Otto Warburg On The Prime Cause & Prevention of Cancer: Respiration of Oxygen in Normal Body Cells vs. Fermentation of Sugar in Cancer Cells

The Prime Cause and Prevention of Cancer
with two prefaces on prevention

Revised lecture at the meeting of the Nobel Laureates on
June 30, 1966 at Lindau, Lake Constance, Germany

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1883-1970

Preface to the Second Revised German Edition of the Lindau Lecture

(The way to prevention of cancer)

Since the Lindau lecture of June 1966 many physicians have examined - not unsuccessfully - the practical consequences of the anaerobiosis of cancer cells. The more who participate in these examinations, the sooner will we know what can be achieved. It is a unique aspect of these examinations that they can be carried out on human patients, on the largest scale, without risk; whereas experiments on animals have been misleading many times. The cure of human cancer will be the resultant of biochemistry of cancer and of biochemistry of man.

A list of selected active groups of respiratory enzymes will soon be published, to which we recently added cytohemin and d-amino-Levulinic acid, the precursor of oxygen-transferring hemins. In the meantime commercial vitamin preparations may be used that contain, besides other substances, many active groups of the respiratory enzymes. Most of these may be added to the food. Cytohemin and vitamin B 12 may be given subcutaneously. (A synonym of "active group" is “prosthetic" group of an enzyme.)

There exists no alternative today to the prevention of cancer as proposed at Lindau. It is the way that attacks the prime cause of cancer most directly and that is experimentally most developed. Indeed millions of experiments in man, through the effectiveness of some vitamins, have shown, that cell respiration is impaired if the active groups of the respiratory enzymes are removed from the food; and that

cell respiration is repaired at once, if these groups are added again to the food. No way can be imagined that is scientifically better founded to prevent and cure a disease, the prime cause of which is an impaired respiration. Neither genetic codes of anaerobiosis nor cancer viruses are alternatives today, because no such codes and no such viruses in man have been discovered so far; but anaerobiosis has been discovered.[8]

What can be achieved by the active groups, when tumors have already developed? The answer is doubtful, because tumors live in the body almost anaerobically, that is under conditions that the active groups cannot act.

On the other hand, because young metastases live in the body almost aerobically, inhibition by the active groups should be possible. Therefore we propose first to remove all compact tumors, which are the anaerobic foci of the metastasis. Then the active group should be added to the food, in the greatest possible amount, for many years, even for ever. This is a promising task. If it succeeds, then cancer will be a harmless disease.

Moreover, we discovered recently[a)] in experiments with growing cancer cells in vitro that very low concentrations of some selected active groups inhibit fermentation and the growth of cancer cells completely, in the course of a few days. From these experiments it may be concluded that de-differentiated cells die if one tries to normalize their metabolism. It is a result that is unexpected and that

encourages the task of inhibiting the growth of metastases with active enzyme groups.

As emphasized, it is the first precondition of the proposed treatment that all growing body cells be saturated with oxygen. It is a second precondition that exogenous carcinogens be kept away, at least during the treatment. All carcinogens impair respiration directly or indirectly by deranging capillary circulation, a statement that is proved by the fact that no cancer cell exists, the respiration of which is not impaired. Of course, respiration cannot be repaired if it is impaired at the same time by carcinogens.

It has been asked after the Lindau lecture why the repair of respiration by the active groups of the enzymes was proposed as late as 1966, although the fermentation of the cancer cell was discovered as early as 1923. Why was so much time lost?

He who asked this questions ignored that in 1923 the chemical mechanism of enzyme action was still a secret of living nature alone.[1] The first active group of an enzyme, "Iron, the Oxygen-Transferring Part of the Respiratory Enzyme" was discovered in 1924[2]. There followed in two decades the discoveries of the O2-transferring metalloproteins, the flavoproteins and the pyridinproteins, a period that was concluded by the "Heavy Metals as Prosthetic Groups of Enzymes"[3] and by the "Hydrogen Transferring Enzymes"[4] in 1947 to 1949.

Moreover, during the first decades after 1923 glycolysis and anaerobiosis were constantly confused, so that nobody knew what was specific for tumors. The three famous and decisive discoveries of DEAN BURK and colleagues[5] of the National Cancer Institute at Bethesda were of the years 1941, 1956 and 1964: first, that the metabolism of the regenerating liver, which grows more rapidly than most tumors, is not cancer metabolism, but perfect aerobic embryonic metabolism; second, that cancer cells, descended in vitro from one single normal cell, were in vivo the more malignant, the higher the fermentation rate; third, that in vivo growing hepatomas, produced in vivo by different carcinogens, were in vivo the more malignant, the higher the fermentation rate. Furthermore, the very unexpected and fundamental fact, that tissue culture is carcinogenic and that a too low oxygen pressure is the intrinsic cause were discovered[6-8] in the years 1927 to 1966. Anaerobiosis of cancer cells was an established fact only since 1960 when methods were developed[7] to measure the oxygen pressure inside of tumors in the living body.

This abridged history shows that even the greatest genius would not have been able to propose in 1923, what was proposed at Lindau in 1966. As unknown as the prime cause of cancer was in 1923 was the possibility to prevent it.

Life without oxygen in a living world that has been created by oxygen[9] was so unexpected that it would have been too

much to ask that anaerobiosis of cancer cells should be accepted at once by all scientists. But most of the resistance disappeared when at Lindau it was explained that on the basis of anaerobiosis there is now a real chance to get rid of this terrible disease, if man is willing to submit to experiments and facts. It is true that more than 40 years were necessary to learn how to do it. But 40 years is a short time in the history of science.[10]

Wiesenhof über Idar-Oberstein, August 1967
OTTO WARBURG

a) In press in Hoppe-Seylers Zeitschrift für Physiologische Chemie 1967. 10 g riboflavin per ccm or 10 g d-Aminolevulinic acid inhibit in vitro growth and fermentation completely but inhibit respiration less. As expected, ascites cancer in vivo is not cured.

Two years after the Lindau lecture LINUS PAULING (Science Vol. 160, Page 265, 1968) proposed to control mental diseases by adding to the food the active groups of respiratory enzymes. But here the experimental basis was lacking. No mental disease is known so far, the prime cause of which is an impairment of the respiration of brain cells.

Preface to the First Edition
(Prevention of endogenous cancer)

Most experts agree that nearly 80% of cancers could be prevented, if all contact with the known exogenous

carcinogens could be avoided. But how can the remaining 20%, the endogenous or so-called spontaneous cancers, be prevented?

Because no cancer cell exists, the respiration of which is intact[1], it cannot be disputed that cancer could be prevented if the respiration of the body cells would be kept intact.

Today we know two methods to influence cell respiration.[1] The first is to decrease the oxygen pressure in growing cells. If it is so much decreased that the oxygen transferring enzymes are no longer saturated with oxygen, respiration can decrease irreversibly and normal cells can be transformed into facultative anaerobes.

The second method to influence cell respiration in vivo is to add the active groups of the respiratory enzymes to the food of man. Lack of these groups impairs cell respiration and abundance of these groups repairs impaired cell respiration - a statement that is proved by the fact that these groups are necessary vitamins for man.[2]

To prevent cancer it is therefore proposed first to keep the speed of the blood stream so high that the venous blood still contains sufficient oxygen; second, to keep high the concentration of hemoglobin in the blood; third to add always to the food, even of healthy people, the active groups of the respiratory enzymes; and to increase the doses of these groups, if a precancerous state[3] has already

developed. If at the same time exogenous carcinogens are excluded rigorously, then most cancers may be prevented today.

These proposals are in no way utopian. On the contrary, they may be realized by everybody, everywhere, at any hour. Unlike the prevention of many other diseases the prevention of cancer requires no government help, and no extra money.

