Supplement to

The Art of Getting Well

DMSO (Dimethylsulfoxide) Treatments in Arthritis

Sources are given in references.

Authors of contributions\quotations are alphabetically arranged; major author, if any, is underlined.

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Several years ago Ronald M. Davis, M.D. demonstrated, in our publication *A Treatment for Scleroderma & Lupus Erythematosus,* the strategic use of DMSO (Dimethylsulfoxide). [See http://www.arthritistrust.org] We've also reported on the good results of DMSO used with and without EDTA (Ethylene Diamine Tetracetic Acid) in our *Chelation Therapy*. [See http://www.arthritistrust.org]

DMSO is a prolific, and inexpensive byproduct of the pulp paper industry, and its medical uses has been reported many times, by many people, but especially in the works of chemist Robert J. Herschler and surgeon Stanley Wallace Jacob, M.D., and also as popularized by Pat McGrady, Sr. in *The Persecuted Drug: The Story of DMSO*[13].

The external and internal use of DMSO by veterinarians for pain in animals has long been accepted. Those humans who've managed to obtain a sufficiently pure variety of DMSO have also benefited from its pain-relieving qualities. Apparently, as Ronald M. Davis, M.D. has learned, when used in an IV over a number of months, even the most hopeless cases of Scleroderma and Lupus Erythematosus can be drastically reversed; while its use (as developed by Ray Evers, M.D. (deceased) in EDTA/DMSO IV's has long been accepted by many holistic physicians for peripheral circulation problems, as well as many other problems all of which are related -- as is Scleroderma and Lupus Erythematosus -- to free radical excess in the human phsyiological systems.

Those of us who've suffered from Rheumatoid Diseases of one or the other of the 75 to 100 differently named collagen tissue diseases know the nature of free radical pathology first hand. We could not, on the best of days, dispute that collagen tissue diseases (Rheumatoid Diseases) generate as a by-product many free radicals which create hob with every working apparatus of our human bodies.

The message to be learned from the pioneer physicians Jack Blount, M.D., Thomas McPherson Brown, M.D., Ronald Davis, M.D., Efrain Olszewer, M.D., Ray Evers, M.D., Stanley Jacobs, Roger Wyburn-Mason, M.D., Ph.D., Gus Prosch, Jr., M.D., Dr. Paul K. Pybus, Fuad C. Sabbag, M.D., Alan Rory Zapata, M.D. and others is that (1) collagen tissue diseases can be licked; (2) the pain of these diseases do not need to be endured; (3) one does not need to destroy self with the use of cytotoxic drugs, gold, penicillamine or long-term corticosteroids to rid oneself of the disease and its effects. Indeed, we have now learned that all of these traditional rheumatic treatments generate more dangeorus free-radicals than the disease itself, thus over-burdening the body even further.

It is clear, in the reports that follow, that Doctors Olszewer, Sabbag and Zapata, along with Jacobs, Evers and Davis, have added new and important knowledge to our persisent search for wellness.

Anthony di Fabio

Control of Free Radicals in Rheumatoid Arthritis and Osteoarthritis

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Figure 1: Relationship Between FR Synthesis (by HLB Test) in Patients with Rheumatoid Disease, Under Antioxidant Therapy Using DMSO

Percent of FR Synthesis			
Therapy	***Before (30.6%)***	***Immediately After (10.6%)***	After 18 Months (13.3%)
40			
30	1111111		
	1111111		
20	1111111		
	1111111		[[[[[[[[
10	1111111	0000000	[[[[[[[[
	1111111	0000000	[[[[[[[[
0	1111111	0000000	[[[[[[[[

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Rheumatoid diseases are some of the most crippling pathologies on earth[1]. Statistically one-third of individuals more than 35 years old suffer from Osteoarthritis, and also a large number of patients with Rheumatoid Arthritis, two of the most common diseases of this group of pathologies.

Rheumatoid Arthritis (RA) is an autoimmune disease, and Osteoarthritis (OA) is a degenerative disease, but both have something in common. They are closely related to Free Radical (FR) synthesis, as an inflammatory disease in RA in the prostaglandin metabolism and synthesis of leukotrienes, and in the destruction of cartilage in patients with OA[2].

Considering that these two pathologies are the most frequently seen in rheumatoid diseases we decided to study a correlation between FR synthesis before therapy, attempting to measure the decrease of FR production using antioxidant therapy with the following objectives:

1. Confirming that there is a relationship between FR synthesis with both diseases;

2. If the antioxidant therapy works not only with clinical improvement but also with an important reduction in the FR production;

3. How we can keep, if possible, the low levels of FR in these patients -- helping to keep the patient asymptomatic for long periods of time.

Methods and Materials

Thirty patients were included, 15 of them with RA, and 15 with OA, with a follow-up of 18 months.

The antioxidant used was DMSO (Dimethylsulfoxide in doses of 5 cc for each therapy together with B complex, Vitamin C, and Magnesium Sulphate) in a five-week, twice-weekly for the first period, and a followup of one bottle monthly for 18 months. As we know DMSO[3] is one of the most powerful antioxidants known, that also blocks the CH-(hydroxyl) FR. (It is well known that the human body does not have any antioxidant enzyme to block its formation.)[4]. DMSO is also an important anti-inflammatory substance, and as we showed in another study, is extremely useful in patients with inflammatory and degenerative diseases.

