Revisiting this question is always helpful to us in clinical practice. Considering the flexion and decompression aspect of decompression flexion distraction adjusting, the benefit of it to disc nutrition needs to be explored. Let's look at the literature on this together.

In chapter 10 of the textbook *Ghosh P: The Biology of the Intervertebral Disc Volume II. CRC Press, Boca Raton, Fl.1988, Adams MA, Hutton WC: Mechanics of the Intervertebral Disc*, Adams makes some important statements on the fluid pumping and diffusion gradients of the intervertebral disc. These may be of importance with spinal decompression manipulation and the nutrition value of stimulating circulation through the disc. Let us look closely at this. On page 66–68, Adams and Hutton state the following:

1. **Changes in the mechanical load acting of the spine cause fluid to flow into and out of the discs, and this fluid pumping can aid the transport of disc metabolites.** Flexed postures expel more fluid from the lumbar discs than does erect postures, with the effect being particularly marked in the nucleus. Glucose supply to disc improves a small amount, but the large molecules may be considerably effected since they diffuse much more slowly. Flexed postures can improve the fluid flow component of the metabolic transport of the discs, at least in the lumbar region. There may be similar benefits from flattening the curvature of the cervical spine.

2. **Diffusion occurs as a result of a chemical concentration gradient, so nutrients diffuse into the disc and waste products diffuse out of it.** Erect postures cause diffusion into the anterior annulus more than the posterior annulus. Flexed postures reverse this imbalance and increase the overall rate of diffusion into the disc. Flexed posture increased diffusion the inner posterior annulus which’s the area of the disc having the poorest nutrient supply in erect posture. It is advisable to change erect posture with sitting postures that flex the lumbar spine. The same conclusions may be applied to the cervical spine, while the thoracic spine would benefit by straightening of the normal thoracic kyphosis.

**WE ARE CREATING INTRADISCAL PRESSURE CHANGE AND INTERVERTEBRAL DISC SPACE HEIGHT WITH OUR DECOMPRESSION ADJUSTING AND PERHAPS THE ABOVE NECESSARY CHANGES ARE CREATED TO AID CIRCULATION AND NUTRITIONAL DIFFUSION INTO THE DISC NUCLEUS PULPOSUS.**

The following paper is one of the few that now begin to discuss nutritional benefit to treat disc degeneration.
TREAT DISC DEGENERATION WITH CHONDROITIN AND GLUCOSAMINE SULFATE

Wim J van Blitterswijk, Jos CM van de Nes, Paul IJM Wuisman: Glucosamine and chondroitin sulfate supplementation to treat symptomatic disc degeneration: biochemical rationale and case report. BMC Complementary and Alternative Medicine 2003;3:2

Nutraceutical therapy for osteoarthritis is mostly shown in treatment of knee osteoarthritis, while virtually no documentation exists on spinal disc degeneration benefit. This paper highlights the potential of chondroitin sulfate and glucosamine sulfate to aid in relieving disc degeneration and its pain production. A case report is given showing a 56 year old man with frequent and recurrent low back pain and ischialgic complaints which existed for more than 15 years. With exercise, he was started on daily 500 mg glucosamine and 400 mg of chondroitin sulfate food supplements, 2 capsules in the morning and one in the evening for 9 months and then 2 capsules in the morning for the remainder of a 2 year period.

The patient felt gradual improvement of the range of motion and functioning of his back with less pain, starting about 6 months after starting taking the supplementation. At the end of two years, he felt his back was stronger and more flexible, and he was capable of withstanding heavier work loads without pain. No adverse reactions were felt.

MRI studies were done at the beginning and at one and two year periods during the supplementation. During the two years, the water content seen on MRI improved in the discs and an L3-L4 disc protrusion seen on the initial MRI decreased as the MRI signal normalized. The height of the L3-L4 disc increased by 5-10%. The L4-L5 disc that showed advanced degeneration showed no change in the two year period, worse or better.

