

Bacterial Infection as the Cause of Scleroderma

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Date: 03-02-06 19:33

Bacterial Infection as the Cause of Scleroderma: A Guide to Antibiotic Therapy

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Paper Type: Review

Please cite as: Cantwell AR Jr, Ganger P: Bacterial Infection as the Cause of Scleroderma: A Guide to Antibiotic Therapy. JOIMR 2006;4(2):1

Published: 2 March 2006

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Abstract

Scleroderma is a disease characterized by thickening and hardening of the skin. While most traditional and established treatments provide temporary improvement at best, remission is rare — and cure impossible. Furthermore, the various medications commonly prescribed have serious side effects. There is urgent need for better treatment.

Scleroderma is erroneously considered an incurable disease of unknown cause. This communication provides evidence implicating infectious bacteria as the cause of scleroderma; and proposes antibiotic therapy as a rational and beneficial treatment for this disease.

Background

Although little known, several researcher-physicians have discovered bacteria in scleroderma. Although these microbes have been reported under different names (acid-fast mycobacteria, mycoplasma, cell-wall deficient bacteria), they all have similar characteristics. They are "pleomorphic", meaning the bacteria have the ability to change their shape and size; and they are difficult to detect and eradicate in body tissues and cells.

Over the last few decades some researchers have shown that Tetracycline drugs are effective against these microbes. An Internet search at the National Library of Medicine's

PubMed website (www.ncbi.nlm.nih.gov), using key words — acid-fast bacteria AND scleroderma — reveals 11 citations associating bacteria with this disease. A **Google.com** search of — acid-fast bacteria + scleroderma — lists dozens of websites discussing bacteria in scleroderma. An Internet search of — scleroderma + antibiotic therapy — refers to various websites (such as www.roadback.org; www.rheumatic.org; and others) providing testimonials from scleroderma patients who have successfully undertaken antibiotic treatment.

Unlike the usual prescribed drugs for scleroderma, tetracyclines are safe and effective, and scleroderma bacteria do not develop resistance to the antibiotic. However, prolonged antibiotic therapy is necessary.

In 1998, the medical journal *The Lancet* published the results of a one-year study, headed by Harvard rheumatologist David E. Trentham testing the tetracycline Minocin (minocycline) on scleroderma patients [1]. Improvement in skin scores (a measurement of hard skin caused by the disease) was noted in all patients, with no serious side effects. Although subsequent studies have not all indicated such a remarkable achievement, more clinical studies using prolonged antibiotic therapy are sorely needed.

Systemic progressive scleroderma is a potentially fatal disease recognized for over 200 years. Scleroderma is considered incurable, but there are thousands of patients who have been helped with antibiotics.

Why hasn't your doctor told you about antibiotic therapy? Bacteria in scleroderma are little-known, and physicians traditionally are slow to accept new ideas that challenge long held medical dogma. And physicians are loathe to prescribe antibiotics, especially in scleroderma, which is widely regarded as non-bacterial in origin.

This report presents evidence for an infectious bacterial agent in scleroderma and a rationale for the use of antibiotic therapy.

The Discovery of the Scleroderma Microbe in 1947

Unusual acid-fast bacteria in scleroderma were first discovered in 1947 by Virginia Wuerthele-Caspe Livingston, M.D. [2]. In a follow-up publication Livingston, along with pathologist Camille Mermod and dermatologist Eva Brodtkin, reported on similar bacteria in the skin of five additional scleroderma patients [3].

Livingston's bacteria were "acid-fast," indicating that they stained in a way similar to the so-called mycobacteria — the bacteria that cause tuberculosis and leprosy (Hansen's disease). In laboratory culture the scleroderma microbe was highly "pleomorphic," meaning the germ appeared in more than one form. Some forms were the size of viruses and invisible microscopically; other forms resembled conventionally-sized bacteria and larger forms resembling spores of fungi and yeasts. In addition, there was evidence that scleroderma bacteria in tissue had lost their cell-wall, thus appearing as pleomorphic cell-

wall-deficient microbes. Such wall-deficient bacteria are similar to what microbiologists also call "mycoplasma" or "L-forms." Livingston's "sclerobacillus" was unlike any microbe previously studied; and it was impossible to classify because of its diversity.

