

RIFE RAY CANCER TREATMENT AND MYCOPLASMAS IN CANCER AND AIDS

Draft paper by Alan Blood, Brisbane Australia, circa 1996

ABSTRACT

This article reviews the theory that mycoplasma and other bacterial organisms may be involved in masking of surface antigens and a number of other immunosuppressive and carcinogenic effects in cancer and in AIDS. The second section discusses the theory of Rife- type electric field oscillators as a medical treatment. We discuss the possibility that modulated induced high frequency oscillating microcurrents on membrane surfaces may remove or disrupt the masking (eg by hCG) of tumour antigens and also of white blood cells. Simultaneously there may be proliferation and hyperactivity of white blood cells caused by cytokine stimulation response. The synergy of these two effects may enhance the restoration of anti-tumour antigen immune recognition and attack in cancer, and possibly other immunosuppressive pathways could also be disrupted. If the bacterial forms associated with cancer or pre-cancer pathology can be successfully attacked in this type of treatment, their immunosuppressive effects could be eliminated as well, although this has not been demonstrated in vivo. It is believed that these hypotheses may account for unconfirmed reports of rapid clinical remissions by Johnson et al prior to WW2, and later by Hamer *22, as well as claims for other types of modulated or pulsed oscillator devices. An appendix section discusses some uncertainties of interpretation of forms observed in live blood, and suggestions for new investigations.

BACKGROUND FACTS ON MYCOPLASMAS

Mycoplasmas have several features which are different from other bacteria. They have no true cell wall. They have a very small number of genes (genome), around 10 times smaller than typical bacteria such as e. coli, and about the same size as the more complex types of virus. There are many classes and species of mycoplasma. They are believed to have had a common ancestor with the Gram- positive Orders of bacteria. Evolutionary studies on rRNA suggest that mycoplasmas may be degenerate forms whose origins may be the hybridization of L-forms of early gram- positive types of bacteria. Mycoplasmas show a large degree of genetic diversity and it seems that they also have a high rate of mutation, possibly because of the loss of protection of a true cell wall. The most researched species is the one which causes atypical pneumonia in humans. Mycoplasmas are also known by the name Pleuro- pneumonia like organisms (PPLo). They can divide like typical bacteria, or alternatively they can assume adult forms where nuclei continue to divide inside a single growing cell enclosed by a sheath. Sometimes multiple adult forms develop partly fused together to give a variety of shapes. At maturity the "adult"

form can burst to release viable elementary bodies or “seed forms”. These can be extremely small, (viable free forms around 0.3 micron), and they can pass through fine filters which do not allow bacterial forms through. For this reason they have been called filterable bacteria. Because of this characteristic, A.Kendall in 1931 incorrectly proposed a theory of a pleomorphic cancer virus, since filterability is a characteristic of virus. Despite the differences from other bacteria, today none of the many types of filterable bacteria are classed as virus, because they all have true cell phases which virus do not have. Some mycoplasmas are harmless commensal populations of mouth or genitals. Others can also be pathogens which can live inside white and red blood cells, especially in AIDS. Sometimes in AIDS the flask- shaped species including newly observed M.Fermentans Incognitus strain can grow fine threads which can bud from the ends.

IMMUNOSUPPRESSION

Since 1920, a number of researchers have isolated and cultured filterable pleomorphic (many- shaped) bacterial forms from a variety of neoplastic tissue *1. Various forms may often be observed in fresh blood in cancer and in AIDS in dark- field microscopy *2. A pin- prick test can gauge cancer risk even before tumour formation (*3), by looking for these forms, as well as spots within red blood cells. Though often dismissed as an opportunistic infection, it may be that micro-organisms play a causative and contributing role in cancer *4,7. The link between pleomorphic bacterial forms and cancer was first indicated by research showing tumour formation in inoculated animals, as well as their presence in microscopic observations of cancer tissue (*1,4).

Some of the observations of cell- like or spore- like forms have been claimed not to be bacteria, but rather membrane fragments, and researchers interested in the Rife approach ought to be aware that in some observations this may well be the case. On the other hand, some forms isolated from cancer, or observed in cancer or AIDS blood are undoubtedly bacterial. Kendall and Rife announced a pleomorphic cancer virus, a theory still supported by Rife “true believers”. This article points out that their early observations of the filterable bacterial forms were not in fact virus, but rather of mycoplasmas or of cell- wall deficient bacteria.

