Protocol for Using Antibiotics

in the Treatment of Rheumatoid Disease

It is important to read the **entire protocol.**

This use of antibiotics for rheumatoid disease

is **different** from the traditional manner

in which antibiotics are prescribed.

This protocol supersedes any previous Road Back Foundation protocols.

It remains based on the publications of Thomas McPherson Brown MD.

as well as current research and treatment experience

covering over 50 years of clinical experience

and has been medically approved.

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Appendix B - Laboratory Tests

1. BACKGROUND (TOC)

Arthritis has been known to exist since prehistoric times. Over 37,000,000 people are currently suffering from some form of rheumatic disease resulting in tens of billions of dollars in health care costs and lost productivity. This protocol provides the means of returning these people to an active, productive life through the safe and effective use of low dose antibiotics.

11. Disease Mechanism (TOC)

This protocol views inflammatory forms of arthritis as a persistent cell-mediated hypersensitivity created by long exposure to antigen derived from a hidden or invisible microbial source (i.e. mycoplasma or closely related bacterial L forms).

It appears that mycoplasma produce their pathogenic effect in man, not by the classical method of invasion and rapid tissue destruction as in lower animals, but by creating a cell-mediated response resulting from long-standing cellular parasitism with gradual sensitization of the host through intermittent antigen release from the cells.

Rheumatoid disease progression to its most advanced stage is the over-reactivity of the host tissues with the development of the hyperimmune or autoimmune state, probably through cell or molecular mimicry. This phase of the disease, characterized by the production of cellmediated antibodies, macroglobulins and the rheumatoid factor appears to be an expression of a second line of defense in the immune system, logically deterring the spread of microbial precursors. **The therapeutic focus must be on eliminating mycoplasma as the key antigenic source.**

Virulence of disease may be determined by biological properties of mycoplasma and include:

1. Generation of hydrogen peroxide radicals by adhering mycoplasma causing host cell membrane damage
2. Competition for and depletion of nutrients which disrupt host cell maintenance function
3. Existence of capsule like, electron dense layers or structures which protect mycoplasma and confer immunoregulatory activities
4. Antigenic variation which causes possible avoidance of protective host immune defenses
5. Secretion or introduction of mycoplasmal enzymes (ATHases, hemolysins, protease and nucleate) into host cell which leads to localized tissue disruption, disorganization and chromosomal aberration
6. Intracellular residence which protects mycoplasmas and establishes latent or chronic states and circumvents mycoplasmacidal immune mechanisms and selective drug therapies.
7. Host genetic markers

It is suggested these agents enter the joint tissue perhaps early in life when the cellular immunity is weak and remain as parasites for many years. Over the years fixed tissue antibodies which represent the defense mechanism of the cells, begin to increase in concentration in the tissues

surrounding the cells that are infected. Finally, when the concentration of these antibodies become sufficiently high, the stage is set for a reaction to occur between the two forces: the extra-cellular and the intracellular mycoplasma.

The beginning of the disease in the clinical sense is when the two forces are thrown together by injury, stress, barometer alterations, or other environmental factors. The initial reaction is one of inflammation in the spaces between the cells and this rapidly extends to produce hot, swollen, tender joints. A reaction is designed to keep the infectious process from spreading. Unfortunately, this protective mechanism also becomes a barrier to the entry of the body's defense forces. Thus it is the nature of the disease to produce its own road block which accounts for the chronic, unrelenting nature of the disease process.

The Hypersensitivity State **(TOC)**

The treatment approach in the microbial hypersensitivity state is determined to be altogether different from standard anti-microbial therapy. In rheumatoid disease, the hyperreactive state itself suppresses microbial antigen replication and accounts for the high degree of localization of mycoplasma foci of involvement. The primary objective of treatment is the suppression of antigen production and at the same time avoiding the sudden release of excess antigen and delayed drug sensitivity by over medication.