In various experiments Livingston and her colleagues injected these pleomorphic bacteria into chicks and guinea pigs. The chicks died. Some of the guinea pigs developed hardening of the skin like scleroderma, and some developed cancer.

Scleroderma is not cancer, but Livingston subsequently discovered similar acid-fast bacteria in various cancer tumors. This research was published in 1950, in the *American Journal of Medical Sciences* in a landmark paper entitled "Cultural properties and pathogenicity of certain microorganisms obtained from various proliferative and neoplastic diseases." The report included a photo of the virus-like form of the microbe magnified 31,000 times, as well as a classic description of the scleroderma microbe [4].

Until her death in 1990 at the age of 83, Livingston claimed that bacteria were the cause of scleroderma and cancer, much to the dismay of the medical establishment. Her struggle against established scientific dogma is recorded in her autobiographical books *Cancer: A New Breakthrough* (1972) and *The Conquest of Cancer* (1984) [5-6]. These books are out-of-print, but are available through Internet used book sources. Further details of Livingston's scleroderma research are recorded in Alan Cantwell's *The Cancer Microbe* (1990) and *Four Women Against Cancer* (2005) [7-8].

Confirmation of the Scleroderma Microbe by Other Researchers

Livingston's discovery of bacteria in scleroderma almost 60 years ago was confirmed in 1953 by researchers at the Pasteur Institute in Brussels, who found the bacteria in nine additional cases [9]. In 1966 Cantwell detected acid-fast bacteria in the tissue sections of a patient with scleroderma [10].

Scleroderma can appear either as a localized or a generalized disease. Over the next two decades Cantwell et al. reported additional cases of acid-fast bacteria and scleroderma, including cases of morphea (localized scleroderma), nodular scleroderma, "pseudoscleroderma," and a fatal autopsied case of progressive systemic scleroderma, in which bacteria were observed in the internal organs and throughout the connective tissue [11-17].

Livingston stressed that the "sclerobacillus" had a particular affinity to connective tissue and to collagen. Scleroderma, along with rheumatoid arthritis and lupus erythematosus, are considered "connective tissue diseases" and "collagen diseases," and diseases of autoimmunity.

Cantwell et al. have reported pleomorphic bacteria in lupus, similar to bacteria found in scleroderma [18-19]; and various investigators have reported mycoplasma infection in

patients with rheumatoid arthritis [20-22].

Research linking mycoplasma to rheumatoid arthritis and scleroderma is simply explained in *The Road Back*, by Thomas McPherson Brown, MD,[23]; and in *Why Arthritis?: Searching for the Cause and the Cure of Rheumatoid Disease*, [24] by Harold W Clark (a microbiologist who worked with Dr. Brown for 35 years). Medical writer Henry Scammell has also authored two books, *The New Arthritis Breakthrough*, and *Scleroderma: The Proven Therapy That Can Save Your Life*, based on Brown's research and the antibiotic therapy he advocated for rheumatoid diseases [25-26].

The Microbiology of Scleroderma

The microbiology of scleroderma is complex. Livingston and her associates claimed the pleomorphic microbe had a "life cycle" including a submicroscopic virus-like phase, undoubtedly related to recently described "nanobacteria." In tissue, in culture, and in the blood these small bacterial forms resemble cell-wall deficient bacteria (mycoplasma and L-forms) [27-28]. Larger growth forms of the bacterium resemble fungus-like spore forms. The largest forms appear as "large body" and "ghost forms" of cell-wall-deficient bacteria. The largest forms in tissue microscopic sections are similar to "Russell bodies."

In the 1890s Scottish pathologist William Russell described variably-sized round pleomorphic fungus-like spore forms in cancer and tuberculosis, which he believed represented "the characteristic organism of cancer." These large forms, now widely recognized and interpreted by pathologists as non-microbial "Russell bodies," have similar characteristics to "large body" forms of cell wall deficient bacteria. "Large bodies" are similar to what Livingston described as fungus-like "globoidal forms" of the scleroderma microbe.