Over the years a variety of names and phylogeny have been suggested for the cancer isolates. Recent film demonstrations by various independent researchers were shown at the World Cancer Congress, including high resolution Somatoscope footage by G. Naessens. The phases of the organisms suggest that at least some of them may be mycoplasmas, but the distinction between these and the cell- wall deficient forms (cwdf) of other bacterial species is still not entirely clear. Other interpretations, ie of host membrane fragments (Url), and of Endobiont forms (Enderlein) need to be acknowledged. (See appendix discussion). Note also the probability of sexual and blood transfusion transmission of the more pathogenic species eg M. Fermentans, M. Penetrans, and M. Pirum and their putative role in AIDS and/ or cancer.

Kleineberger- Nobel noted that mycoplasma (PPLO) could proliferate in a stressed host

*6. Observations suggest that a lowering of immune competence as a result of an episode of grief can lead to cancer in an 18 month timeframe *3. We can reasonably postulate that mycoplasma or other bacterial proliferation subsequent to immune depression may induce cancer. Evidence of carcinogenic effects and a variety of immunosuppressive effects from mycoplasmas have been presented in the literature, and hCG secretion by other species isolated from cancer have been reported by Acevedo et al

Naessens has postulated that in a healthy host the so-called "Somatid" organisms are found as a commensal population of underdeveloped coccoid and sometimes rod forms whose growth factor secretions are controlled by humoral response. He claims that where immune function becomes sub-optimal, an increase in the concentration of the secretions may stimulate development of the advanced multinuclear adult forms, and may also stimulate host cell growth *7. Naessens claims that his observations lead him to the conclusion that once a tumour achieves a "critical mass", nearby white blood cells appear to become paralyzed, and thus the immune system cannot recognize or challenge the abnormal cells. He attributes this effect to the secretion of "Co-carcinogenic K-factor" by the tumour mass. This interpretation is echoed in findings that NK activity is suppressed via hexosamine formation via deacetylase from N-acetyl aminosugars in tumour cells.

It has been reported that human growth factors can stimulate kinase release within human cells leading to oncogenic expression. Some mammal and prokaryote growth factors are quite similar, and it has been suspected that "mimic" bacterial growth factors may induce cancer subsequent to local chronic infection. Indeed mycoplasma secretions have induced nuclear transformations in animal fibroblast cell lines in vitro (*4,20), although the mechanism is unknown. In one such study viral infection and DNA transfection had been ruled out *9. The question of whether mycoplasma growth factors can be oncogenic must be considered, however there is no conclusive evidence to verify this theory.

Transformed (cancer) cells should normally be attacked by white blood cell mediated immune response, but we assume that they may proliferate where antigenicity is masked. A healthy immune system can recognise and attack transformed cells, but on the other hand most human tumours tend to attract only weak immune response. This paradox has been one of the central unsolved mysteries of cancer research. Recently research has isolated a cancer cell protein antigen which has been named malignin. Also an antibody level test was developed *16. Other reviews confirm some antigen expression of antigens on cancer cells coded by genes which are normally dormant. It was noted however that the malignin antigen could often be covered over by polysaccharide substances, thus reducing the antibody contact or white cell recognition site contact with the malignin antigen. This observation raises the questions a) to what extent are mycoplasma polysaccharide secretions, eg galactan, involved in the masking phenomena, especially prior to tumour formation; and b) to what extent are hCG secreting bacteria or their cell-wall deficient forms responsible for antigen masking and local immune suppression.

Cancer cells secrete their own growth factor but require a certain critical level of factor concentration in the interstitial fluid to trigger division. It has been demonstrated that cultured cancer cells need a critical mass of neighbours (in culture tests) before they can

build up sufficient concentration to trigger cell division*9. Therefore it is not unreasonable to postulate that in some circumstances a single transformed cell may not proliferate alone without a boost from bacterial secretions which mimic host growth factors.

In 1972 V Livingstone- Wheeler discussed the discovery of “Choriogonadotropin”- like secretions from cultured human cancer pleomorphic bacterial isolates. Human Chorionic Gonadotropin (hCG) is secreted by the trophoblast and chorion in pregnancy. It is also secreted by human cancer cells. Cancer research has pondered two important questions in regard to this and other similarities between zygote cells and cancer cells. One question is in relation to the mechanism of how the zygote brings about maternal immune tolerance; herein may lie the secret of the apparent non- immunogenicity of cancer tissues. The other question relates to how the chorion induces blood vessel growth, and whether this can help us understand angiogenesis.