Effective treatment has evolved to be the converse of the treatment of standard infection. The dosage of medication is relatively low instead of high, it is generally interrupted instead of sustained, and the treatment is usually long-term.

In all microbial hypersensitivity states, the causative agent virtually goes underground as the tissue reactivity becomes manifest. Thus, in the highly reactive state, very little anti-microbial medication is needed to further control the disease. **And if too much is given, the body begins to react against the medicine itself and defeats the purpose of the treatment. This is the main reason for the intermittent treatment.** All bacterial hypersensitivity states require intermittent antigen suppressing treatment as exemplified by tuberculosis, rheumatic fever and brucellosis.

Treating the Microbial Cause **(TOC)**

The fundamental treatment goal in the induction of a sustained remission is to control and suppress antigen production. The final objective has been the ultimate elimination of the microbial antecedent.

In the primary objective, the suppression of antigen production, dosage needs to be tailored to the individual patient to avoid the sudden release of excess antigen and delayed drug sensitivity by over- medication. The degree of anti-mycoplasmal medication may need to be reduced to the minimum, such as minocycline, 50 ma. once or twice a week, gradually increasing according to patient tolerance in these individuals.

An important guideline in successful treatment has been the avoidance of over- medication with paradoxical worsening. Too much medication can cause a delayed hypersensitive reaction to the drug itself and induce a flare of the arthritis with the development of symptoms closely mimicking the disease (Herxheimer Reaction). A therapeutic balance can be readily reestablished by the temporary interruption of the treatment for a week and then restarting at the same low dose.

Disease Characteristics **(TOC)**

The toxic substances characteristically present in inflamed tissues as well as toxins from organism die-off escape into the blood stream and are transported to the liver where they are destroyed. As they pass through the system, they react at the point of greatest antigen accumulation and may affect the body in a devastating fashion because of their persistence:

In the bone marrow, these toxic materials interfere with formation of blood and therefore anemia is commonly observed in severe rheumatoid arthritis and cannot be corrected until the disease has improved

•I**n the central nervous system** these toxins promote depression, inability to concentrate, loss of memory, loss of interest in one's vocational effort, mental irritability and epiloidal type seizures

•I**n the muscular system these** toxins are responsible for excessive fatigue and persistent weakness which is difficult to overcome by physical means
• Gland functions are also depressed
• There is an effect upon **the digestive system** and many other basic physiological processes
• E**very organ system** may be involved at one time or another

Those patients in whom arthritis has a severe onset but in whom it subsides rapidly without residual change and who subsequently have recurrent, similar episodes, seem to be less likely to develop destructive joint changes than patients in whom the onset is gradual with smoldering involvement. In the course of time, through the continuity of the anti-mycoplasma treatment method, there is a gradual reduction in the many sites of joint inflammation and the toxic substances generally diminish.

Focal Infections **(TOC)**

It is imperative patients be examined for other sources of antigen in intra-vascular or extra-vascular fluids in various intercellular sites, or, more grossly, in various cysts, fibrotic cavities and other remote area (sinuses-allergies, genitourinary tract, gut, pelvic area, digestive tract, teeth, etc.). Each of these different locations for the antigenic source creates a new set of variables but the primary problem - bringing the anti-infectious drug into direct contact with the microorganism - is common to all.

Focal infections are known to activate arthritis. It is possible that tissue invisible L-forms left in the wake of an infection, can perpetrate the inflammatory reaction through continued antigen releases rather than microbial invasion. The infectious tie-in becomes progressively more tenable

when bacterial variants (frequently streptococcus, L-forms) derived from focal infections are found. Anti-streptococcal antibodies cross react with mycoplasma protein (heart myosin, tropomysin and mycoplasmal adhesions). These variants must be treated often simultaneously with the original mycoplasma infection, but using a different antibiotic to achieve effectiveness.