"Large bodies" can be sometimes be observed in tissue microscopic sections of scleroderma [29] and lupus erythematosus, as reported and illustrated by Cantwell, in Gerald Domingue's (editor) *Cell Wall-Deficient Bacteria* on page 326 and 336 [30]. More information on William Russell and Russell bodies can be found on the Internet by [Googling](#) key words: Russell body + Cantwell.

The Recent Discovery of Bacteria in Stomach Ulcers:

Implications for Scleroderma Research

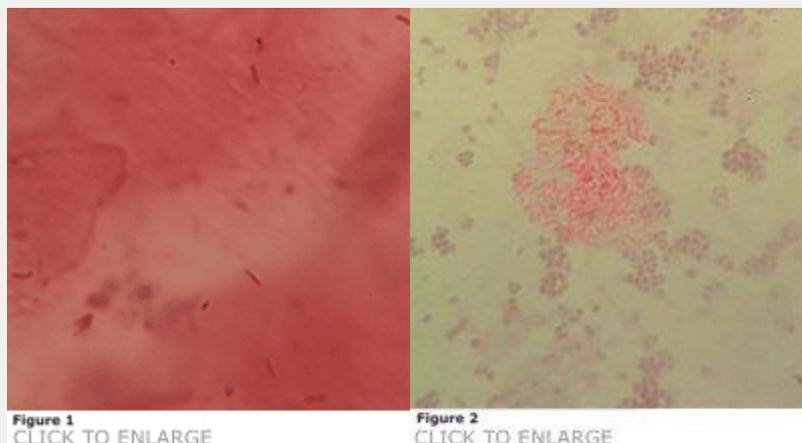
In 2005 the **Nobel Prize** in Medicine was awarded to two Australian researchers who discovered in the 1980s that most stomach ulcers were not due to diet, spicy food, alcohol, stress, or psychiatric disorders — but rather to stomach bacteria. The idea of bacteria causing stomach ulcers was previously considered heresy because doctors were convinced that bacteria could not possibly survive in the acid environment of the stomach. Now it is accepted that *Helicobacter pylori*, a microbe found in both normal and in ulcerated stomachs, is the cause of most stomach ulcers. An effective antibiotic treatment for ulcers has been devised. What was essential in the discovery of stomach Helicobacter was the use of a special tissue stain to properly identify these bacteria in ulcer tissue.

To further illustrate how one physician can be correct — and the rest of his colleagues wrong — is the case of A. Stone Freedberg, a 96-year-old American pathologist who found similar bacteria in stomach ulcers in 1940, years before the Australians. Unfortunately, he was discouraged from continuing his research by doubting colleagues; and also by subsequent studies that supposedly proved that such an infection was not possible. Thus, Freedberg's discovery of ulcer bacteria was ignored for decades, much to the detriment of ulcer sufferers.

The Microscopic Appearance of Bacteria in Scleroderma

In searching for bacteria in scleroderma it is necessary to use a special "acid-fast" stain when coloring the tissue biopsy specimens for microscopic examination. This acid-fast stain is a traditional stain used to detect tuberculosis-type and leprosy microbes, also known as mycobacteria. The "routine" hematoxylin-eosin stain used by pathologists does not show these microbes clearly. Livingston believed the scleroderma microbe was closely related to the TB-causing mycobacteria; and she emphasized that the acid-fast stain was the key to identify these microbes in tissue and in laboratory culture.

By use of the acid-fast stain and the highest magnification (oil-immersion at a power of 1000X) of the light microscope, one can detect in scleroderma tissue the tiny, round, pleomorphic, variably acid-fast coccoid forms, which are the most common forms of the microbes found within the cell (intracellular) and outside the cell (extracellular). The acid-fast, red-staining rod (stick-like) forms characteristic of TB bacteria have been demonstrated in scleroderma, but they are extremely difficult to demonstrate because of their rarity. Figure 1 shows rare acid-fast rods in the skin of a fatal case of systemic scleroderma [10, 11]. Figure 2 shows the appearance in culture of a pleomorphic unidentified microbe (acid-fast rods and non-acid-fast round cocci) cultured from the patient's hardened skin. Figure 3 shows large clear yeast and fungus-like "ghost-like" forms, and large solid-staining forms in the fatty portion of the skin from the same patient. Such large pleomorphic forms resemble the appearance of certain growth stages of cell-wall-deficient and "L-forms" of bacteria, and forms known to microbiologists as "large bodies."



