

The Rheumatoid Disease Foundation
Old Harding Road, Rt. 4, Box 137 Franklin, TN 37064
(615) 646-1030
(Now 7376 Walker Rd, Fairview, TN 37062; 615-799-1002)

The Rheumatoid Disease Foundation
Summary of Research to December 31, 1986

We believe that we have made significant progress during the past two years, and that we are verging on great news in every research direction. One more year of excellent research could well see the most significant progress of all.

My summary — of the research reports submitted by our outstanding professional scientists — does not necessarily reflect their interpretation and/or views. I am a lay person in these research fields, and therefore may have summarized our results incorrectly.

Whether or not you read my summary and whether or not you agree with my views therein, please read the research reports attached herewith. By going to the original source you will surely be obtaining the scientists' correct views of their own work.

I am wholly responsible for this summary.

The Rheumatoid Disease Foundation will appreciate your corrections and/or suggestions.

Cordially,

Perry A. Chapdelaine, Sr.

May 15, 2004 Memo:

Sad to say that we lost our fundraiser in the middle of excellent progress untangling the biochemical causation of rheumatoid disease. Suggestions made in this report have not, to our knowledge, ever been followed up.

Cordially,

Perry A. Chapdelaine, Sr.

The Rheumatoid Disease Foundation
by **PERRY A. CHAPDELAINÉ, SR.**
Executive Director/Secretary

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

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 sulpha drugs; 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 books¹ and numerous articles¹. This eminently acceptable medical physician and researcher became the genius who first developed an 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 results 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, named differently and not previously recognized as stemming from the same source. Of course, there are also other causes for the same symptoms.

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 protozoon 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 conference²⁷, where he received a standing ovation. No one followed up on his discoveries until Robert Bingham, M.D. reported the findings in a magazine²⁴; and Jack M. Blount, Jr., M.D. tried metronidazole (chemically related to then unavailable clotrimazole) on himself²⁴.

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 theory²: 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 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 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, 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 reports³, it apparently can also happen under appropriate nutritional regimes. The 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 body⁴. 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 against Candidiasis⁸) 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 injections⁵”, 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 studies⁶ 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 *Candidia albicans*⁸ that can present similar symptoms.

If there were a placebo affect (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 treatments⁹.

The Rheumatoid Disease Foundation prays that all patients will be among those who respond.

The Movement is Growing

I founded and chartered *The Rheumatoid Disease Foundation* 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.)

Eugene S. Wolcott, M.D. become Senior Vice-Chairman. Dr. Paul K. Pybus became the *Foundation's* Chief Medical Advisor. He had worked with Roger Wyburn-Mason as Roger's house physician (England) many years earlier, and held 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. Jack Blount after serving nearly four years as Chairman, became Chairman Emeritus. 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 physicians¹⁰ located in many different countries (but chiefly in 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, B.S. assisted by protozoologist Robert J. Neff, Ph.D. Tony Chapdelaine later became an M.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 17 different foreign countries, and hopefully there will be Chapters to serve localities with free-treatment for the indigent one day.

So What Have We Learned?

First and foremost, we believe 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 preceeded 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. 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!

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 protozoon theory of the causation of RD. After many nights of work with Stamm, back in the nineteen fifties, he was finally convinced by Stamm that a protozoon 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 insure that no foreign bacteria would go through the minced samples into their collection jar. By so using these antibiotics, he insured the creation of what has come to be known as Cell Wall Deficient (CWD) organisms¹².

To be aware of the fact that he had created Cell Wall Deficient organisms, Wyburn-Mason would have had to know about them, and the scientific field had not clearly defined them until 1974 by Domingue, Schlegel and Woody¹³.

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.

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 they tell clinicians whether or not patients suffer from 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 deficiency²³.

The study of Cell Wall Deficient organisms is not an unknown field, but simply an esoteric specialty that has yet to be integrated into routine 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 also 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 nuclei³⁰.

Kwang Jeon, Ph.D., concluded that no amoeba were present in knee effusion samples submitted from our referral physicians³¹.

Dr. Paul K. Pybus and A.H. Davies, Ph.D. at first thought they were viewing amoebae, but later concluded they viewed macrophages³².

Brian Susskind, Ph.D. also concluded through use of both synovium and other tumour samples, that only macrophages were present, not amoebae³³.

It is easy to understand, then, how it was that Roger Wyburn-Mason and Stamm — after repeatedly “viewing” protozoons in their cultures, after pursuing world literature on protozoons² 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 protozoons 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”, 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 much better than present rheumatology practices; i.e. it works 78% to 95% of the time, depending on patient group and physician practices⁹.
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 to Ca/Mg et. al. to RD.¹⁴

EDTA also inhibits PLA2¹⁴, thus explaining to some extent why EDTA chelation therapy gives temporary relief (not to mention the presumed cleaning up of “free radicals” generated during the inflammatory process¹⁵. — We would guess that DMSO, properly used, also temporarily cleans up presumed free-radi-

cals, but also contributes beneficially to the change in ratio of HDL to LDL²⁸⁾

4. Clotrimazole kills a very wide spectrum of protozoons in the test tube, as opposed to metronidazole and tinidazole¹⁶. Tinidazole and clotrimazole can be metabolized by either human enzymes or intestinal micro-flora; Metabolization of metronidazole relies solely on intestinal micro-flora¹⁷. This may explain why the first treatment of metronidazole may be effective, but not the second: during 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 again¹⁸. (We presume “good guys” micro-flora includes *Lactobacillus acidophilus* and *Lactobacillus bifidus*, but more research needs performed to be certain, or more information needs gathered.)