If the patient has an **elevated ASO titre** and/or strong history of streptococcal infection, ampicillin, 250 ma. is prescribed to he taken once daily (preferably in the evenings and not at the same time as the tetracycline). This is continued until the ASO titre becomes normal after which the patient is **monitored for recurrence.**

III. Treatment (TOC)

Treating Patients with Severe or Long Standing Disease (TOC)

Treatment must be directed on two fronts: the hypersensitivity state and the microbial cause. The inflammation is treated with a constant dosage of anti-inflammatory drugs and the suppression of the microbial infection with low dose, intermittent antibiotics. Since long-term treatment is required, continuous low dose, intermittent antibiotics are more viable for patient safety and can be continued for years.

Washout period (TOC)

If the patient has been or is still on strong anti-rheumatic drugs (especially those which tend to build up in the liver like methotrexate or gold) or drugs which sensitize the digestive tract, a washout period of 4 to 6 weeks might be considered to avoid a reaction such as colitis. Low dose (<10 ma) prednisone can be used to maintain the patient during washout.

Anti-inflammatories (TOC)

'The first step is to reduce the inflammation with anti-inflammatory medications to prepare the way for the antibiotic action.

Enteric coated aspirin can be useful when given two to three tablets, three to four times a day. Other anti-inflammatory drugs may be useful as well. **Because of the individualized patient response to NSAIDs, choice of drug will depend upon individual patient response.**

People who have native allergies such as hay fever, asthma etc. often tolerate the phenylbutazone derivatives rather than the other, non-steroidal anti-inflammatory drugs. In these highly allergic individuals, antihistamines and even corticosteroids in **very small doses** (less than 5 ma. a day) can be helpful.

Beginning the Therapy (TOC)

Patients must be advised that the treatment is extremely slow and gradual and may take six months to a year before they can really see much improvement. In more severe or long-term patients it can take much longer (25 years).

A proper chemotherapeutic approach must recognize that tetracyclines and erythromycins are effective against many mycoplasma infections; some are resistant to one strain or the other. Mycoplasma testing to isolate the strain may be necessary (See Appendix A).

Patients with severe or long-standing disease are started with a series of daily intravenous or intramuscular antibiotic treatments for a period ranging from one to three weeks. Clindamycin is given to eradicate long-standing L, forms of bacteria resident in the gut, respiratory tract, genitourinary tract and other areas to allow greater permeability of the tetracycline family of antibiotics and diminish the variables of disease. Clindamycin is concentrated in the phagosomes of the neutrophils, and therefore accumulates at the site of inflammation.

Clindamycin IM - 300 ma. clindamycin can be administered intramuscularly (IM) once daily for one to two weeks followed by 300 ma. weekly, monthly or at 6 week intervals as needed and tolerated by the patient. Because it remains in the tissues longer, a lower dose is effective.

Clindamycin IV - The IVs should be started at a low dose (300 ma) and gradually increased as needed to avoid the development of resistance in the bacterial L-forms that might be present. If this resistance develops, the patient will not respond as well to the antibiotic therapy.

IV therapy is begun gradually at 300 ma. given in 250 cc 5% dextrose solution administered by IV drip over a 45 minute period for the first two days. The next two days, the dose is increased to 600 ma. and finally to 900ma on subsequent days if no adverse reaction is observed.

IV or IM therapy with clindamycin is continued at spaced intervals according to the patient's need. It can be given once weekly or twice a month again titrated to patient need. If weekly or monthly IVs are not possible for the patient, then a series can be administered at more widely spaced intervals such as every six months and later on an annual basis until the laboratory values return to normal.

Oral Clindamycin - Some physicians have had success using clindamycin orally (i.e. 1200 mg..) in a single weekly dose instead of in IV or IM

Tetracycline Therapy **(TOC)**

Following the initial clindamycin IV or IM course, oral minocycline or doxycycline is most commonly prescribed, continuing the periodic clindamycin as an adjunct to therapy. Care should be taken not to administer any antibiotic drug at too high a dosage too fast to avoid an allergic reaction by the patient.