Wiesenhof, August 1966
Otto Warburg

The Prime Cause and Prevention of Cancer

(Revised Lindau Lecture)

By OTTO WARBURG
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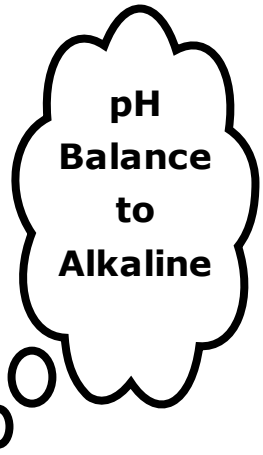
Note by DEAN BURK: Adapted from a lecture originally delivered by O. Warburg at the 1966 annual meeting of Nobelists at Lindau, Germany. O. Warburg won the Nobel Prize in Medicine in 1931 for his discovery of the oxygen-transferring enzyme of cell respiration, and was voted a second Nobel Prize in 1944 for his discovery of the active groups of the hydrogen transferring enzymes. Many universities, like Harvard, Oxford, Heidelberg have offered him honorary degrees. He is a Foreign member of the Royal

Society of London, a Knight of the Order of Merit founded by Frederick the Great, and was awarded the Great Cross with Star and Shoulder ribbon of the Bundesrepublik. His main interests are Chemistry and Physics of Life. In both fields no scientist has been more successful.

There are prime and secondary causes of diseases. For example, the prime cause of the plague is the plague bacillus, but secondary causes of the plague are filth, rats, and the fleas that transfer the plague bacillus from rats to man. By a prime cause of a disease I mean one that is found in every case of the disease.



Cancer, above all other diseases, has countless secondary causes. But, even **for cancer, there is only one prime cause. Summarized in a few words, the prime cause of cancer is the replacement of the respiration of oxygen in normal body cells by a fermentation of sugar. All normal body cells meet their energy needs by respiration of oxygen, whereas cancer cells meet their energy needs in great part by fermentation.** All normal body cells are thus obligate aerobes, whereas all cancer cells are partial anaerobes. From the standpoint of the physics and chemistry of life this difference between normal and cancer cells is so great that one can scarcely picture a greater difference. Oxygen gas, the donor of energy in plants and animals is dethroned in the cancer cells and replaced by an energy yielding reaction of the lowest living forms, namely, a fermentation of glucose.

The key to the cancer problem is accordingly the energetics

of life, which has been the field of work of the Dahlem institute since its initiation by the Rockefeller Foundation about 1930. In Dahlem the oxygen transferring and hydrogen transferring enzymes were discovered and chemically isolated. In Dahlem the fermentation of cancer cells was discovered decades ago; but only in recent years has it been demonstrated that cancer cells can actually grow in the body almost with only the energy of fermentation. Only today can one submit, with respect to cancer, all the experiments demanded by PASTEUR and KOCH as proof of the prime causes of a disease. If it is true that the replacement of oxygen-respiration by fermentation is the prime cause of cancer, then all cancer cells without exception must ferment, and no normal growing cell ought to exist that ferments in the body.

An especially simple and convincing experiment performed by the [US] Americans MALMGREN and FLANEGAN confirms the view. If one injects tetanus spores, which can germinate only at very low oxygen pressures, into the blood of healthy mice, the mice do not sicken with tetanus, because the spores find no place in the normal body where the oxygen pressure is sufficiently low. Likewise, pregnant mice do not sicken when injected with the tetanus spores, because also in the growing embryo no region exists where the oxygen pressure is sufficiently low to permit spore germination. However, if one injects tetanus spores into the blood of tumor-bearing mice, the mice sicken with tetanus, because the oxygen pressure in the tumors can be so low that the spores can germinate. These experiments

demonstrate in a unique way the anaerobiosis of cancer cells and the non-anaerobiosis of normal cells, in particular the non-anaerobiosis of growing embryos.

The Fermentation of Morris Hepatomas

A second type of experimentation demonstrates a quantitative connection between fermentation of tumors and growth rate of tumors.

If one injects rats with cancer-inducing substances of different activities, one can create, as HAROLD MORRIS of the National Cancer Institute in Bethesda has found, liver cancers (hepatomas) of very different degrees of malignancy. Thus, one strain of tumor may double its mass in three days, another strain may require 30 days. Recently DEAN BURK and MARK WOODS 3), also of the National Cancer Institute, measured the in vitro rates of anaerobic fermentation in different lines of these hepatomas, and obtained a curve (Fig. 1) that shows a quantitative relationship between fermentation and growth rate, and therefore between fermentation and malignancy, in these various tumor strains. The fermentation increases with the malignancy, and indeed the fermentation increases even faster than the malignancy.

Special interest attaches to the fermentation of the most slowly growing hepatomas, because several investigators in the United States believed that they had found *) that such tumors had no fermentation; that is that anaerobiosis cannot

be the prime cause of cancer.

*) For example see C. H. BÖHRINGER SON, Ingelheim am Rhein, the factory Work-Journal "Das Medizinische Prisma" , Vol. 13, 1963. Here a lecture of VAN POTTER (Madison, Wisconsin) is reprinted where owing to the slow-growing Morris-tumors anaerobiosis as prime cause of cancer is rejected and the lack of "intracellular feeding back" is claimed to be the real cause of cancer.

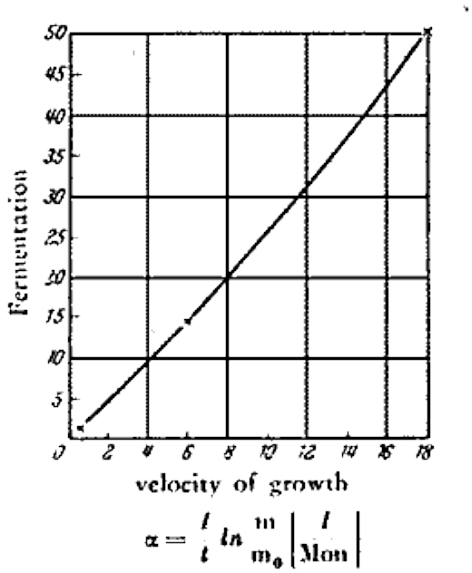


Fig. 1. Velocity of growth and fermentation of the Morris-Hepatomas, according to DEAN BURK and MARK WOODS

DEAN BURK and MARK WOODS saw immediately from their curves that in the region of the zero point the rate of fermentation was so small that it could no longer be measured by the usual gross methodology employed by the aforementioned workers, whereas in the same region the smallest growth rate was always easily measurable. BURK and WOODS saw, in other words, that in the region of the zero pint of their curves the growth test was more sensitive

than the usual fermentation test. With refined and adequate methods for measuring fermentation of sugar (glucose) they found, what any physical chemist after a glance at the curve would realize, that even the most slow-growing Morris hepatomas fermented sugar.

The results of DEAN BURK and MARK WOODS were confirmed and extended by other workers with independent methods. PIETRO GULLINO, also in Bethesda, developed a perfusion method whereby a Morris hepatoma growing in the living animal could be perfused for long periods of time, even weeks, by means of a single artery and single vein, and the blood entering and leaving any given tumor could be analyzed. GULLINO found with this method that the slow-growing Morris hepatomas always produced fermentation lactic acid during their growth. This was in contrast to liver, where, as known since the days of CLAUDE BERNARD, lactic acid is not produced but consumed by liver; the difference between liver and Morris tumors in vivo is thus infinite (+ vs. -). GULLINO further found that tumors grow in vivo with diminished oxygen consumption. In summary, GULLINO's findings indicate that the slow-growing Morris hepatomas are partial anaerobes. SILVIO FIALA, a biochemist at the University of Southern California, found that not only did the slow-growing hepatomas produce lactic acid, but also that the number of their oxygen-respiring grana was reduced.

The slow-growing Morris hepatomas are therefore far removed from having refuted the anaerobiosis of tumors.

On the contrary, they are the best proof of this distinctive characteristic. For forty years cancer investigators have searched for a cancer that did not ferment. When finally a non-fermenting tumor appeared to have been found in the slow-growing Morris tumors, it was shown to be a methodological error.

Transformation of Embryonic Metabolism into Cancer Metabolism

A third type of experiment, from the institute in Dahlem with coworkers GAWEHN, GEISSLER and LORENZ, is likewise highly pertinent. Having established that anaerobiosis is that property of cancer cells that distinguishes them from all normal body cells, we attacked the question, namely, how normal body cells may become transformed into anaerobes 6)7)8).

If one puts embryonic mouse cells into a suitable culture medium saturated with physiological oxygen pressures, they will grow outside the mouse body, in vitro, and indeed as pure aerobes, with a pure oxygen respiration, without a trace of fermentation. However, if during the growth one provides and oxygen pressure so reduced that the oxygen respiration is partially inhibited, the purely aerobic metabolism of the mouse embryonic cells is quantitatively altered within 48 hours, in the course of two cell divisions, into the metabolism characteristic of fermenting cancer cells. Fig. 2 illustrates the very simple experimental procedure involved.

