Elbow dysplasia is a common cause of forelimb lameness in juvenile medium- and large-breed dogs; the most common dysplastic lesion of the elbow joint is FMCP. Despite a wealth of literature describing the purported mechanism of formation of this lesion, the true pathogenesis of the osteochondral changes preceding FMCP remains poorly defined. Early data suggested that FMCP was a result of osteochondrosis. However, findings of more recent studies have countered this theory and instead suggest that primary supraphysiologic overload of the medial aspect of the coronoid process in association with joint incongruity results in formation of an FMCP. In support of the latter theory, a histomorphometric study of excised coronoid processes from dogs with FMCP revealed that diffuse damage and fatigue microfracture of the subchondral bone both preceded overt cartilage fissuring and paralleled in severity the gross pathological findings of the fragment. As well as diffuse subchondral microfracture in dysplastic specimens, adjunctive osteocyte loss and increased bone porosity have also been described.

Clinical diagnosis of FMCP can be challenging because evidence of overt fragmentation is rarely provided via conventional radiography. A recent study involving concurrent computed tomographic and arthroscopic assessments of elbow joints in dogs revealed that this dual approach provides the most accurate assessment of pathological changes in the coronoid process in vivo. However, because of both the accessibility and cost associated with advanced imaging procedures, conventional radiography remains the most commonly used imaging technique with which a diagnosis
is achieved. Assessment of secondary adaptive and degenerative changes are useful in aiding diagnosis.13 One such change, that of ulnar trochlear notch sclerosis, has been recently quantified as a change in radiopacity affecting the craniodistal aspect of the trochlear notch in dogs with FMCP.13 Results of that study indicate that an increase in ulnar trochlear notch bone radiopacity accompanies FMCP. These findings appear to contradict aspects of the aforementioned histomorphometric study13 of the MCP and associated subchondral bone in dogs, in which osteocyte loss and increased porosity rather than an increase in bone microarchitecture were detected in this region. Although sagittal and parasagittal osteochondral regional BMDs of the elbow joint in clinically normal dogs have been quantified,14 no studies have directly compared the topographic distribution of BMD in normal and fragmented coronoid processes to our knowledge. Such information would be useful for defining whether regional differences in BMD and thus differences in loading characteristics of fragmented coronoid processes differ from those of unaffected MCPs. The purpose of the study reported here was to objectively quantify BMD in MCPs of dogs with and without FMCPs by use of DEXA and to establish how previously reported regional decreases in subchondral porosity10 relate to changes in BMD in this region.

Materials and Methods

Dogs and osteochondral sample collection—Osteochondral samples were obtained from dogs that underwent unilateral or bilateral subtotal coronoid ostectomy as a treatment for FMCP (Figure 1). Fifty osteochondral FMCP samples were collected from 31 dogs. Among the dogs, there were 17 Labrador Retrievers, 6 Rottweilers, 3 Border Collies, 2 Boxers, 1 Bulldog, 1 Airedale Terrier, and 1 Flat-Coated Retriever. Osteochondral samples were obtained from 12 dogs that had unilateral FMCP and 19 dogs that had bilateral FMCP (1 sample/affected joint). Of the 31 dogs, 24 were male (15 were neutered) and 7 were female (2 were neutered). The weight of the FMCP group dogs ranged from 15.0 to 35.4 kg (mean ± SD weight, 32.4 ± 8.83 kg). Age at which forelimb lameness and signs of elbow joint pain became noticeable in these dogs ranged from 5 to 71 months (mean age, 22.9 ± 19.28 months).

Ten control osteochondral samples of the MCP were obtained from 5 healthy Greyhounds (1 sample/forelimb) that were free from forelimb disease and that were euthanatized (via IV administration of an overdose of pentobarbital sodium) for reasons unrelated to the study. At the time of euthanasia, weights of the control group dogs ranged from 30.0 to 36.0 kg (mean weight, 31.8 ± 2.49 kg); the age range of these dogs was 14 to 47 months (mean age, 7.28 months).

Bone samples were harvested and stored in buffered 10% formol-saline solution prior to analysis. Prior to sample collection following euthanasia, craniocaudal and flexed mediolateral radiographic views of both elbow joints were obtained for all dogs with and without FMCP. All radiographic views were scored for osteophytes according to the International Elbow Working Group protocol.15 In this scheme, a score of 0 indicates no radiographic evidence of osteoarthritic change and a score of 3 indicates maximal osteophyte diameter > 5 mm.

Bone samples were mounted in 8-mm-thick blocks of expanded polystyrene for DEXA analysis. Areal BMD analysis of all subtotal coronoid ostectomy fragments was performed by use of a densitometry scanner and software (small animal application); BMD values were calculated by dividing bone mineral content by skeletal area. Each
polystyrene sheet was analyzed by use of the DEXA machine and software prior to placement of samples on it to confirm a mineral content reading of zero for each sheet. All samples were mounted and analyzed in an identical manner in a proximodistal orientation and then subsequently in a mediolateral orientation. Each osteochondral fragment was labeled on the polystyrene block, and digital photographs of each block of samples were taken at the time of analysis as a record of fragment order and orientation.

An AOI for measurement of mBMD of 0.03 cm² was defined, and data from each osteochondral sample were collected in 0.03-cm² increments with the first measurements at the level of the radial incisure for both orientations of analysis. Data were collected to within 2 mm of the osteotomized surface of each fragment.

**Statistical analysis**—All data were assessed for normality and confirmed to not deviate from a normal distribution. A paired t-test was used to assess for a difference in mBMD between right and left limb control osteochondral samples in the proximodistal or mediolateral orientations. Mean BMD data from left and right limb control osteochondral samples were then combined to yield a mean control BMD value for each AOI in both proximodistal and mediolateral orientations. Data from both left and right limb FMCP osteochondral samples were combined to yield a mean FMCP BMD value for each AOI in both proximodistal and mediolateral orientations.

![Figure 2](image-url)
Unpaired t tests were performed to assess for differences in age and body weight of the dogs in the 2 groups as well as differences in mBMD of the control and FMCP osteochondral fragments for each AOI in both proximodistal and mediolateral orientations. In addition, unpaired t tests were performed to assess for differences in axial and abaxial mBMDs in control or FMCP samples. Significance was defined as a value of $P < 0.05$.

**Results**

Results of unpaired t tests indicated that the weights or ages of the control and FMCP group dogs did not differ significantly ($P = 0.96$ and $P = 0.87$, respectively). Radiographic views of each elbow joint from which osteochondral samples were obtained were scored according to the International Elbow Working Group protocol. For all elbow joints in the control group dogs, the score was 0, indicating no evidence of osteoarthritic change bilaterally. For the FMCP group dogs, 2 elbow joints were assigned a score of 3, 18 elbow joints were assigned a score of 2, 12 elbow joints were assigned a score of 1, and 18 elbow joints were assigned a score of 0. Dual-energy x-ray absorptiometry revealed no significant ($P = 0.19$) difference in mBMD between left and right limb osteochondral samples in the control group.

By use of DEXA, the proximodistal and mediolateral regional mBMDs in control and FMCP samples were mapped (Figures 2 and 3). Unpaired t tests performed on the mean proximodistal or mediolateral AOI data from the control and FMCP groups revealed a significant ($P < 0.001$) difference in BMD between groups in both orientations. Unpaired t tests performed to compare the axial and abaxial data from the control or the FMCP group revealed a significantly lower mBMD in the axial region of samples in each group ($P = 0.01$ and $P = 0.04$, respectively).

**Discussion**

Results of the study reported here indicated that there are distinct regional variations in mBMD within and between MCPs of dogs with and without FMCP. Analysis of the regional mBMD of the control group revealed variation in mBMD in both proximodistal and mediolateral orientations. On the basis of proximodistal regional mBMD values, the central articular portion of the MCP had the highest mineral density. This region extends sagittally throughout the body of the MCP from the region caudal to the radial incisure caudally toward the base of the trochlear notch. Mean BMD was approximately 50% higher in the abaxial portion of the MCP than the value along the axial margin. If BMD varies as a consequence of bone loading, then this suggests that load transfer from the medial portion of the humeral condyle is predominantly through the abaxial portion of the MCP.

Proximodistal regional mBMD analysis of the FMCP samples revealed a similar pattern of BMD distribution to that observed for the control samples; however, compared with the control group findings, mBMD values for the FMCP group were decreased throughout the entire articular region. This difference in proximodistal regional mBMD between the control and FMCP groups was significant. Also, comparison of mediolateral regional mBMD values revealed a significant difference between the 2 groups. Assessment of the FMCP group mBMD values revealed a region of greater BMD in the centrocaudal articular portion of the bone, similar to that observed in the control group. In the FMCP group, mean BMD values for the axial portion of the MCP and the radial incisure were decreased, compared with the value for the abaxial articular margin (as determined for the control group). This pattern of mBMD again suggests that loading is predominantly through the abaxial portion of the FMCP.

The topographic variation in mBMD observed both within and between control and FMCP osteochondral samples in the present study is an interesting finding. It has previously been purported that supraphysiologic loading of the MCP, either because of a short radius or ulnar trochlear notch dysplasia, may result in FMCP. However, if overloading of the MCP does occur, then a...
pathoadaptive increase in bone density specifically in the axial portion of the MCP where fragmentation occurs (reflecting excess loading in this region) may be expected. Such a change was not evident in the FMCP-affected dogs in our study. On the basis of topographic variation in proximodistal mBMD in the control group and assuming that BMD is increased in association with loading, MCP loading would be expected to be directed through the centroaxial portion of the MCP rather than through the axiocranial portion of the process where fragmentation typically develops.