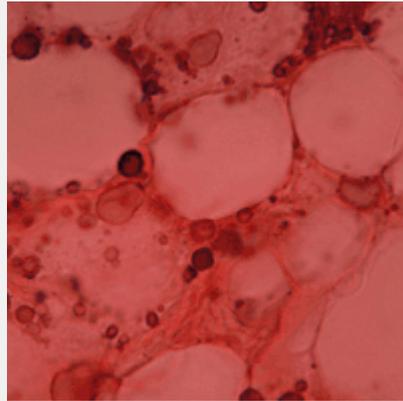


Figure 3
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Figure 4 shows coccoid forms in another fatal scleroderma case, and Figure 5 shows the pleomorphic cocco-bacillus cultured from the skin. Similar coccoid forms were seen throughout the connective tissue and in some of the internal organs at autopsy of the patient (Fig 6). During life a positive identification of the microbe was not possible. However, cultures of the skin at autopsy grew *Mycobacterium fortuitum*, an acid-fast "atypical" non-tuberculous species of mycobacteria [14].

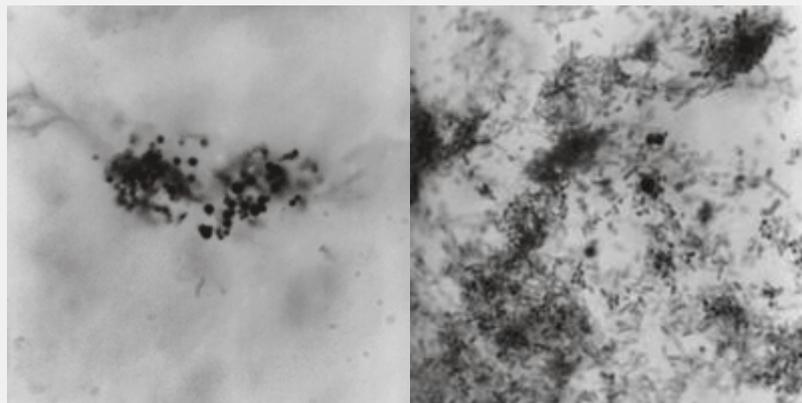


Figure 4
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Figure 5
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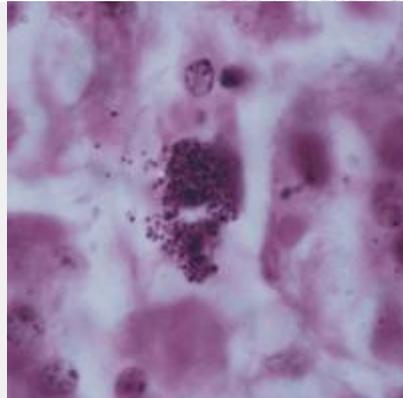
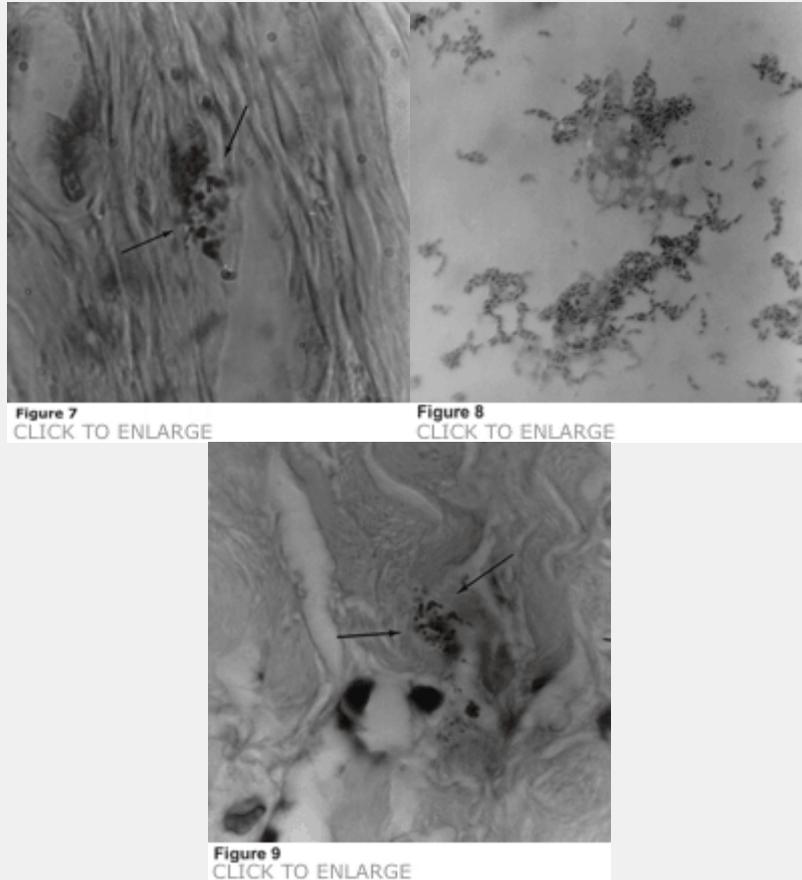


