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Editorial

Freedom of the Press

This is the third quarterly number of the first year of this new medical journal. It has been called "controversial" as well as "helpful in my medical practice." "Revolutionary" is one of the more quotable descriptions received to date. But as yet, none of the articles have been challenged by letters or papers contradicting or opposing the opinions and findings of the many authors who have contributed new methods and theories of the treatment of the rheumatoid diseases. This Journal is a published Forum for medical expression, protected by the First Amendment to the Constitution of the United States. Here physicians can announce their discoveries or ideas to stimulate others to investigate, challenge, and support or contradict their work. There is no "stamp of approval" nor endorsement by the Academy of Rheumatoid Diseases, the Journal, nor its editor on these articles. They stand or fall on their own merit with the test of time. But letters from readers are welcome, and with your permission, will be published in later numbers of the Journal.

The Rheumatoid Disease Foundation [Now The Arthritis Trust of America]

The Rheumatoid Disease Foundation was chartered as a non-profit, charitable organization in the State of Tennessee, October 13, 1982, and received its retroactive tax-exempt status from IRS March 29, 1983.

The purpose of The Rheumatoid Disease Foundation is:

1. To disseminate the scientific findings of Professor Roger Wyburn-Mason, that the *limax amoeba* is the source-cause of most forms of Rheumatoid Diseases; [although it appears now that this theory may have been incorrect, the treatments developed from it have been successful, Ed.S.C.]
2. To contract with professional scientific and medical organizations for research and developmental studies related to the cure and/or remission of Rheumatoid Diseases;
3. To fund basic research with such professional organizations;
4. To provide free and/or contributory treatment to needy victims of Rheumatoid Diseases;
5. To solicit funds from the general public in support of the above programs.

For Further information, a list of doctors knowledgeable about the Arthritis Fund's protocols and research, or a list of books and articles helpful in treating and managing arthritis for both consumers and physicians, write to:

The Soviet Health Care System
An Orthopedic Study Tour of the Soviet Union

by Robert Bingham, M.D.

In Moscow, we visited General Hospital No. 7. Each hospital serves a district of the city and has satellite clinics within walking distance of everyone in each community. All Soviet medical and hospital care is free, including drugs and appliances.

The out-patient clinics are 85 percent staffed by women doctors with general practice training. No one is admitted directly to the hospitals except by ambulance or station wagon type vehicles from the clinics. Outpatient care and home nursing services were reported to be "very good" but lacking in expensive medicines such as some types of antibiotics. There is an almost total absence of "disposables," syringes, needles, plastics, and paper. Glass, metal, rubber, and cotton goods are washed, sterilized and reused as in this country fifty years ago.

The hospital was not crowded, but there were very few empty beds, as elective surgery is only by reservation, with a waiting list from a few weeks to several months for most procedures. Four to six patients occupied each ward, with no privacy curtains o[r] chairs for visitors. The equipment in the operating rooms, the emergency rooms, intensive care, and x-ray equipment was adequate and well made, but there were no conveniences such as electric beds, telephones, television, or intercoms. There was an abundance of hospital employees — doctors, nurses, aides, and housekeeping personnel. The extreme cleanliness of the hospitals, and of all Russia for that matter, was very impressive. In the two weeks, we did not

see a discarded bottle, can, scrap of paper, or even a cigarette butt.

The doctors we met were very well informed of the most modern techniques and medicines. They have available translations of medical journals and articles from all over the world and pride themselves on "keeping up." They are proud of some advances they have made in medical knowledge. In orthopedic surgery, they demonstrated a bone plate with two curved falanges to secure fixation of fracture fragments in two planes. Beginning the third post-operative day, they apply a transparent green varnish-like substance over the wound. This does away with bandages, tape, dressings, and permits direct visual inspection of the suture line. It excludes contamination and possible external infection. They called the preparation, "alizarin-green."

The surgeons work from 8:30 a.m. to 2:30 p.m., five days a week, and take calls on rotation nights and week-ends. A second shift staffs the hospital in the evenings and a third shift at night. They have a month of vacation each year and a month for post-graduate work, study, and research. They can retire at 55 years of age, but have a greater income if they continue to work part-time. There is no private practice. Physicians are paid "a little less than coal miners and a little more than teachers." Nothing is bought for credit, but if they place their money at 5% in the bank, they can "save enough to buy a small car in two years."

In Leningrad, we were taken to a typical rehabilitation institute. It is located in the suburbs, adjacent to a lovely green forest. It was more attractive in furnishings and decorations than the hospital and was only a few years old. The patients had disabilities which prevented them from returning to work after discharge from the hospital for their acute care, operation, injury, or illness. The average patient stay varied from one to six months. The physical therapy and occupational equipment was excellent and very complete, including therapeutic pools, a swimming pool, gymnasium, and a small basketball court. They have skis for winter exercise and bicycles for the summer. Especially good were the shops where patients constructed wood furniture or metal appliances for the hospital, and women worked in sewing rooms making gowns and repairing linens.

This was the only phase of the Soviet medical system which seemed superior to our own, the availability of free convalescent and rehabilitation hospitals and services. Since there is no unemployment in the Soviet Union, each worker is considered a "national asset," and every effort is made to return a disabled person to some type of job after his rehabilitation treatment.

For "rest and recreation," we flew south to the Black Sea port of Sochi. Here on the pebbled beaches over 300,000 loyal Communist workers and their wives enjoy free or discounted vacations for being outstanding in their jobs. From dawn to dusk, they throng the shore to absorb enough sunshine to last through the winter. Concerts, the circus, restaurant dining, and strolling in the parks and boardwalk occupy their time. But for sick and disabled, there are other facilities. The natural hot mineral waters made this the spa city for the former Russian nobility. Now the various labor unions have acquired the stately mansions or have built their own impressive sanitaria.

Our tour group was shown patients bathing in the pools, using arm and leg baths, drinking the water, inhaling the vapors, irrigating the sinuses and other body orifices, and lying in hot mud packs. All those interviewed through the interpreters claimed "great improvement." The chief benefits seemed to be improved circulation for patients with arthritis and less post-operative stiffness in those recovering from joint surgery.

For all of us, the visits to the magnificent museums and art galleries, the former palaces, the opera, ballet, concerts, and folk dancing and singing would make the trip worth the effort and expense. But the experience of seeing the Russian people, friendly, helpful, healthy, and well-fed, very hard-working and intensely patriotic in love for their country gave us an insight to their way of life.

As to politics? Only one doctor spoke freely and openly. The communist party is small, estimated at seven to fifteen percent of the adult population. The word "Soviet" means "committee," and everything in a citizen's life is decided by little soviets, layer upon layer; red tape, laws, and regulations have taken away all personal freedoms and are giving a few back as favors to the privileged class. But the accomplishments of the government in development of heavy industry, mining, oil, power sources and modernization of the country in the past 65 years are unequaled in the world. They also have a strong and almost controlling military-industrial complex — but they say it is for "peace, not war." Can you believe, they want to be "strong to prevent any country, or combination of countries stopping this program of progress"? The young Communist wants the Soviet Union to be the "greatest nation in the world." For this, they sacrifice. And they may make it.

Rheumatoid Disease — Discoveries of Roger Wyburn-Mason, M.D., Ph.D.

by Perry A. Chapdelaine, Sr.

Roger Wyburn-Mason was a research physician and protozoologist. During his life-time, he received the highest possible academic grades in every degree undertaken. He was involved with the testing of sulfa dugs; was the first to identify a viral form of cancer; had two nerve diseases named after him; was a specialist on nerves and also did research on cancer; and he wrote a number of definitive medical books[1] and numerous articles.[1] This eminently acceptable medical physician and researcher became the genius who first developed and effective theory and treatment for Rheumatoid Diseases.

Over a life-time of brilliant work, he concluded that a certain kind of commensal organism, an amoeba (*limax amoeba:* a one celled, free-living animal organism which he named *Amoeba chromatosa*), creates conditions inside the body that result in damage, and this damage presents itself in many different forms depending upon which tissues are infected.

If one has a genetic susceptibility toward the products of this presumed amoeba, or its toxins, or resulting proteins from the dead amoeba, then, over a time-period, one can and usually does have one or more symptoms: Rheumatoid Arthritis, Bursitis, Ankylosis, Spondylitis, Psoriasis, Lupus, Rheumatic Heart, Carpal Tunnel Syndrome — in all about 100 different symptoms not previously recognized as stemming from the same source.

What He Did

Professor Roger Wyburn-Mason claimed to have isolated out a *limax amoeba* from human tissue and sera by taking advantage of the fact that the organism is attracted to heat. He cooled one side of a collection of specially prepared tissue and heated the other side. The amoeba migrated to the warm side, also passing through a very fine filter. The collection of amoebae was then grown in the laboratory. He claimed that some were placed back in animal tissue, where exactly the same cellular lesions were observed as found in Rheumatoid Disease patients. He concluded that the protozoan had escaped attention because they looked under the microscope very much like human macrophages.

