) was calculated for each of the 5 muscle groups for all subjects. The group muscle mass is represented as a percentage of hemipelvic muscle mass.

Table 2: Correlation coefficients for muscle pairs within each group of muscles.

Group	Muscle	r	P value
1	Superf glut / Mid glut	-0.61	0.016
	Deep Glut / Piriformis	0.83	<0.001
	Middle gluteal / Tensor fascia lata	-0.53	0.043
	Deep gluteal / Tensor fascia lata	-0.61	0.016
	Piriformis / Tensor fascia lata	-0.61	0.016
2	Biceps Femoris / Semimemranosus	-0.72	0.003
	Semimemranosus / Semitendinosus	-0.47	0.074
3	Internal Obturator / Gemelli	-0.59	0.022
	Internal Obturator / Quadratus femoris	-0.54	0.038
4	Vastus medialis / Rectus femoris	-0.72	0.002
	Vastus medialis / Cranial sartorius	-0.51	0.05
5	Adductor longus / External obturator	0.85	<0.001
	Gracillis / Adductor magnus et brevis	-0.77	<0.001
	Adductor longus / Adductor magnus et brevis	-0.57	0.026
	Pectineus / Adductor magnus et brevis	-0.54	0.038

r – Coefficient correlation (Correlation was considered significant if $P \leq 0.05$.)
Listed are muscle pairs having a $|r| > 0.40$.

DISCUSSION

This study demonstrates that low muscle mass in small and medium sized dogs is not associated with bony changes characteristic of CHD, as reported in larger breed dogs. To facilitate comparison of the present results to previous published data, a 2-fold HMI is referred to as the pelvic muscle mass index (PMI). The PMI of the present cohort is lower than previous data (9.70±1.60 herein vs. 16.2±1.5 in greyhounds and 9.9±0.8 in German shepherd dogs [GSD] with CHD) (9).

Riser *et al.* examined the association between CHD and pelvic muscle mass in 95 dogs divided into six groups based on frequency of occurrence of CHD (8). The mean PMI of the entire cohort, which included greyhounds, GSD with CHD and July hound with subluxated hips were 9.9, 14.2 and 9.0, respectively. Based on these results, the authors defined critical index levels predicting development of normal and dysplastic hip joints. A PMI ≤10.89 was predictive of CHD, with sensitivity of 86% (8). If this cutoff was to be applied to the present cohort, changes associated with CHD would have been expected to be common, as 10/15 of the dogs had PMI<10; yet, changes consistent with CHD were not identified in any of the dogs dissected herein. This suggests that the above-mentioned previously published cutoff, applicable to large breed dogs, cannot be applied to small to medium sized mixed breed, non-chondrodysplastic dogs.

The GMI in this study was lower than data published for greyhounds and greyhound-GSD cross dogs with or without CHD, however, they are comparable to those of both normal and dysplastic GSD dogs, excluding the femoral nerve-innervated muscle group in normal GSD dogs [(9), Table 3].

The contribution of each muscle group to the total hemi-pelvic muscle weight (GMI) in this study was fairly consistent (Figure 2). Furthermore, results of the contribu-

Table 3: Comparison of Group muscle mass index for large dogs[a] with and without hip dysplasia and small and medium size dogs without hip dysplasia.

Muscle Group	Mixed Breed Dogs (n=15) (Present study)	Greyhounds (n=5)[a]	GSD Normal hips (n=12)[a]	GHXGSD Normal hips (n=5)[a]	Normal hips[a] Mean (±SD)	GSD Dysplastic (n=23)[a]	GHXGSD Dysplastic (n=7)[a]	Dysplastic Hips[a] Mean (±SD)
Cranial and Caudal Gluteal Nerve	14.54(±1.17)	13.64	14.21	13.90	14.07(±0.39)	14.47	14.69	14.58 (±0.16)
Sciatic Nerve	35.73(±1.72)	38.29	36.43*	39.00	37.36(±1.54)	36.93	37.20	37.07(±0.19)
Femoral Nerve	28.45(±1.49)	26.74	29.77*	26.40	27.84(±1.57)	29.44	28.09	28.77(±0.95)
Obturator Nerve	21.28(±0.73)	21.37	19.56	20.60	20.70(±0.84)	19.10	20.05	19.58(±0.67)

GH-Greyhound, GSD-German Shepherd Dog, GHXGSD – German Shepherd Dog-Greyhound crossbreeds.[a] data reported by Cardinet *et al.*

tion of each muscle group to the total hemipelvic muscle mass were similar when the GMI values were calculated based on previously reported data (9). It is noteworthy that the contribution of each muscle group remained consistent regardless of presence or absence of CHD, and possibly decreasing the load on painful hip joints induced a generalized muscle atrophy, where the proportional contribution of each muscle group to the total hemipelvic muscle mass remained constant. Therefore, possibly the proportional contribution of each muscle group to the total hemipelvic muscle mass is tightly genetically controlled.

The IMI was calculated in this study to evaluate individual muscle contribution in its muscle group. The gemelli muscle IMI showed the highest CV (0.36), while IMI CVs of the remaining individual muscles were ≤ 0.21. Likely, the weighed mass of the gemelli muscle, being the muscle with the lowest mass, was highly adversely affected by dissection-related inconsistencies. There is no consensus definition of what constitutes a high individual muscle mass CV; nevertheless, considering the body weight range of the individual dogs in this study, we believe that the individual muscle variation among dogs might be small. The IMI was not reported in previous studies. Therefore, we were unable to derive it from previously reported data.