If one then brings such cells, in which during their growth under reduced oxygen pressure a cancer cell metabolism has been produced, back under the original high oxygen pressure, and allows the cell to grow further, the cancer metabolism remains. The transformation of embryonic cell metabolism into cancer cell metabolism can thus be irreversible, and important result, since the origin of cancer cells from normal body cells is an irreversible process. It is equally important that these body cells whose metabolism has thus been transformed into cancer metabolism now continue to grow in vitro as facultative anaerobes. The duration of our experiments is still too limited to have yielded results of tests of inoculation of such cells back into mice, but according to all previous indications such cells will later grow as anaerobes upon transplantation into animals.

Increase partial pressure of Oxygen pO2

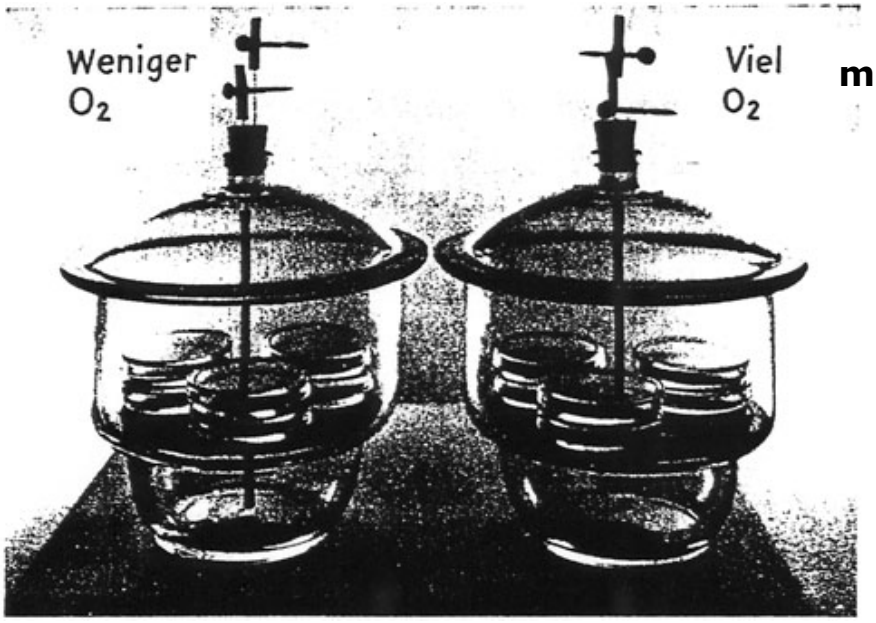
In any case, these experiments belong to the most important experiments in the field of cancer investigation since the discovery of the fermentation of tumors. For cancer metabolism, heretofore, measured so many thousand of times, has now been induced artificially in body cells by the simplest conceivable experimental procedure, and with this artificially induced cancer metabolism the body cells divide and grow as anaerobes in vitro*).

*) The experiments were at once repeated, when they were published, of course without acknowledgment. See for example Th. Goodfriend, D. M. Sokol and N. O. Kaplan, J. molecular Biol. 15, 18, 1966.

In recent months we have further developed our experimental arrangements so that we can measure manometrically the oxygen respiration and fermentation of the growing mouse embryonic cells during the metabolic transformation. Fig. 3 shows the experimental arrangement. We find by such experiments that 35 percent inhibition of oxygen respiration already suffices to bring about such a transformation during cell growth**). Oxygen pressures that inhibit respiration 35 percent can occur at the end of blood capillaries in living animals, so that the possibility arises that cancer may result when too low oxygen pressures occur during cell growth in animal bodies.

**) These experiments show, like the curve of Dean Burk and Mark Woods in Fig. 1, that it is more correct to designate tumor cells as "partial anaerobes" rather than "facultative anaerobes". A body cell is transformed into a tumor cell if only a part of the respiration is replaced by fermentation.

less O2



more O2

Fig. 2. Method to transform embryonic metabolism into cancer metabolism by decreasing the oxygen pressure

(Weniger O_2 = Less O_2 Viel O_2 = Much O_2)

The induction of cancers by solid materials injected into animals is a further experimental indication of this possibility. If one implants discs of solid substances under the skin of rats, the discs will soon be surrounded by capsules of living tissue that will be nourished with blood vessels from the hypodermis. Sarcomas very frequently develop in these capsules. It is immaterial whether the solid discs are chemically plastics, gold, or ivory, etc. What produces the cancer is not the chemical nature of the solid discs, but the special kind of blood nourishment supplied to the tissue encapsulating the discs. This blood provision varies with the site and in adequacy within a given animal, and induces cancer from the low oxygen pressure in the encapsulating disc.

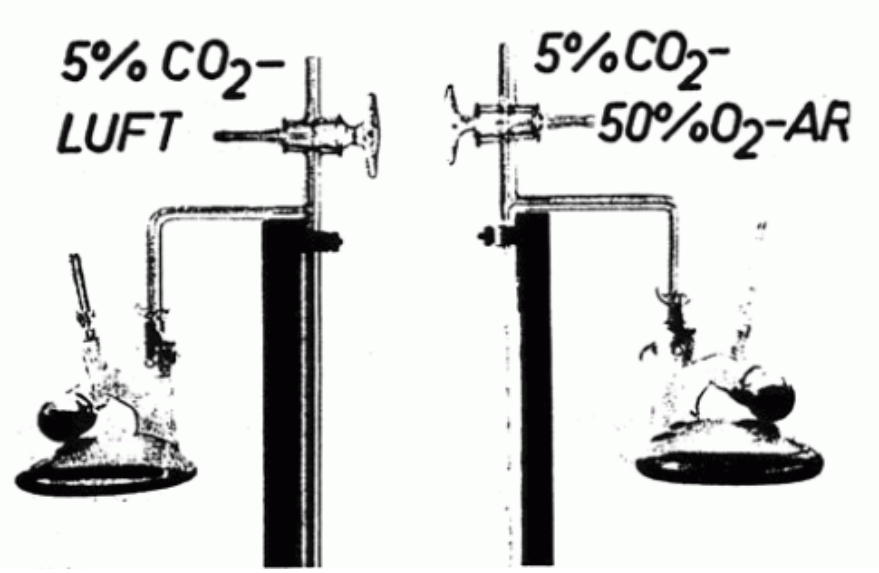


Fig. 3. Method to measure manometrically respiration and fermentation during the transformation of embryonic into

cancer metabolism*)
(Luft = Air)

*) The vessels are not shaken, because shaking inhibits growth. Therefore, the oxygen pressure in the liquid phase at the bottom of the vessels is much lower than in the gasphase. For example, when the oxygen pressure in the gas phase was 2000 mm H2O it was at the bottom of the vessels 130 mm H2O. (O. Warburg, A. Geissler and S. Lorenz, Zeitschr. für Naturforschung 20b, 1070, 1965.)

Thermodynamics

If a lowered oxygen pressure during cell growth may cause cancer, or, more generally, if any inhibition of respiration during growth may cause cancer, then a next problem is to show why reduced respiration induces cancer. Since we already know that with a lowering of respiration fermentation results, we can re-express our question: Why does cancer result if oxygen-respiration is replaced by fermentation?

The early history of life on our planet indicates that life existed on earth before the earth's atmosphere contained free oxygen gas. The living cells must therefore have been fermenting cells then, and, as fossils show, they were undifferentiated single cells. Only when free oxygen appeared in the atmosphere - some billion years ago - did the higher development of life set in, to produce the plant and animal kingdoms from the fermenting, undifferentiated

single cells. What the philosophers of life have called "Evolution créatrice" has been and is therefore the work of oxygen.

The reverse process, the dedifferentiation of life, takes place today in greatest amount before our eyes in cancer development, which is another expression for dedifferentiation. To be sure, cancer development takes place even in the presence of free oxygen gas in the atmosphere, but this oxygen may not penetrate in sufficient quantity into the growing body cells, or the respiratory apo-enzymes of the growing body cells may not be saturated with the active groups. In any case, during the cancer development the oxygen-respiration always falls, fermentation appears, and the highly differentiated cells are transformed to fermenting anaerobes, which have lost all their body functions and retain only the now useless property of growth. Thus, when respiration disappears, life does not disappear, but the meaning of life disappears, and what remains are growing machines that destroy the body in which they grow.

