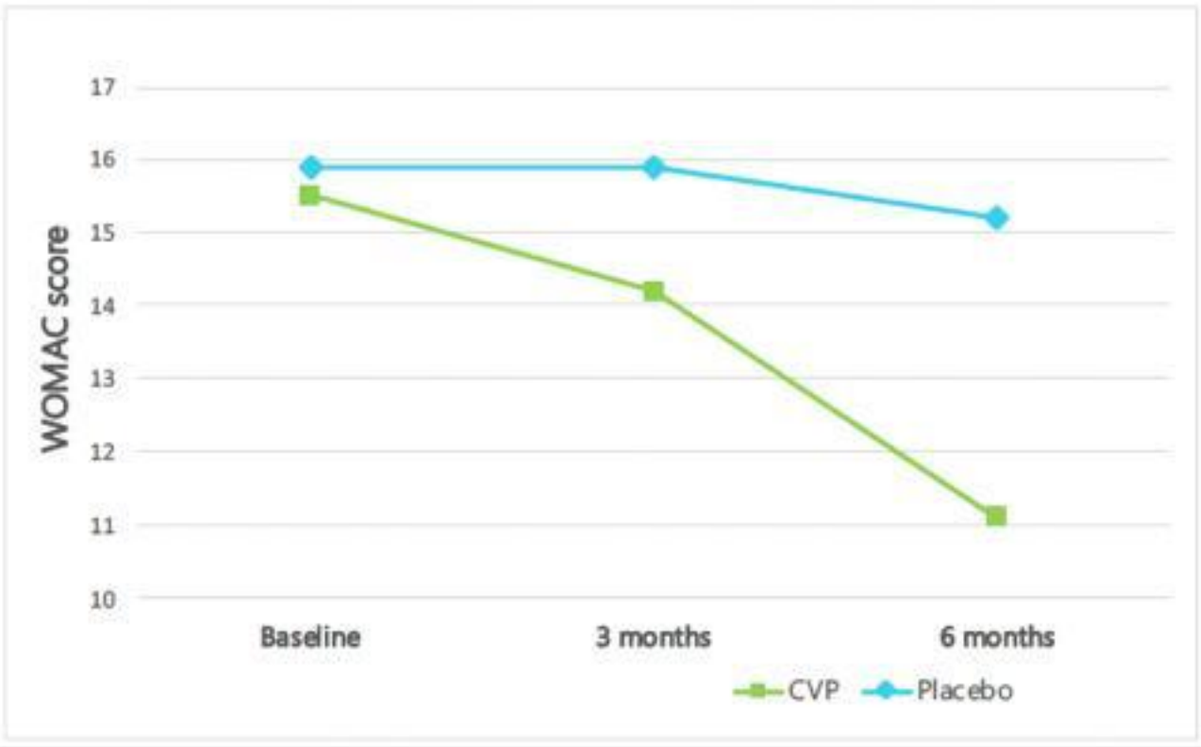
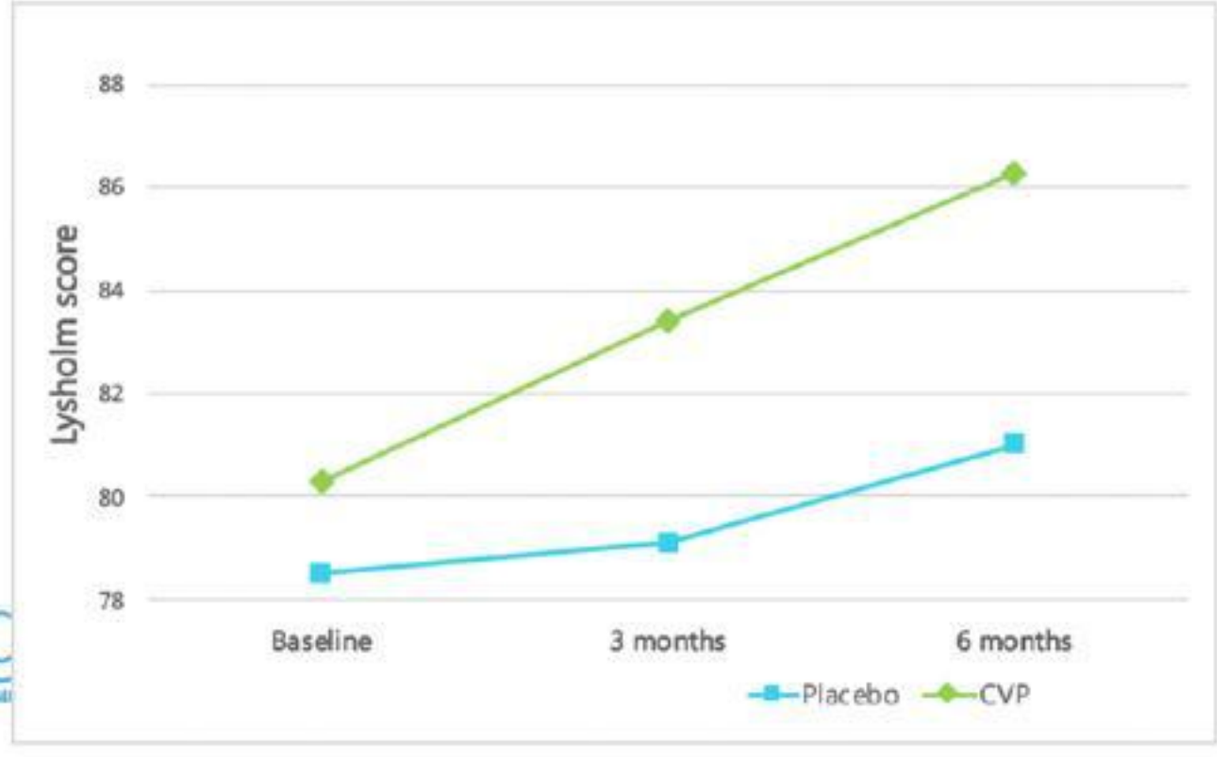
JOINT HEALTH