CG- like secretions from bacteria may play some role in immune suppression. H F Acevedo reported a direct correlation between hCG titer in normal pregnancy, chorionic carcinoma and hydatid mole versus Immunoglobulin levels and a number of other immune cell standards. He has also demonstrated a correlation of human cancer cell lines to metastasize in nude mice models to the degree of hCG expression of the cell line. Later we discuss Acevedo’s isolation of CG secreting ”abnormal” bacteria from cancer.

At the point of tumorigenesis, before there is any significant tumour cell secretion, we may postulate that bacterial secretions are involved in the prevention of immune response against newly transformed cancer cells *7. According to a review by Macomber, Chorionic Gonadotropin- like substances (*19) contain sialic acid residues which may increase membrane negative charge by adhesion to cell surfaces of cancer, trophoblast, sperm and T- cells. This results in reduced immunogenicity of the above cells and of the mycoplasmas. CG adhesion to white blood cells may interfere with receptor binding in immune recognition. A role of electrostatic repulsion has been postulated as inhibiting white blood cell response against transformed cells*4. Other immunosuppressive pathways are briefly discussed.

At this point I would remind the reader that the dominant research finding is that cancer cells secrete hCG. Therefore much of the relevant research on hCG in cancer will have been investigating this connection. However the possibility of a similar model of immunosuppression arising from CG secretion of bacterial origin has generally not been considered.

Bogoch recently completed research which developed a useful malignin antibody test. Malignin is a tumour antigen which seems to be universal to most cancer types. The protein antigen is part of the cell membrane. Bogoch notes that this protein antigen is often covered over by polysaccharide substances, implying antigen masking. High titers of antibody correlated well with survival. Conversely, low titers correlated with progressive disease and mortality. In some cases the marker was subsequently shown to be present before clinical observation of cancer. It is not clear whether general

immunosuppression causes a failure to mount an immune response against tumour antigens, or whether antigen masking is to blame. It seems likely that both play some role in the pathology of cancer, and that they are interrelated.

Instead of a covering cell wall, mycoplasmas can protect themselves by making a slime covering. They can secrete considerable quantities of polysaccharide substances. Some reviews have noted what appears to be the ability of mycoplasmas to alter their secretion products. Also some lipid-associated membrane proteins can change the expression and size of the antigen molecule with high frequency. A variable expression genetic system has been elucidated for this type of control of membrane antigens. It is possible that because these antigen molecular patterns are not constant, they can to some degree elude immune response. Other workers have reported that some mycoplasma galactan polysaccharide secretions bind antibodies, and inhibit phagocytosis. Other research notes the detection of unusual products in mycoplasma infected helper T-cells. It is probable that if the helper cells fail to secrete IL-2, (a required "second message") that there can be no T-cell activation against tumour antigens. Some mycoplasma species can cause membrane damage via cytoadsorption by localised peroxide and N-O secretion. Some "flask-shaped" species can live inside white blood cells to escape immune attack *9. Naessens has also filmed the migration of small rod forms between red cells. Note that epithelial basal membrane degradation has been suggested as a likely first step in angiogenesis, followed by growth factor-like chemotactic signals from the tumour inducing blood vessel capillary growth. A question arises regarding a possible contributing role of local damage and / or growth promotion by mycoplasmas in angiogenesis. The outgrowth of threads from the flask-shaped species may also be pertinent to this question.

Once established, many types of cancer cells create their own immunosuppressive effects, eg secretion of substances similar to p15-E retroviral proteins, which have anti-immune effects probably including blocking macrophage chemotaxis pathways *4, 24. In addition, tumour secretion of histamines has been shown to be immunosuppressive, and more importantly, angiogenic. (This is now treated with Cimetidine in bowel cancer at Sydney's St George hospital) *17. Tumour lactic acid metabolism also creates gross effects and liver load. The immunosuppressive effects of tumour secretion make it difficult to achieve a healing response, and are the target of various approaches in immunotherapy. But even before tumour formation, we can postulate that mycoplasma proliferation may have triggered and supported the initiation of cancer. Therefore methods to improve immune strength, eg nutrition, should be considered as a preventative approach *3.