He studied a number of medicines and found several that would stop the progress of Rheumatoid Disease. He announced these findings in 1964 at a scientific conference,[27] where he received a standing ovation. No one followed up on his discoveries until Robert Bingham, M.D., reported the findings in a magazine[24]; and Jack M. Blount, Jr., M.D., tried metronidazole (chemically related to then unavailable clotrimazole) on himself.[24]

Whether or not the amoeba is ever verified to be the cause, we are certain of this: his treatment works!

If You Are a Rheumatoid Disease Victim — How Does the *Presumed* Amoeba Affect You?

According to Roger Wyburn-Mason's theory[2]: To those of us who are genetically susceptible to the amoeba and its products, the organism is dangerous and very damaging.

The amoeba is found freely floating in the air, in water we drink, in ponds, swimming pools, health spas, and in some foods. It is almost impossible to stay away from the amoeba, although there are some things that can be done to minimize risk of exposure.

When the amoeba is killed inside the human body with an antiamoebic, the body responds by creating "flu-like symptoms." These symptoms can include: Itching, ringing in the ears, bronchitis symptoms, coughing, nocturnal muscle spasms, bone pain, bitter or metallic taste, temporary memory loss, sleepiness, depression, palpitations, frequent urination, burning sensation during urination (clotrimazole does this, sans Herxheimer), pain in joints and flu-like symptoms, no appetite, flushing of skin and reddish patches, general malaise, fever, vomiting, nausea, diarrhea, headache, heavy perspiration especially at night.

A patient will not have all of these symptoms, but only those where the amoebae has been quietly working — and such symptoms are clear evidence of a genetic susceptibility to the amoeba and its products.

The above symptoms are titled the "Jarisch Herxheimer reactions," and they are also found when killing other organisms, as in the treatment of Tuberculosis, Syphilis, Leishmaniasis, Leprosy (Lucio's phenomenon). From anecdotal reports,[3] it apparently can also happen under appropriate nutritional regimes.

Jarisch Herxheimer theory and interpretation states that the above symptoms may be found whenever an organism more complex than a simple bacteria is killed inside the human body.[4] Whether or not the presence of an inimical organism, and its death, is both a necessary and sufficient condition is not known.

When you've gone through the Jarisch Herxheimer (which may be very mild or very heavy), you should be well — except for damage already done — until you've gotten reinfected with the organism.

Related Treatments

A physician may decide to simply give a patient antiamoebics or to give the medicines at the same time he is treating other problems. Nutrition, physical exercise, and allied treatments (such as Candidiasis[8]) that supplement overall well-being are very important. Why? Because they are designed to improve the immunological system — the ability to fight the presumed amoeba in a natural way. It is assumed that the "stronger" an RD victim's immunological system, usually the longer they can go before receiving more antiamoebics.

The RD victim may also receive a second treatment, called "intra-neural injections,"[5] which is very effective in controlling the pain of Rheumatoid Disease as well as the pain of Osteoarthritis.

How Many People Get Results?

Our experience in open studies[6] shows that results are obtained from 78% to 95% of the patients treated for Rheumatoid Disease *using our treatment protocol*.[7]

These results differ greatly because different physicians select patients differently, and they may also include in their clinical study some patients that are not affected by the presumed amoeba, but in fact may be affected by other organisms, such as *Candida albicans*[8] that can present similar symptoms.

If there were a placebo effect (belief) factor involved, our results would not be greater than about 30%, the same results that trained Rheumatologists get with various "accepted" but often dangerous treatments.[9]

The Rheumatoid Disease Foundation [now The Arthritis Fund] prays that all patients will be among those who respond.

The Movement is Growing

I founded and chartered the Rheumatoid Disease Foundation [now The Arthritis Fund] as a non-profit, charitable, IRS tax-exempt organization on October 13, 1982 with a number of lay people (notably treasurer Frederick Binford and vice-treasurer Donald Vansant and others now resigned) and five physicians on the Board of Directors: Robert Bingham, M.D., Jack M. Blount, M.D., Gus J. Prosch, M.D., Dr. Paul K. Pybus, Eugene S. Wolcott, M.D., and Professor Roger Wyburn-Mason, M.D. (Roger Wyburn-Mason died, and two resigned: Gus J. Prosch, M.D., and Robert Bingham, M.D.)

Eugene S. Wolcott, M.D., stayed on to become Senior Vice-Chairman. Dr. Paul K. Pybus became the Foundation's Chief Medi-

cal Advisor — he had worked with Roger Wyburn-Mason as Roger's house physician many years earlier, and holds a deep respect for Wyburn-Mason's skill and knowledge. Jack M. Blount, M.D., became the Rheumatoid Disease Foundation's Chairman until succeeded by John Baron, D.O., recently. Jack Blount is now Chairman Emeritus, after serving nearly four years. Interestingly, Eugene S. Wolcott, M.D., was my family physician for twenty years, having treated my wife, myself and ten children. Like many other physicians, he joined the Board only after having tried our treatment on patients and having observed the results on me and others.

Since then hundreds of physicians[10] located in many different countries (but chiefly the U.S.) have skeptically tried our treatment and have been quite impressed with results.

We are now funding double-blind studies on the use of one antiamoebic (clotrimazole) at Bowman Gray School of Medicine and have funded or are funding other scientific studies at the Medical College of Virginia and Vanderbilt University. Dr. Kwang Jeon, protozoologist, University of Tennessee, has done some work without pay, as has Dr. Paul K. Pybus and pathologist A. H. Davies, Ph.D., (South Africa) and medical student, Tony Chapdelaine, assisted by protozoologist Robert J. Neff, Ph.D.

Tens of thousands of concerned citizens have joined with this Foundation, in spreading the word, and by sending in their contributions.

We are represented in more than 9 different foreign countries, and there will be Chapters to serve localities with free-treatment for the indigent one day, hopefully soon.

So What Have We Learned

First and foremost, we believe (but cannot prove) that we have learned that Roger Wyburn-Mason, M.D., like Semmelweis, had the wrong theory but the correct solution. Many will remember that Semmelweis preceded Pasteur's germ theory by his theory of the "odor of death" and thus brought child-bed fever deaths down drastically. For this wrong theory, right treatment — and the savings of lives that embarrassed others in the medical profession who had a higher death rate — he was cast out of his medical association.

Science, you may remember, grows by development of theories which, when they work, are accepted whether or not they make sense. Later, with refinements, they may be changed, so long as the changed theories also work and usually work better. Please keep in mind John W. Campbell's statement which defines proper scientific method as thus: *A theory need not be correct, it need only work*![20]

When Wyburn-Mason worked with Admiral Stamm, protozoologist, according to his now deceased wife, Joan Wyburn-Mason[11,27] Roger himself could not accept the protozoan theory of the causation of RD. After many nights of work with Stamm, back in the fifties, he was finally convinced by Stamm that a protozoan was the culprit. Stamm, remember, was an eminent and well-published protozoologist.

When Roger invented his thermotropic separating device, he sterilized his samples by the use of penicillin and streptomycin to ensure that no foreign bacteria would go through the minced samples into their collection jar. By so using these antibiotics, he ensured the creation of what has come to be known as Cell Wall Deficient (CWD) organisms.[12]

To be aware of the fact that he had created Cell Wall Deficient organism, Wyburn-Mason would have had to know about them, and the scientific field had not clearly defined them until 1974 by Domingue, Schlegel, and Woody.[13]

Note, further, that without access to electron microscopy — unavailable to either Wyburn-Mason or Stamm — had they known about Cell Wall Deficient organisms, they could not have studied them, nor observed the cell-wall striping effects of antibiotics on common bacteria that they thought to kill by use of antibiotics to prevent experimental contamination in the amoebae thermotropic separation device.

A further note of strong interest: well-trained protozoologists of today will often observe Cell Wall Deficient organisms, or colonies of them, under ordinary microscopes, and conclude that they are viewing amoebae![12]

And—

Even today, 1987, there are virtually no clinical laboratories that have the expertise and training to isolate Cell Wall Deficient organisms from human tissues and to study them, nor can anyone determine the implications of the existence of such organisms inside the immunologically deficient patient, nor can anyone determine whether or not such organisms contribute to immunological deficiency.[23]

The study of Cell Wall Deficient organisms is not an unknown field, but simply an esoteric specialty that has yet to be integrated into the routine of physician knowledge and practice.

