

Molecular Characterization of Arthritis in Dogs

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Osteoarthritis (OA), or degenerative joint disease, is the most prevalent joint disorder in dogs, affecting as many as 20% of adult dogs and is a major cause of pain and physical disability in our older pets. Excess body weight or obesity is considered a predisposing risk factor, with even a mild degree of excess body weight considered detrimental. Currently, more than 25% of dogs seen by veterinarians are overweight or obese.

Osteoarthritis involves disruption of metabolic homeostasis within the articular chondrocyte, resulting in pathologic degradation of articular cartilage, inflammation, and pain. This imbalance between anabolic and catabolic factors involves many factors at the molecular level. Numerous OA gene-expression studies, taken together, suggest that inflammatory pathways play a critical role in the chondrocytes response to injury and subsequent progression toward repair or toward arthritis. The ability to identify and track multiple pathways in arthritic chondrocytes has greatly improved our understanding of the disease process, and can provide markers to assess potential efficacy of interventions, including nutrition.

Nutritional intervention to prevent or reduce excessive body weight lower the risk for development of OA as well as reduce symptoms in afflicted dogs. Further, certain nutrients have been shown to have a more direct impact on the biochemistry of OA. Functional genomics, cellular biochemistry and in-vivo clinical investigations were used to examine and confirm the impact of oral supplementation with the anti-inflammatory fatty acid, eicosapentaenoic acid (EPA), on OA in canines. EPA displaced arachidonic acid in tissue phospholipid and served as alternative substrate for COX-2 enzymes. Also observed was altered expression of OA related genes, with subsequent in vivo reductions in matrix metalloproteinases and inflammatory mediators, clearly linking this nutritional intervention to specific clinical markers and observations.