But why oxygen differentiates and why lack of oxygen dedifferentiates? Nobody would dispute that the development of plants and animals and man from unicellular anaerobes is the most improbable process of all processes in the world. Thus there is no doubt, that EINSTEIN descended from a unicellular fermenting organism - to illustrate the miracle, molecular O2 achieved. But according to the thermodynamics of Boltzmann,

improbable processes require work to take place.

It requires work to produce temperature differences in a uniformly temperatured gas; whereas the equalization of such temperature differences is a spontaneous process that does not require work. It is the oxygen-respiration that provides in life this work, and dedifferentiation begins at once when respiration is inhibited in any way. In the language of thermodynamics, differentiation represents a forced steady state, whereas dedifferentiation - that is, cancer - is the true equilibrium state. Or, illustrated by a picture: the differentiated body cell is like a ball on an inclined plane, which, would roll down except for the work of oxygen-respiration always preventing this. If oxygen respiration is inhibited, the ball rolls down the plane to the level of dedifferentiation.

But why respiratory energy and not fermentation energy can differentiate, whereas in general, for example in growth, respiratory energy and fermentation energy are equivalent? Obviously, there would be no cancer if there were not this discrimination of fermentation energy, that is, if fermentation like respiration could differentiate. Then, when respiration is replaced by fermentation, fermentation would take over differentiation, and a high state of differentiation would be maintained even in the fermenting body cells.

Chemistry

Physics cannot explain why the two kinds of energy are not

equivalent in differentiation; but chemistry may explain it. Biochemists know that both respiration energy and fermentation energy do their work as phosphate energy, but the ways of phosphorylation are different. If one applies this knowledge to carcinogenesis, it seems that only oxidative phosphorylation but not fermentative phosphorylation can differentiate, a result, that may in future explain the mechanism of differentiation.

Yet Biochemistry can explain already today why fermentation arises, when respiration decreases. Figure 4 shows that the pathways of respiration and fermentation are common as far as pyruvic acid. Then the pathways diverge. The endproducts of fermentation is reached by one single reaction, the reduction of pyruvic acid by dihydro-nicotinamide to lactic acid. On the other hand, the endproducts of the oxidation of pyruvic acid, H2O and CO2, are only reached after many additional reactions. Therefore, when cells are harmed, it is probable that first respiration is harmed.

In this way the frequency of cancer is explained by reasons of probability.

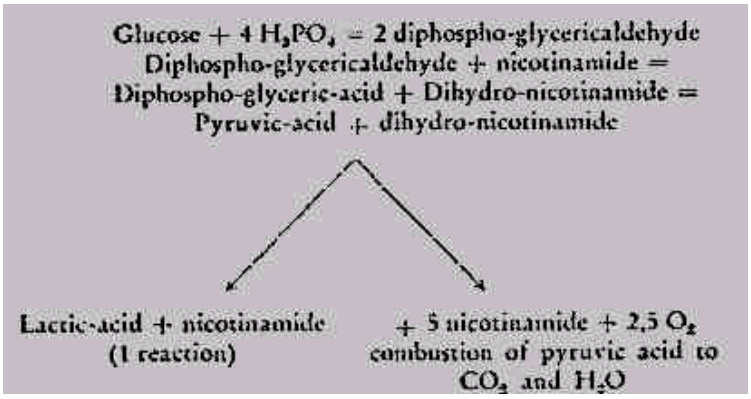


Figure 4

To sum up:

1. Impairment of respiration is [more] frequent than impairment of fermentation because respiration is more complicated than fermentation.
2. The impaired respiration can be easily replaced by fermentation, because both processes have a common catalyst, the nicotinamide.
3. The consequence of the replacement of respiration by fermentation is mostly glycolysis, with death of the cells by lack of energy. Only if the energy of fermentation is equivalent to the lost energy of respiration, is the consequence anaerobiosis. Glycolysis means death by fermentation, anaerobiosis means life by fermentation.
4. Cancer arises, because respiration, but not fermentation, can maintain and create the high differentiation of body cells.

To conclude the discussion on the prime cause of cancer, the virus-theory of cancer may be mentioned. It is the most cherished topic of the philosophers of cancer. If it were true, it would be possible to prevent and cure cancer by the methods of virology; and all carcinogens could be eaten or smoked freely without any danger, if only contact with the cancer virus would be avoided.

It is true that some virus-caused cancer[b)] occur in animals,

but no one sure human virus-cancer has been observed so far, whereas innumerable substances cause cancer without viruses in animals and man. Thus viruses do not meet the demands of Pasteur, that is must be possible to trace the prime cause in every case of the disease. Therefore science classifies viruses as remote causes of cancer, leading to anaerobiosis, the prime cause that meets the demands of Pasteur.

b) The chicken Rous sarcoma, which is labeled today as a virus tumor, ferments glucose and lives as a partial anaerobe like all tumors. O. WARBURG, Bioch. Zeitschrift 160, 307, 1925; F. WIND, Klinische Wochenschrift, Nr. 30, 1926.

Many may remember how anaerobiosis as prime cause of cancer was recently disputed emphatically, when one single cancer - the slow Morris hepatomas - was believed (wrongly) to lack in fermentation. In contrast the virus theory is adhered to although all cancers of man are lacking in virus-origin. This means the surrender of the principles of Pasteur and the relapse into bygone times of medicine.

Applications

Of what use is it to know the prime cause of cancer? Here is an example. In Scandinavian countries there occurs a cancer of throat and esophagus whose precursor is the so-called Plummer-Vinson syndrome. This syndrome can be healed when one adds to the diet the active groups of respiratory

enzymes, for example: iron salts, riboflavin, nicotinamide, and pantothenic acid. When one can heal the precursor of a cancer, one can prevent this cancer. According to ERNEST WYNDER 3) of the Sloan-Kettering Institute for Cancer Research in New York, the time has come when one can exterminate this kind of cancer with the help of the active groups of the respiratory enzymes.

It is of interest in this connection that with the help of one of these active groups of the respiratory enzymes, namely nicotinamide, tuberculosis can be healed quite as well as with streptomycin, but without the side effects of the latter [c)]. Since the sulfonamides and antibiotics, this discovery made in 1945 is the most important event in the field of chemotherapy generally, and encourages, in association with the experiences in Scandinavia, efforts to prevent cancer by dietary addition of large amounts of the active groups of the respiratory enzymes. Since there can scarcely be overdosage, such experiments can do no harm.

c) V. CHORINE: C. R. sci. Paris, 220, 150 (1945). – H. FUST and A. STUDER, Schweizerische Z. für allgemeine Pathologie, Band 14; Fasc 5 (1951).

I would like to go further and propose always making dietary additions of large amounts of the active groups of the respiratory enzymes after successful operations when there is danger from metastatic growths. One could indeed never succeed in redifferentiating the dedifferentiated cancer cells, since during the short duration of human life

the probability of such a back-differentiation is zero. But one might increase the respiration of growing metastases, and thereby inhibit their fermentation, and - on the basis of the curve of DEAN BURK and MARK WOODS obtained with the Morris hepatomas - thereby inhibit the growth of metastases to such an extent that they might become as harmless as the so-called "sleeping" cancer cells in the prostates of elderly men.

A Second Example of Application

The physicist MANFRED VON ARDENNE has recently attacked the problem of the therapy of cancer. ARDENNE discovered that cancer cells owing to their fermentation, are more acid – inside and on their surface – than normal cells and hence are more sensitive to high temperatures. On this basis, he and his medical colleagues have treated cancer patients, after surgical removal of the primary tumors, by raising the body temperature of the patients to about 109° Fahrenheit for an hour, in the hope that the metastases will then be killed or their growth so slowed up as to become harmless. It is not yet decided whether this idea can be described as a practical success. But the provisional work of ARDENNE is already of great significance in a field where hopes of conventional chemotherapy have been dimmed but might be brightened by combination with extreme or moderate hyperthermy.

A third application. According to an estimate by K. H. Bauer of the Cancer Institute in Heidelberg, at least one

million of the now living twenty five million male inhabitants of West Germany will die of cancer of the respiratory tract; still more will die from other cancer. When one considers that cancer is a permanent menace, one realizes that cancer has become one of the most dangerous menaces in the history of medicine.