Be it further noted that Robert Neff, Ph.D., through separate studies using knee effusions and other techniques, concluded that Roger Wyburn-Mason and protozoologist Stamm had used faulty filtering equipment and insufficiently differentiating microscopic equipment, so that they were unable to differentiate microscopically between host cells and amoebae, and that more likely he observed blood cells that persisted but did not grow or divide, further, that it seems probable that the structures he called cysts were damaged and clumped host cell nuclei.[30]

Kwang Jeon, Ph.D., concluded that no amoeba were present in knee effusion samples submitted from our referral physicians.[31]

Dr. Paul K. Pybus and A.H. Davies, Ph.D., at first thought they were viewing amoebae, but later concluded they viewed macrophages.[32]

Brian Susskind, Ph.D., also concluded through use of both synovium and other tumor samples, that only macrophages were present, not amoebae.[13]

It is easy to understand, then, how it was that Roger Wyburn-Mason and Stamm — after repeatedly "viewing" protozoans in their cultures, after pursuing world literature on protozoan[2] and seeing therein much that corroborated and explained, *and especially after developing an "anti-amoebic" treatment that worked spectacularly for the first time in world history on 80% of those Rheumatoid Disease victims treated,* that they felt their case was closed, that protozoans were proved.

What Do We Know For Sure?

Thanks to many funded and unfunded researchers (and chiefly to the synthesis of Dr. Paul K. Pybus,[11] our chief medical advisor) from what we seem to know through our research to date, we can guess at the following facts:

1. Our treatment works 48% to 65% better than present rheumatology practices; i.e. it works 78% to 95% of the time, depending on patient group and physician practices.[6,9]

2. If a placebo effect were involved, this percentage would not be greater than 30%.[9]

3. Clotrimazole inhibits formation of phospholipase (PLA2), in a calcium dependent manner. PLA2 precursors the arachidonic cascade. Further, note that an under/over concentration of Ca or Fe ion determines the quantitative nature of the dependency, thus explaining to some extent the nutritional relationship of Ca/Mg et al. to RD.[14]

EDTA also inhibits PLA2,[14] thus explaining to some extent why EDTA therapy gives temporary relief (not to mention the

presumed cleaning up of "free radicals" generated during the inflammatory process.[15] —We would guess that DMSO, properly used, also temporarily cleans up presumed free-radicals, but also contributes to the change in ratio of HDL to LDL[28,35]).

4. Clotrimazole kills a very wide spectrum of protozoans in the test tube, as opposed to metronidazole and tinidazole.[16] Tinidazole and clotrimazole can be metabolized by either human enzymes or intestinal micro-flora; metabolization of metronidazole relies solely on intestinal micro-flora.[17] This may explain why the first treatment of metronidazole may be effective, but not the second: during the process of being metabolized by intestinal micro-flora, it also kills off the "good-guys." When micro-flora is replaced, or taken with metronidazole, the treatment often becomes effective again.[18] (We presume "good guys" micro-flora includes *Lactobacillus acidophilus*, *Lactobacillus bifidus*, and *Streptococcus faecium*, but more research needs to be performed to be certain, or more information needs to be gathered.)

5. Clotrimazole kills *Candida albicans*.[8,19]

6. Clotrimazole stimulates cortisol.[14,35] (Perhaps a means of getting marginally deficient patients weaned away from external cortisone?)

7. Metronidazole kills over-active macrophages according to work by Paul K. Pybus and A.H. Davies (1st reported), and seems to be corroborated by Kwang Jeon, Ph.D. reports.)[21]

8. Clotrimazole does not kill over-active macrophages.[11]

9. Various nutritional substances affect the disease state and the progress of wellness. (Copper, Boron, selected fats, sugars, various other vitamins and minerals, various diets that work or harm, et al.)[22]

10. *Candida albicans* often spreads with the presumed *Amoeba chromatosa* under the same rules related to "weakening" of immunological system, and probably ought to be treated simultaneously, if suspect.[8]

11. There is a relationship between allergenic responses from various antigens and RD symptoms.[22] (See various treatments based on "allergenically clean" clinics, pure water fasts, bio-detoxification programs developed by L. Ron Hubbard, now implemented by Zane Gard, M.D., San Diego, CA, et al.)

12. Our treatment protocol includes different "anti-amoebics" which affect amoebae differently, according to environment, concentration and other factors, according to *in vitro* chemosensitivity studies.[34] This has been presumed to explain varying effects *in vivo* as due to varying body chemistry and varying genus, species and strains of amoebae. Varying organisms may still be involved and so may varying body-chemistries.

So, What is Our Direction of Search Now?

1. The Bowman Gray Medical School Rheumatoid Arthritis Study on use of clotrimazole in double-blind trials goes onward. Whether or not Roger Wyburn-Mason's theory is correct, the treatment works, and we must establish through double-blind means that it is both safe and effective (FDA criterion).

2. At the suggestion of many (Pybus, Neff, Franson, Susskind, Jeon, others, and because of negative results in reproducing the Stamm/Wyburn-Mason protozoan, we should concentrate primarily on the bio-chemical connections involving principally clotrimazole and metronidazole. For example, Smith at Bowman Gray has taken randomized knee effusions which have been supplied to Franson[35] at Medical College of Virginia. Franson and Susskind are cooperating in developing further Franson's breakthroughs related to clotrimazole, and Susskind's further findings. Experimental results will be eminently useful and publishable.

3. Robert J. Neff, Ph.D.,[30] and Kwang Jeon, Ph.D.,[31] both have interesting suggestions potentially fruitful for further research that should decidedly be followed up.

Kwang Jeon would "test the hypotheses that infective amoebae are directly or indirectly involved in the manifestation of arthritic symptoms and that anti-amoebae drugs such as Imidazole compounds cause the remission of reducing the secondary effects of amoebae on other cells such as synovial cells. For example, Imidazole compounds may act on altered synovial cells in the joints of rheumatoid arthritis patients to prevent the production of rheumatoid factors, thus reducing chronic inflammation in arthritic patients." He would further "examine synovial fluid samples from arthritis patients for the presence of possible infective agents such as amoebae, study the effect of Imidazole compounds on cultured synovial cells, with special emphasis on the viability of synovial cells *in vitro* and subsequent production of immune complexes, and compare growth behavior of synovial cells from patients treated with drugs, and use animal models such as susceptible rats to examine the *in vivo* effect of Imidazole on synovial cells."[31]

Robert Neff, Ph.D., would suggest determining "the concentration of amoebae antibodies in both the synovial fluid and serum of RA patients. The enrichment of the antibody might be determined by comparing the antibody concentration with the concentration of a non-immune constituent such as human serum albumen; determine if amoebae antibody complexes with antigen and C fragments are present in the phagosomes of leukocytes of a series of both RF plus and RF minus RA patients. If present, determine if the antibodies are enriched in the phagosome aggregates as compared to other antibodies or other proteins found in the same synovial fluid; determine if neutrophils, present in both synovial fluid and peripheral blood, are already activated to attack amoebae and if the attack is mediated by amoebae antibodies; determine the complement pathway/s present in synovial fluid in serum."[30]

4. Brian Susskind, Ph.D., suggests: "Therefore clotrimazole, levamisole, tinidazole, and metronidazole may yet be found to subserve similar immuno-modulatory mechanisms [as cyclosporin and levamisole] in rheumatoid arthritis. Further studies are necessary to determine if clotrimazole exerts modification of the inflammatory response at one or more specific sites, and if it acts as a general immuno-suppressant or as an immuno-potentiator under selective conditions. Hence, the paramount importance of correlating *in vitro* data with an experimental *in vivo* system in order to determine which effects are relevant to the drug's therapeutic activity. Complete understanding of the clotrimazole's immuno-modulating activities will also lead to the design of more effective protocols."[33]

5. Richard C. Franson, Ph.D., states that "We have demonstrated clearly that clotrimazole inhibits the human synovial fluid PLA2 (as well as other neutral-active and calcium ion dependent PLA2s) in a calcium ion-dependent fashion. That is, the lower and more physiologic levels of calcium ion ... clotrimazole produced dose-dependent inhibition. Because membrane phospholipids contain the bulk of arachidonate (the precursor for prostanoids and leukotrienes) in the SN-2 position of the molecule, the ability of this molecule to act as an anti-inflammatory agent was proposed. Tinidazole and histamine had little or no effect on enzyme activity; similar results were obtained [with] metronidazole ... . The mode of action appears to bind clotrimazole to the phospholipid substrate since centrifugation studies of drug plus substrate *E. coli* resulted in cosedimentation of both components leaving no inhibiting activity in the supernatant fraction. ...

"...we are continuing the search for endogenous regulators of what we believe is a proinflammatory PLA2 in SF [synovial fluid]. It is clear from these studies that both inhibitory lipids and proteins are present in synovial fluid that moderate the expression of PLA2.