Failure to achieve proper drug titration in either the continuous anti-inflammatory or intermittent anti-microbial medications may result in disturbance of the rheumatoid state of balance and promote clinical worsening (the Jarisch-Herxheimer Reaction).

Severe or Long-standing Disease

Patients with severe or long-standing disease are started on a low dose of oral minocycline or doxycycline ranging, according to patient tolerance, from 50- l 00 mg or tetracycline 250 ma. once daily one to two days per week. Titrated to patient tolerance the dose should be increased to a working standard dose of minocycline or doxycycline 100 mg once daily or tetracycline 250 ma. twice daily Monday, Wednesday and Friday.

If the medication tends to aggravate the condition, it is spaced differently, maybe to once a week; or twice a week, and gradually increased to the M-W-F dosage. Some patients are so highly sensitized to drugs that they can only tolerate minocycline or doxycycline 25-50 ma. Once every two weeks or even once a month, but with continued titration of the dosage, it is possible to work up to the optimum standard dosage of minocycline or doxycycline 100 ma. once or twice daily, Monday. Wednesday and Friday without flaring the disease.

Less Severe, Early Disease **(TOC)**

For patients with less severe or early disease, the IV or IM treatment may not be required as they experience the same result using oral medication exclusively. **The optimum standard dosage** for these patients is minocycline or doxycycline 100 ma. once daily, Monday, Wednesday and Friday or tetracycline 250 ma. twice daily Monday, Wednesday and Friday.

Unlike standard anti-microbial therapy, this method requires the application of new principles of drug administration with low dosage properly spaced, clinically titrated and most often given over a long period of time. **Because absorption takes place from all levels of the alimentary tract from the stomach onwards, but is never complete, the larger the dose the lower is the proportion of it absorbed.** The mechanism responsible for the decreased absorption appears to be twofold:

1. Tetracycline solubility is better in the more acid solution of the stomach; less so in the alkaline solution of the intestine.

2. Tetracyclines also tend to combine with divalent metals, of which calcium is likely to be present in the largest amount.

Important Note about Lupus & Minocycline **(TOC)**

Lupus patients may want to use another tetracycline other than minocycline. A few studies have shown that minocycline can cause lupus like symptoms in some patients and the PDR states that IV and oral minocycline can exacerbate the symptoms of lupus in some patients. This may be a Herxheimer expression.

IN ADDITION, an association has been shown between M. hominis and lupus. M. hominis is resistant to erythromycin so this also should be avoided when prescribing for lupus patients. 18 Ginsburg, 19 Cassel1 M. hominis is susceptible to clindamycin in vitro, possibly making it an effective adjunct to tetracyclines in patients testing positive for M. hominis. (See laboratory information.)

Increasing the Dosage (TOC)

Two basic methods of evaluation are considered as guides to the proper intervals for increasing the antibiotic dosage.
I) The patient should be watched and questioned carefully during the first months of treatment for subjective (invisible) and objective (visible) exacerbation of disease symptoms following administration of the medication. As long as a definite flare related to therapy (see section on Herxheimer) is noted, the dosage is not increased, regardless of laboratory findings. Usually this association does not persist beyond four weeks to six months, at which time the dosage is increased, if laboratory indications are present. In general, the initial dosage of antibiotic is maintained for three to six months despite the frequent feeling that larger doses would speed recovery.

2) Blood tests are followed at four to twelve week intervals until the patient stabilizes when every six months is often enough. As long as signs of improvement continue, the dosage of antibiotic is not changed. If the laboratory evidence of improvement fails to occur or stabilizes at an abnormal level, an increase in dosage, in frequency or a change in route of administration is considered.

