

improvement, owner satisfaction questionnaire (which included six questions about patients response to the treatment and life quality) and all adverse effects to treatments recorded (Aes).

Results were analyzed by the SPSS 20.0 program. The nonparametric Kruskal-Wallis and Mann-Whitney tests were used to compare non-categorical variables and crosstabs with contingency coefficient to evaluate the categorical ones.

C. Results: A total of 42 males and 8 females, with mean age of 74 ± 39 (8–135) months and mean weight of 36.6 ± 11.9 (20–66.2) kg were included in the study. No differences were appreciated between groups in these variables.

Mild OA was present in 3 animals from the PRGF group, 3 and 10 animals of the PRGF and aMSC groups respectively, presented moderate OA, and 17 animals presented severe OA in both groups. OA degree radiologically evaluated did not vary within groups, and there was no improvement in time. However, in the rest of variables (functional limitation, joint movement, joint flexion and extension degree, owner's and veterinarians VAS, joint movement range, muscular atrophy and patient's life quality) a clear improvement has been seen since the first month of study in both groups, maintaining up to six months, although the aMSC group obtained better results at 6 months than the PRGF group in joint movement ($p=0,022$), flexion degree ($p=0,0034$), extension degree ($p=0,02$), owners VAS ($p=0,044$), veterinarians VAS ($p=0,00$) and range of movement ($p=0,003$). There were no adverse effects present during the study.

D. Conclusions: OA is one of the most common causes of lameness in dogs. In this study both PRGF and aMSC treatments have demonstrated to improve functional limitation, joint movement and pain feeling even without radiological improvement and with absence of adverse effects. Both AMSC and PRGF therapy are new means of treatment and create big hopes to overcome cartilage regeneration, maintaining or improving joint function and structure.

213 CARTILAGE REPAIR IN AN OSTEOCHONDRAL DEFECT IN A RABBIT MODEL

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Purpose: Despite the fact that mesenchymal stem cells (MSC) offer clinical potential for osteoarthritis applications, retaining sufficient numbers of functional MSC at the site of injury for optimal repair still continues to be a major challenge. One method of overcoming this limitation is to create an artificial extracellular matrix or scaffold to hold the cells in place. Previous research suggests that biomaterials possessing an elastic modulus between 2–50MPa are suitable for functional cartilage repair. To this end, the main aim of this study was to examine the effect of scaffold mechanical properties on cartilage repair in a rabbit model in vivo.

Methods: Two three-dimensional (3D) scaffold structures fabricated from different biomaterials were selected; a 55/45 wt% polyethylene oxide terephthalate/polybutylene terephthalate (PEOT/PBT) scaffold created by 3D fiber deposition with a compressive modulus of 3.6 MPa and a bilayered PGA/PLGA+CaS composite construct (TruFit™) with a compressive modulus of 50 MPa. Using scanning electron microscopy, the 3D architecture of the scaffolds was visualized and the porosities measured using volume displacement. Upon characterization of rabbit MSC morphology, growth kinetics and tri-lineage differentiation potential, the optimal cell seeding density and attachment conditions were evaluated. Cartilage repair was examined in a 3x3x3 mm osteochondral defect in male White New Zealand rabbits in accordance with ethical guidelines and approval, with 3 groups, empty defect (n=3), empty scaffold (n=6) and MSC seeded scaffold (n=6). After 6 weeks, tissue repair was assessed using toluidine blue staining to evaluate tissue morphology and a modified ICRS scoring system using 3-blinded reviewers to grade cartilage repair.

Results: The 3D architecture of the scaffolds was comparable with structures previously used for cartilage repair, with porosities of 76% measured for the PEOT/PBT scaffold and a porosity gradient from 63% to 97% observed for the bilayered TruFit™ construct. Rabbit MSC were shown to have a fibroblastic morphology and were capable of osteogenic, adipogenic and chondrogenic differentiation. Optimal cell attachment was observed for 1 million cells/scaffold in combination

with 50µg/ml fibronectin. There was evidence of repair in the empty defect, however, although not significant, cartilage regeneration was improved and degenerative changes were reduced in the presence of the scaffolds (Fig 1 and 2). In terms of scoring, no statistical difference was observed for both scaffolds, in terms of thickness of repair tissue or integration with native tissue. Seeding the PEOT/PBT scaffolds with MSC appears to produce lower scores for degenerative changes in the repair tissue and adjacent tissue when compared to the empty, contralateral control with no cells (Fig 2). In contrast, seeding the TruFit™ scaffold with MSC does not appear to improve degenerative affects. Moreover, there are bone cysts visible in the subchondral bone.

Conclusions: In summary, two scaffolds with mechanical properties at both ends of the materials property spectrum were analyzed. Although, both scaffolds revealed interesting, albeit different results, neither construct produced an optimal result. Thereby, suggesting that cartilage repair is a multifactorial problem, which is not modulated by mechanical properties alone.

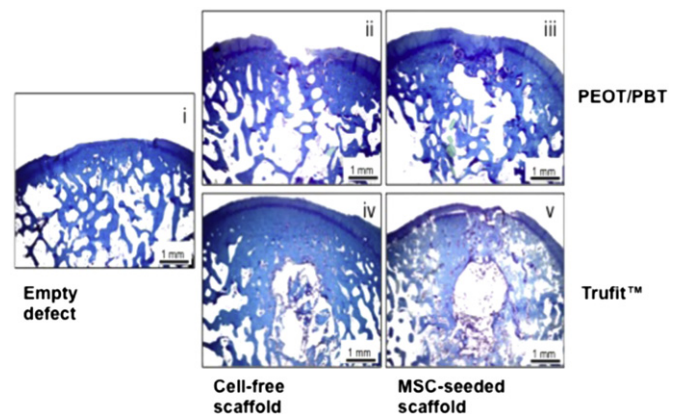


Figure 1. Representative images showing toluidine blue staining in osteochondral defect.

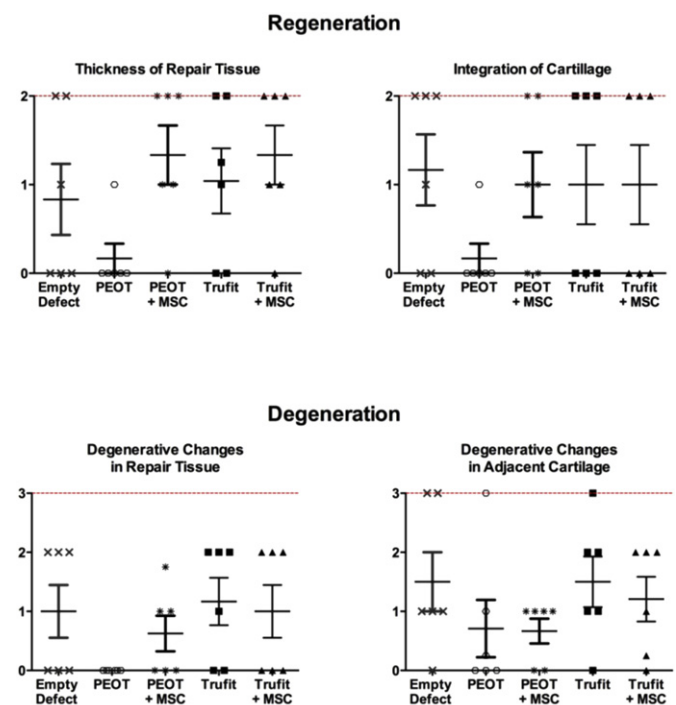


Figure 2. Histological scoring for (A) Regeneration (B) Degeneration, Red line indicates highest score possible.