We believe that clotrimazole is an additional modulator and that the very interesting studies that Dr. Susskind now pursues with respect to the drug's effect on monocytes may be membrane-lipid mediated and thus be directly related to our basic observation of phospholipase inhibition.[35]

6. Lida Mattman, Ph.D., of Wayne University suggests relating, if possible, knowledge of Cell Wall Deficient organisms to Rheumatoid Disease.[23]

Reason: If you will look on pages 38 and 39 of *Rheumatoid Diseases Cured at Last* (Third Ed.),[24] you'll read mention of work performed by Marmor and Warren. They isolated a heat resistant RNA molecule from *active* Rheumatoid Disease synovium. On injections in mice and chickens, active RD was created. Isolates taken from these were again passed through other mice and chickens and these produced active RD.

On first reporting this satisfaction of Koch's Postulates, attempts to reproduce their study failed. According to Lida Mattman, when Warren and Marmor pointed out that it required synovium from "active" RD victims, the study was indeed replicated as reported. Yet no one seems to have followed up on this lead.

Dr. Lida Mattman, herself, has taken Cell Wall Deficient *propiono* bacteria (common on skin) and on injecting it into chicken eggs, has created chickens with Rheumatoid Disease.[12]

Dr. Lida Mattman speculates that: A heat resistant RNA molecule may ride piggy-back on the Cell Wall Deficient organism and, on entering human tissue, sets up the "genetic sensitivity" to the Cell Wall Deficient organism.[12]

Thus, it is clear that either an RNA molecule or a Cell Wall Deficient bacteria, or some combination of both can cause Rheumatoid Disease in certain animals.

This would also suggest a correlation nicely with Thomas Brown's,[26] M.D., (Arthritis Clinic of Northern Virginia, P.C.) thesis that a mycoplasmic bacteria is involved with gorillas, and presumably humans (Cell Wall Deficient bacteria are often protozoan in appearance, just as they may be mycoplasmic in appearance).

And the effects of Cell Wall Deficient bacteria inside the human body would begin to explain peculiarities of the immunological envelope as well as disease states.[29]

7. And while it may be an insignificant point — no stone should be left unturned. During my recent visit to Phillip Hoekstra, III, and Lida Mattman, Ph.D., Hoekstra, using a darkfield microscope, showed in my own blood both the existence of a Cell Wall Deficient *Candida albicans* and a strange appearing leukocyte. He made a photo of the leukocyte, stating that he had noted a strong correlation on viewing this kind of abnormal leukocyte in all RD victims. The photo will be made available to our physicians and scientists for further possible correlations.

Therefore, as can be seen, The Rheumatoid Disease Foundation [now the Arthritis Fund] started with a hypothesis that works very well but — by keeping an open mind toward all possibilities — has made much progress in understanding the reasons for the treatment's successes and, like any good research orientation, can now point to areas of research that are likely to conclude our understanding of Rheumatoid Disease [to] the benefit of all.

The Rheumatoid Disease Foundation [The Arthritis Fund] has done more good for those afflicted with Rheumatoid Disease, and it has made more progress in conquering Rheumatoid Disease — with less money — than any other organization in history, starting with Roger Wyburn-Mason's apparently faulty hypothesis that led him to the world's first correct treatment.

It is believed that we are on the virtual threshold of understanding all, and had we not gotten ourselves involved with an unethical fund-raiser, our financial plight would not have suffered, as our understanding has grown.

Others Helped

If you are a patient taking antiamoebics, or are about to be treated with them, or if you are a physician about to treat patients with our protocol, you got where you are because others shared their knowledge and resources to let you know there was a cure, or at least control and probable remission.

If others had not benefited, you would not be reading this today, or administering our treatment to others today.

Can You Help Others?

There are literally millions of good, decent folks of all ages — young and old — who need to be treated for crippling Rheumatoid Disease. They need to know that there is help, that others are well, that the disease can now be conquered and the terrible scourge brought to an end.

You Can Help

If you are a Rheumatoid Disease victim, you can help by getting yourself well, telling others about your recovery, working with newly founded local Chapters to raise funds to help others get well, writing to influential people, contributing funds to support our research, buying and distributing literature and books and by your thoughtful suggestions.

If you are a physician, you can help by many of the same activities described above, but also by telling other physicians about us and letting your patients know about us — especially through solicitation materials available through our office that you can give to those you treat, using our treatment protocol.

It's Up to You

How fast do you want the disease to disappear from the Earth's face?

It's up to you!

Tell folks about us — get them well — support our research and/or a local chapter — everything and anything, no matter how small, will get us there!

What If You Have Further Questions or Wish to Donate?

If you wish to donate or desire more information, write to our National Office, at The Arthritis Fund, 5106 Old Harding Road, Franklin, TN 37064, Fax/phone the same: start sending *before* you hear our signal (615) 646-1030.

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The Free-living Amoebic Causation and Cure of Activity in Rheumatoid and Auto-Immune Diseases

by ROGER WYBURN-MASON

Editor's Note: This is from the last manuscript by Doctor Roger Wyburn-Mason. While it repeats information previously published, it also includes material inserted shortly before his death and an updated bibliography to 1979. As a basis for the research work at three schools of medicine and the clinical practice of over 250 physicians throughout the world and the formation and function of the Rheumatoid Disease Foundation [now The Arthritis Trust of America], it has historical as well as medical value.

However, numerous species of *free-living amoebae* are known. Most fall into two genera, *Acanthamoeba* and *Naegleria* and some are pathogenic to man and animals; they are found on the surface soil preferring warm, moist conditions and proliferate in warm stagnant pools and at the bottom of rivers and lakes, particularly around the entry sites of warm effluents. They have been found in the domestic water supply, in human feces and in unpasteurized milk. Pathogenic free-living amoebae are readily isolated from chlorinated swimming pools, potable water, sewage and human nasal and throat cavities. They often contaminate tissue cultures. In inimical conditions, they form hollow spherical cysts which are present in the air in most parts of the world and can easily be found on agar plates exposed to air. Free-living amoebae prefer warm surroundings, and they tend to migrate from cool environments to body temperature, a property known as thermotropism.[1]

All terrestrial animals and plants and those inhabiting fresh water and also probably the sea, live in a world surround by many species of free-living amoebae, which certainly pass into the mammalian respiratory passages as cysts or trophozoites in the gastrointestinal tract of many animals, including man, since they are found in their feces. As the organisms are motile, it would be unreasonable to suppose that, once they had entered the orifices of man or other warm-blooded animals, they would not migrate under the thermotropic influences into the body tissues. Since the amoebae may prove to be either non-pathogenic to animals, the same must also apply should the organisms reach human tissues.[1]

Recently it has been shown[10] that the sera of all humans, including that of the cord blood, contain antibodies to either *Acanthamoeba* or *Naegleria*, indicating *universal* present or past infection of man and the newborn with these organisms. Textbooks on protozoology state that "unspecified types of amoebae have been isolated at times from every tissue in the body,"[3] or "there is hardly an organ in the body from which somebody has not obtained amoebae."[4] Thus, *all human bodies appear to contain free-living amoebae* somewhere in the tissues. A few cases of leisons due to species of such organisms have been described in plants and man, in particular amoebic meningo-encephalitis.[5,6]

The whole syndrome resembles syphilis. Waldenstrom and others, indeed, state that "if the spirochaete had not been discovered, syphilis could be taken to be the ideal model of an autoimmune disease. The variety of tissue reaction antibodies, the widespread lymphocytic tissue damage and the vasculitis are characteristic features."[21] Rheumatoid disease closely resembles the rheumatic manifestations in leprosy[22] which may present with an acute arthritis affecting one or a number of joints, polymyositis, skin leisons, fever raised ESR, etc., with increase in circulating gammaglobulins and positive serological tests for autoantibodies, RF and ANF, as in rheumatoid disease. This is an immune complex syndrome with antigen provided by disintegrating *M. leprae.* The reaction may be precipitated by antileprosy drugs, a reaction known as Lucio's phenomenon, which is identical in nature with the Herxheimer reaction. The syndrome confirms the deductions made regarding rheumatoid disease. Such observations prove that every tissue in the body may contain unsuspected free-living amoebae, which, if pathogenic, may cause tissue infiltration by lymphocytes with germinal centers and often plasma cells in genetically susceptible subjects as governed by their tissue types. They are the source of Glynn's previously postulated unknown chronic antigenic stimulation,[23] as the cause of rheumatoid disease.

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The Use of Ionic Copper in the Treatment of Arthritis

by SELDON NELSON, D.O.

Editor's Note: Dramatic clinical improvements in some cases of rheumatoid disease have resulted with the use of new resin coated copper granules. This paper covers the rationale and techniques of treatment. Copper is considered a trace mineral, and in this form, it is used as a dietary supplement, as rheumatoid patients are so often deficient in this substance. Normal and healthy persons usually show no reaction to these doses of copper, and no severe effects or reactions have been reported. Much clinical testing and follow-up reports must be obtained before the method is recommended for general use. Trial amounts may be obtained from the author of this paper.

