®

**Free Living Amoeba & The Effects of Anti-amoebic Drugs on Rheumatoid Disease**

The Cause of Rheumatoid and Autoimmune Diseases?

by ROGER WYBURN-MASON
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**Historical Note**

*A letter from Professor Roger Wyburn-Mason*
*(Formerly published in* The Journal of the Rheumatoid Disease Foundation, *Volume 1, Number 1)*

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Rheumatoid Disease is a generalized condition, not just one of joints and muscle spasm. The occurrence of rheumatoid granulomatous nodules subcutaneously, at the sites of pressure or even on the meninges or sclera or of rheumatoid lung, heart, liver, and kidney lesions or of involvement of the parotid and lacrimal glands and skin lesions can only be found in a systemic pathology.

The autonomic neurogenic cause of the disease was exploded many years ago by the fact that complete sympathectomy was repeatedly found to have no effect on the disease. This is not a "one-of" finding, but has been repeatedly confirmed. The nervous system may be involved in producing the inflammatory changes in rheumatoid disease.

Years ago I showed that inflammation in a tissue is dependent on the integrity of the unmyelinated C fibers of the posterior nerve roots and mixed peripheral nerves. If these are destroyed, as in gunshot wounds, leprosy, tabes or syringomyelia then injury to the part normally supplied by these nerve fibers results not in inflammation but in necrosis. While in the condition of causalagia resulting from injury to the median or sciatic nerves, mild trauma in the painful area may result in an exaggerated inflammatory response as compared with that in normal tissues of the patient. Inflammation depends on antidromic impulses passing down from the spinal cord to the inflamed area through these special nerve fibers, in the case of rheumatoid arthritis, to the region of the joints.

Such cases as the following:

All were fit middle-aged men, ploughing the dry fields on windy days — one in the Middle West of USA, one in Ontario and one in Rhodesia. During the ploughing, there was a great deal of dust being blown about from the dry surface soil and this was inhaled by the subjects. During the next night, all three were wakened by drenching night sweats, general malaise and next morning were found to have temperatures of 105° F. Every joint in the body was painful, swollen and immobilized even including the cryoarytenoids and temporo-mandibular. They had a cough, sputum, severe headache and muscular aching. All were admitted to the hospital and eventually found to be suffering from acute rheumatoid disease. In spite of intensive treatment, their symptoms only very gradually diminished over the next 3-4 months, but they were left at this time with severe pain and swelling of the joints which did not respond to any treatment over the next year or more. These cases are typical of severe infection and ***NOT*** of a disturbance of the autonomic nervous system. The origin of their infection would seem to lie in something inhaled from the copious surface soil dust (which contains free living amoeba).

I isolated free-living amoebae from all the body tissues in cases of active rheumatoid disease, cultured them from the laboratory, found that antiamoebic substances killed them and then treated cases of active rheumatoid disease with various antiamoebic substances.

Incidentally, the eminent protozoologists Kofoid and Swezy, working in their laboratory at the University of California (LA) in 1922 found the same organism in the bone marrow of cases of rheumatoid arthritis and suggested its aetiological relationship to the disease some 40 years before my work. They reported this in a zoological journal which never reached the medical profession.

Furthermore, any substance which in vitro kills the organism when given to active cases of rheumatoid arthritis often produces a transient Herxheimer reaction, that is an exaggeration of the inflammatory changes of rheumatoid arthritis and often the appearance of lesions in previously unaffected tissues, just like mercury exaggerated the symptoms of syphilis. When given to healthy subjects, these antiamoebic drugs have no such effect, and Herxheimer reactions do not occur when antibiotics or antiviral substances are used against sufferers from bacterial or virus diseases. This observation alone shows the presence in rheumatoid arthritic lesions of an organism more complex than a bacterium, namely an amoeba. This is the complete proof of the amoebic causation of rheumatoid arthritis. [Subsequent studies were unable to confirm this amoeba theory, but strongly suggested that the treatment worked to normalize or stop auto-immune activity of macrophages which happened to resemble amoeba. Thomas McPherson Brown, M.D. felt that he'd isolated mycoplasm from arthritic joints. Historical research clues lead to a conclusion that perhaps many different organisms can stimulate a tissue sensitivity to themselves, thus creating arthritic symptoms. Ed.]

Local anesthetics have two effects — they are anti-protozoal and also paralyze the unmyelinated C fibers, the discharge of which is responsible for inflammation. Both these effects could explain some of the benefits from procaine therapy in rheumatoid disease.

**Free-Living Amoeba**

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Numerous species of the universally-found free-living amoeba

are known. Most fall into two genera — *Acanthamoeba* and *Naegleria* — and some are pathogenic to man and animals. In inimical conditions, they form hollow spherical cysts, which are present in the air in most parts of the world and can readily be found on agar plates exposed to air. Free-living amoeba prefer warm surroundings and tend to migrate to an environment at body temperature (thermotropism).

All living beings are surrounded by many species of these free-living amoeba which pass into mammalian respiratory passages as cysts or trophozoites. These must also be present as trophozoites in the gastrointestinal tract of many animals, including man, since they are found in their feces. These organisms are motile and, once they enter an orifice, they migrate under thermotropic influences into body tissues.[20]

Recently it has been shown[3] that the blood contains antibodies against *Acanthamoebae* and *Naegleria*, indicating universal infection of man and the newborn with these organisms. Textbooks on protozoology state that unspecified types of amoebae have been isolated from every tissue in the body[6]; there is hardly an organ in the body from which somebody has not obtained amoebae.[2] Lesions due to species of such organisms have been described in a few cases of plants and man, namely amoebic meningo-encephalitis.[1,16]

In 1922, the eminent protozoologists, Kofoid and Swezy, reported the presence of free-living amoeba in the bone marrow of rheumatoid arthritis patients without dysentery or *E. histolytica* in the feces. These organisms were distinguished from human cells by mitotic processes and contained only 6 chromosomes rather than the normal 46 of human cells. They showed a single blunted pseudopodium and numerous vaculous, suggesting a causal relationship between infection and the arthritic process.[5, 9-14]

### Anti-amoebic Substances

Recently I have shown[19, 21-23] that free-living pathogenic or non-pathogenic amoebae can be made to migrate out of human tissue, including those of the newborn and fetus, by using the property of thermotropism. Large numbers are found in the affected tissues of all patients with rheumatoid disease, in extra-articular tissues affected by autoimmune disease, in normal feces, in uncooked butcher's meat, and in surface soil. Smaller numbers may be recovered from some tissues of apparently healthy humans, when they are presumably of non-pathogenic nature; they appear identical with those found by Kofoid and Swezy. These findings have been confirmed in laboratories in various parts of the world.

In the laboratory, these organisms can be cultured in "amoeba saline," into which a culture of *E. coli* has been introduced. Various antiamoebic substances found effective in killing the organisms when added to the cultured cells include bile salts (1% solution), 4-aminoquinolines, copper sulfate (very dilute solutions), metallic copper, gold salts, emetine, dehydroemetine, pentamidine, and levamisole (which contains an imidazole group). Particularly effective are the 5-nitroimidazole group of drugs — including metronidazole, tinidazole, ornidazole and nimorazole — which possess a wide spectrum of antiprotozoal as well as antiamoebic activity.

Since the organisms are not numerous and look like macrophages or lymphocytes, they can be recovered in spite of the fact that they are not usually observed in affected tissues stained by ordinary methods. This is a feature repeatedly observed in laboratory animals experimentally infected with free-living amoebae and recalls the situation with syphilitic lesions before stains for *Treponema pallidum* were discovered. The organisms in rheumatoid arthritis, however, can be demonstrated in tissue sections by immunofluorescent staining using sera containing appropriate antibodies to the organisms and by studying their mitosis and chromosome content in marrow biopsy material.

### The Effect of Anti-amoebic Drugs on Active Rheumatoid Disease

Rheumatoid disease is not limited to joints but may involve any tissue of the body. The same histological changes in joint capsules are found in extra-articular lesions and consist of lymphocytic infiltration, formation of germinal follicles, and often plasmocytosis accompanied by arteritis, arteriolitis, or endarteritis. Many of the extra-articular lesions constitute so-called auto-immune diseases but also include Sjogren's Syndrome, bone marrow infiltrations, thymic lesions, and granulomatous nodules. Symptoms typical of an infection — fever up to 40° C, sweating and raised ESR — may be present. Any of the extra-articular or autoimmune lesions may occur in any combination with or without arthropathy.

I have shown that any substance which kills the free-living amoebae *in vitro*, when administered to cases of active rheumatoid disease, may cause a rapid disappearance of the inflammatory changes around the joints and elsewhere in the body. Complete cure is obtained in early cases. But more commonly these drugs may induce a transient exaggeration of the inflammatory changes around the joints and elsewhere; and often, inflammatory lesions in a part of the body not previously affected will appear. This may be accompanied by influenzal symptoms, sweating, pyrexia, lymphadenopathy rise in ESR, and eosinophilia. This reaction is also seen in cases of active rheumatoid disease treated with gold salts[18] and levamisole.[15] Various countries — notably the U.K., United States, Holland, and New Zealand — have confirmed this reaction in cases of active rheumatoid disease treated with anti-protozoal substances.

### The Herxheimer Reaction

This phenomenon, first described by Herxheimer[8] in cases of syphilis treated with mercury, also occurs in diseases due to organisms more complex than bacteria when drugs that kill the causative organism in the tissues are administered. This "Herxheimer reaction" is due to the liberation of irritant and antigenic substances from the dying organisms and is not observed in healthy persons or in rheumatoid patients given antibiotics. Its occurrence in rheumatoid disease (including those of autoimmune lesions) treated with various antiamoebic drugs proves that a causative pathogenic amoeba is present in the affected tissues. [Since anti-amoebic medication is also known to kill friendly bacteria, increased accumulations of acetaldehyde byproducts from population explosions of *Candida albicans* could also account for what was thought to be a Herxheimer reaction. Ed.] After administration of antiamoebic drugs (especially 5-nitroimidazoles), evidence of rheumatoid disease activity usually completely disappears in both joints and extra-articular tissues within 3 to 6 months. Therefore, autoimmune lymphocytic and humoral reactions are not the primary disturbance in rheumatoid and autoimmune diseases; they are the cellular-antibody response to infection and its antigens and contribute to the tissue damage.

The whole syndrome resembles syphilis. Indeed, Waldenstrom and others[17] 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 damage, and the vasculitis are characteristic features."

Rheumatoid disease also closely resembles the rheumatic manifestations in leprosy.[4] This disease may present with acute arthritis affecting one or a number of joints, polymyositis, skin lesions, fever, raised ESR, and other signs; there will also be an increase in circulating gammaglobulins and positive serological tests for autoantibodies RF and ANF, as in rheumatoid disease. This is an immune complex syndrome with antigens provided by disintegrating *M. Leprae*. The reaction known as Lucio's phenomenon, which is identical to the Herxheimer reaction, may be precipitated by antileprosy drugs.

The syndrome confirms deductions made regarding rheuma-

toid disease, proving that every tissue in the body may contain unsuspected free-living amoebae. If pathogenic, they 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 also the source of unknown chronic antigenic stimulation previously postulated by Glynn[7] as the cause of rheumatoid disease. [See "The Herxheimer Effect," http://www.arthritistrust.org.]

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## The Free-Living Amoebic Causation and Cure of Activity in Rheumatoid Diseases

*by Roger Wyburn-Mason*

*(Formerly published in* The Journal of the Rheumatoid Disease Foundation, *Volume 1, Number 2)*

A reactive arthritis may occur with gastrointestinal infection with bacteria, such as *Campylobacter jejuni*, Salmonella, Shigella and Yersinia, in patients with HLA-B27 tissue types and disappear with successful treatment of the infection (Kosanen et al. 1980). Between 1922 and 1952, numerous publications described *E. histolytica* bowel infection with or without dysentery as associated with a condition practically identical with rheumatoid disease. Both conditions are cured by emetine without any exaggeration of the joint symptoms. Yet it is not unusual for sufferers from rheumatoid arthritis to show *E. histolytica* or its cysts in the feces and never in the rheumatoid lesions.

All terrestrial animals and plants, and those inhabiting fresh water and the sea, live in a world surrounded by many species of free-living amoebae. These may pass mammalian respiratory passages as cysts or trophozoites and may be present as 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 reasonable to suppose that once they had entered the orifices of man or other warm-blooded animals, they would migrate under thermotropic influences into some body tissues. Since amoebae may be either non-pathogenic or pathogenic to animals, the same may apply should the organisms reach human tissues.[1]

In 1922, the eminent protozoologists, Kofoid and Swezy, in California, reported the presence in the bone marrow in cases of rheumatoid arthritis, without dysentery of *E. histolytica* in the feces, of a free-living amoeba distinguished from human cells by its mitotic processes which contained only 6 chromosomes as compared with the normal 46 of human cells. It showed a single blunted pseudopodium and numerous vacuoles. They suggested an aetiological relationship between the infection and the arthritic process.

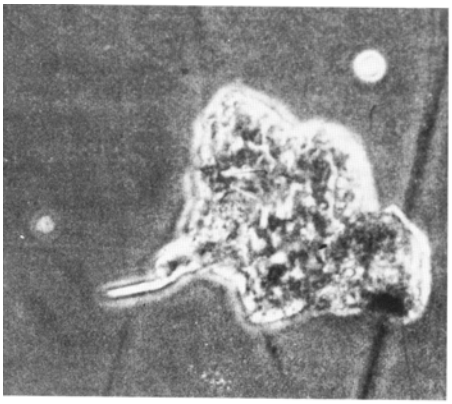
In numerous recent publications, the speaker[14, 18] has shown that, using the property of thermotropism, free-living amoebae pathogenic or non-pathogenic, can be made to migrate in varying numbers out of human tissues, including those of the newborn and fetus (Figs. 1). They are also found especially in the affected tissues of patients with rheumatoid disease, in malignant tumors, in normal feces, in uncooked butcher's meat, and in surface soil.

They may be recovered in small numbers from some tissues of apparently healthy humans, where they are presumably of non-pathogenic nature. They appear identical with those found by Kofoid and Swezy. These findings have been confirmed in various parts of the world, including Vanderbuilt University, U.S.A., the Oncological Research Institute, Bratislva, Czechoslovakia, Waikato Hospital, New Zealand, the Hospital for Special Surgery, and St. Vincent's Hospital, New York and in England.[1]

These organisms can be cultured in the laboratory in "amoeba saline" into which a culture of *E. coli* has been introduced. The effect of various antiamoebic substances in killing the organisms can then be studied by adding them to the cultured cells. Those which do so include one percent solutions of bile salts, 4-aminoquinolines, very dilute solutions of copper sulfate, metallic copper, gold salts, emetine, dehydroemetine, pentamidine, levamisole (which contains an imidazole group), and in particular the 5-nitroimidazole group of

drugs, including metronidazole, clotrimazole, tinidazole, ornidazole and nimorazole. All of these possess a wide spectrum of antiprotozoal, including some antiamoebic activity.

The organisms have been recovered with great difficulty because they are not observed in affected tissues stained by ordinary methods. They are not numerous and look like macrophages or lymphocytes. This is a feature repeatedly observed in experimental infections with free-living amoebae in laboratory animals. (This recalls the situation in syphilitic lesions until stains for the *Treponema pallidum* were discovered). The organisms in rheumatoid arthritis can, however, be demonstrated in tissue sections by immuno-fluorescent staining using sera containing appropriate antibodies to the organisms and by studying their mitosis and chromosome content in marrow biopsy material.



*Photograph of a* Limax amoeba *which emigrated from the malignant tissue in a case of carcinoma of the bronchus (unstained). (X1500). Note single spike-like pseudopodium, typical of many species of Naegleria*.*

Figure 1

**Rheumatoid Arthritis, a Generalized Disease**

Rheumatoid Arthritis is not solely one of joints but may involve any tissue of the body. The same histological changes in the joint capsules may be found in extra-articular lesions and consist of inflammatory lymphocytic infiltration, formation of germinal follicles and often plasmocytosis accompanied by arteritis, arteriolitis, or endarteritis. Many of the extra-articular lesions constitute so-called "auto-immune diseases," but also include Sjogren's syndrome, bone marrow infiltrations, and thymic lesions with or without myasthenia, fever up to 40° C, sweating and raised ESR, typical of an infection. Any of the extra-articular of AI lesions may occur in any combination with or without arthropathy.

From a study of the world literature, it seems that some of the extra-articular lesions may involve:

1. Exocrine glands often producing enlargement and dilatation of the ducts. It may involve the lacrimal and salivary glands (Sjogren's syndrome, R.A. in miniature), breast (cystic mastitis), pancreas (lymphocytic pancreatitis, which may exhibit calcification), liver (active chronic hepatitis, primary biliary cirrhosis), gall bladder and bile ducts (chronic cholecystitis and stenosing cholangitis), and kidneys (chronic nephritis, pyelitis).

2. Endocrine glands, including the thyroid (lymphocytic or Hashimoto's thyroiditis), adrenals, parathyroids, thymus (with or without myasthenia) and pituitary.

3. Polymyositis, myasthenia, bursitis, tenosinovitis, or rheumatoid nodules in any tissue.

4. Mucosal inflammation followed by atrophy, which may involve the gastro-intestinal tract producing atrophic stomatitis, pharyngitis, esophagitis, gastritis, or coeliac disease and ulcerative colitis, or in the respiratory tract leading to atrophic rhinitis, Eustachian salpingitis, laryngitis, or bronchitis.

5. Fibrosing alveolitis, pulmonary nodules, lung fibrosis, or pleuritis.

6. Peri-, myo-, or endo-carditis.

7. Bone marrow infiltrations with various disturbances of blood formation.

8. Paget's disease of bone, spondylitis.

9. Lymphadenopathy or splenomegaly with reactive lymphoid hyperplasia.

10. Choroiditis, uevitis, retinitis, scleritis.

11. Various skin lesions, including icthyosis, dermatitis, leukoderma, and melanoderma.

The serum may or may not contain RF, various auto-antibodies, ANF, increase in gammaglobulins and usually a raised ESR.

**The Effect of Antiamoebic Drugs on Active Rheumatoid Disease**

The author has shown that when any substance which kills the free-living amoebae *in vitro* is administered to cases of active rheumatoid disease or its extra-articular manifestations, it may cause a rapid disappearance of the inflammatory symptoms around the joints and elsewhere in the body.[1] This includes coeliac disease, ulcerative colitis, cystic mastitis, lymphocytic thyroiditis, etc. Myasthenia gradually disappears and in early cases[11], complete cures may be obtained. These drugs often induce an Herxheimer reaction that is a transitory exaggeration of the inflammatory changes around the joints and elsewhere. This may be accompanied by influenzal symptoms, sweating, pyrexia, lymphadenopathy, rise in ESR, and eosinophilia.

This reaction has been reported by others in cases of active rheumatoid disease treated with gold salts[18] and levamisole[19], (which kill free-living amoebae). Such a phenomenon, first described by Herxheimer[20] in 1902, when cases of syphilis were treated with mercury, also occurs in syphilis treated with penicillin and in other diseases due to organisms more complex than bacteria when drugs which kill the causative organism in the tissues are administered. This reaction is due to liberation of irritant and antigenic substances from the dying and dead organisms. The Herxheimer reaction in cases of active rheumatoid disease treated with antiprotozoal substances has been confirmed in various countries, notably in U.K., U.S.A., Holland, and New Zealand.[1] It is not observed in healthy persons so treated or in rheumatoid diseases given anti-biotics. It's occurrence in rheumatoid disease, including "autoimmune" lesions during treatment with various antiamoebic drugs *proves* the presence in the *affected tissues* of a *causative pathogenic organism*. [See "The Herxheimer Effect," http://www.arthritistrust.org.]

By administration of antiamoebic drugs, especially 5-nitromimidazoles, to cases of rheumatoid disease for 3-6 months, the evidences of disease activity usually completely disappear in joints and extra-articular tissues. "Autoimmune" lymphocytic and humoral reactions are thus not the primary disturbance in rheumatoid and "autoimmune" diseases, but are the cellular-antibody response to the infection and its antigens and contribute to the tissue damage.

**Practical Details of Treatment of Rheumatoid and Related Diseases**

A simple method of treatment of rheumatoid disease is to administer 2 grams on 2 successive nights of one of the 5-nitroimidazoles for a 70 Kg patient. In some cases, the organism , as judged by the severity of the Herxheimer reaction, may prove to be susceptible to one or another of the 5-nitroimidazoles.

In order to prevent any severe Herxheimer reaction, the patient may be given an anti-inflammatory drug. The drug may remain at an effective level in the blood for four or more weeks while the inflammatory reaction is dying down.

If it is necessary for a further dosage of the antiamoebic drug to be given, wait until three months after the original doses to give the induced inflammatory response [time] to die down. Often only a single treatment is necessary and improvement may continue over the course of a year or more with return of a normal ESR and disappearance of all signs of disease activity. [See "The Roger Wyburn-Mason, M.D., Ph.D. Treatment for Rheumatoid Disease," http://www.arthritistrust.org.]

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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 surrounded 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 wide-spread 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. [See "The Herxheimer Effect," http:www.arthritistrust.org.]

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