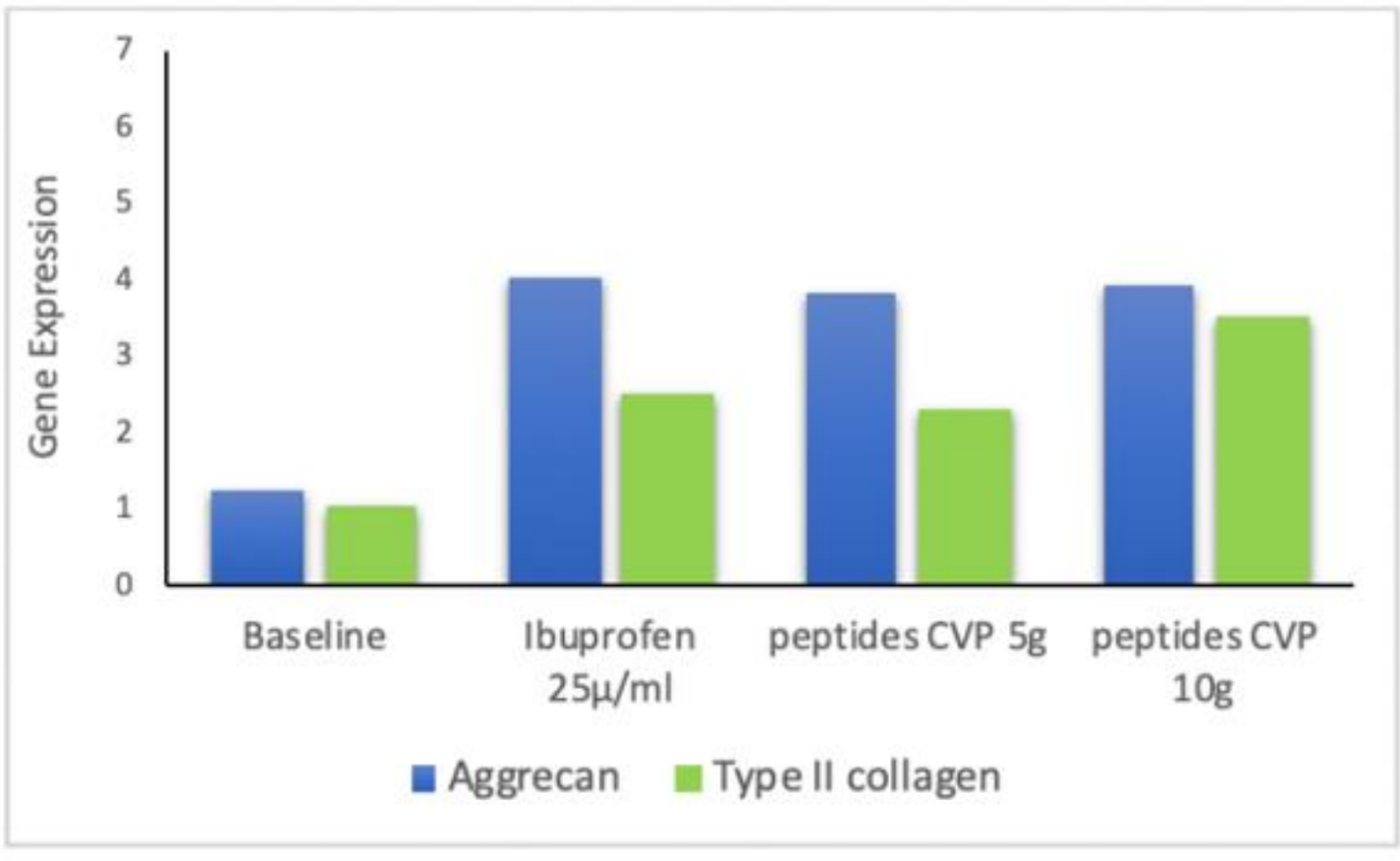
In healthy joints, the cartilage matrix composition is regulated by chondrocytes (joint cells) through a finely balanced process of synthesis and turnover, which ensures joint lubrication and cartilage matrix renovation. When these processes are disrupted, causing an imbalance involving matrix-degrading enzymes, which attack aggrecan and collagen resulting in the deterioration of cartilage structure and function

