



Gerontology: Prolonging Health Through Nutrition

This article was condensed and adapted from the following manuscripts published in the Proceedings of the 2001 Purina Nutrition Forum:

- Gerontology and Longevity: An Introduction by Drs. D.P. Laflamme and M.R. Kelly
- Influence of Body Condition on Canine Osteoarthritis by Drs. G.K. Smith, R.D. Kealy, D.N. Biery and D.F. Lawler
- Phytochemicals in Bone and Joint Metabolism by Drs. B.A. Watkins and Y. Li
- Glucosamine and Chondroitin Sulfate in the Prevention and Management of Osteoarthritis by Dr. M.A. Anderson
- Eat to Your Heart's Content: Nutritional Modulation of Cardiac Disease by Dr. L.M. Freeman

While maximum lifespan appears to be predetermined by genetics, how much of that potential lifespan is survived can be influenced by numerous factors, including environment and nutrition. Much progress has been made in extending the average lifespan over the past century by attempting to modulate these factors. While data on dogs and cats is sporadic and difficult to interpret, it appears that the percent of cats aged 10 years or older increased from about 10% in 1983 to about 40% in 1995. ¹ Likewise, the percent of dogs aged 10 years or older increased from about 28% in 1983 to 37% in 1995. ¹ In order to continue this progress, research is in full swing in the areas of gerontology and geriatrics.

The field of geriatrics deals with diseases of old age and their treatment, while gerontology deals with the aging process itself. "Aging" is the progressive decline in an organism's internal condition, leading to an increasing risk of death. ² A goal of nutritional gerontology is to decrease the rate of aging, or to delay the decline in function associated with age, resulting in enhanced quality of life.

Much of the research in gerontology and geriatrics has been either in humans or animal models of human conditions. While changes in pet animals do not totally parallel those seen in humans, physiologic changes and age-associated diseases are similar (Table 1). ^{1,3-7} For example, many of the diseases of old age or common causes of death among the elderly are similar between humans and pet dogs or cats. ^{1,8-10} After adjusting chronological age for physiological age, the age of onset of many of these conditions, e.g. cancers or renal failure, are similar between dogs and humans. ^{1,11,12} Thus, some of the emerging findings from other species may be applicable to dogs and cats.

Impact of Early Nutrition on Late-Life Disease

Classical nutritional deficiencies result in poor growth and development, depressed immune function, decreased cognitive function, and other problems. There is now an emerging recognition that nutrient imbalances early in life can alter physiological function and induce disease later in life. For example, inadequate maternal protein intake results in increased susceptibility to lung damage from air pollutants and an increased frequency of glucose intolerance and diabetes mellitus in the aging adult offspring. 13-15 Reproducing bitches consuming marginal protein intake produced apparently normal and healthy puppies. However, despite normal nutrition after birth, these puppies demonstrated compromised immune function many months later. 16

How a puppy is fed during its first year can alter its skeletal structure and influence later life musculoskeletal disease. The risk of developing canine hip dysplasia (CHD) can be increased by overfeeding puppies. In a study investigating the effects of lifelong food restriction (25%) in Labrador retrievers, the frequency and severity of CHD and osteoarthritis (OA) were altered by food intake. Using the OFA method, CHD was diagnosed in 7 of 24 restricted dogs and 16 of 24 full-fed dogs. 17 By age 52 weeks, differences between the groups for frequency and severity of OA already were statistically significant. 18 Hip dysplasia has long been understood to be a disease of complex inheritance. However, environmental factors can influence the expression of diseases of complex inheritance, and in these investigations, food consumption and body weight were found to be such factors. These findings support a clinical recommendation to avoid excessive food intake in growing dogs, particularly in breeds prone to CHD.

Impact of Nutrition on Osteoarthritis

The foregoing studies demonstrated that controlling body weight during growth reduced the development of CHD in susceptible dogs. Food restriction was continued in adulthood in these dogs, resulting in a decrease in the prevalence of OA in all joints evaluated. By age 5 years, the percent of dogs affected with hip joint OA was decreased from 52% in full-fed dogs to 13% in restricted dogs. 18 By 8 years of age, 45% of full-fed dogs had OA in two different joints whereas only 5% of restricted dogs were similarly affected. 19 Only 9% of full-fed dogs had no evidence of OA, but 23% of the restricted dogs were free of OA.