Correlations were examined to further characterize the individual muscles within groups. We hypothesized that within a muscle group, there would be no correlation (r=0) in muscle mass between pairs of muscles with broadly similar functions. The *r* values varied between |0.41| and |0.85|, and although reflecting only moderate correlations between muscle pairs, they are nevertheless valuable in assessing potential causation.

Pairs of muscles with broadly similar functions were generally negatively correlated. There was a negative moderate correlation between the masses of the gracillis and adductor magnus et brevis muscles (r = -0.77), indicating that approximately 60% of the decrease in weight of the adductor magnus et brevis muscle mass might be due to an increase of the weight of the gracillis muscle. Exceptions to this were seen in Group 1, where the deep gluteal muscle mass was quite strongly and positively (r = 0.83) correlated with the piriformis muscle mass. This possibly supports the theory that the piriformis muscle is not a separate muscle in the dog, but is rather part of the deep gluteal muscle.

The adductor longus muscle mass had a rather strong positive correlation (r = 0.85) with the external obturator muscle mass. We were unsure as to the biological significance of this finding, as these two muscles have different functions. Nevertheless, it may be speculated that this correlation reflects the situation in a healthy joint. This relationship should be examined in future studies in dogs with CHD.

This study has several limitations. First, this cohort, including small to medium sized dogs with large variations in body weight that should be expected to show low frequency of CHD, limits conclusions which can be drawn. Another limitation is absence of hip joint radiography, which is considered the gold standard in diagnostic test for CHD. We assumed that as we used adult dogs, sufficient time would have passed to allow for the easy identification of dysplastic joints at necropsy. Nevertheless, we were unable to identify any osteophyte formation at the joint capsule insertions, or other abnormalities upon careful inspection of the joint capsule, labrum and capital ligament.

In conclusion, the contribution of hemiplevic individual muscle and muscle group mass in small-medium dogs were characterized. The previously suggested parameters defining the relationships of hemiplevic muscle mass in large breed dogs predisposed to CHD might not be suitable for small to medium sized mixed breed dogs, in which pelvic muscle mass is lower, even in absence of CHD.

ACKNOWLEDGEMENTS

The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

REFERENCES

1. Aura, O. and Komi, P.V.: Effects of muscle fiber distribution on the mechanical efficiency of human locomotion. Int.J. Sports. Med. 8 Suppl 1:30-37, 1987.
2. Brown, S.H.M. and McGill, S.M.: Muscle force-stiffness characteristics influence joint stability: A spine example. Clin. Biomech. 20:917-922, 2005.
3. Chleboun, G.S., Howell, J.N, Conatser, R.R. and Giesey, J.J: The relationship between elbow flexor volume and angular stiffness at the elbow. Clin. Biomech. (Bristol, Avon) 12:383-392, 1997.
4. Huxley, A.F.: Muscle structure and theories of contraction. Prog. Biophys. Biophys. Chem. 7:255-318, 1957.
5. Miller, A.E.J., Macdougall, J.D., Tarnopolsky, M.A. and Sale, D.G.: Gender Differences in Strength and Muscle-Fiber Characteristics. Eur. J. Appl. Physiol. Occup. Physiol. 66:254-262, 1993.

6. Smith, K.G., Leighton, E.A., Karbe, G.T., et al: Pathogenesis, diagnosis, and control of canine hip dysplasia, in Johnston, S.A. and Tobias, K.M. (eds.): Veterinary Surgery Small Animal (ed 2nd), Vol. 1. St. Louis, Missouri, Elsevier, 2018.
7. Riser, W.H., Cohen, D., Lindqvist, S., Mansson, J. and Chen, S.: Influence of early rapid growth and weight gain on hip dysplasia in the German Shepherd Dog. J Am Vet Med Assoc. 145:661-668, 1964.
8. Riser, W.H. and Shirer, J.F.: Correlation between Canine Hip Dysplasia and Pelvic Muscle Mass – a Study of 95 Dogs. Amer. J. Vet. Res. 28:769-777, 1967.
9. Cardinet, G.H., Kass, P.H., Wallace, L.J. and Guffy, M.M.: Association between pelvic muscle mass and canine hip dysplasia. JVMA. 210:1466-1473, 1997.
10. Ihemelandu, E.C., Cardinet, G.H., Guffy, M.M. and Wallace, L.J.: Canine Hip-Dysplasia – Differences in pectineal muscles of healthy and dysplastic German Shepherd Dogs when 2 Months old. Am. J. Vet. Res. 44:411-416, 1983.
11. Lust, G., Ross, G.E., Geary, J.C. and Ross, G.E. Jr.: Changes in Pelvic Muscle Tissues Associated with Hip Dysplasia in Dogs. Am. J. Vet. Res. 33:1097-1108, 1972.
12. Hermanson, J.W.: The muscular system, in Evans HE, de Lahunta A (eds): Miller's anatomy of the dog (ed 4th), Vol. St. Louis, Missouri, Elsevier, 2013, pp. 185-281.