While one case does not prove that neutraceuticals have caused remarkable disc improvement, it does document healing of a disc degeneration with reduction of a disc protrusion and pain relief and increased flexibility and strength. This would not be expected in a patient with no intervention, especially at nearly 60 years of age.

Biochemical facts about the disc. The matrix mass of the intervertebral disc is primarily glycosaminoglycan (GAG), which is a long chain of sugar units, negatively charged by carboxyl and sulfate groups. These charged groups are neutralized by cations, which, in turn, attract and retain large quantities of water my osmotic forces. Chondrotin sulfate is an abundant disaccharide unit in the GAG polymer. With aging there is a shift from chondroitin sulfate 4 to chondroitin 6 sulfate and degeneration sets in. Nutritional diffusion into the disc depends on exercise to facilitate the hydrostatic pressure within the disc.

It is postulated that the inability of chondrocytes to make sufficient and full-sized extra matrix molecules may be caused by age related dedifferentiation of sugar units and...
sulfate groups to the growing GAG chains. Such loss of nutrient sugar supply to the chondrocytes could lead to disc degeneration. Supplementation with chondroitin sulfate units perhaps supplants this deficiency and increases the sulfated GAG content. The implication is that glucosamine and chondroitin may promote GAG synthesis in case of cartilage degeneration. Chondroitin sulfate has been found to reach synovial fluid and joint cartilage when orally administered.

In vivo studies have confirmed that exogenous (radiolabeled) glucosamine and chondroitin sulfate are both taken up by chondrocytes and indeed used to build their extracellular matrix. Administration of sufficient glucosamine may boost GAG synthesis in degenerated chondrocytes if endogenous glucosamine is a limiting factor, and/or if glycosyltransferases are downregulated, e.g. by interleukin 1B. Chondroitin sulfate may help to restore the impaired sulfation of degenerated GAGs. Glycosamine is about 90% absorbed leading to an availability of 12-44% and is persistently incorporated into articular cartilage. Chondroitin sulfate is bioavailable from 5-15% with peak levels in the plasma reached between 2 and 5 hours after oral administration with significant accumulation found upon multiple dosing.

Glucosamine and chondroitin sulfate are chondroprotective agents by inhibiting enzyme breakdown of the cartilage matrix and the production of inflammatory mediators (prostaglandin E2, nitric acid and other enzymes). Chondroitin sulfate is anti-inflammatory and inhibits bradykinin or chymopapain induced proteoglycan depletion from articular cartilage. When combined, glucosamine and chondroitin sulfate are more effective than taken alone, allowing significant drops in NSAID use in osteoarthritis patients.

In summary, oral glucosamine and chondroitin sulfate can pass the gastrointestinal tract and can indeed reach articular cartilage, probably also the intervertebral disc, where it may have at least a chondroprotective effect, and, quite possible, a regenerative effect. More investigation is necessary.

Further discussion on the nutritional aid to reversing or preventing disc degeneration is covered in the following paper I wrote in answer to a doctor who stated that most research on nutritional treatment of cartilage was done on the knee. Please see my answer as follows:

The majority of studies favor the use of glycosaminoglycan in the treatment of osteodegenerative arthritis of peripheral joints such as the knee; however the facet joints of the spine are synovial lined joints as are those of peripheral joints. Synovial lined joints with their hyaline and fibrocartilage are shown to benefit from the anti-inflammatory benefits of glycosaminoglycan. Let’s discuss the literature on disc metabolism influence with glycosaminoglycan.
At the outset, I would like to share a two volume text that represents, to me, the finest treatise on the biochemistry of the intervertebral disc, and from which some of my material on this subject will be taken. The text is entitled *The Biology of the Intervertebral Disc* Volumes I and II, by Peter Ghosh, B.Sc., Ph.D., A.R.I.C., F.R.A.C.I., Director, Raymond Purves Research Laboratories at Royal North Shore Hospital of Sydney and Associate Professor, Department of Surgery, University of Sydney, St. Leonards, N.S.W., Australia, published by CRC Press, Inc. Boca Raton, Fl., 1988.