Many experts agree that one could prevent about 80% of all cancers in man, if one could keep away the known carcinogens from the normal body cells. This prevention of cancer might involve no expenses, and especially would require little further research to bring about cancer prevention in up to 80 percent *).

*) Since this estimate was published, some thought 80% even too low. Yet prevention remained taboo and early diagnosis was the only consolation that was offered.

Why then does it happen that in spite of all this so little is done towards the prevention of cancer? The answer has always been that one does not know what cancer or the prime cause of cancer [might] be, and that one cannot prevent something that is not known.

But nobody today can say that one does not know what cancer and its prime cause [may] be. On the contrary, there is no disease whose prime cause is better known, so that today ignorance is no longer an excuse that one cannot do more about prevention. That prevention of cancer will come there is no doubt, for man wishes to survive. But how long prevention will be avoided depends on how long the

prophets of agnosticism will succeed in inhibiting the application of scientific knowledge in the cancer field. In the meantime, millions of men must die of cancer unnecessarily.

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A comment by Wilhelm H on the relationship of cancer researcher Dr. Johanna Budwig with Otto Warburg, Szent-Györgyi and other illustrious scientists:

The lecture of Warburg is very interesting. It is obviously very good since he was the discoverer of the importance of

oxygen in cell respiration. Yet it was also a sad witness of his closed mind towards Dr. Budwig's discovery. He gave the lecture some 15 years after Dr. Budwig had found the missing link which he had unsuccessfully searched for. Dr. Budwig was by this time in full swing of healing cancer patients with her Oil-Protein Diet. Here is what Dr. Budwig said:

"I assumed that Professor Warburg would recognize the significance of my discovery regarding the essential fatty acids and their role in the electro-kinetic power of the cell. In 1952 I repeatedly sent him my works and wrote to him. He refused to meet with me to discuss it."

Official history unscrupulously omits Dr. Budwig's contribution, and telling the truth tarnishes some shining names. Warburg was not the only Nobel winner to ignore Dr. Budwig's vital discovery. Other giants of scientific history missed the significance of "fat."

Dr. Budwig is quoted in an interview in the following manner: "Szent-Gyorgyi dealt with my published works, but he never acknowledged me," she said bitterly. "For example, he ignored me in his book Electronic Biology and Cancer, even though he was fully aware of my discovery."

More on Budwig's relationships to other scientists at Linseed and Fatty Acids: Johanna Budwig. For a thorough introduction to the subject of Johanna Budwig's natural healing protocol for cancer and other degenerative disease, see Dr. Budwig's Healing Diet & Protocol, including the

complete list of Healing Cancer Naturally articles on Dr. Budwig's protocol. Unsolicited visitor's comment: "I have been educating myself on the Budwig protocol and your site is by far the most informative."

Also see Sugar and Cancer and Cancer Bacteria (breast cancer).

While the animal research (vivisection results) quoted by Dr. Warburg may indeed be transferrable to humans in that in the cited cases humans react similarly as did the animals, in the great majority of cases the opposite is true, see Animal Experimentation Unscientific: Physicians Convincingly Argue That Animal Testing Seriously Impedes Progress in Human Medicine While Vivisection Industry Profits.

Introduction and complete list of Healing Cancer Naturally articles on ”known”, suspected and probable causes of cancere

Suppressed and forgotten cancer microbe research could hold the key
to the cause and cure of this dread disease

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Despite a century of cancer research the cause of breast cancer remains unknown. Age, diet, stress, hormone factors, genetic predisposition, and cancer viruses are all suspected as possible causative factors, but totally ignored are infectious bacteria which have been implicated in breast cancer, as well as other forms of cancer.

Over the past century physicians such as James Young, John Nuzum, Virginia Livingston, and numerous other researchers have reported on the existence of a specific microbe associated with cancer. The microbe is a ubiquitous, pleomorphic organism with a complex life cycle. A review of this literature suggests that the cancer microbe plays an important role in the development of malignancy. Because the etiology of cancer remains unknown, this research deserves further examination and reaappraisal.

William Russell (1852-1940) and ”the parasite of cancer”

A century ago when major diseases like tuberculosis, leprosy, and syphilis were discovered to be bacterial (not viral) infections, many physicians suspected bacteria might also cause cancer. At the close of the nineteenth century (when the science of microbiology was in its infancy), many different microbes (variously called ”cancer coccidia,” ”sporozoons” and ”cancer parasites”) were cultured from cancer. A few of these microbes produced cancer tumors when injected into animals, but many did not.

On December 3, 1890 William Russell, a pathologist in the School of Medicine at the Royal Infirmary in Edinburgh, gave an address to the Pathological Society of London in which he outlined his histopathologic findings of ”a characteristic organism of cancer” that he observed microscopically in fuchsine-stained tissue sections from all forms of cancer that he examined, as well as in certain cases of tuberculosis, syphilis and skin infection (1).

The parasite was seen within the tissue cells (intracellular) and outside the cells (extracellular). The size of Russell’s parasite ranged from barely visible, up to ”half again as large as a red blood corpuscle.” The large size of some of these bodies suggested a fungal or yeast-like parasite. Russell provisionally classified the parasite as a possible ”blastomycete” (a type of fungus); and called the round forms ”fuchsine bodies” because of their bluish-red staining qualities.

The idea of a cancer germ (especially one that could also be

identified in TB and syphilis) was received cautiously. Nine years later in 1899, in another report on ”The parasite of cancer” appearing in The Lancet (April 29), Russell admitted that finding cancer parasites in diseases other than cancer was indeed a ”stumbling block.” By this time a considerable number of scientists concluded that Russell bodies were merely the result of cellular degeneration of one kind or another. Furthermore, no consistent microbe was cultured from tumors; and the inoculation of these microbes into animals produced conflicting and often negative results (2).

Russell was trained as a pathologist, not as a microbiologist, and he avoided getting into the bacteriologic controversies regarding various microbes grown from cancer. He simply concluded, ”It seems almost needless to add that there remains abundant work to be done in this important and attractive field.”

After three years’ work at the New York State Pathological Laboratory of the University of Buffalo, Harvey Gaylord confirmed Russell’s research in a 36 page report titled ”The protozoon of cancer”, published in May, 1901, in the American Journal of the Medical Sciences. Gaylord found the smallest round forms, which were the size of ordinary staphylococci, as well as the larger spherical bodies which were the size of fungal spores (3) .Russell’s 1899 paper ended his writings of a cancer parasite, but his discovery quickly became known to pathologists as ”Russell bodies.” These bodies continue to fascinate researchers and

physicians (like myself) up to the present time.

When Russell died at the age of 89 in 1940, the British Medical Journal published a large obituary noting that he was universally respected and had served at one time as President of the Royal College of Physicians. No mention was made of his ”cancer parasites” or his ”bodies”, except to remark that ”in his earlier years Russell devoted much time to the study of the cancer cell. (For further details, see ”The Russell body: The forgotten clue to the bacterial cause of cancer” at www.rense.com/general44/russell)

The microbiology of cancer

The idea of a cancer parasite was finally dismissed in 1919 by noted American pathologist James Ewing. In his popular textbook, *Neoplastic Diseases*, he declared: ”Few competent observers consider it (the parasitic theory) as a possible explanation in cancer.” In Ewing’s opinion, cancer did not act like an infection. Therefore, he concluded that microbes couldn’t possibly cause it. He wrote: ”The general facts of the genesis of tumors are strongly against the possibility of a parasitic origin.” [4] Subsequently, few doctors dared to contradict Ewing by investigating bacteria in cancer.

Nevertheless, during the 1920s a few persistent physicians like pathologist John Nuzum of the University of Illinois College of Medicine; surgeon Michael Scott from Butte, Montana; and obstetrician James Young of Edinburgh, Scotland, continued to publish research showing that

bacteria were implicated in breast cancer and other forms of cancer.

Working independently of one another, all three researchers cultured unusual bacteria from breast cancer, as well as from breast cancer tumors in mice. The peculiar growth of the ”pleomorphic” cancer germ defied the established laws of microbiology by its ability to change shape and form, depending on how it was cultured in the laboratory, as well as the amount of oxygen supplied for growth and the age of the culture.

At first, the germ was barely visible as tiny round coccal forms. Later, these cocci enlarged into rod-shaped bacteria, which could connect together to form chains resembling a fungus. Small cocci could also enlarge into larger yeast and fungal-like spore forms.