Livingstone- Wheeler used autologous vaccines against the patient's own pleomorphic bacterial isolates along with BCG (an anti-TB serum) as cancer therapy *11. In 1982 success in cancer control was reported in animal vaccination with CG-beta subunit with tetanus toxoid and adjuvants *9. It may also be possible to develop vaccines with isolates from sarcoid tissue and in Kaposi's sarcoma in AIDS *27. Cantwell has reported acid-fast bacteria in KS. Recent studies have been in conflict as to whether mycoplasmas can be isolated from KS tissue. Various other means could be considered to therapeutically

attack mycoplasmas, eg specific antibiotics or drugs. Naessens developed a method to inhibit some cancer cell secretions by lymphatic injections of 714X camphoramine *7. Mycoplasmas are inhibited by anionic detergents *18, and therefore saponific plants may be useful. Perhaps this is why yucca, a cactus, has become a traditional remedy. Natural substances which stimulate NK activity have been reported, as well as substances which stimulate T- cell activity against tumour cells probably by IL-2 stimulation.

THE USE OF ELECTRIC FIELD OSCILLATORS IN MEDICINE

In the 1920's Royal R.Rife of San Diego (*21, 22) developed an audio- pulsed radio frequency electric field oscillator which produced an audio intermittent or square wave modulated r.f. oscillating electric field set up in an electrode gap within a large spherical gas plasma tube output fired by an overmodulated a.m. signal *12. In the area near the tube, the so- called "Rife Ray" was claimed to be able to kill or affect motility of various pathogens at modulations of audio frequencies specific to each species. It could be that this claim may in fact only apply to wall- less or abnormal- walled organisms. Rife made visual and film observation through a special high magnification micropolariscope of his own design. In some cases Rife claimed to have observed membrane rupture of bacteria. In 1995 Bare produced film showing the process of rupture of some Paramecia in a sample using a variant design of the Rife device over 45 seconds *13. However this film should not be interpreted as fully verifying Rife's claims, since it was necessary to visually track forms of abnormal morphology to capture the rupture event, and other cells in the sample did not rupture. Modified Phanotron gas tubes are now available which have been driven by conventional 100W transmitter/ linear amp/ tuner * 13. The most powerful c 1935 Rife design was reported to be driven by a 500 W transmitter with a 8000 V signal. Whether this Voltage was an r.f. peak value or power supply value is not clear.

A suitable variation to this type of device for experimental cell research would be the adaptation of an electrophoresis unit by driving it with audio- pulsed r.f., and using a non conductive sucrose medium. Alternatively an air gap between 2 electrodes could be employed. Bare's design employs a straight gas tube which is externally wrapped by cable from a transmitter. Each cable end is tied off into circle or loop ends, and a gap remains between each of these loops, creating a dipole.

We could assume that an electron or negatively charged particle in the body of the patient experiences an attraction to the anode. The amplitude of the force of attraction varies at r. F. Hydrogen ions and other positive ions will experience forces in the opposite direction. Therefore we assume that in the area near the tube, all charges will experience some induction due to local electric field perturbation at r.f. We also assume that most induced current flow will occur on membrane outer surfaces, just like r.f. current concentrated on the outside surface of a cable. Because the audio modulated intensity falls away to a low or zero amplitude for about half the audio cycle, membranes experience a switching from surface current or excited state to relaxed state at audio frequency. This may equate to an audio oscillation of charges from interior to exterior of the local membrane. Various

speculations as to the mode of biological effect follow, but note that testing of various claims has not been undertaken.

Recent research has suggested that at least some ion pumps utilizing ATP run at fixed frequencies from 1kHz to 1 Mhz *19. The claimed Rife mortal oscillatory rate effect on bacteria (m.o.r. effect) may conceivably be caused by ion pump failure by electrical resonance when subject to synchronized oscillator output. On the other hand such a phenomenon could in theory adversely affect animal cells, but the treatment was claimed to be safe for humans. However claims have been made by Crane of parasite killing (ie worms at 20 Hz modulation). Post- treatment effects of weakness or illness have been reported, but have been attributed to toxin release, or may be a symptom of fever- like immune hyperactivity.

An alternative explanation may be that postulated by Pappas, ie that the plasma membrane exhibits different electrical resistance depending on the direction of current flow, and that the efficaciousness of pulsed h.f. magnetic or electric field oscillators lies in generating ion dispersion in tumour cells such that there is an increment of one- way charge movement at each modulation pulse. Thus cells revert to normal type membrane potentials, and thus lose the mitotic condition at low potential, as well as becoming immunogenic *28. Pappas also claims that weak- walled and wall- less bacterial samples have been killed by his coil magnet device. This latter recent observation seems to be in agreement with Rife's claims of success in killing pleomorphic forms. (These were of major interest to A.Kendall and E. Rosenow). It may be that other claims by Rife that a whole library of bacteria and virus could succumb to his treatment at individual mortal oscillatory rates may simply not be true. On the other hand models of immune stimulation may be plausible.