This text points out that the matrix of the disc consists of two major macromolecules – proteoglycans and collagen. The proteoglycan is mainly responsible for the high water content of the gel like nucleus and is responsible for the load carrying function of the disc. Proteoglycans are the core protein to which one or more glycosaminoglycans are attached. In the human disc, the glycosaminoglycan is majority composed of chondroitin sulfate, keratin sulfate and hyaluronate. The water content of the disc correlates well with the glycosaminoglycan content. The mature nucleus pulposus contains the highest concentration of keratin sulfate of any connective tissue. Disc degeneration consists of the loss of proteoglycan in both the nucleus pulposus and annulus fibrosus with the concomitant loss of water content.

Ghosh reported on studies of the rabbit model that disc cells respond to depletion of their extracellular matrix in degeneration by initiating a repair mechanism. He cites Bradford who showed the repair mechanism of the nucleus pulposus to follow, within 3 months after administering chemonucleolysis, with normal proteoglycan formation seen at 6 months.

Ghosh wrote on pages 148 and 149 that there are few therapeutic measures available which have been satisfactorily shown to modify the constitution of the intervertebral disc. Information on the mechanism by which dissolution of disc proteoglycans can lead to the biosynthesis of a new matrix still remains unresolved. The mechanisms controlling cell division and matrix production in the disc and how these processes are influenced by the introduction of exogenous enzymes into the system is important. Enzymes that inhibit degeneration of the disc proteins could lead to favorable metabolic replenishment of the matrix proteoglycans and restore positive biomechanical properties to the disc.

Support for the regeneration of disc matrix is shown in a paper entitled *Reduced Degradation of Disc Proteoglycans in vivo, By Systemic Administration of Arteparon published in the J of Bone and Jt. Surg Br 1988;70B:166 by TC Cole, P Ghosh and TKF Taylor* It was pointed out that, while anti-inflammatory drugs were commonly used in the management of spinal arthropathy, their influence, if any, on degenerative processes within the intervertebral disc had not been established. In this study, a group of aged beagle dogs, which are prone to disc prolapse, were administered a semisynthetic polysulfated glycosaminoglycan used in the treatment of osteoarthritis termed Arteparon. It was subcutaneously given at 2.0 mg/kg twice weekly for 6 months. Two months prior to sacrifice, all animals were administered NaSo4 to label their disc proteoglycans isotopically. A control group of age matched beagles was similarly treated with this
isotope. At necropsy, disc proteoglycans were isolated, purified and analysed. The proteoglycans isolated from the Arteparon treated group showed higher aggregation of proteoglycans in both the anulus fibrosus and nucleus pulposus compared to the control group. The proteoglycans isolated from the drug treated group were found to be larger and more capable of forming a larger proportion of aggregates in the presence of excess hyaluronic acid. This study showed that the systematic administration of glycosaminoglycan decreased the rate of proteoglycan turnover in the aged discs of these animals. It is proposed that the action of glycosaminoglycan is to inhibit enzymatic action against various neutral proteinases and lysosomal hydrolases, all of which have been shown to degrade proteoglycans extracellularly and intracellularly. Arteparon did indeed enter the disc tissues as noted by the negative charge and molecular weight of approximately 10,000 Daltons present within the disc tissue after administration. This shows that the glycosaminoglycan did enter and localize within disc tissues to allow inhibition of degradation.

Ghosh does point out that patients do not present before disc degeneration becomes symptomatic and in these instances the introduction of enzyme inhibitors, such as Arteparon, into the disc may be too late to reverse the matrix damage that has already occurred. Further work is needed for the definitive answer to this question.