Nuzum grew his ”micrococcus” from 38 of 41 early breast cancers, and from the cancerous lymph nodes and metastatic tumors resulting from spread of the cancer to other parts of the body.[5-6] During his 6 years of intensive bacteriological study, he learned the microbe could pass through a filter designed to hold back bacteria, indicating that some forms of the microbe were as small as the size of some viruses. With special stains he detected these small round coccoid forms within the breast cancer tumor cells. Although Nuzum couldn’t produce cancer tumors in mice, he was able to induce breast cancer tumors in 2 of 5 dogs injected with the microbe.

In a dangerous human experiment he injected the groin of a 70-year-old man with the bacteria he cultured from breast cancer. After 62 injections over an 18-week period, a skin cancer formed in the man's groin. This experiment showed that breast cancer microbes were also capable of producing a different kind of cancer, such as skin cancer. [6]

Young found his microbe in 16 cases of breast cancer, and in two mice with breast cancer. He identified "spore forms" and clumped "spore balls" in microscopic sections prepared from the mouse tumors. [7-8]

Scott described three stages in the life cycle of his parasite: rod forms, spore or coccus-like forms, and large spore-sacs resembling a fungus. [9-10] He treated cancer patients with an effective antiserum against these microbes, and spent the rest of his life trying to alert his colleagues to the infectious cause of cancer. But the antagonism of the medical profession to Scott's cancer parasites and his antiserum was overwhelming, and he died a forgotten man.

Virginia Livingston and the cancer microbe

During the last half of the twentieth century cancer microbe research was barely kept alive by a quartet of women, now all deceased. The published research of Virginia Wuerthele-Caspe Livingston-Wheeler (a physician), Eleanor Alexander-Jackson (a microbiologist), Irene Diller (a cellular biologist) and Florence Seibert (a chemist) provides indisputable evidence that bacteria are implicated in cancer.

Livingston, who never let the male-dominated medical profession intimidate her, independently discovered the cancer microbe in the late 1940s and never stopped talking about it until her death in 1990, at the age of 84. Aided by Alexander-Jackson, who supplied the bacteriologic expertise, they became an unstoppable research team. [11-15]

The two women found a special stain (the acid-fast stain) that allowed the microbe to be recognized in culture and within the cancer tumor. Like the researchers back in the 1920s, they confirmed the microbe was filterable; and electron microscopic photos provided further proof that the filterable forms were indeed viral-size. Livingston named the microbe "Progenitor cryptocides", which greatly angered cancer experts, microbiologists, and American Cancer Society spokespersons, all of whom insisted the cancer microbe did not exist!

In the 1950s Irene Diller of the Institute for Cancer Research at Fox Chase, Philadelphia, discovered fungus-like microbes in cancer cells. Joining forces with the Livingston team, Diller worked with specially bred mice with a proven cancer incidence. By injecting them with microbes cultured from breast cancer and other tumors, she was able to more than double the cancer incidence of the mice. [16]

She injected healthy animals with cancer bacteria. When cancer tumors developed she successfully cultured the microbe from the tumors—thus proving that these bacteria were implicated in the production of cancer. Utilizing

Livingston's methods, Diller also grew the microbe from the blood of cancer patients.

Florence Seibert and cancer bacteria

In the early 1960s Florence Seibert became so impressed with Diller's research that she quit retirement to help prove that bacteria cause cancer. Back in the 1920s Seibert devised a method to make intravenous transfusions safe by eliminating contaminating ubiquitous bacteria. Later, as one of the foremost authorities investigating the chemistry and immunology of the acid-fast bacteria that cause tuberculosis, she perfected the skin test for tuberculosis that has been used worldwide ever since. In 1938, she was awarded the famed Trudeau Medal, the highest prize given to tuberculosis research.

Experiments conducted by Seibert and her research team showed these acid-fast and TB-like cancer microbes were not contaminants because they were able to isolate bacteria from every piece of tumor (and every acute leukemic blood) they studied. [17]

In her autobiography, *Pebbles on the Hill of a Scientist*, published privately in 1968, she wrote: "One of the most interesting properties of these bacteria is their great pleomorphism. For example, they readily change their shape from round cocci, to elongated rods, and even to thread-like filaments depending upon what medium they grow on and how long they grow. This may be one of the

reasons why they have been overlooked or considered to be heterogeneous contaminants...And even more interesting than this is the fact that these bacteria have a filterable form in their life cycle; that is, that they can become so small that they pass through bacterial filters which hold back bacteria. This is what viruses do, and is one of the main criteria of a virus, separating them from bacteria. But the viruses also will not live on artificial media like these bacteria do. They need body tissue to grow on. Our filterable form, however, can be recovered again on ordinary artificial bacterial media and will grow on these. This should interest the virus workers very much and should cause them to ask themselves how many of the viruses may not be filterable forms of our bacteria."

Seibert's provocative papers, some published by the prestigious Annals of the New York Academy of Sciences, should have caused a stir. But with the quartet slowly closing in on the infectious cause of cancer, funds from previous supporters (like the American Cancer Society) suddenly dried up. All cancer microbe researchers eventually discovered that studying cancer bacteria was the kiss of death as far as funding was concerned. And without adequate funding, this type of cancer research was made more difficult.

But coming from thirty years of research into the acid-fast bacteria that cause tuberculosis, Seibert knew that the discovery of a pleomorphic and acid-fast microbe in cancer was tremendously important. She fervently believed that

knowledge of this microbe would be instrumental in developing a possible vaccine and more effective antibiotic therapy against cancer. In *Pebbles* she confided: "It is very difficult to understand the lack of interest, instead of great enthusiasm, that should follow such results, a lack of certainty not in the tradition of good science. The contrast between the progress made in tuberculosis where we know the cause, where we have good general diagnostic tests, where we have a vaccine and effective antibiotic controls, and that made in cancer with the millions invested, is very striking. Some dedicated scientists should indeed find it rewarding to confirm or deny these painstaking and time-consuming experiments, for the sake of establishing the first necessary step in the important problem of the etiology of cancer."

Like the other women, Seibert observed the virus-like forms of the cancer microbe within the nucleus of the cancer cells. She theorized this infection could disrupt and transform nuclear genetic material that could lead to malignant change. Even though cancer microbes might appear to be simple and common microbes, their ability to infiltrate the nucleus of cells meant they were far from harmless.

In 1990, at the age of 92, Florence Seibert was inducted into the National Women's Hall of Fame, along with Barbara Jordan (Government), Billie Jean King (Athletics) and Margaret Bourke-White (Arts). When she died the following year her passage was noted in *Time* and *People* magazines, and in major newspapers like *The Los Angeles Times*. All the

obituaries mentioned her contributions to the safety of intravenous fluids and her great achievement with the TB skin test. But not a word was written about her cancer microbe research, to which she devoted the last thirty years of her life.

Breast cancer and cancer-associated bacteria

Each year 190,000 American women are diagnosed with breast cancer. And the prognosis is still dismal for women whose breast cancer has spread to the lymph nodes and beyond. Yet the medical establishment remains adamantly and irrationally opposed to cancer microbe research. It is perhaps understandable from an economic viewpoint that the medical profession would not welcome a proposed infectious cause of cancer that would challenge the highly lucrative multibillion-dollar cancer industry.

Physicians confidently ignore cancer bacteria because they have been carefully taught in medical school that there are no significant bacteria detectible in cancer. They still believe that cancer microbes represent contaminant bacteria or bacteria of no significance. Thus, published reports of cancer microbe research are rarely cited and the subject remains virtually unknown.

The idea of a microbe with virus, bacteria, and fungal-like stages is also anathema to most doctors. However, over the past several decades the study of cell-wall deficient bacteria and ”mycoplasma-like” bacteria (which are both bacterial

and viral-like) indicates that microbes indeed have a complex life cycle. In 1919, when Ewing offered his damning opinion of "cancer parasites", none of these microbiologic peculiarities was recognized!

In some instances, cancer microbe research appears to be deliberately suppressed. For example, the National Cancer Institute informs viewers about Virginia Livingston and states: "There is no scientific evidence to confirm her theories of cancer causation or to justify her treatments." (bccancer.bc.ca/PPI/UnconventionalTherapies/LivingstonTherapy.htm) Obviously, this official judgment is not accurate because, as we have noted, Livingston's discoveries have been confirmed by many competent scientists.