When an increase in dosage is indicated, it is accomplished either by decreasing the interval between doses or the dosage itself is increased and administered at the same intervals. In a converse manner, if it appears that the drug-induced flare is excessive, dosage may be decreased by the same increments or by prolonging the interval between doses. This process of adjustment or titration is continued until the patient reaches the optimum standard dose of minocycline or doxycycline 100 ma. once or twice daily Monday, Wednesday and Friday or tetracycline 250 ma. twice daily Monday, Wednesday and Friday.

Injecting the Joint (TOC)

Intra-articular injections of clindamycin have been very effective when the reactive state of the joint is so intense that penetrance is not achieved by the oral or IV/IM route. The inflammation must be reduced in most instances for maximum clindamycin effect - the usual treatment plan for **large joints,** clindamycin 2 cc (300 mg.) plus dexamethasone I cc (4 mg). A reduced amount of the same combination of these medications is used for **smaller joints.** A ratio that has been found effective is either 2/3 to 1/3 or 1/2 to 1/2). This is one instance where the temporary blocking effect of corticosteroids becomes important.

The Herxheimer (drug related flare) (TOC)

A general aggravation of symptoms is sometimes seen following onset of therapy and is more likely to occur if the disease is severe. This flare may be subjective (invisible) or objective (visible), or both, and may occur several hours or even up to two to four weeks after the medication is started.

Those patients who test low in hematocrit and serum albumin levels and high globulin levels prior to treatment have the most intense flare on a given dosage of antibiotic.

The Herxheimer flare is the first indication that the antibiotic is reaching its target, and therefore considered a good sign.

When the severity of the arthritic condition begins to lessen, either from a spontaneous improvement or as a result of the continued treatment with carefully measured doses of antibiotic, a greater tolerance for the antibiotic, is noticed and larger doses are tolerated without a return of the Herxheimer flare reaction. If however, the dose has been increased too rapidly at any time, the initial flare reaction may occur again.

In the evaluation of these exacerbations, the physicianmust remain aware of the frequent disease flares from other causes, and he should attempt to differentiate from those which may be drug induced.

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Differentiating between a Herxheimer, an RA flare and an allergic reaction to the drug

Laboratory tests can help differentiate between a worsening of disease (RA flare), a Herxheimer reaction to microbial toxins, and an allergic reaction to medication.

1) WBC will **elevate** in a Herxheimer and **lower** in a flare.

2) A **Herxheimer** will also exhibit a coincidental **elevation of SED rate, gamma globulin and total globulin, and a fall in serum albumin and hematocrit.** Patients who exhibit this flare reaction accompanied by **anemia, depression of serum albumin, elevated total globulin and gamma globulin** are probably reflecting a **more intense reaction pattern to anti-L substances** than in hematologically mild cases.

3) **A marked increase in eosinophils** (for instance about 30%) is an indication of an **allergic reaction to the drug.**

First signs of improvement **(TOC)**

There is usually little objective improvement in patients during the first three months of therapy. In the ensuing three to nine months the improvement, when it occurs, is quite gradual. The course of events is similar to, although sometimes slower than that noted with gold, methotrexate or chloroquine therapy.

The first sign of improvement is usually a lessening of duration of morning stiffness although the initial onset may be as severe as usual. The patient notices a general feeling of well-being lasting initially for perhaps an hour or two and gradually increasing to more good days and fewer bad days with a longer time span in between.

As anti-mycoplasma therapy continues, toxic substances are gradually reduced and normal functions begin to return. Strength increases, blood count rises to its normal level, mental acuity, ability to concentrate, a return in interest in work and a lessened sense of irritability all become noticeable dividends. All these symptoms may improve remarkably with rheumatoid remission, even after having been present for years.