Two double-blind placebo-controlled clinical studies[2] revealed that an 8 g daily intake of **collagen peptides formulated in Collagen Vital Power** (hereafter CVP) **significantly improved joint comfort and functionality**. During the 6-month study, women with diagnosed knee osteoarthritis (X-ray assessment + quantifying using the Kellgren-Lawrence grading system) were randomly assigned either 8 g of peptides CVP or a placebo. Their knee joint function was assessed before (baseline) and after supplementation (3 and 6 months) with standardized score systems (WOMAC for general joint discomfort and function and Lysholm for more specific functions when walking, jumping and stair climbing).





In another study, Benito-Ruiz and colleagues[3] have **proven the effects of CVP collagen peptides on the gene expression of 2 major constituents of the extracellular matrix of the cartilage: aggrecan and type II collagen.** Chondrocytes were collected from articular cartilage and mixed with collagen peptides CVP at different concentrations. Cells were seeded and cell growth was measured until 8 days. mRNAs were extracted from the chondrocytes (Messenger RNA, is a molecule encoding a chemical «blueprint» for a protein product). The concentration of total mRNA was measured by optical density. Gene expression was then measured by quantitative PCR (quantitative Polymerase Chain Reaction, is a laboratory technique used to amplify and simultaneously quantify a targeted DNA or RNA molecule). This has shown that CVP collagen peptides specifically enhanced expression of aggrecan and type II collagen mRNA, and thus the chondrocyte gene expression of the cartilage extra cellular matrix components. In this study, it has a similar effect on joint cells as Ibuprofen, a usual medication against inflammation.



Other studies have also proven that subjects with the greatest joint deterioration benefited the most from the effects of collagen peptides[4] or that **collagen peptides were clinically even more efficient than glucosamine** in reducing joint pain and improving joint functionality[5].

More recently, in 2017, a study made in the Center for Musculoskeletal Research, University of Rochester Medical Center, New York USA(6), has proven hydrolyzed collagen type 1 (CVP) is chondroprotective and anti-inflammatory in murine posttraumatic osteoarthritis (meniscalligamentous injury on the knee).
At various time points post-injury, hydroxyproline assays were performed on blood samples to confirm hCol1 delivery, and joints were harvested for tissue and molecular analyses were performed, including histomorphometry, OARSI and synovial scoring, immunohistochemistry and mRNA expression studies.
In the CVP peptide supplemented group chondroprotective effects were observed in injured knee joints, with dose-dependent increases in cartilage area, chondrocyte number and proteoglycan matrix at 3 and 12 weeks post-injury. Preservation of cartilage and increased chondrocyte numbers correlated with reductions in MMP13 protein levels and apoptosis, respectively. Supplemented mice also displayed reduced synovial hyperplasia that paralleled a reduction in Tnf mRNA, suggesting an anti-inflammatory effect.
These findings establish that in the context of murine knee PTOA, daily oral consumption of CVP peptides is chondroprotective, anti-apoptotic in articular chondrocytes and anti-inflammatory.

