The
SURVIVAL FACTOR
In
NEOPLASTIC AND VIRAL
DISEASES

An Introduction to
Carbonyl and Free Radical Therapy

A Study o f the Phenomena of the Free Radical, the Double Bond, and its Alpha Placed Hydrogen Atom in the Pathogenesis and Correction o f Neoplastic, Viral and Bacterial Diseases

By

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PREFACE

The Global Natural Health Advocate's effort to represent this material was substantial, gleaned from various sources, and compiled to accurately represent original form. This being said, form is continuity of Koch's work and a print style reflective of the time these were first issued. Form is retaining page numbering, photos, evidentiary items, and other minutia to provide researchers with landmarks for further research. Some characteristics are only practical in electronic formats. While there is careful editing of all materials, there exists the possibility of content error. There are many technical terms, specific chemical names, some of which are not currently in use, and other obscure nomenclatures. As time permits an extensive glossary of terms and a concordance index will be generated. This will require completion of the entire electronic rendering of topics, therefore time and energy awaits.

Computerized data facilitates corrections of unintentional transcriptional error. There will be a shakedown phase, but at some point, content and form should be quite consistent with originals. Issuance of this compendium will use computer applications that may also evolve over time, but as a legacy provide a foundation for an architecture actually derived in part from Dr. Koch's work. Eventually, paper, bound, printings will be available on request. This latter, is not a priority, but once the initial phases are complete are the least difficult to accomplish. Since this is an anthology of Fredrick Koch work, it has commentary, addendums, and some biographical material.

Commentary is designed to communicate Koch's work to lay persons, professionals, including researchers. However, researchers are not the primary target, educated laity and alternative practitioners are. Researchers could have approached this subject matter a long time ago with objectivity and earnestness, but they have not. More is the pity, it appears that ordinary people and practitioners will have to force feed a recalcitrant system into reclaiming respectability by judicious review of a now lapsed method.

Frederick Koch knocked on the doors of science, medicine, and academia with bible in hand to share his work. The result was disastrous for him professionally and personally. However, despite attack from the very people he sought to communicate, he continued to perfect his process. Some did pay attention and their statements were clear and distinct.

Well versed in the printed disparagements of Dr. Koch's work, it was with a heavy heart that I first set out to visit him. I resented spending the time which must be taken from my own practice in order to do this, for that year had been the most industrious and the most profitable in my experience. **Bluntly I asked him if he could cause the disappearance of cancer, sufficiently often, to make it clear this was caused by his treatment.** He assured me he could do so, inviting me to return as often as I desired and to follow some cases through their recovery period. I adopted his suggestion, going to his clinic often, following up cases I had seen treated, and learning from personal observation that he was helping cancer, in a fashion unmistakable. His records were abundant, correctly kept, and through the months I found they were never altered or falsified.

Always he wished to give his discovery to humanity, and lie desired the cooperation of others in improving what lie had so well commenced. Therefore it was with his consent, and for this purpose that I took Honorable Forbes Godfrey, M.D., Minister of Health in the Government of Ontario to meet Dr. Koch, and to observe his work.

The first visit lasted three days, and three months later we both returned. For the next two years he went himself, somewhat frequently. He took supplies home with him, and used the treatment in the homes of his own patients. He was thrilled by what he was observing, and profoundly stirred, when this diligence in following up the actual work, enabled him to arrive at a decision, and gave him tile right to hold an opinion. He expressed his opinion, that radium did not offer any promise of solving the cancer problem, while praising the merits of the Koch treatment, and he strongly opposed the Purchase by the government, of a supply of radium. He had a clear right to his opinion that the Koch treatment had outstanding virtues, since he had seen recovery of several cases of cancer during its use, and he was happy to announce, "Repeatedly Dr. Koch has stated he is willing and desirous of revealing his formulas to medicine just as soon as his treatment has had proper investigation and endorsement by a medical body." (Toronto Star, April 21, 1930.) This was the position he maintained as Minister of Health through a general election which the government won, he being reelected in his own constituency.[1]

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[1] **THE CHEMISTRY OF NATURAL IMMUNITY**, (revised edition) William Frederick Koch, Ph. D., M.D., CHRISTOPHER PUBLISHING, HOUSE, