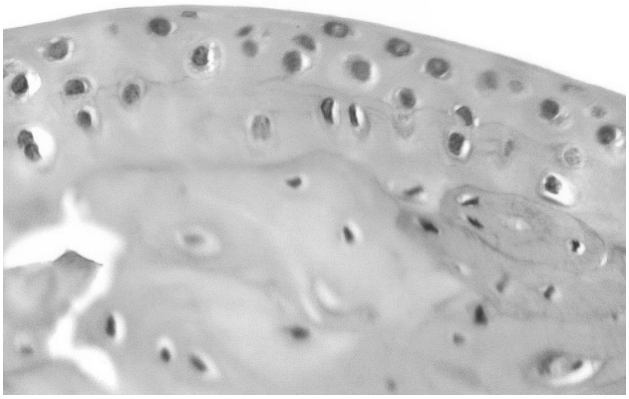

Fig. 2. Knee joint, articular cartilage, normal control, ank^{+}/ank^{+} mouse. Figure width = 100 μm.

capsule and periosteum occurs, followed by osseous metaplasia and ultimately ankylosis. With age, the sequence of histologic changes proceeds progressively to involve the proximal joints as well.

Conclusions: The ank^{-}/ank^{-} mouse demonstrates chondrocyte hypertrophy, joint space calcification and excessive peri-articular ossification, all features that occur to more or less extent in osteoarthritis. As these features develop and progress over 18 weeks of life, we conclude that the ank^{-}/ank^{-} mouse is a useful model for investigating the pathogenesis and regulation of these articular morphologic features of osteoarthritis.

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CHONDROGENESIS OF INFRAPATELLAR FAT PAD AND SYNOVIAL MEMBRANE CELLS IN COMPARISON TO ARTICULAR CARTILAGE CHONDROCYTES

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Purpose: Cartilage is frequently damaged and only has a limited capacity for repair. There is growing interest in the use of cell based therapies for enhancing cartilage repair. Autologous chondrocytes have been used in the repair of focal articular cartilage defects, although there are issues with donor site morbidities. There has been recent interest in the use of undifferentiated adult progenitor cells from other sources, which under appropriate culture conditions can be differentiated into chondrocytes. Such cells can be sourced from a variety of tissues, including the synovial membrane (SM) and the infrapatellar fat pad (IPFP). In this study we identified the chondrogenic potential of canine cells sourced from the SM and the IPFP, and compared their potential with articular cartilage chondrocytes (ACC). We hypothesised that IPFP and SM derived cells would have comparable chondrogenic potential in comparison to ACC in an *in-vitro* chondrogenesis model.

Methods: IPFP, SM and articular cartilage were harvested from the femoropatellar joint of 4 skeletally mature dogs (aged 3-5 years) which had no signs of joint disease. Cells were isolated by collagenase digestion and proliferated for two passages in monolayer culture. 3D cell aggregates were formed from 500,000 cells by centrifugation. The cell aggregates were placed in chondrogenic media and cultured for 2 weeks. Chondrogenesis was assessed by: Wet weight, Histologically (H&E, safranin O staining and collagen-II immunohistochemistry), Gene expression (real-time PCR quantification of collagen-I, -II, aggrecan), and Biochemically (Total glycosaminoglycan (GAG) and DNA content).

Results: ACC derived cell aggregates were significantly bigger than aggregates derived from either IPFP or SM cells ($p<0.05$), with the mean wet weight of the aggregates being 5.2mg for the ACC cells, 1.2mg for the IPFP cells and 1.1mg for the SM cells. Histologically the IPFP and SM cell derived aggregates showed significantly lower matrix accumulation in comparison to the aggregates from ACC cells, and stained much more poorly for both GAG and Collagen-II. Similarly, both total GAG and DNA content of the aggregates were significantly lower in both the SM and IPFP cell derived aggregates in comparison to the ACC cell derived aggregates. Average GAG content per cell aggregate was over five fold lower in aggregates derived from SM cells, and three fold lower in aggregates derived from IPFP, in comparison to those derived from ACC. ACC derived cell aggregates expressed high levels of both collagen-II and aggrecan genes, although collagen-I gene expression was of a similar magnitude to that of collagen-II expression. Average ratio of collagen-II to collagen-I gene expression per cell aggregate was over 500 fold lower in aggregates derived from SM cells, and nearly a 100 fold lower in aggregates derived from IPFP, in comparison to those derived from ACC.

Conclusions: Under the culture conditions used in this study, cell aggregates derived from ACC were able to produce a cartilage like matrix with the cells showing gene expression indicative of a chondrogenic phenotype. However, the ACC cells showed similar levels of collagen-I gene expression in comparison to collagen-II gene expression indicating a failure to fully recapitulate the phenotype of cells found within hyaline articular cartilage. Using the cell extraction protocol and culture conditions used in this study, neither SM nor IPFP derived cells demonstrated ability to produce a cartilage like matrix, nor did these cells demonstrate a gene expression profile consistent with chondrogenic differentiation.

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PREVALENCE OF RADIOGRAPHIC OSTEOARTHRITIS OF KNEE AND LUMBAR SPINE, AND ITS ASSOCIATION WITH PAIN: THE RESEARCH ON OSTEOARTHRITIS AGAINST DISABILITY (ROAD) STUDY

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Purpose: Although osteoarthritis (OA) of knee and lumbar spine is a major cause of disability in the elderly, few epidemiologic studies have been performed. We established a large-scale nationwide clinical study called ROAD (research on osteoarthritis against disability) in 2005 to clarify the environmental and genetic backgrounds. We began the study by creating a comprehensive and systemic database including clinical and genomic information in two cohorts of urban and rural areas. From their baseline data, we investigated the prevalence of radiographic OA of knee and lumbar spine, as well as its association with the respective local pain.

Methods: We recruited 1,885 inhabitants older than 50 years (mean age=73.5 yrs.): 1,030 (355 men & 675 women; mean age=76.9 yrs.) from the urban and 855 (317 men & 538 women; 69.3 yrs.) from the rural areas. The radiographic severity of OA was determined according to the Kellgren/Lawrence (KL) grade (0-4) at femoral-tibial joints of bilateral knees and at intervertebral spaces from L1/2 to L5/S1 of the lumbar spine by a blinded orthopaedic surgeon. Logistic regression analysis was performed after adjustment for age to determine the association.

Results: Prevalence of radiographic OA (KL> or = 2) in either