Figure 6
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Figure 7 illustrates coccoid forms in the skin of a rare case of nodular scleroderma, and Figure 8 shows the pleomorphic bacteria cultured from the skin [15].

Figure 9 shows coccoid forms in the skin of localized scleroderma, also known as morphea. Further details of these cases are recorded in the literature. [10-11, 14-16].



Despite various reports in peer-reviewed medical journals, the discovery of bacteria in scleroderma remains largely unknown and ignored by most physicians and scleroderma researchers.

Treating Scleroderma with Long-Term Antibiotics

The treatment currently recommended for chronic progressive systemic scleroderma is largely ineffective. For a disease to be treated in the best possible way, it is imperative that the cause be known. That is why the wider recognition of a bacterial agent in scleroderma and antibiotic therapy against these infectious agents is so important. Tetracyclines inhibit bacterial protein synthesis, and have anti-inflammatory and other actions [31]. Minocycline has recently been shown helpful in lessening calcification in scleroderma [32].

The discovery of acid-fast bacteria in scleroderma also calls into question therapy with steroids (prednisone, cortisone) and other immunosuppressive therapies that are usually contraindicated in the presence of known infection with acid-fast mycobacteria. For example, treating tuberculosis with steroids only worsens the disease.

In a complex and chronic disease such as scleroderma, multiple drug therapy will probably be necessary (as is the case in the treatment of TB) — and more than one antibacterial agent might eventually prove more beneficial. It is also likely that new antibiotics (and possibly a vaccine) will need to be developed to combat the pleomorphic bacteria in scleroderma.

At present, it is helpful to note the experiences of scleroderma patients who have been benefited by antibiotic therapy. One such patient is co-author Pat Ganger.

Pat Ganger's Successful Battle with Systemic Scleroderma

I first developed symptoms of systemic scleroderma in December 1982, when I experienced persistent tingling in my hands. Shortly afterward, my lower legs became swollen.

Initial blood work showed a sedimentation rate of 16 (indicating inflammation) and an ANA (anti-nuclear antibody) titre of 1:200, with a speckled pattern "consistent with scleroderma." An electromyogram showed possible myopathy (muscle disease). My reflexes were normal; and after additional testing, the neurologist assured me my problem was not neurologic. A rheumatologist said my pulmonary function test was "not bad, considering," but I was never shown a copy of the results. I was also underwent a veinogram, but I was not allowed to see the results of that test either.

As my symptoms worsened with hardening of the skin in many areas, I consulted with two dermatologists, one of whom suspected scleroderma. An internist claimed my illness was "rheumatic," but he was undecided as to which exact disease it was. Finally, a skin biopsy of hardened skin on my chest revealed microscopic changes typical of scleroderma. I was diagnosed with progressive systemic scleroderma in September 1983, but I had no idea what scleroderma was.