Rife claims effective modulation values at 1927 Hz for carcinoma and 2008 Hz for sarcoma. I was interested to read Gianni Dotto (who developed the patented Dotto Ring in 1975 in USA) mention 2000 Hz frequency as a sort of cell self- tuning frequency, but no explanation was expounded. Recently I studied nerve impulse action potentials in unmyelinated nerve cell axons. Na channels open quickly, followed more slowly by K channels which counter the Na flow- induced potential change. (These types of channels are Voltage- gated). It just so happens that peak depolarization occurs 0.5 ms after the initiation of the action potential. Therefore a 2000 Hz modulated excitation would trigger Na flow like a diode current, but K flow would not get a chance to "kick in".

In addition to the concept of an antibiotic effect, it has been noted that various electrical oscillatory applications can cause the stimulation (*13) of white blood cells, probably by causing cytokine secretion, similar to what the blood does near the site of a wound. This effect may be of particular importance to encouraging immune recovery by recognition and response to cancer cells. It has been claimed that after electrotherapy white blood cells are observed to undergo rapid multiplication and to become hyperactive for about 18 hours until dying off.

It may be that binding sites or electrostatic bonds of CG- like secretions and possibly

other polysaccharide substances may be broken by induced or transduced electric oscillating currents. If this is the case, then the combined effect of white cell hyperactivity and their access to newly exposed tumour antigens may engender an immune recovery.

In trying to develop a theory to explain the claimed anti-cancer effects of oscillators of quite different designs and outputs developed independently by various different innovators, it has seemed to me most likely that in all cases an immune recovery is engendered somehow by means of induced microcurrents. The role of hCG secreted by tumour cells, and the possible role of other bacterial secretions, must be prime suspects in immunosuppressive mechanisms. The dispersion of these slimy coatings by modulated h.f. local membrane microcurrents would firstly expose tumour antigens, and secondly the coatings on T- cells would also be dispersed. Activation of the helper T-cells would be necessary to provide IL-2 “second message”. This may occur naturally, or alternatively IL-2 therapy may prove a useful adjunct. Also IL-2+ genetically engineered anti-tumour activated autologous CD8+ T-cell therapy as outlined by D M Pardoll may be useful. Wheeler’s vaccine method may be of use, but her clinic was closed down by an ever- vigilant FDA. Modern research is also investigating vaccine therapy against cancer.

In searching for a means to kill Kendall’s pleomorphs, Rife learned to force the aggregation of viable filtrates isolated from cancer tissue by an r.f. stressing technique, using a corkscrew or helical plasma tube as a test tube holder, and driven with unmodulated r.f. Thus a sample of filtrate in a test tube could be positioned so that one electrode was above and the other below the sample. The aggregation or clumping occurs because cells stick together under r.f. fields eg in r.f. electrophoresis. In the case of filtrates of wall- less organisms it is likely that the elementary bodies will completely fuse. The motile aggregates could then be imaged, and a mortal audio resonant frequency was determined by tests with the Rife Ray. Tumour formation after inoculation was claimed to have been prevented by Rife Ray treatment. In hindsight the latter conclusion may be open to criticism. It is true that inoculation of cancer bacterial isolates will induce tumours, and that effective antibacterial treatment post- inoculation may prevent the tumour growth. However the link between the cancer microbes and human cancer is not universal as Rife and Kendall were tempted to believe. Kendall’s culture technique required a pork gut medium, and we could suspect that his culture organisms were contaminants. However their capacity to induce tumours by inoculation is still significant. Modern research has verified that pure cultures of mycoplasma isolates do indeed cause tumours by inoculation.

AIDS DISCUSSION

A significant number of AIDS patients are infected with mycoplasmas, and it was thought that these were opportunistic infections after HIV immunosuppression. A new strain named *M. Fermentans Incognitus* has been discovered in some 17 % of cases. CD-4 binding sites on the membrane of *M. Incognitans* have been discovered, which means that the virus can be transported by *Incognitans*, and it has been argued that the AIDS

syndrome may in at least some cases be a co-infection based on the sexual transmission of mycoplasmas. A revealing series of experiments with monkeys shows that inoculation with HIV-1 alone will not kill monkeys, probably because this virus is human specific. However when injected with *M. Incognitus* alone, monkeys showed wasting syndrome, and death within 7 to 9 months. One in vitro experiment showed that a cell line infected with both *M. Arginini* and HIV-1 showed HIV-1 expression at a rate 40 times greater than a control with HIV-1 only. If this is the case in vivo in AIDS, the mycoplasma co-factor link should not be ignored. We may postulate mycoplasma pathogenicity and various immunosuppressive effects as contributing to the disease process. Therefore the therapeutic treatment of mycoplasmas in AIDS eg by specific antibiotics may be useful.