According to the research by the late Dr. Roger Wyburn-Mason, the cause of rheumatoid arthritis and some other chronic and rheumatoid diseases may be an amoeba parasite, *amoebae limax* or *Naegleria.* These organisms, or whatever is eventually proved to be a cause for these conditions, are sensitive in varying degrees to various antibiotic substances.

Basic research has been done which indicates that the causative agents or rheumatoid diseases are susceptible of being destroyed by minute amounts of pure metallic copper. For example, even water contaminated with *limax amoebae* can be cleansed by running through copper pipe. Since it "is not possible to run a person thru a pipe of copper," the copper ion must be given to a person in another form. While Dr. Wyburn-Mason reported some success with copper sulfate, less toxic forms have been sought by clinical investigators.

It has been found that pure metallic copper can be prepared and administered in the form of granules in microgram quantities on an ion exchange resin.* This is successfully used as a nutritional supplement in patients deficient in this element, which appears to make them more susceptible to chronic illnesses of the rheumatoid type.

Copper is an essential trace mineral in human diets, consumed daily in some foods, and contributes to health in the formation of new blood cells, the red blood cells and the leukocytes which help the patient to fight infections.

Some physicians have found that when copper of this type is used in microgram quantities, it is a very conservative treatment, obtaining therapeutic benefits in ridding the body of toxic parasites such as amoebae without risking toxic reactions or serious side effects.[1] Of course, as with any heavy metal, taking too much copper or for too long a time can produce adverse effects which can easily be detected and avoided or treated.[2]

Ceruloplasmin and serum copper levels are indicators for therapeutic and also toxic levels of copper permitting periodic evaluations by the physician. As with any method of arthritis treatment, if effective results are obtained, certain baseline laboratory values, including a SMAC-24, a CBC, and serum copper levels and ceruloplasmin levels must be determined for later reference.

Contraindications to this treatment may be any abnormal neurological signs or symptoms, although some success with this therapy has been seen in one neurological disease where it seems that copper deprivation may be a factor.[3]

The Treatment Program

The following protocol is utilized in treating patients with active rheumatoid disease:

When the patient has signed an informed consent to a new type of food supplement to correct a probable essential trace mineral deficiency, a test amount of the ionic copper granules can be given in the office. This may be as few as 5 [granules or micrograms?] or as many as 20, although 15 is the average amount for a 150 pound adult male.

As one becomes familiar with the treatment, a "feeling" of the proper amount for each patient will develop. In a short time, clinical judgment will determine the initial dose and the amount to be increased each day, usually divided into three equal doses, taken on the tongue and washed down with a half glass or more of water.

The first treatment program will take about six weeks and the patient should have a favorable response, which may be from moderate relief and improvement of signs and symptoms to a complete or permanent remission.

In addition to the copper granules, patients may take their customary medicine for arthritis discomfort, and a biologically active nutritional supplement is also used.[4]

As with all antibiotic therapy, the substance used is usually given to achieve a specific blood level. For the use of copper as a nutritional supplement and to build up the natural resistance of the body to the infective agent, no definite blood levels have been determined. They may be different for different patients. Clinical observations along with the specific blood levels for the particular patient will act as a guide should it seem desirable to repeat the program.

When the therapeutic level of copper in the blood is reached, then the susceptible microorganisms, whose presence is the probable cause of the disease, are killed by the chemical activity of copper ions. This is an all-encompassing phenomenon, and it affects the entire population of microorganisms in question. But the killing of the susceptible microorganisms may, and usually does, result in the production of a *Herxheimer reaction.* (The patient may feel that the arthritis is getting worse, or that a flare-up or aggravation of the disease is occurring. It should be explained to the patient that this is an "expected reaction" probably caused by release of toxic substances from the killed pathogenic organisms, or the amoebae in rheumatoid arthritis, and not by live microorganisms of any exacerbation of the disease.)[5]

The extent of the Herxheimer reaction is directly related to the number of the microorganisms being destroyed, the area of the body that has been affected by the rheumatoid disease, the rate of release of toxins from the dead microorganisms and the patient's own resistance or sensitivity to foreign proteins.

Rarely, some patients experience a severe reaction appear "really sick." But the whole secret of success with this treatment with copper granules is to get the patients past the Herxheimer reaction with a minimum of discomfort and apprehension about the apparent flare-up of the disease.

This is usually best accomplished by getting past the stage of the reaction as quickly as possible, as opposed to stringing out the process and prolonging the agony.

Start the patient on whatever level that it appears can be comfortably handled. For example: Prescribe an initial dose of 10 granules. This is to be taken three times a day the first day. Then increase the total dose by 5 granules each day, observing the reactions and tolerance. If this does not provoke a reaction, then increase the daily total by 10 granules per day until the patient is taking 75 to 80 granules per day. This level is maintained for 10 to 14 days. Then, the maximum dose is achieved by going to 90 granules a day. Then the Herxheimer reaction should be safely passed, permitting the patient to take 90 granules 2 times a day for two weeks, then the lesser dose of 100 granules once a day for two weeks. Then the copper medication is stopped and the clinical and laboratory evaluations repeated to judge the state or progress and recovery of the patient.

The treating physician should expect and look for signs of a

Herxheimer reaction in his patient once the copper granule treatment has been started. It may be very mild and immediate, taking only a few seconds, or it may develop later, several hours to several days to manifest itself. Since it is due to the killing of the microorganisms responsible for the disease, it is a clinical confirmation of the diagnosis of rheumatoid disease as well as an indication that the patient will benefit for the treatment with improvement or complete recovery.

The reaction to the copper granules is *not a drug* reaction. When the granules are given to a control who is a healthy subject, no Herxheimer reaction occurs.

If the patient has had a treatment with gold therapy, penicillamine or cortico-steroids currently or recently, or for long periods of time, the usual physiological Herxheimer response may be altered. In some of these patients, it may be entirely absent. This probably indicates that the normal immunological responses of the patient's body has been altered by these drugs. It may also indicate an acquired resistance of the pathological organism responsible for the disease to antibiotic agents.

What should the patient be told to expect in the form of a Herxheimer reaction? These symptoms may occur in order of their frequency and severity —

1. A dry, "funny" or metallic taste in the mouth.
2. Increased aching and pain in the joints.
3. Muscle fatigue and a "burning" sensation.
4. Loss of appetite, nausea, occasional vomiting.
5. Diarrhea or constipation, cramping, gas.
6. Some tissue or joints swelling, redness, local heat and inflammation.
7. Increased muscle and joint stiffness.
8. Low fever and night sweats.

Other rare signs and symptoms may temporarily appear, mimicking other rheumatoid diseases: Skin manifestations, eruptions, scaling, eczema, and psoriatic appearing lesions. If the organisms have been lodged in the tissues of the central, peripheral or autonomic nervous systems, there may appear neurological or sensory symptoms, including the special functions of vision, hearing, taste, and smell.

Since Rheumatoid diseases are systemic in nature, the endocrine tissues seem to have an affinity for the organisms causing the infection. In the Herxheimer reaction, there may be significant changes, such as alteration of the menstrual cycle, decreased need for insulin in diabetes, less thyroid requirement in hypothyroidism, and reduction in the signs and symptoms of endometriosis. Headaches are not uncommon, and psychological changes may be noticed temporarily such as unexplained anger, depression, irritability, listlessness, fatigue, etc. The patient must be reassured regarding these phenomena, and they may be reduced or prevented by appropriate treatment. Complete suppression, however, removes a significant clinical observation which the physician uses as a treatment guide.

However, in some patients, it may be necessary to treat it or to suppress the Herxheimer reaction to help the patient get past this stage of treatment with less discomfort and less physical disability. This may be done in one of several ways. First, the daily dose of copper granules may be reduced by one half or more for two or three days, or until the uncomfortable symptoms subside, then beginning again on a lower level. Second, the medication may be completely halted for a week or so, then begun with a covering dose of symptomatic medications. Third, medications may be taken along with the copper granules without conflict or weakening of their therapeutic effect. These may include analgesics such as aspirin and the non-steroidal anti-inflammatory drugs, muscle relaxants and antiemetics. Fourth, an initial dose of depo-steroidal drug may be administered once a week until the Herxheimer symptoms have subsided or are past.[6]

Then, when the Herxheimer reaction has been suppressed, the disease should be treated more vigorously, increasing the dose of the copper granules to 30 granules three times a day and up to 90 granules twice a day for two weeks, then 100 granules once a day for two weeks. Then the medication — or food supplement — as it should be considered, is stopped. By this time, the patient's body has been saturated with a high and normal amount of copper sufficient to control the active form of the disease.

The philosophy behind the copper granule use in rheumatoid disease is to restore normal tissue levels of copper and then increase these to tolerance to inhibit and kill microorganisms responsible for these chronic systemic infections. While it will be important to continue research on the nature of these infectious forms, protozoa, mycoplasmas, or viruses, it *is not necessary* to identify the cause to get a good treatment result.