In addition to reducing the prevalence and severity of OA through food restriction, correction of obesity can reduce lameness and improve mobility. 20 Dogs vary considerably with respect to individual maintenance energy requirements, so it is not feasible to specify a universal energy intake to achieve the benefits observed in the restricted dogs. However, it is recommended that growing puppies and adult dogs be fed lifelong to maintain lean body conformation (Body Condition Score of 4 or 5 on Purina's 9-point BCS system) to minimize the development of OA with advancing age.

The most controversial treatment of OA in the past decade has been disease-modifying agents. Research has focused on slowing the progression of cartilage degradation while promoting

cartilage matrix synthesis. 21 Many "nutraceutical" products are now available that purport to have a positive effect on the articular cartilage matrix, increasing hyaluronic acid synthesis, inhibiting catabolic enzymes, and encouraging normalization of the synovial membrane and cartilage matrix. 22 Among these are omega-3 (n-3) fatty acids, glucosamine, chondroitin sulfate, and several others.

Dietary long-chain polyunsaturated n-3 fatty acids also may be beneficial in controlling OA and rheumatoid arthritis. Much of the bone and cartilage damage in these conditions is induced by eicosanoids, lymphokines and free radicals, which also influence cell proliferation and stimulate collagenase and protease secretion. 23 Inflammatory cytokines inhibit chondrocyte proliferation 24 and induce cartilage degradation, for which part of the response may be mediated by PGE2. 25 Dietary n-3 fatty acids, especially DHA and EPA, can reduce production of the pro-inflammatory factors IL-1, IL-2, and TNF in cartilage tissue. 26

Studies evaluating dietary intervention with n-3 fatty acids have shown modest, but rather consistent beneficial effects of these fatty acids in joint disease. 27-31 Since PGE2 activation may play an important role in cartilage biology, collagen and proteoglycan synthesis, 32 dietary fatty acids also may be important for supporting joint repair. Further investigations are needed to describe the effects of nutraceutical fatty acids and the ratio of n-6/n-3 fatty acids on cartilage biology, joint disease and ligament healing.

Glucosamine, an amino-monosaccharide, is a precursor to the disaccharide unit of glycosaminoglycans (GAGs), which comprise the proteoglycan ground substance of articular cartilage. 22 Glucosamine provides the raw materials for the biosynthesis of glycosamino-glycans and modulates chondrocyte genetic expression for fibronectin, stromelysin and proteoglycan synthesis. 33 Chondrocytes obtain preformed glucosamine from the circulation (or synthesize it from glucose and amino acids); therefore, adequate levels in the body of glucosamine or its precursors are needed for synthesis of glycosaminoglycans in cartilage. In clinical trials with human OA patients, orally administered glucosamine resulted in significant improvements in joint mobility and pain reduction. 34-37

Chondroitin sulfate (CS) is the predominant GAG found in articular cartilage. CS inhibits cartilage breakdown both in vitro and in vivo; it appears to work by promoting prostaglandin levels. 38-40 Clinical studies in human beings have shown CS to be effective in reducing symptoms related to OA in the knees and hips. 41-43 While canine studies evaluating glucosamine or CS alone have not been published, preliminary studies indicate a combination of glucosamine and CS was effective in reducing OA in dogs following cranial cruciate disease and inflammatory synovitis. 44

Impact of Nutrition on Management of Age-Related Diseases

The most common "age-related" diseases of dogs and cats include arthritis, cardiovascular disease, cancers, chronic renal failure, and thyroid disorders. These diseases also occur in human beings, with typical age of onset similar between species when adjusted for "physiologic age" (Table 2). 12

The process of choosing an appropriate diet for an animal, especially those with chronic disease, involves examining the patient, the diet and the owner's feeding practices, and considering all the issues at hand. It is important to assess a number of different factors to determine which diet or diets might best suit a particular patient.

Nutrition and Cardiovascular Disease

In general, the nutrients of concern in cardiac patients are calories, sodium and chloride, protein, potassium, and magnesium. However, patients with cardiac disease vary tremendously in terms of clinical signs, laboratory parameters and food preferences, and these all affect diet selection.

Desc:

Your pet's diet is closely related to his life span. This article goes into detail on how nutrition effects your pet's health.