A rheumatologist tried numerous medications: steroids, colchicine, diuretics, Motrin, Feldene, and eventually d-penicillamine, a popular scleroderma treatment at that time. Methotrexate, sometimes used as a cancer treatment, was not used for rheumatic disease until years later.

Despite this therapy, I continued to decline. I developed weakness, joint pain, and curled fingers covered with painful ulcers. Sleeping was painful as my shoulders, hips and knees were pressure points, and I began to have gastrointestinal reflux when I lay flat. Four pillows and padding under the sheets did little to help, and I could sleep only about 20 minutes at a time before waking in pain and having to change position.

My skin continued to harden, and swallowing problems escalated. The skin of my face tightened, especially around the mouth, which made eating difficult. I no longer recognized myself in the mirror. Dressing was difficult. I was unable to reach behind my back to tuck in a blouse or reach my feet to put on shoes and socks. It was increasingly difficult for the technician to draw blood due to hardened skin on both arms, thickened blood vessel walls, and scarring of the popular puncture places. At this time a chest x-ray revealed scarring consistent with scleroderma, although I was not yet short of breath.

My disease had become life-threatening with internal organ involvement, and I experienced a steady decline over the next several years. My rheumatologist explained this was the normal progression for scleroderma. He told me if I became one of the rare, lucky ones I might go into remission, but if I did, I would not get better, just stop getting worse. I was resigned to this poor prognosis, but my husband was not. He encouraged me to read, learn, and fight, but I was just too tired to deal with anything but my disease.

In 1985 I started a local scleroderma support group and began to educate myself about my illness. Through one of the women in my group I learned about Thomas McPherson Brown, MD, and his use of tetracycline antibiotics to treat rheumatoid disease. I knew I would die if I stayed on my present course, so feeling I had nothing to lose, I went from my home in Ohio to Arlington, Virginia, and began antibiotic therapy at his clinic with a five day course of intravenous clindamycin.

What a shock! I experienced an immediate improvement. The pain, hard skin, ulcers, fatigue, tingling, swallowing difficulties and reflux, all slowly disappeared. Within four years of antibiotic therapy, I was in remission with all my symptoms reversed, except my curled fingers.

A letter from The Cleveland Clinic in January 1995, states that other than the finger contractures, "there was essentially no other evidence of previous scleroderma" and that I was in remission. However, there was old evidence of scarring in my lungs.

A miracle? No. A fluke? No. Based on years of research, Dr. Brown, a rheumatologist and a microbiologist, believed scleroderma and other closely-related rheumatoid diseases were caused by a tiny bacterium called a mycoplasma [20,23]. Tetracycline, the antibiotic he chose to treat rheumatoid patients, was the safest and most effective available. Some 40 years and tens of thousands of patients later, his treatment is still being used to give patients back their quality of life, but many people don't know about it.

Although I never met Dr. Brown (he was ill with bone cancer when I began treatment at his clinic, and died several months later) I feel I know him through his patients who shared their experiences with me. They all loved him because he promised to help them, and he delivered on that promise.

Various researchers continue to implicate mycoplasma forms of bacteria in arthritis, as reviewed by Schaefer et al. and Johnson et al. [33-34]. On the [PubMed](#) website there are 248 citations when keywords "mycoplasma AND rheumatoid arthritis" are typed-in, but only a handful of references to "mycoplasma and scleroderma." My blood tests for the presence of mycoplasma were positive for both *Mycoplasma pneumoniae* and *Mycoplasma salvarium*, so I continued antibiotic therapy with oral tetracycline. Later, I continued with Minocin (the brand name of minocycline), as well as clindamycin intravenously, once a month, for the first four years.

Once I achieved remission and a reversal of most of my symptoms, I continued with just the oral antibiotic. It is 23 years since my diagnosis. I remain on oral Minocin and am healthy, active and most grateful to Dr. Brown for his pioneering work in devising an antibiotic therapy, based on his research of an infectious agent in scleroderma.