5. Clotrimazole kills *Candida albicans*¹⁹.

6. Clotrimazole stimulates cortisol.²⁰ (Perhaps a means of getting marginally deficient patients weaned away from external cortisone?)

7. Metronidazole kills over-active macrophages according to work by Dr. 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 macrophages¹¹.

9. Various nutritional substances affect disease state and 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 infection of the presumed *Amoeba chromatosa* under the same rules related to “weakening” of immunological system, and probably ought to be treated simultaneously, if suspect⁹.

11. There is a relationship between allergenic responses from various antigens and RD symptoms²². (See various treatments based on “allergenicly clean” clinics, pure water fasts, bio-detoxification programs 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 studies³⁴. 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 microorganisms 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 experiment, 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 at Medical College of Virginia. Franson and Susskind are cooperating in developing further Franson’s breakthrough’s 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-amoeba drugs such as Imidazole compounds cause the remission by 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 those not treated with drugs, and use animal models such as susceptible rats to examine the *in vivo* effect of Imidazole compounds on synovial cells.³¹”

Robert Neff, Ph.D. would suggest determining “the concentration of amoeba 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 amoeba 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 amoeba 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 immunosuppressant 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 protocols²³.”

5. Lida Mattman, Ph.D. of Wayne University suggests relating, if possible, knowledge of Cell Wall Deficient organisms to Rheumatoid Disease²³.

Reason: If you will look on pages 38 and 39 of *Rheumatoid Diseases Cured at Last*²⁴ (3rd ed.) 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 Postulate 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 a Cell Wall Deficient organism and, on entering human tissue, sets up the “genetic sensitivity” to the Cell Wall Deficient organism¹².

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’s²⁶, 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 protozoon 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 states²⁹.

6. And while it may be an insignificant point — I feel that no stone can 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 leucocyte. He made a photo of the leucocyte, stating that he had noted a strong correlation on viewing this kind of abnormal leucocyte in all RD victims. I asked that the photo be made available to our physicians and scientists for further possible correlations, and so it shall.

Therefore, as can be seen, *The Rheumatoid Disease Foundation* has 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, and thus benefit all.

The Rheumatoid Disease Foundation 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 suffer, as our understanding has grown.

Others Helped

If you are a patient taking antiamoebics, or about to be treated by 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, help found and 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 patient 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 Rheumatoid Disease Foundation*, 7376 Walker Road, Fairview, TN 37062 (formerly Rt. 4, Box 137, Franklin, TN 37064).

References

1. Wyburn-Mason, Roger. *The Causation of Rheumatoid Disease and Many Human Cancers — A New Concept in Medicine, A precis and Addenda*, *The Rheumatoid Disease Foundation*, Rt. 4, Box 137, Franklin, TN 37064, 1983.

2. Wyburn-Mason, Roger. *The Causation of Rheumatoid Disease and Many Human Cancers — A New Concept in Medicine*, Iji Publishing Co., Tokyo, Japan, 1978.

3. Numerous personal letters.

4. Pybus, Paul K. *Intraneural Therapy*, unpublished manuscript. Also see Haraldur Gudjonsson. "The Jarisch-Herxheimer Reaction" *Experimental Studies on Humans and Rabbits*, Stockholm, 1972.

5. Pybus, Paul K. (Paul Notrik, pseudonymously) *The Control of Pain in Arthritis of the Knee*, [based on Wyburn-Mason neural theory of 30 years earlier] *The Rheumatoid Disease Foundation*, Rt. 4, Box 137, Franklin, TN 37064, 1984.

6. See Bingham, M.D., Prosch, M.D., Dr. Pybus, Simoons, Ph.D.: "Imidazole Compounds for the Treatment of Rheumatoid Disease," presented at American Academy of Medical Preventics, November 16, 1985. Also see William Renforth, M.D. "Metronidazole Cures Rheumatoid Arthritis," *Historical Documents in Search of the Cure for Rheumatoid Disease*, both published by *The Rheumatoid Disease Foundation*, Rt. 4, Box 137, Franklin, TN 37064, 1985.

7. William Renforth, M.D. should be credited with independent research using metronidazole in the treatment of Rheumatoid Disease. See Item 6, above.

8. Crook,-William, M.D. *The Yeast Connection*, Professional Books, PO Box 3494, Jackson, TN 38301, 1986. C. Orian Truss, M. D. *The Missing Diagnosis*, The Missing Diagnosis, PO Box **26508**, Birmingham, AL 35226, 1982.