It has been possible to measure clinical and laboratory parameters for the degree of disease control over a five year period. During this period over 70 percent of patients treated showed sustained improvement after five years. **A major advantage of this approach has been the freedom to treat early rheumatoid arthritis in a basic manner with success.**

Continuing Treatment During: (TOC)

Improvement

Most patients need to continue treatment indefinitely. Medication should be continued until the laboratory findings return to normal values for at least three months. Then gradually the IV treatment is discontinued, and if no adverse effects are observed, the oral tetracycline derivative is lowered. For some patients this treatment provides a permanent remission and no further medication is needed. Still other patients need to stay on a maintenance dose to keep the disease under control. If medication is discontinued too soon a rebound effect may occur which can be more severe than the original disease. If symptoms should return, a short course of tetracycline derivative may be sufficient to put the patient back in remission. Repeat courses at short intervals may be needed and will usually reestablish the remission for an indefinite period. **For some patients, the hypersensitivity has probably cascaded into a form of molecular mimicry and as such, the auto-immunity becomes the target. Antibiotics remain effective, although rotation to other drugs with higher tissue concentration (such as azithromycin) may be necessary to block receptor binding of the mimicry molecules.**

Drug Rotation (TOC)

Thomas McPherson Brown, MD, originator of antibiotic therapy, found rotating the antibiotic every 4-5 years even within the same classification, decreased the possibility of developing patient tolerance to the drug. After a rotation period, the original drug may be re-instituted.

Side Effects (TOC)

The tetracycline derivatives and crythromycin are both highly effective and safe anti-mycoplasma substances. It is characteristic of microbial hypersensitivity states that the proper use of anti-microbial agents is in general **surprisingly free from drug complications** unless the medications possess sensitizing properties or innate toxicity's. It is of additional interest that mycoplasma, unlike bacteria, do not possess a cell wall, only a thin covering membrane. Thus, **long-term exposure to anti-mycoplasma substances would not be expected to create mycoplasma resistance which is usually dependent upon cell wall enzymatic activity. In over five decades of use, no ill effect from medication has been experienced,** except the

emergence of delayed sensitivity as with all drugs in this field, but on a level that is very easy to manage.

Candida is not usually a problem, but in some patients it can become severe. **Acidophilus** should be prescribed as a preventive and testing should be done regularly to prevent an overgrowth from occurring. The candida, if persistent, should also be treated concurrently with the rheumatoid disease.

Laboratory Tests **(TOC)**

Patients stay on medication until all laboratory tests return to normal. Some patients indicated they began to feel better long before their laboratory results improved. The converse can also be true. (See Appendix B)

This protocol is compiled largely from the published works of Thomas McPherson Brown, M.D., the physician who pioneered and developed this treatment over fifty years ago.

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Appendix A - Mycoplasma Response to Drugs **(TOC)**

Mycoplasma Strain	Susceptibility to	Resistant to
M. pneumoniae 14 strains found in respiratory/ urogenital tracts & synovial fluid	erythromycin tetracycline, doxycycline oxytetracycline clarithromycin A63075 temafloxacin, difloxacin ciprofloxacin	
U. urealyticum 28 strains found in urogenital/ respiratory tract & synovial fluid	temafloxacin, difloxacin (somewhat) ciprofloxacin (some strains) ofloxacin doxycycline, tetracycline	erythromycin (5 strains) clarithromycin A63075 tetracycline – some strains (Baseman & Tully)
M. hominis 20 strains found in urogenital/ respiratory tract	tetracycline, doxycycline clindamycin temafloxacin, difloxacin	erythromycin (all strains) clarithromycin A63075 cetracycline – some strains (Baseman & Tully)
M. fermentans var. *incognitos* NIH 713-001-084 found in urogenital tract & synovial fluid	tetracline, doxycycline clindamycin	erythromycin (incognitis, NIH713-001-084)
M. penetrans GTU 54-6A1	erythromycin tetracycline, doxycycline clindamycin zithromycin, clarithromycin ofloxacin	
M. purim 70-159 found in urogenital tract	tetracycline, doxycycline clindamycin zithromycin, clarithromycin ofloxacin	
M. orale found in the (mouth) & respiratory tract		