The copper granules permit the patient to be treated at a variable rate according to his own tolerance to the signs and symptoms of the Herxheimer reaction. The rate of "kill-off" of the microorganisms is directly related to the amount of copper granules used. Each copper granule will kill a certain amount of susceptible microorganisms. Ten granules will kill ten times as many. Fifty granules will kill fifty times as much. So the rate at which the patient can be treated, and his or her disease controlled, is dose related.

No serious reactions have developed with this treatment. There are no contraindications known to date. And there are no drug sensitivities or sensitivity problems with copper granules, since it is a normal physiological trace mineral in the human body.

Conclusions

Metallic copper in pure ionic granules has been successful in treating the rheumatoid diseases when used as a dietary supplement and increasing the amounts up to tolerance of the patients to Herxheimer reaction symptoms. No adverse or metal toxicity reactions have occurred to this form of copper in the amounts recommended. Recovery and improvement in rheumatoid signs and symptoms with this new method suggest its importance as a new road to health for the patient.

* Copper micro-granules were supplied as MEIRA™ Cu by Midwest Metabolic, Inc., 1435 East Grand River Avenue, Williamston, MI 48895.

Arteriosclerosis
A Vital Message to My Patients

Gus J. Prosch, M.D., and Wyatt C. Simpson, M.D.

We sincerely desire that all our patients and their families enjoy the best health possible. During the past 2-3 years, there has been an explosion of research and knowledge that can vitally effect the health of each of us. This paper was written in an effort to teach you, inform you, and convince you to change some of your eating habits so that you and your family will enjoy a healthier life and fewer health problems. Some authorities may question some of our conclusions, but when we daily observe the health of our patients improve from following these suggestions, in the name of *Truth* we must speak out and share this information with you.

Today's number one health hazard is arteriosclerosis or hardening of our arteries. This causes heart attacks, strokes, and peripheral vascular disease (usually in the legs) which leads to more misery and suffering than any other disease known today. This does not count the expenditure of billions of dollars and the loss of millions of days of productive work for the American work force.

Before 1900, this disease was hardly known and was extremely rare. In fact, the first "heart attack" was described in the medical literature in 1910. Dr. Paul Dudley White (President Eisenhower's heart specialist) saw a heart attack for the first time in 1929. The disease began with the advent of hydrogenated oils (margarine) and the processing (refining) of our grain foods such as wheat, corn, rye, barley, oats, etc., where all the vital fatty acids are removed from these grains. The food companies must remove these fatty acids so that the grain foods do not turn rancid and spoil, otherwise the foods would not last long on the shelves of our super-markets. Our great-great grand parents and their parents had very little arteriosclerosis even though their diets included foods known to be high in cholesterol such as eggs, butter, lard, and "sow-bellies," etc. However, they did not eat any hydrogenated oils, and their grain foods were home ground and not processed.

We have known for 20 years that the dietary cholesterol cannot be the cause of arteriosclerosis for several reasons. First of all, the dietary cholesterol in the stomach is broken down into its tiny component parts and although some is absorbed through the intestinal wall, most of our cholesterol in our system is manufactured by our own body. The problem of arteriosclerosis develops because our bodies do not use the cholesterol properly that our bodies make. Also, Iceland Eskimos, whose diet by the way is ten times higher in cholesterol than our diet, have very little arteriosclerosis. They should be "dying like flies" if dietary cholesterol intake caused arteriosclerosis. But they do not suffer from heart attacks, strokes, and poor circulation in their extremities unless they move to more civilized areas of the world and begin eating as we do. The two things these Eskimos eat differently from us are: (1) they do not eat any hydrogenated oils and (2) they eat a great deal of cold water ocean fish which are very high in fatty acids.

In the early 1940s, when the Germans overran Norway, the incidence of arteriosclerosis, cancer, and schizophrenia was quite high in that country. The Germans took away all the margarine from the Norwegians, and the incidence of these diseases dropped significantly. After the Germans left and Norwegians again began to eat their margarine, the incidence of these diseases increased to their former levels.

In America, we are developing arteriosclerosis at earlier ages than ever before even though there is a greater effort on the part of most of us to decrease our cholesterol intake in our diets. Autopsies performed on soldiers killed in the Korean War showed approximately 30% of these young men suffered from advanced atherosclerosis. About twenty years later, in the Viet Nam War, autopsies performed on soldiers killed showed approximately 60% suffered from advanced arteriosclerosis. We must do something about this trend, and that is the purpose of this paper.

Recent research has proven that all hydrogenated oils block the chemical pathways that are necessary for our bodies to use the cholesterol that our bodies manufacture. Also, our bodies must have certain essential fatty acids (now being removed from our foods) to assimilate and use our cholesterol as well as to manufacture certain hormone-like chemicals called prostaglandins, our cells cannot function properly, and they will be subject to disease. We believe that this is one of the main reasons that we are seeing an explosion of many chronic degenerative diseases such as Arteriosclerosis, Arthritis, Diabetes, Lupus, Schizophrenia, Multiple Sclerosis, Asthma, and numerous others. Other conditions that can benefit from these dietary changes include hyperactivity and learning disabilities, premenstrual syndrome, systemic yeast infections as well as many skin disorders and allergies.

What You Can Do!

1. Totally avoid all hydrogenated oils as in margarine, cooking oils, deserts (doughnuts, cookies, cakes, etc.) and deep fried foods such as French fries, corn and potato chips, etc. You can use cold pressed oils found at health food stores for cooking, but do not use extremely high temperatures. If the cooking oil label doesn't state "cold pressed," it is probably hydrogenated. You should read all food labels and avoid those that have hydrogenated oils as ingredients. Don't cook foods with high temperatures as all oils over 350 degrees become hydrogenated. Cook longer at lower temperatures.

2. Increase the essential fatty acids in your diet.

   a. Eat cold water ocean fish 3-4 times per week such as salmon, cod, mackerel, sardines, (pour off hydrogenated oils), water packed tuna. Warm ocean fish (snapper, flounder, perch, etc.) is second best. Fresh water fish (cat fish, trout, etc.) contain the smallest amount of the fatty acids.

   b. Try to eat 3 teaspoons of 1 tablespoon of *virgin* (not pure) olive oil daily (as on salads) but keep refrigerated after opening bottle.

   c. As a snack food, walnuts are very high in fatty acids.

   d. Only eat breads and cereals that have "100% whole wheat or whole grain" written on the package. Most brown breads are not whole grain but have coloring added. Avoid processed or refined cereals or white flour products such as breads, crackers, macaroni, spaghetti, noodles, etc. You can get these foods as whole grain from health food stores.

3. With any chronic illness at all, you should follow the above plus add the following supplements and follow the additional instructions.

   a. Purchase some salmon oil capsules (Maxepa) at a health food store and take 4-6 capsules daily. Extreme care must be exercised in locating this product as it is made only in England and many health food store products claiming this ingredient only contain soy oil. *Efamol* is one acceptable brand and *Nature's Way* is another.

   c. Decrease your red meat intake since red meats contain arachidonic acid which can provide too much of a bad prostaglandin plus a very bad substance called leukotrienes which will aggravate many disease conditions.

   d. Avoid all sugars, sweets, deserts, and all white flour foods.

   e. Get a good hypoallergenic, non-yeast multiple vitamin and mineral tablet and take 3-4 tablets per day. Be sure you get at least 500 mg. vitamin C, 50 mg. B-3 and B-6, 50 mg. zinc, 100 mcg. selenium, and 400 mg. of magnesium in your supplements. The above mentioned vitamins are necessary in the fatty acid chemical

reactions.

If you and your family will follow the above recommendations in your dietary habits, you will enjoy a longer, healthier life with much less chance of developing any chronic degenerative disease. You may pass the information on to friends and relatives to help them enjoy a happier and healthier life.

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Arthrosis

by M. BELY, M.D.

Editor's note: Dr. Bely presents an excellent description of the pathologic processes in degenerative arthritis and a report on experimental osteoarthritis produced in rats by sodium fluoride. The relation of fluoridated water to the human disease is under further investigation in Hungary.

Osteoarthritis is a degenerative process leading to progressive damage of the articular cartilage and secondary disintegration of the articular surface of bones. Several factors are known to have a role in the pathogenesis of the disease in secondary arthrosis. If the etiologic factor is unknown, the term idiopathic or primary arthrosis is used.