9. Klippel, John H., M. D., Decker, John L., M. D. Eds.: *Clinics in Rheumatic Diseases* Vol. 9/No.3, W.B. Saunders Company Ltd., December 1983.

10. See *The Rheumatoid Disease Foundation* Physician and Scientist Referral listing.

11. Personal Communication from Dr. Paul K. Pybus.

12. Personal Conversation with Lida Mattman, Ph.D., Wayne University, Detroit, MI and Phillip Hoekstra, III, Ph.D., Thermascan, Inc., 21519 Harper, St. Clair Shores, Detroit, MI, 48080.

13. Domingue, Gerald J. "Naked Bacteria in Human Blood", *Microbia*, Tome 2, No. 2, 1976.
14. Franson, Richard; Moseley. "Relation between Calcium Requirement, Substrate Charge, and Rabbit Polymorphonuclear Leukocyte Phospholipase A2 Activity, *Biochemistry*, American Chemical Society, 17, 4029, 1978. Franson, R., R. Dobrow, J. Weiss, P. Elsbach, W.B. Weglicki. "Isolation and Characterization of a phospholipase A2 from an inflammatory exudate," *The Journal of Lipid Research*, Vol. 19, No. 1, Jan. 1978. Franson, Richard C., Drove Eisen, Robert Jesse, Carmine Lanni. "Inhibition of Highly Purified Mammalian Phospholipases A2 by Non-Steroidal Anti-Inflammatory Agents," *Biochem. J.* 186, 633-636, 1980. Franson, R. J. Weiss, L. Martin, J.K. Spitznagel, P. Elsbach. "Phospholipase A Activity Associated with the Membranes of Human Polymorphonuclear Leucocytes," *Biochem. J.* 167, 839-841, 1977. Franson, Richard C. "Intracellular metabolism of ingested phospholipids, *North-Holland Biomedical Press, Knight (ed) Liposomes: From Physical Structure to Therapeutic Applications*, Chapter 12, pp. 349-380, 1981. And many other related papers with Franson.
15. Cranton, E. *Bypassing Bypass*, Stein and Day, 1984; also see *The Chelation Answer*, Morton and Walker, publisher unknown to author but available at most stores.
16. Chapdelaine, Tony. Unpublished Vanderbilt University research paper, available through *The Rheumatoid Disease Foundation*, 1985.
17. Personal Communication.
18. Personal Communication with various Rheumatoid Disease Victims.
19. Personal Communication from Dr. Paul K. Pybus.
Also see *Physicians Desk Reference*.
20. See Franson studies, Item 14, above.
21. Personal Communications: Dr. Paul K. Pybus, Kwang Jeon, Ph.D., University Tennessee, Dept. Protozoology.
22. Randolph, Theron G., Ralph W. Moss, Ph.D. *An Alternative Approach to Allegies*, Bantam Books, 1982. Also, for nutritional components, read most everybody, everywhere.
23. Mattman, Lida. *Cell Wall Deficient Organisms*, Chemical Rubber Company, 1976.
24. de Fabio, Anthony, *Rheumatoid Diseases Cured at Last*, *The Rheumatoid Disease Foundation*, Rt. 4, Box 137, Franklin, TN 37064, 3rd ed., pp. 38-39, 1985.
25. Personal Communication with Tony Chapdelaine.
26. Brown, Thomas, M.D. "The Cure and the Controversy," *The Washington Post Sunday* April 22, 1984.
27. Wyburn-Mason, Joan. *Dedication, Love and Humour*, *The Rheumatoid Disease Foundation*, Rt. 4, Box 137, Franklin, TN 37064, 1985.
28. Personal trials and lab tests.
29. Personal communications, Tony Chapdelaine.
30. Neff, Robert J., Asbell-Gillespie, Deborah, Chapdelaine, Tony, "Isolation and cultivation of soil amoebae from fluids and tissues of patients with rheumatoid disease," Unpublished final report on a research grant from *The Rheumatoid Disease Foundation*, November 8, 1986.
31. Jeon, Kwang, Ph.D. "Effect of anti-amoeba drugs on synovial cells of arthritic joints," Unpublished research proposal, March 5, 1985, together with personal conversations and letters.
32. Davies, Dr. A.H., Paul K. Pybus. Unpublished paper (untitled) together with personal letters and conversations, 1985-86.
33. Susskind, Brian, Ph.D. Unpublished final report on a research grant from *The Rheumatoid Disease Foundation* and research proposal, letter November 6, 1986.
34. Neff, Robert J., Chapdelaine, Tony, "Preliminary report on drug research involving *Acanthamoeba* and *Naegleria*," Unpublished final report on a research grant from *The Rheumatoid Disease Foundation*, July 14, 1984.

The Rheumatoid Disease Foundation is a project of *The Roger Wyburn-Mason & Jack M. Blount Foundation for Eradication of Rheumatoid Disease, Inc.*, a Tennessee chartered, non-profit, charitable, IRS tax-exempt organization.