In order to measure the FR production we used the method HLB (Heitan-La Garde-Bradford)[5] that determines the presence of ROTS (reactive oxygen toxic species), mostly formed by FR, that plays a role in virtually all disease states and metabolic dysfunctions. The production of ROTS affects the basic cellular structures and metabolic pathways and also reacts with blood constituents to form various by-products which can be seen as morphological changes in the blood. The specific morphological changes in the blood vary as a function of the pathological condition; in this case on Rheumatoid pathologies, strength of the immune system, specific ROTS as well as level of ROTS being generated. The study includes the extracellular matrix formed by: proteoglycan (as chondroitin sulphate, dermatan sulphate), hyaluronic acid, collagen and elastin. Each of these substances may be degraded by enzymes specific for each substance. The degradation enzymes are as follows: chondrotinase (degrades condroitin sulphate), hyaluronidase (degrades hyaluronic acid), collagenase (degrades collagen) and elastase (degrades elastin).

In patients with Rheumatoid pathologies, we can find, as seen in induced-experimental arthritis studies in rats, the activity of collagenase increase in skin, bone, liver, kidney, and spleen, while that of hyaluronidase decreased significantly in liver, kidney and spleen[6]. When traumatized normal pig synovia was cultured with normal pig cartilage for 14 days the breakdown of cartilage collagen and proteoglycan was accompanied by the appearance of both collagenase and proteoglycanase[7]. Studies in patients with inflammatory Rheumatoid Arthritis indicated an increase of prolyl hydroxylase in 70% of those with active disease[8].

The measuring of ROTS was done in the first visit of the patient to the office, a second measure was done after any one of the infusions of DMSO, and the third evaluation after 18 months in order to measure its antioxidant effect in long-term therapy in maintenance doses.

Results

In a recent study[9,10] we showed that antioxidants are extremely important in order to control the synthesis of FR, as well as the symptoms of patients with Osteoarthritis. As we know FR are closely related to degeneration in OA, and also in the inflammatory response in patients with RA.

From the beginning we have used in patients with rheumatoid diseases the antioxidant DMSO (Dimethylsulfoxide) that has an important role over the OH-FR, instead of the popular antioxidant EDTA, that we use in patients with cardiovascular diseases as we show in our other studies[11,12].

Our protocol in the last 5 years includes: one infusion twice weekly for 5 weeks, followed by one weekly or each 15 days, followed by one infusion monthly for as long as 1 or 2 years. With this protocol we have been successful in over 85% of patients with OA, and over 77% in patients with RA, with long-lasting effects, without using any other oral or parenteral non-steroidal or steroidal anti-inflammatory.

The 30 patients included in this study were regular patients in our clinic and were evaluated three times in order to see if it was possible to use the HLB test as a measuring method of FR, as well as DMSO as an optimum antioxidant. The results obtained are represented in Figure 1, where we find an initial average FR measuring 30.6% of the patients included, with an important and significant decrease of FR production after DMSO administration, obtaining lower levels with an average of 10.6%. That represents a 66% decrease in patients before beginning the DMSO therapy, and keeping the patients in monthly applications we obtained an average of 13.3% of FR synthesis. That represents 52% decrease than the patients had in the beginning, and 12% higher than patients after any DMSO infusion.

It is important to verify that the higher values were obtained in patients with RA, and the lowest in patients with OA.

This study was done by: Centro Internacional de Medicina Preventiva, Rua Compevas 211 Perdizes, Sao Paulo 1501. Brazil; Tel: (011) 623000.

References

1. Beary J. III. Ed. *Manual of Rheumatology and outpatient orthopedic disorders*. A Little Brown 1981.

2. Decker J., Scott T. Eds. *Perspectives in Rheumatology*. Curr Med Lit Ltd. 1984.

3. Wood D. and J. and JU., Pharmacology and Biochemical considerations of DMSO. *Annals of the NY Acad of Sci.* 1982.

4. Gorog P, Kovacs I. Antiarthritic and antithrombotic effects of topically applied DMSO. *Annals of the NY Acad of Sci.* 1982.

5. Bradford, Allen, Cullen: *Oxidology*. The R. Bradford Fdtn. Los Altos, CA 1985.

6. Kuberampata T. et al. Effect of Adjuvant Arthritis on collangenase and certain lysosomal enzymes in relation to the catabolism of collagen. *Agents Actions* 10 78 1980.

7. Crossley M. et al. Biochemical and Pharamcological Studies on Synovium Cartilage Interactions in Organic Culture. *European Journal of Rheumatology and Inflammation* 5 15 1982.

8. Acta Univ. Ouluensis, Series A, 85(1979). Savulainen E.R. Enzymes of Collagen biosynthesis in diseases of the liver and connective tissues. *Chyem Abst* 9 1. 1729 lit. 1979.

9. Olszewer et al. Evaluacion clinica de pacientes con artrosis, sometidos a tratamento com DMSO, mesoterapia y mucopolisacaridios. Vol. 15, pp 14-17. Junio de 1991.

10. Olszewer E. A new approach to Rheumatoid diseases *Townsend Letter for Doctors*. October 1991. Letter.

11. Olszewer E. Carter J. EDTA Chelation Therapy in Chronic

Degenerative Disease. *Med. Hyp.* 27, 41-49. 1988.

12. Olszewer E., Carter J., Sabbag F. A pilot double blind study of Na-Mg EDTA in peripheral vascular disease, *JNMA* Vol. 82, N 3, March 1990.

13. Pat McGrady, Sr., *The Persecuted Drug: The Story of DMSO*, The Nutri-Books Corp., Box 5793, Denver, CO 80217, 1979.