Interestingly, Beck claims that transduction electrotherapy prevents the capacity of the HIV virus to attach to the CD-4 surface receptors of helper T-cells * 14. An interesting article appeared in an Australian newspaper describing how a farmer suffering the long term effects of Ross River virus was pushed into an electric fence by a playful calf only to discover that his symptoms were relieved. A neighbour with the same affliction thought he'd try his luck, and was also rewarded with respite of symptoms! Unfortunately it is unlikely that any "anecdotal" claims of this kind will attract any follow-up research.

The mycoplasma- cancer link may be disputed on the grounds that less than half of cancer or AIDS cases have yielded mycoplasma isolates. However we contend that the question of a link in some cancer pathways is still open. Even if the Mycoplasma infections in AIDS are indeed late-comers to the disease, their contribution to the disease may be significant.

CLINICAL NOTES

In his cancer treatment Rife set a protocol of 3 minute exposures with 3 day rests in order to allow toxin elimination. There may be some danger of kidney failure or lesion haemorrhage in cases of breakdown of tumour masses, *23 . In many cases surgery or the other tumour destructive therapies may be indicated prior to Rife treatment. For bleeding it may be appropriate to administer Tributyrate *26. Like other forms of electrotherapy, there is a danger of induced heart attacks as well as epilepsy. Electrotherapy may therefore be generally be considered contra-indicated for patients with a history of these conditions. Rife claimed that he detected no harmful effect from his oscillator to humans. However there may be unknown hazards, and there have been reports of frequencies which affect intestinal flora. Possible effects on early pregnancy should also be considered. Where live blood observation indicates pre-cancer pathology, Rife treatment may similarly unmask the bacterial forms and T- cells, thus engendering anti-mycoplasma immune response. A reduction of mycoplasma population, particularly of the advanced phases, could eliminate the source of immunosuppression, which would be considered a major cancer risk factor.

Because the emphasis is on immune response, various natural therapies, exercise,

vegetable juices, anti-oxidant vitamin and mineral supplements, herbs, nutritional protocol, substitution of refined salt for sea salt, etc, medical immunotherapies and even psychotherapy are considered necessary adjuncts to the Rife therapy. Conventional immunosuppressive therapies (ie X ray and chemotherapy) may counteract the desired immune response in the short to medium term, but could be considered nonconcurrently. Surgery, on the other hand, may be indicated as an early treatment, and would be expected to assist the process of immune recovery. Critics may suggest that these comments constitute a danger to the public should they be tempted to avoid seeking qualified treatment. Such debate is dealt with by other commentators. However there is no excuse for the ongoing failure of the “establishment” to grant funding to undertake impartial investigation, eg for the Wheeler vaccine, nor for the many “snow jobs” that have recently been exposed, eg on Sheridan’s Entelev.

A number of products using skin- contact electrodes (transduction devices) go by the name of Rife devices, but in fact use only audio currents. These have been reported to be of use in pain relief in arthritis at 5000 Hz, and would be expected to stimulate white blood cells, but have not been demonstrated to be of use against cancer. “Hulda Clark Zappers” use high frequency unmodulated currents, supposedly against a pathogenic fluke parasite. Although her theory is most likely flawed by misinterpretation of radionic signatures, it may nevertheless come to be shown as a useful therapy. I have thought she may be reading “slime” rather than liver flukes (or reading AIDS, which has often mistakenly been the case). Various other designs have been produced including magnetic field oscillators, some of which are legal to use and have demonstrated physiological effects. Because of the prevalent prejudice in the scientific establishment, there appears to be no peer reviewed literature to prove or disprove the various claims, and no doubt no adequate funding for such research. Inevitably this boils down to “assignment of priorities”, and the prejudice becomes invisible. If anyone has relevant literature or data, please let me know!