Without Dr. Brown's treatment, many patients would have become permanently crippled or dead, myself included. My rheumatologist expected this to happen. When I returned to show him how well I had done on antibiotic therapy that he did not and could not recommend, he refused to make eye contact with me. As he stared at my file, he merely said, "I don't think the treatment will hurt you."

In the early 1990's I was astonished to discover an old article in a scleroderma newsletter from 1985 by a California dermatologist claiming a microbial cause of scleroderma! His research was along the same lines as Dr. Brown's, and although they were unaware of each other's research, both had ascertained that scleroderma was caused by a microbe. Both physicians published their research in reputable and peer-reviewed medical journals. Just a few years later *The Lancet* would publish the one-year study showing improvement of scleroderma with antibiotic therapy [1].

The more I recovered, the more I wanted to make this information available to patients who might benefit from it. In the early 1990's I met Carol Lange, who had been a rheumatoid arthritis patient of Dr. Brown's for 24 years. We became good friends, and with several others, we started an organization called The Road Back Foundation to disseminate that information.

Our goal was to undertake extensive study of the published research to help us understand our disease. For 10 years we read hundreds of books and scientific papers and communicated with many patients, sharing what we learned in the Foundation's newsletter published from 1993 until 2000.

Little by little, our understanding grew as evidence in the medical literature accumulated for a microbial cause and antibiotic treatment for scleroderma, rheumatoid arthritis, and other rheumatic diseases. We also learned of many scleroderma and rheumatoid arthritis patients who were personally helped by it.

When Carol and I left The Road Back Foundation in March of 2000, we discussed what remained to be done. Henry Scammell's and Harold Clark's books are good introductions to antibiotic therapy, but they leave some unanswered questions for both patients and physicians. We decided to write a handbook for physicians and patients that would contain all the information we had gleaned over our decade of research. Filled with references to

hundreds of scientific papers, it explains the microbe in scleroderma and other rheumatoid diseases, how the various disease symptoms develop, and how to begin antibiotic therapy under a doctor's supervision. Readers say it is well-written and easy to understand. The book, *Solving the Puzzling Problem of Arthritis*, is available on [Amazon.com](https://www.amazon.com) [33].

Final Thoughts on Antibiotic Therapy for Scleroderma

Progressive systemic scleroderma is a life-threatening disease. A 1995 Canadian study by Abu-Shakra and Lee showed 61% of systemic scleroderma patients died within 9 years [34].

Although more studies are urgently needed, recent reports attest to the efficacy of tetracyclines in rheumatoid arthritis and scleroderma. With the many daily advancements in medicine, we remain puzzled as to why antibiotic treatment for these diseases remains largely unknown or ignored, despite solid research supporting these findings [35-36].

Yet another interesting and provocative report is a recent study attempting to treat scleroderma with immunotherapy in the form of a heat-killed vaccine derived from delipidated and deglycolipidated *Mycobacterium vaccae*, a non-tuberculous species of mycobacteria [37]. This experimental vaccine has been used to prevent tuberculosis and leprosy in the Philippines, and also to treat various forms of cancer. This new research deserves to be evaluated in light of Virginia Livingston's highly controversial belief that acid-fast TB-like mycobacteria are implicated in the etiology of scleroderma and cancer.

It is endlessly and erroneously repeated that the cause of scleroderma is unknown, and that there is no evidence to support a bacterial etiology. As a result, physicians cling to the belief that the body is attacking itself through "autoimmune" mechanisms; and doctors persist in advocating conventional therapies for systemic scleroderma, which are widely regarded as ineffective.

We believe the body is not attacking itself, but is desperately trying to rid itself of disease-producing microbes that are not recognized by the scientific community.

Antibiotic therapy with tetracyclines has helped many patients with scleroderma and rheumatoid arthritis. Patient postings at <http://rheumatic.org/medhist.htm> and other websites confirm this. Hopefully, this communication will provide information and encouragement to patients considering antibiotic therapy, based on solid research showing a microbe in scleroderma.

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SCLERODERMA ANTIBIOTIC TREATMENT WEB SITES

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