Results of a recent histomorphologic study in dogs have indicated that there are decreased osteocyte numbers and increased porosity in the axial portion of MCPs, compared with findings in unaffected MCPs. The comparatively decreased mBMD in this region detected in the FMCP samples in the present study support those findings. Together, the data from the 2 studies are suggestive of regional axial osteoporosis of the FMCP in dogs.

Studies in humans have revealed that the compressive strength of bone is proportional to the square of its apparent density, and this fact has been used to estimate bone strength on the basis of densitometric examination. To the authors’ knowledge, no similar studies have been performed to investigate the compressive strength of canine bone as a function of BMD. However, extrapolation from the human studies would predict that compressive strength of the axial portion of the MCP in dogs is reduced, compared with that of the abaxial surface. If an elbow joint becomes incongruent during a dog’s development such that proximodistal loading of the MCP transiently increases, then mechanical overload could result in preferential subchondral failure of the axial portion of the MCP because of the decreased bone densities in this region of the coronoid process. Such a theory is supported by the fact that the axial mBMD value in the present study was approximately 50% less than that of the abaxial value, which would infer a 4-fold decrease in compressive strength of the axial portion of the MCP.

Data obtained from an in vitro study of canine femurs indicate that microcrack accumulation impairs the mechanical properties of bone by reducing its elastic modulus. Microcrack formation is an integral feature of FMCP development.10 Reduction in compressive tolerance of the axial portion of the MCP because of decreased BMD in this region may predispose to microcrack formation. This in turn reduces the elastic modulus of the bone. Thus, for a given stress during loading, the strain within the axial portion of the MCP may be increased, thereby perpetuating further microcrack formation, increasing bone porosity via subsequent remodeling of those fatigue fractures, and resulting in a progressive reduction in BMD in this region.

The findings of our study appear to contradict those of a previous radiographic investigation in which ulnar trochlear notch radiopacity was quantified in Labrador Retrievers with FMCP.22 In that radiographic study, sclerotic change was greatest in the craniodistal aspect of the ulnar trochlear notch in FMCP-affected dogs. The radiographic data predicted increases in BMD of FMCPs; however, histomorphometric analysis findings and the results of the present study do not support this. Thus, for ulnar trochlear notch sclerosis to develop in the area previously described,3 the adaptive increases in loading that induce sclerotic change must be occurring through the ulnar trochlear notch proximolaterally rather than through the MCP per se. A reduction in the BMD of an FMCP would infer a corresponding decrease in compressive strength of the MCP, with the potential for preferential transfer of load proximolaterally along the ulnar trochlear notch rather than through the MCP. This change in vectoral compressive force could result in increased proximolateral ulnar trochlear notch loading and adaptive sclerotic change specifically within this region of the ulna rather than within the MCP.

In the present study, DEXA analysis was used to obtain quantitative measurements of osteochondral density. Similar techniques have been used in an in vivo study of knee joint osteoarthritis in humans. Other means of quantitative assessment of bone density include computed tomographic osteoabsorptiometry, which has been applied in assessments of human elbow joints.24 However, studies to evaluate the diagnostic potential of that imaging technique in veterinary medicine are currently lacking. Historically, assessment of canine ulnar trochlear density has been subjective and involved observer evaluation of radiographic views. However, standard radiographic projections do not facilitate direct visualization of the MCP and observer assessment of trochlear sclerosis has recently been shown to be unreliable,25 suggesting that alternative quantitative means of assessing bone density of dysplastic canine elbow joints should now be considered.

A limitation of our study was a lack of control breed-matched osteochondral samples with which to make a direct comparison between FMCP-affected and nonaffected dogs. Nevertheless, comparisons between MCPs of FMCP-affected dogs and Greyhounds without FMCPs were informative and allowed determination of patterns of bone density distribution of the MCP in FMCP-affected elbow joints and in joints in a breed in which elbow dysplasia is not common. The Greyhounds were retired racing dogs that were euthanized for reasons unrelated to the study; as such, an increase in BMD, as is the case with human athletes,26,27 may have developed in regions of the control dogs’ MCPs. However, significant asymmetric variation in ulnar bone density (as a consequence of adaptive remodeling) was not evident between limbs of dogs in the control group.

Results of the present study indicated that mBMD is reduced in osteochondral samples of MCPs in FMCP-affected dogs, compared with mBMD in samples of MCPs in Greyhounds without FMCP. In dogs with and without FMCP, topographic differences in mBMD of the MCP were detected; the BMD of the centroaxial region of the process was significantly decreased, compared with the findings in the abaxial region. These differences in BMD may influence compressive strength within the MCP and predispose to osteochondral failure in the cranioaxial portion of the MCP in dogs.

a. PIXIImus scanner, Lunar, Madison, Wis.

b. GraphPad Instat, version 3.06, Graphpad Software Inc, San Diego, Calif.

References