The degenerative process takes place in the articular cartilage, consisting of chondrocytes and intercellular matrix. The intercellular matrix is formed by a structure of collagen fibrils embedded in a proteoglycan matrix. The collagen fibrils have a characteristic orientation. Originating in the border-line between bone and cartilage, the fibrils run vertically upward to the surface of the articular cartilage, there bend and run further parallel with the surface, forming a dense layer, the so-called lamina splendens. The fibrils of the tangential zone, that run tangentially to the chondrocytes, are named "interterritoreal fibrils." The other part of the collagen fibrils — the territoreal fibrils — are organized circularly around the chondrocytes. The territoreal fibrils form sequences of linearly arranged microspheres.

This particular collagen structure provides the special biomechanical characteristics of the articular cartilage. The vertical fibers ensure resistance against twisting, tracting shearing stress, the lamina splendens serves as a shield, the linearly arranged microspheres resist against pressing forces. The lower zone of the articular cartilage is sclerosed, so the physio-chemical properties of this zone are similar to the characteristics of the subchondral bone tissue, providing firm connection between bone and cartilage.

Four zones can be distinguished from each other in the articular cartilage according to the orientation of collagen fibrils:

IV: Lower, sclerotic zone.

III: Vertical zone.

II: Zone of bending.

I: Zone parallel with the surface.

The other constituent of the intercellular material is the so-called matrix. The matrix consists of aggregates, composed by proteoglycans bound to molecules of hyaluronic acid. The proteoglycans are mucopolysaccharides (new name: glycosaminoglycane) bound to carrier proteins. Binding proteins bind the proteoglycans to molecules of hyaluronic acid. the mucopolysaccharides — strongly hydrophilic due to their negative charge — have a main role in the biomechanical properties of the cartilage. Their great water binding capacity (they can bind as much as 10,000 times larger amounts of water than their own) provides the elasticity and load bearing potential of the cartilage.

The chondrocytes are responsible for the balance of matrix, synthesized by them.

According to a generally accepted principle, the metabolic disturbance of chondrocyte activity is in the center of the pathogenesis of arthrosis in the case of primary arthrosis. The synthesizing activity of chondrocytes decreases, and probably abnormal matrix structures are also generated. A part of chondrocytes becomes degenerated, so enzymes, further damaging the structure of matrix get released:

— mucopolysaccharidase, splitting the mucopolysaccharides off from their carrier proteins;

— protease, breaking up the carrier and binding proteins;

— hyaluronidase, decomposing the molecules of hyaluronic acid, that keep the proteoglycane aggregates together;

— collagenase, damaging the bridges of collagen fibrils, that collapse after all.

The fragments of articular cartilage cause synovitis; the enzymes, released during the inflammatory process further increase the enzymatic destruction of chondroid tissue. Because of the damage of chondroid tissue, the surface of the articular cartilage becomes incongruent, so the remaining congruent surface gets relatively overloaded (unchanged load presses a smaller intact surface). The relative overload further increases the destruction of the articular cartilage. The degenerative process is a so-called vicious circle. The cause that starts the vicious circle is known in secondary arthrosis.

For example, in the case of syringomyelia, or tabes dorsalis, the vicious circle is started by the overload of articular surface due to the disturbance of bathyesthesia of joints. In the case of haemarthrosis, positive ions accumulate in the joint, so the negative charge of mucopolysaccharides becomes neutralized. The mucopolysaccharides, therefore, lose their water-binding capacity, so the elasticity and load-bearing potential of the articular cartilage decreases.

In the case of ochronosis, a pathologic metabolite, the homogentisine acid destroys the chondrocytes, the synthesizing potential of chondrocytes decreases, and enzymes further damaging the matrix get released.

Four clinical-radiological stages of arthrosis are distinguished:

Stage I: Mild clinical symptoms appear. Discrete sclerotization of the cotyloid cavity can be seen on the X-ray picture of the affected joint, the articular space and the condyle remain intact.

Stage II: The movability of the joint decreases because of the pain at the start of a movement, and the rigidity of the joint. There appear small cysts in the cotyloid cavity, and fine osteophytes on the X-ray picture.

Stage III: The movability of the joint becomes significantly limited. Secondary inflammation may occur. Cysts can be observed either in the cotyloid cavity or in the condyle on the X-ray picture. The articular space becomes irregularly narrowed.

Stage IV: The joint is more or less stiff, immobile. Secondary inflammation often takes place, signs of muscle decompensation are observable. Severe deformation of the joint can be disclosed on the roentgenogram: deformation of the condyle and acetabulum, cystic degeneration, detritus in the articular cavity, bizarre osteophytes, extremely narrow articular space.

The synovial membrane and cartilage of the joint and the subchondral bone tissue are inseparables [and] form an interdependent functional unit. The above mentioned vicious circle process leading to increasing destruction of the joint is accompanied by reactive synovitis. Secondary changes occur in the subchondral bone tissue too. The incongruence of the surface of the articular cartilage leads to the overload of the intact, congruent parts of the joint. The subchondral bone trabecules may collapse; therefore secondary necrosis of bone tissue in smaller fields may occur. The osteonecrosis increases the incongruence, leading to relative overload of the intact areas.

This process is a vicious circle too. The synovial membrane, articular cartilage and subchondral bone tissue form a functional unit. Injury of any of these components during an illness leads to the impairment of the other structures of this functional unit.

For example, in the case of primary arthritis, secondary destruction of the cartilage and osteonecrosis takes place; or at primary necrosis of the subchondral bone tissue, secondary destruction of the cartilage and reactive synovitis occur.

Cases of primary arthritis accompanied by secondary bone and articular impairment, due to mycosis, metabolic diseases, and autoimmune processes are interpreted.

Cases of aseptic bone necrosis due to trauma or systemic diseases (osteonecrosis due to sickle cell anemia, dysbaric trauma, steroid administration, etc.) are interpreted.

If the aseptic necrosis occurs in the subchondral region, impairment of the articular cartilage and synovial membrane can be disclosed in every case.

The histologic differential diagnosis is very important because the therapy and prognosis is different in osteo-arthrosis of different origins.

Changes in the Collagen Structure of Bone Tissue in Experimental Fluorosis

Introduction

According to experi[ments] in human [physiology], about 10% of the whole preexisting bone tissue is reorganized in a year.[12] This perpetual process of rebuilding, remodeling the bone tissue is due to the action of multicellular functional units (BMU, BRU or BSU), consisting of osteoclasts and osteoblasts. It is generally accepted fact, that sodium fluoride causes enlargement of the whole bone mass. There is no confirmed and generally accepted theory in the literature yet as to how NaF influences bone tissue whether the enlargement of the bone mass is due to increased osteopoesis (stimulation of osteoblasts)[4,5,8,9,13,14,16,18] and/or to decreased bone absorption (blockade of osteoclasts)[1,2,7,10,11,13,15,17,19]. Authors agree that the formed bone is inferior to normal, the matrix is irregular[4,6,10,18] the collagen structure of the newly formed bone tissue differs from normal,[10] and the mineralization is enhanced[4,8,10,11,13,14,18].

The aim of our experiments was the investigation of the changes of collagen structure in experimental fluorosis.

Material and Methods

The experiments were performed on 45 female rats in 3 groups. Fifteen animals were given 0.5 mg, another 15 animals received 5.0 mg of sodium fluoride intraperitoneally, daily, for 3 months. Fifteen animals — the control group — received physiological saline solution in the same way.

X-ray pictures were taken of the killed animals. Histologic investigation was performed on both femurs and on the third, fourth and fifth lumbar vertebra of the animals. The material, fixed in 1% formalin solution was decalcified. The decalcifying agent consisted of 24 ml of 85% formic acid, 50 ml 35% hydrochloric acid, and 125 mg distilled water, (imbedded in paraffin, serially sectioned, and stained with picrosirius red).[2]

The regularity of collagen fibrils of the preexisting bone tissue was measured by a polarization optic method according to Brace-Kohler in 550 nm monochromatic light using an Opton Standard microscope. The measurements were performed on the corticalis and spongiosa of both femurs and vertebrae using 5 visual fields in each case. Ten measurements were made in all fields. The average of retardation values, characterizing the regularity of collagen fibrils was calculated. Analysis of significance was performed between the retardation values obtained according to T and Welch (modified T) tests.

Results

The retardation values measured in the spongiosa and corticalis of the femurs and vertebrae are represented.

The regularity of collagen fibrils in the corticalis and spongiosa of femurs and vertebrae decreased as compared to normal. In the case of daily administration of 0.5 mg NaF, the observed difference is significant.

Administering 5 mg NaF daily for 3 months, the regularity of

collagen fibrils significantly decreased as compared to normal in the corticalis and spongiosa either of the femur, or of the vertebrae.

Discussion

The intercellular matrix of bone tissue consists of a collagen structure, embedded in proteoglycan aggregates. The process of formation and mineralization of the inter-cellular matrix are in a close relation. Isolation injury of any of these components is inconceivable. During the recent investigation, irregularity of the preexisting bone tissue's collagen structure could be disclosed by a specific topooptic method.