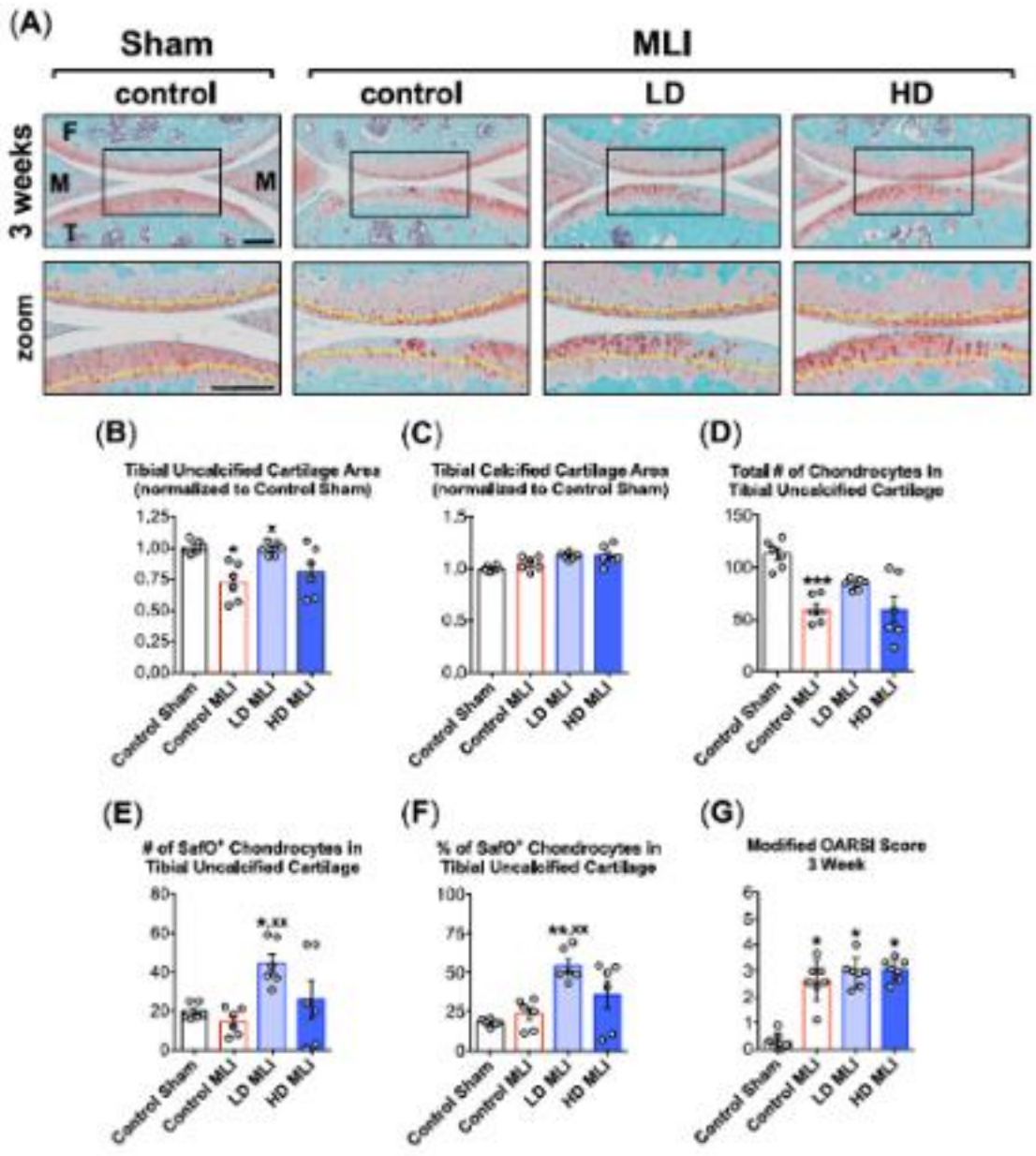


Fig. CVP=hCol1 is chondroprotective in the early stages of murine PTOA. Panel (A) presents an array of representative 40x Safranin O/Fast Green stained sagittal sections (40x) from the medial compartment of sham and MLI joints 3 weeks post-injury under various treatment conditions (control = vehicle, LD = 3.8mg hCol1/day, HD = 38mg hCol1/day). Joint structures are labeled (F = femur, M = meniscus, T = tibia) and the black box denotes the area shown in the zoomed images, where the tidemarks are denoted with a yellow dashed line. Black scale bars depict 100μm. Cartilage architecture was evaluated using the Osteomeasure System to determine the tibial uncalcified cartilage area (B), tibial calcified cartilage area (C), the number of chondrocytes in the tibial uncalcified cartilage (D), and the number (E) and percentage (F) of Safranin-O positive (SafO+) chondrocytes in the tibial uncalcified cartilage. OARSI scoring of the sections analyzed by histomorphometry was also performed (G). For histomorphometry and cell counting, symbols (joint. For OARSI Scoring, symbols () represent the average score for each joint based on scoring of 3 sections/ joint by four observers. Bars in all graphs represent the average for each experimental group (± SEM, N = 6). Significant differences between experimental groups in the histomorphometry data (B-F) were identified via one-way ANOVA with a Tukey's multiple comparisons post-test (*p<0.05, **p<0.01, ***p<0.001 compared to Control Sham; xp<0.05, xxp<0.01 compared to Control MLI). Significant differences between experimental groups in the OARSI data (G) were identified via a Kruskal-Wallis Test with a Dunn's multiple comparisons post-test (*p<0.05, compared to Control Sham).