The public are often warned to avoid unproven therapies, and perhaps rightly so. However in view of the acknowledged poor results of orthodox treatments, it must be in the public interest to properly investigate new alternatives. Other authors have pointed out that this logic conflicts with corporate and professional interests. While this unfortunately may have tended to be the case at certain times in many countries, it is to be hoped that compassion and common sense will prevail in the future.

APPENDIX TO DISCUSSION ON BACTERIAL FORMS IN CANCER

A good deal of controversy and misinterpretation has dogged this study, from as far back as Bechamp over 100 years ago, and is still unresolved. Research collated in V. Livingstone- Wheeler’s “Microbiology of cancer” contains various speculations as to the classification of these organisms, including mycobacteria, mycoplasma, Actinomycetales, as well as the then mysterious L-forms or cell- wall deficient forms (cwdf). Wheeler’s “folly” was to declare a new species called Progenitor Cryptocides, What appeared to be a definitive elucidation was later presented by Acevedo et al, who reported the isolation

and identification of a number of non-mycoplasma bacterial species from cancer *29. The American Cancer Society publication CA in its criticism of Wheeler made an unreferenced comment to the effect that Wheeler's organism was not a new species, but a collection of common and rare known types.

A closer reading of the abstract of Acevedo's paper is warranted. The paper reports the expression of hCG- like material, or fragments thereof, from 7 bacterial species isolated from cancer, and cwd forms of 2 bacterial species from non- cancer patients. (I might add at this point that mycoplasmas, had they been present, would not have been isolated by standard culturing techniques.) From Acevedo: " Electron microscopy of these 9 strains" (including comparison of CG negative controls of these strains (sic)) "revealed morphological alterations in the bacterial cell walls and cytoplasmic material and/ or bizarre forms of reproduction in 6 of the 9 strains expressing hCG- like material including the 2 cwd variants."

Majnarich and Wheeler reported the transmission of CG+ characteristic between bacterial species which suggested the transmission of a CG+ plasmid. While this suggestion may be verified in future work, we may speculate on a role of mycoplasmas as a possible CG+ plasmid vector. Acevedo's description of morphological and reproductive alterations in his CG+ isolates begs the question of the mechanism of this manifestation. Is this an effect of CG on the cell? The latter question could be reasonably simply tested by adding CG+ extracts to CG-neg strains in vitro. For the purposes of the current discussion we shall assume that this is not the case. I have found no literature to suggest CG production in mycoplasma studies. However I would like to point out that mycoplasmas have been shown to cause nuclear transformations of hamster fibroblast cells. Also persistent mycoplasma infection showed multi- stage malignant transformation in animal embryo cells which was reversible up to a certain point upon disinfection. Hemolytic activity has been described, including a role of H₂O₂ and NO. Intracellular mycoplasmas have been shown to take up a position near the nucleus. Plasmid transfection studies in mycoplasmas has shown "co-integrate structures". Perhaps mycoplasma supernatants and sonicates could be added to Acevedo's CG neg strains to determine any effects. Other studies could probe for CG plasmids or gene amplification in the CG+ strains, as well as in mycoplasma isolates from cancer. Here we note a reported capacity of mycoplasmas to "take" entire sequences in plasmid transfection in "co-integrate structures" (with the genome), implying their capacity as vectors for a whole range of genetic material of host origin, and possibly indirectly as infective vectors for cancer.

It is also relevant to point out that critics have stressed that any isolates from cancer may merely be opportunistic infections, and this may also be the case for Acevedo's isolates. This argument is countered by the induction of tumours by inoculation, and claims by some oncologists that the blood forms are to be found before any sign of tumour. Of the 7 strains isolated from cancer by Acevedo, none were reported to be cwdf. How can we reconcile this finding with the extensive list of reports of extremely pleomorphic forms by other workers? This conundrum has been neatly side- stepped by critics who imply that all such observations are the result of "contaminations". This thinking may have some justification due to the infamous ability of mycoplasmas to contaminate bacterial

and cell cultures. Also the early special serum supplemented media developed by Wheeler's associates cannot be considered as contaminant free. However the live blood observations and inoculation experiments are overlooked by the critics, and therefore the whole issue should again be thrown open to debate and research. The divide between proponents of the bacterial link to cancer versus a powerful orthodoxy in denial are extreme. The ACS critic claimed that he had never encountered the presence of bacteria as described by Wheeler in cancer tissue, and that they simply "do not exist".