In addition, Livingston has written three books on the cancer microbe: *Cancer: A New Breakthrough* (1972), *The Microbiology of Cancer* (1977), and *The Conquest of Cancer* (1984). [18-20] More recent books on bacteria in cancer include Alan Cantwell's *The Cancer Microbe* (1990) [as well as his *Four Women Against Cancer: Bacteria, Cancer & the Origin of Life* published in 2005] and *Can Bacteria Cause Cancer?* (1997) by David J Hess. [21,22]

Next

Introduction and complete list of Healing Cancer Naturally articles on "known", suspected and probable causes of cancer

Breast Cancer and Cancer Bacteria ctd.
Suppressed and forgotten cancer microbe research could hold the key
to the cause and cure of this dread disease
2004 by Alan Cantwell, Jr., M.D.
ctd. from previous page

As discovered by Virginia Livingston, the acid-fast stain is the essential stain to detect cancer bacteria in histopathologic microscopic tissue sections from breast cancer. Using acid-fast staining techiques, bacteria have been identified in breast cancer, lymphoma, Kaposi's sarcoma (the so-called "gay cancer" of AIDS) and other forms of cancer. [23-25] Figure 1-5 show bacteria identified in breast cancer and in the metastasis to the skin. Figure 6-8 show the appearance of Staphylococcus epidermidis cultured from the breast tumor metastasis to the skin. All these microphotographs are from a woman who died of breast cancer at age 40, one year after her breast cancer and several positive lymph nodes were removed. A careful

perusal of these photographs reveals that the cocci cultured from the tumor are similar, if not identical, to the coccoid forms seen in the original breast cancer tissue. Smaller numbers of microbes were also identified in ”normal” and cancer-free breast tissue removed at the time of surgery. This suggests that the bacteria are not ”secondary invaders” because they are identifiable in areas *before* the tissue has been invaded by cancer cells. [23]

Click on each figure to see a larger, higher-resolution rendering.

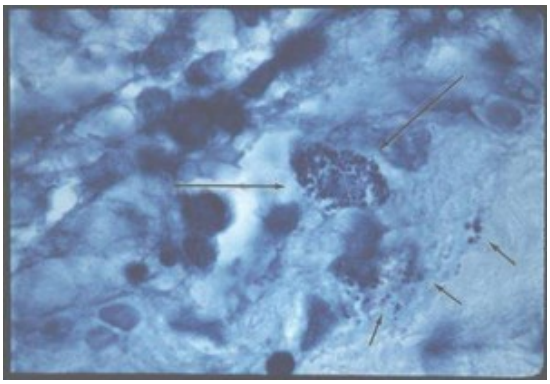
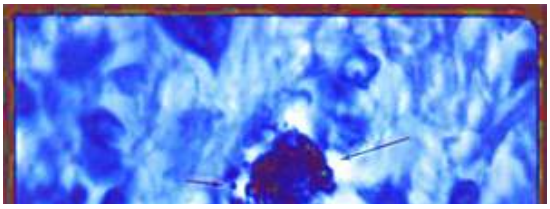


Fig. 1: Histopathologic tissue section from infiltrating ductal carcinoma of the breast. Long arrows point to a cluster of intracellular coccoid forms; short arrows point to scattered extracellular coccoid forms. Intensified Kinyoun’s (acid-fast) stain; magnification x 1000, in oil.



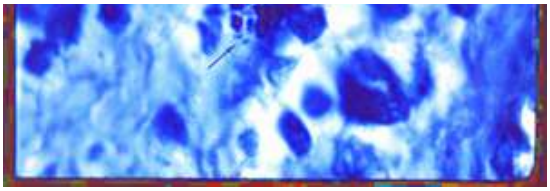


Fig. 2: Tissue section of breast cancer. The long arrow points to a collection of variably-sized round & coccoid forms tightly packed around a cell nucleus. The larger round forms have the appearance of Russell bodies. Short arrows point to tiny coccoid forms resembling the size of ordinary staphylococci at the periphery of the cell. Intensified Kinyoun's (acid-fast) stain; magnification x1000, in oil.

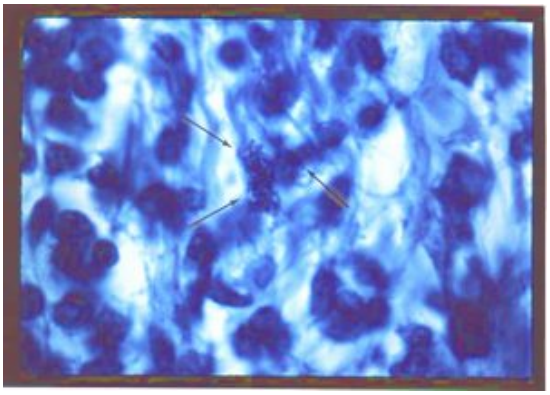


Fig. 3: Tissue section of breast cancer. Arrows point to a focus of tiny extracellular coccoid forms. Intensified Kinyoun's (acid-fast) stain; magnification x1000, in oil.

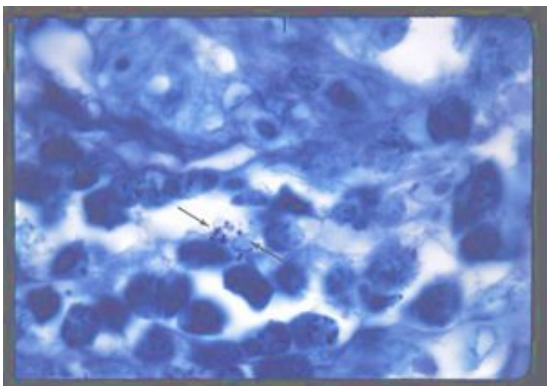


Fig. 4: Tissue section of breast cancer. Arrows point to a small focus o extracellular coccoid forms scattered among the cancerous cells. Intensified Kinyoun's (acid-fast) stain, magnification x1000, in oil.

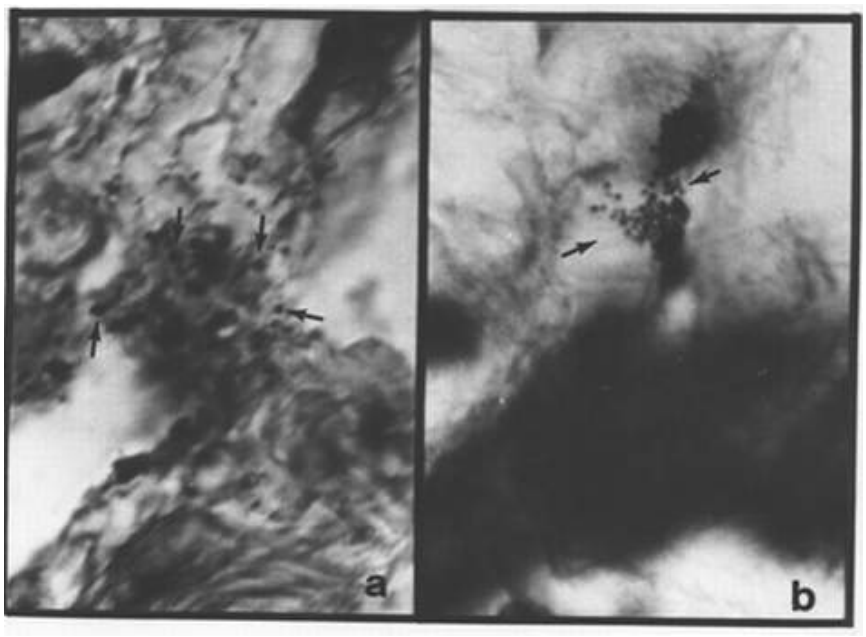


Fig. 5 A, B: Black and white photo of tissue section of skin showing metastasis of breast cancer to the skin. Arrows point to intra and extracellular collections of coccoid forms in the dermis of the skin. Intensified Kinyoun's (acid-fast) stain, magnification x1000, in oil.