M. salivarium found in the (mouth,) respiratory & urogenital tract		
M. hyorhinis found in synovial fluid *M. bucale* found in respiratory tract		Had this study been done later than 1981, minocycline would probably have been added to most susceptibility lists
M. arthritidis found in synovial fluid		

LIST FOUND: Cassell & Cole, **NAME OF ARTICLE.** *NEJM* ,304:2 1981
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Appendix B - Laboratory Testing **(TOC)**

ESR, hemoglobin, platelet count, gamma globulin, C-reactive protein, rheumatoid factor, ANA, MCF, candida and ASO are some of those usually monitored in these patients. However, no test is perfect in all cases. Situations can change, interfering factors can change test results, as can some drugs, and healthy people can have positive results and sick people can have negative results.

A major advantage of this approach has been the freedom to treat early rheumatoid arthritis successfully in a basic manner with antibiotics.

ESR - Erythrocyte Sedimentation Rate The ESR is a less reliable guide to the degree of basic rheumatoid activity because of the frequent fluctuations associated with minor environmental changes. The pattern formed by a series of ESR tests in a given individual is more useful in interpreting the disease activity than in a single determination.

GAMMA GLOBULIN, Lymphocytes When the **gamma globulin level** is elevated it provides an excellent index of the degree of rheumatoid activity.

Another index of the state of the rheumatoid process, which may be diagnostically useful in some patients, is an absolute increase in Lymphocytes.

RH - Rheumatoid Factor Rheumatoid factor, although frequently positive in arthritic patients, **has been negative in a number of patients diagnosed with severe disease (as high as** 20%). These patients have demonstrated improvement when treated with tetracycline derivatives.

PERCENTAGE OF PRESENCE OF RF IN SOME RHEUMATIC DISEASES

Classic RA 50%
Early or Atypical RA 50%
Juvenile RA 20%
Infectious Diseases 10%
Elderly Persons 25%
Healthy Adults 5%

ANA - Antinuclear Antibody Abnormal ANA is considered an indication of rheumatic disease. A negative ANA is sometimes interpreted as eliminating lupus or scleroderma as a possible diagnosis. The higher the titer, the morespecific the test is to lupus.

There are many kinds of ANAs, some specific to certain rheumatic diseases:
Anti -SCL-70 - scleroderma
Anti-Centromere - CREST scleroderma
Anti-DNA - lupus
Anti-RA33 - RA

PERCENTAGE OF PRESENCE OF ANA IN SOME RHEUMATIC DISEASES

Systemic Lupus Erythematosus 95-100%
Diffuse Scleroderma 75-80%
Sjogren's Syndrome 40-75%
Rheumatoid Arthritis 25-60%
Juvenile RA 15-30%
Dermatomyositis, Polymyositis 10-30%
Healthy Adults ~5%

DISEASE ASSOCIATED WITH ANA PATTERNS
Peripheral exclusive to lupus
Homogenous, Diffuse lupus, related CT
Nucleolar scleroderma (SSc)
Speckled, Irregular mixed CTD (SLE & SSc)

MCF - Mycoplasma Complement Fixation Mycoplasma complement fixation test (MCF) identifies antibodies to specific strains of mycoplasma. A positive test justifies using antibiotics as a treatment, but can also be an aid in choosing the antibiotic prescribed. Low titers are significant, and in humans, are seldom high. Often during treatment the MCF becomes strongly

positive sometimes showing as much as a fourfold increase before dropping again.

PCR - Polymerase Chain Reaction PCR is a highly specific test which identifies organisms, such as mycoplasmas by their DNA.

ASO - Anti-streptolysin -O This test for the presence of streptococcus is elevated in many patients with rheumatic disease. It has been noticed with some patients, that an elevated ASO titer can mean response to treatment is compromised. Some patients cannot tolerate any titer above negative. Treatment with ampicillin until a negative titer is achieved can speed treatment response to antibiotic therapy, and failure to treat the strep concurrently can sometimes delay the positive response to antibiotic therapy.