The investigations disclosed that the regularity of the collagen structure of preexisting, differentiated, lamellar bone decreases, so fluoride exerts its effect not only on the newly generated (newly formed woven) bone tissue, but also changes the collagen structure of the preexisting bone too. In our opinion, these changes can be considered as part of the toxic effect of fluoride exerted on osteocytes. The changes in collagen structure are certainly followed by damage to the matrix (proteoglycan aggregate). We are planning the selective investigation of this field.

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Nutrition, Prostaglandins and Arthritis

by Harold E. Buttram, M.D.

Editor's Note: This is the first of two articles by Dr. Buttram describing the importance of diet and nutrition in arthritis diseases. The role of prostaglandins is discussed. Recommendations are made for the reduction of animal fats and increased intake of the leafy green vegetables to combat disease. Evening primrose oil and northern fish liver oils may be useful in treatment. The second article will appear in the April, 1987 issue of the Journal.

Prostaglandins — What Are They?

Just as protein metabolism is regulated by enzymes, the *lipids, the fat tissues of the body are regulated by hormone-like substances called prostaglandins.* Prostaglandins are manufactured by virtually all tissues of the body. They are not stored in the body but are produced as needed. Their extreme potency is reflected in the fact that most biological effects are brought about by extremely minute quantities, expressed in parts per billion.

There are three major classes of prostaglandins: PGE1, PGE2, and PGE3.

The PGE1 Series: Figure 1. These are derived from a family of vegetable oils known as the *Omega-6 oils,* which are from Southern or warmer climates. *Linoleic acid* is the predominant fatty acid in these oils, which include corn, soy, sunflower, safflower, cottonseed, and peanut oils.

Defects in formation of PGE1 hardly ever result from dietary deficiencies in the U.S., because the Omega-6 oils are in great abundance in the typical American diet. However, the processing of linoleic acid to PGE1 is dependent on the enzyme, Delta-6 Desaturase, which may be inhibited by a number of conditions which will be described later.

Defects in formation of PGE1 are almost always due to reduced activity or inhibition of Delta-6-Desaturase enzyme.

PGE2 Series: It is this series which is the villain of our story, in the sense that it may be the source of tissue injury and tissue inflammation in such conditions as rheumatoid arthritis, premenstrual syndrome, asthma, psoriasis, and eczema, as well as a number of other conditions. It also causes increased clumping (hyperaggre[ga]tion) of platelets which can lead to blood clots an various forms of vascular disease.

The fatty acid, *arachidonic acid,* is the source of the PGE2 series and its various products, which include *thromboxane* and the *leukotrienes.* The *leukotrienes* are the most powerful inflammatory substances known to man, being 1,000 to 10,000 times more inflammatory than histamine. *Thromboxane* has a powerful action in causing vascular spasm and platelet clumping.

Now all of this may sound very technical to a non-professional reader, and such it is, but *the important point is this: arachidonic acid, which is the prime source of inflammatory substances in the body, is derived in large measure from animal fats, primarily red meat.* Therefore, diet has a great deal to do with the treatment of inflammatory conditions, such as rheumatoid arthritis, eczema, and even asthma.

Aspirin, cortisone, and other non-steroidal agents have been used many years for treatment of inflammatory conditions, but only recently have we learned how they act to reduce inflammation. They do this by inhibiting various steps in formation of the PGE2 prostaglandins. However, these substances may have side effects, which at times can be quite serious. Although these medications may at times be required, depending on the judgment of a physician, there are safer approaches.

Inflammatory reactions may be reduced by enhancing production of PGE1 and PGE3 series of prostaglandins, both of which

have strong inhibiting actions of the inflammatory PGE2, and also by reducing the dietary arachidonic acid, which is the source of inflammatory reactions. These things can be done through nutrition, as will be shown later.

Delta-6 Desaturase, Control Tower for the Prostaglandins:[2] The technical name of the enzyme, Delta-6 desaturase, is of little importance to most of our readers. But it is of immense practical importance to realize that there is a single enzyme which controls and regulates the formation of both the PGE1 and PGE3 series of prostaglandins, the two series which are essential for balancing the PGE2 series and inhibiting potentially tissue-damaging reactions from the latter.

Unfortunately, the Delta-6 Desaturase enzyme is not terribly hardy or durable, and it is subject to defects. The list of factors which have been found to reduce the efficiency of this enzyme is a very long one, but the most important are the following:

— High intakes or high blood levels of cholesterol.

— Hydrogenated oils, which are a source of trans fatty acids.

— Adrenaline (epinephrine) released from adrenal glands during stress.

— Excessive alcohol (about 10% of all calories consumed by people in North America is in the form of alcohol).

— Diabetes.

— Atopy (allergy). Atopy is an inherited susceptibility to certain diseases such as hay fever, eczema, and asthma. About one person in 5 or 6 in North America is atopic. Epidemiological studies have shown that some conditions other than the three listed above are also associated with atopy. Over 70% of women with premenstrual syndrome and over 70% of hyperactive children come from atopic families. It is now known that people with atopy have a defect in the functions of Delta-6 Desaturase.

— Vitamin and mineral deficiencies, notably pyridoxine (vitamin B6), zinc, and magnesium.

Nutritional Strategies in Management of Inflammatory Disorders

Reduction of Dietary Arachidonic Acid: Considering that powerful inflammatory substances such as leukotrienes and thromboxane are bio-synthesized from the fatty acid, arachidonic acid, it would appear rational to reduce the intake of dietary arachidonic acid, thereby reducing the source of these substances. Practical application of this principal today is most frequently found in treatment of rheumatoid arthritis, but it would probably be beneficial for other inflammatory conditions as well.

Since arachidonic acid is richest in animal fats, nutritional treatment of inflammatory conditions should include a reduction or elimination of red meats along with restriction of eggs, milk, and other dairy products.

Enhancement of PGE3 Series: In contrast to the omega-6 oils, which are abundant in the typical American diet, the omega-3 fatty acids are commonly deficient. The precursor of PGE3 series, *alpha-linolenic acid,* is found in green leafy vegetables, in small amounts in soy oil and wheat germ oil, and large amounts in linseed oil. The preformed eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) are abundant in northern seafood, which we recommend as a regular part of the diet.

Marine lipids do have a powerful effect in suppressing inflammation, by their inhibitory action on the "arachidonic acid cascade." This action is mediated largely by eicosapentaenoic acid (EPA). Studies have shown marine lipids to be beneficial in such diverse conditions as rheumatoid arthritis, migraine headaches, nephritis, and asthma.[5,6] In one of these studies, 17 patients with rheumatoid arthritis were placed on a diet low in saturated fats with daily supplements of 1.8 grams of EPA (commercially available as MaxEPA)[7]. After 12 weeks, these patients demonstrated favorable results with reduction in the number of tender joints and reduced morning stiffness as compared with control patients. Another study demonstrated a reduction in proinflammatory leukotrienes in diets enriched with fish-oil derived lipids.[8]

PGE3 Series: This series of fatty acids comes predominantly from colder, Northern climates in the form of seeds, nuts, lentils, and northern seafood. The omega-3 oils are generally more highly unsaturated than the omega-6 oils. Because of this, the omega-3's tend to have a lower freezing point so that they are better adapted to plants and animals in colder climates. Northern foods which provide significant sources of the omega-3's include wheat (in the wheat germ), flax seeds (source of linseed oil), navy beans as well as kidney, red and pinto beans, walnuts, chestnuts, and seafood such as salmon, cod, mackerel, pilchards, and sardines.

Even today, many nutritionists and physicians fail to recognize the critical importance of dietary omega-3 fatty acids, because all early experiments in fatty acids were done in rats, which do not require the omega-3's for normal development. It is now known that the omega-3's are the predominant fatty acids in the human brain, and that they are essential for normal brain development and normal immunological function in the human.

Some nutritional authorities today believe that omega-3 deficiencies contribute to many of the major mental and physical diseases in America today, including learning disabilities and behavioral disorders in children. There is every reason for believing this to be the case when one considers that dietary intake of omega-3 fatty acids in the U.S. has been reduced about eighty percent in the last century as a result of (1) milling (refining) of grains, (2) hydrogenation of oils, and (3) selective use of southern oils.

Current scientific interest and recognition of the omega-3 fatty acids and their necessity for human health began in the early 1970s with observations of the Greenland Eskimos, whose native diets, consisting largely of fish, seal, and whale, were very high in the omega-3's. Among the Eskimos, it was observed that such conditions as vascular disease, heart attacks, hypertension, obesity, rheumatoid arthritis, eczema, asthma, and diabetes were uncommon or even unknown. Ongoing research today indicates that the omega-3's lower blood cholesterol and triglycerides prevent blood clots, retard development or atherosclerosis, lower blood pressure, ease skin disorders such as eczema, aid in brain development, and relieve inflammatory conditions such as arthritis.

[References missing]