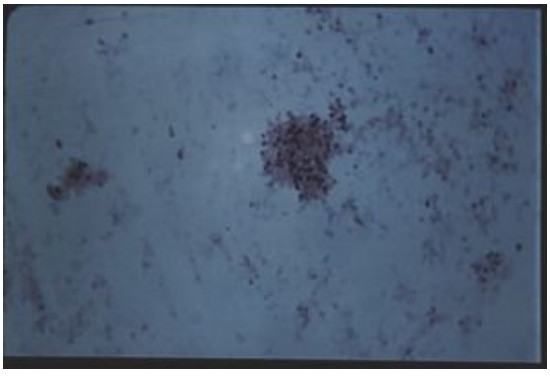


Fig. 6: Gram's-stained smear of Staphylococcus epidermidis cultured from the metastasis of the breast cancer to the skin, illustrated in fig. 5. The bacteria are Gram-variable; Some of the forms stain purple, the typical color of "Gram-positive" staphylococci. Other cocci stain pink, suggesting poorly-staining, possible cell-wall-deficient forms of staphylococci.

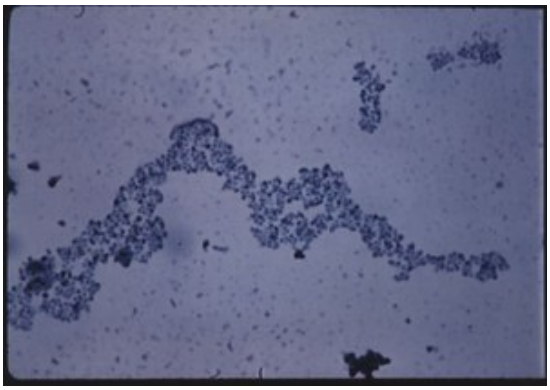


Fig. 7: Same culture as Fig. 6, but showing the appearance of the staphylococci when stained with the Ziehl-Neelson (acid-fast) stain. Note that the size and shape of the staphylococci are identical in size and shape to the small coccoid forms seen in the original breast tumor (Figures 1-4) and in the skin tumor metastasis (Figure 5). Magnification x1000, in oil.

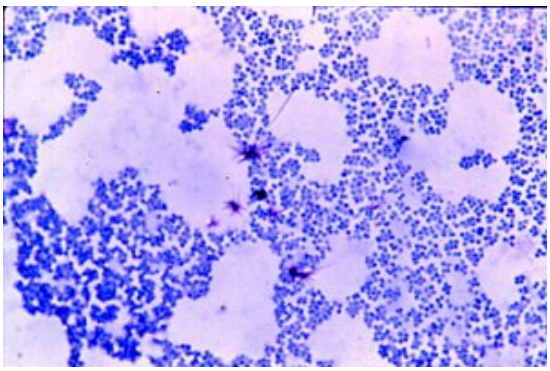


Fig. 8 : Ziehl-Neelson (acid-fast) stain of Staphylococcus epidermidis cultured from a metastatic skin lesion from breast cancer. Note the large dark-stained granules from which acid-fast (red and pink) thin, sharp ”spicules” emerge. In their 1970 paper [13], Livingston and Alexander-Jackson showed exactly the same type of acid-fast spicule growth in culture from the urine of a cancer patient (their Figure 12A). (Their research regarding "a specific type of organism cultivated from malignancy" was presented at the New York Academy of Sciences in November 1969.)

Radical treatment and the need for more bacteria research

The current lack of knowledge about the cause of breast cancer has resulted in the recommendation of some very expensive and death-defying treatments for this horrendous disease. Bone marrow transplants, which carry a 5% death rate, are being proposed as a routine treatment, at a minimal cost of $100,000 per patient.

As described in Karen Stabiner’s *To Dance with the Devil: The New War on Breast Cancer* (1997), the procedure is not pretty. [26] First, a catheter is placed in a woman’s chest to deliver the drugs. A surgical treatment is then performed to scrape out bone marrow from her pelvis, followed by 7 days of growth hormone injections. Then starts days of intravenous chemotherapy that can cause kidney and bladder damage. A catheter is placed in the bladder, followed by a round of intravenous BCNU, or carmustine, a drug that makes a

woman feel like she is falling down drunk. Patients become sleepy, sullen, disoriented, agitated, and angry. Loss of bowel control and vomiting are common. After all this, women are put into isolation because the white count drops precipitously, making her vulnerable to all sorts of infections. There may be inexplicable spiking fevers and rashes, and the inevitable loss of hair. After three weeks, patients are allowed to go home where they are told to watch for "interstitial pneumonitis," a potentially fatal aftereffect if not diagnosed and treated early.

Bone marrow transplant for breast cancer is not guaranteed, nor is it considered a cure. Women have been known to die of cancer three months after the procedure, proving that some patients do not respond to chemotherapy no matter how high the dose.

Even with radiation, chemotherapy and surgery, the cost of dying of cancer is not cheap. At the price patients are paying, physicians should not have the luxury of being ignorant about cancer microbe research, particularly when these microbes can be identified in cancer tumors.

With 40,000 American women dying annually from breast cancer, it is time medical science reevaluated the parasite of cancer that James Ewing so casually dismissed in 1919. Perhaps if he hadn't been so adamant about cancer microbe research, his colleagues might have been able to do more to save him when he himself eventually died of bladder cancer.

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Biography:

Dr. Cantwell is a retired dermatologist, and an AIDS and cancer researcher. He is the author of "The Cancer Microbe", "AIDS and the Doctors of Death" and "Four Women Against Cancer: Bacteria, Cancer & the Origin of Life".
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other items) through my Amazon links (US, UK & Canada) and take advantage of Amazon's (often) low prices.

"This article was originally published by Patient Focus, Inc. The mission of Patient Focus is to build humanity back into cancer treatment and make it more bearable while a cure for cancer continues to escape us. Patient Focus has a keen interest in research pertaining to the microbiology of cancer. Also please note that Dr. Cantwell's work regarding cancer-associated bacteria is not related (in his view) to the research of Hulda Regehr Clark and her theories of cancer causation."

Addendum by Dr. Alan Cantwell
regarding a BBC News report of October 10, 2004, on the subject of "Antibiotic (doxycycline) can 'turn off cancer'" (see below):

"For more than a century a small group of researchers, including myself, have implicated bacteria in cancer (see my book, THE CANCER MICROBE, Aries Rising Press). Now it turns out that a common antibiotic -- doxycycline -- can turn off a gene in mice that leads to liver cancer.

Let's hope it doesn't take another century for scientists and physicians to follow up on this, and to explain why they keep ignoring cancer-causing bacteria. For more information on "cancer microbes" -- [do an internet search] and type in

those exact words."

Antibiotic can 'turn off cancer'

adapted from BBC MMIV

Scientists have shown that a common antibiotic can turn off cancer cells in mice, offering hope of new treatments for cancer patients.

The antibiotic worked by turning off a gene called Myc, which is known to trigger cancer.

Mice remained cancer free for as long as they took the drug. When it was stopped they developed liver cancer, the Stanford University team found.

Cancer experts said the Nature study held promise for human cancer drugs.

Cancer switch

The findings might also apply to cancers of the breast, bowel and prostate, the researchers hope.

This is because all of these cancers, as well as liver cancer, begin in cells that line the body called epithelial cells.

According to Cancer Research UK, the gene may contribute to as many as one in seven cancer deaths.

"Drugs blocking Myc might be effective cancer treatments

in the future." Dr Elaine Vickers from Cancer Research UK

The Stanford scientists studied mice whose liver cells had been altered to carry a modified Myc gene known to cause cancer.

Myc controls cell division. Unlike the normal version of the gene, the modified version stayed permanently switched on, meaning cells were constantly dividing and some became cancerous.

Feeding the mice the antibiotic doxycyline turned the faulty Myc gene off so cancer growth was blocked.

When the researchers stopped the doxycycline the mice developed aggressive liver cancer.

Reintroducing doxycycline into their feed not only turned Myc back off, blocking further cancer growth, but it also turned the cancer cells back to normal.

Reversing cancer 

MMS
Cancer, Virus,
Bacteria, Parasites

Lead researcher Dr Dean Felsher said: "The exciting thing is you can turn cancer cells into something that appears to be normal."

But he said even though the cells looked normal, they still had the ability to become cancerous if the antibiotic were to be stopped.

This could explain why some cancers come back after

people have had chemotherapy, he said.

"This is a terrible cancer. Anything that is encouraging in liver cancer may be important," he said.

Dr Elaine Vickers, science information officer for Cancer Research UK, said: "The Myc gene is known to be overactive in many types of cancer."Estimates suggest that the gene may contribute to as many as one in seven cancer deaths.

"This research is very interesting.

"It adds to the weight of evidence suggesting that drugs blocking Myc might be effective cancer treatments in the future."