Mycoplasma research has isolated various strains from some cancer tissue, and from blood in AIDS, but not in a majority of cases. I would very much like to see Naessens' organism genotyped. To ensure that any putative mycoplasmas are not discounted yet again as suspected contaminants, it may be appropriate to employ a micromanipulator to individually select the pleomorphs in blood. Also I would like to see Acevedo's work repeated with an emphasis on the culturing requirements of mycoplasmas.

SOMATID CYCLE

Under high resolution light microscopy, Naessens has defined a 16 stage life-cycle for the Somatid, much of which appears similar to mycoplasma phases. However there are controversial differences to Kleineberger- Nobel's PPLO cycle. Dancing dots are shown in Naessens' films which he assigns as Phase 1. These were said to contain no DNA and were stable when heated, whereas chylomicrons (lipid micelles) were not. If these particles are membrane fragments, their composition would be a mix including proteins which may be stable under the heat test. In orthodoxy, the smallest viable elementary bodies are around 0.2 micron, around the size of Naessens' Phase 2. The phase 1 would seem unlikely to be a bacterial precursor.

The Somatid cycle as observed in in vitro culture were reported to go from the "spore" (phase 2, normally kept underdeveloped in vivo by antibody attack against its growth factors) through to rod form (pathological in vivo) which grows out to a mycobacteria-like form with which goes on to develop a bubbled cytoplasm and later bursts. The released substance was said to form "levurid" or yeast like forms which grow out to 4 to 7 micron spherical forms. (In vivo films showed forms like this attached to red cells or free in the plasma in cancer pathology). These grew out in culture to larger long bulbous forms which peristaltically ejected the cytoplasmic content at maturity, and left the sheath or thallus behind. These thalli could be observed in blood in advanced cancer.

Kleineberger- Nobel's work focussed on *M. Pneumoniae*. She describes only one bursting stage at maturity, and therein lies an apparent discrepancy. It is possible that Naessens has isolated more than one species, or grown contaminants. The larger adult phase resembles the flask-shaped species of mycoplasmas, and it may have grown out more slowly. Alternatively, changes in culture conditions over time may have given rise to altered morphology of later maturing forms. The observed thalli in pathology may arise from either bursting form. Naessens commented on different in vitro morphology; a snake-like long thin form was filmed in vitro. It may be that Naessens cycle is indeed basically accurate. Since mycoplasma research has been far from comprehensive, this is

not entirely unlikely.

Demonstrations of hypotonically stressed or heat stressed healthy red blood cells showed the outgrowth of chains, which could coalesce into more spherical forms, and both forms could break away. Similar outgrowths were also demonstrated by W J Clifford by the addition of minute dilutions of toxic metals to blood in isotonic solution. It was claimed that similar phenomena could be observed in cancer, and Naessens assigned the chain shapes as bacterial phase Somatids. W Url argues that these outgrowths consist of the cell membrane, but not any bacterial form. Naessens' film also clearly shows spots in red cells and these were assigned as cancer or pre- cancer indicators. These have also been noted as "sclerotic inclusions" in the work of Enderlein.

ENDERLEIN'S ENDOBIONTS

The Enderlein school describes various forms observed by dark- field microscopy in cancer blood, but I have not studied this material in detail. Endobiont lifeforms are purportedly symbioses of *Aspergilla* and *Mucor*, which can "copulate" from assigned lower forms to a series of higher forms (Beilin). A system of medical therapy including Homoeopathics have been developed to treat cancer via the treatment of the Endobionts. Enderlein developed inoculations of lower forms to induce devolution of native forms. In common with Naessens, Enderlein also ascribes a very small precursor; the protit. The tiny dancing forms would seem to have lively motion, which Naesens attributes to electrostatic repulsion. Perhaps this characteristic caused Enderlein in the 1950's to believe them to be cell precursors. However the weight of modern orthodox opinion would go against such an interpretation. Some blood pictures show these dancing dots, and others, even in advanced cancer, do not. In some cases such forms are seen only after treatment with 714X. I have heard naturopaths ascribe these forms to a result of leaky gut syndrome.

Although the pioneering work of many early researchers may have led them to advance theories which we may view as uninformed or even arcane, we must acknowledge the detail of their observation and consider the general thrust of their findings. The history of science shows many "rediscoveries" of the research of scientists which had fallen into obscurity. The approaches of Livingstone- Wheeler, Rife, and many others may well set the stage for a new wave of therapeutic approaches. As ever, a self-annointed hierarchical priesthood stands in the way of progress. I would like to end this paper with a salient quote from Fleming. "Penicillin sat on the shelf for ten years while I was called a quack".

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