Pearce et al wrote in J Ortho Research 5, 198, 1987 that the proteoglycan changes within the disc with degeneration reveal a low proteoglycan concentration throughout the lumbar discs. It is proposed that low proteoglycan concentration in all the discs of a spine precedes degeneration. In the early stages of degeneration, there is excessive degradation of proteoglycans with an accompanying response on the part of the cells to repair their matrix. In later stage degeneration of the disc, further proteoglycan loss occurs although some ineffective repair process may still be operative. Pearce, interestingly, proposes that the degenerative process is not confined to the degenerate disc, but is widespread, although at different stages of the pathological process, throughout the lumbar spine.

It is very interesting to explore the possibilities of slowing or preventing disc degeneration. Kaigle wrote in Backletter, 1997, 12[?] that disc degeneration begins in the second decade of life and prevention strategies must occur early to disrupt this degenerative cascade. We know that aging decreases the chondroitin sulfate and sulfate content of the white fibrocartilage of the disc and prevention may be good treatment. The fibrocartilage of the knee and intervertebral disc are the same tissue. The use of glycosaminoglycan (chondroitin sulfate) as an anti inflammatory agent for the knee can be looked upon as the same for the disc.

study, 92 patients were studied in a randomized, placebo-controlled, peer-reviewed, clinical study with glucosamine sulfate in patients with lumbar spondyloarthrosis pain of six weeks duration. It showed that 51% improved with glucosamine sulfate versus 28% with placebo. The effect lasted beyond the end of treatment and was safe for the patient.

*Naylor et al reported in Orthopedic Clinics of North America 1975;6:1 in a paper entitled Enzymic and immunological activity in the intervertebral disc,* that study of the disc components by chemical analysis, radiograph crystallography, and electron microscope showed that in disc degeneration there is a fall of the total sulfate (both chondroitin sulfate and keratin sulfate) with age. *Happey et al reported in Biochemical Aspects Of Intervertebral Discs in Aging and Disease in Jayson M, ed. Lumbar Spine and Back Pain, New York: Grune & Stratton, 1976;318* that there is a gradual diminution of the sulfate content of the disc with aging and that degeneration of prolapsed discs usually contain less than half the sulfate values of the normal disc.

While the literature is not as great on the anti-inflammatory benefits of glycosaminoglycan for white fibrocartilage of the disc, there is some with good concepts in place. Certainly the facet joint osteoarthritis of the spine benefits as other synovial lined cartilage joints. Future work will show the benefit for disc. I am diligent in the quest for this clinical proof. Thank you for the opportunity to study this subject.

**HISTORY & CURRENT USE OF DISCAT by Cox**

In 1966, following reading the book entitled *Biochemistry of the Disc,* by Cole and Ghosh, I started to give my osteoarthritic patients chondroitin sulfate. Also, in the early 1970’s I started using it for intervertebral disc degeneration patients based on this textbook. Today we see the heralding of the use of glucosamine and chondroitin sulfate for degenerative disc disease with documented biopsy and MRI proof of regeneration of disc with increased signal intensity on MRI, increased disc height, and reduction of disc protrusion. In 1966 I developed Discat formula for use with my own patients based on the original Cole and Ghosh research. Over the years I have reformulated Discat until today it is called Discat Plus and is glucosamine and chondroitin sulfate with the minerals found in the intervertebral disc; based on the Donnan Equilibrium principle of the disc. I give my patients 2000 mg of Discat Plus a day for the first three months and then decrease it to 1000 mg per day for chondroprotection against degeneration and arthritis. Go to the website chiro-manis for further information on my use of Discat Plus in treating disc patients. The above papers support my study and efforts in developing this formula.

Attached is my recommendation to all my patients for nutritional support, "Complete Spinal Nutrition," with full explanation of what each does for the patient. The addition of the new FLAX PLUS FIBER formula has been a source of relief for many patients with colon conditions. The Disc & Joint Pain Relief Complex is herbal pain relief in back pain.

Respectfully submitted,
James M. Cox, D.C., D.A.C.B.R.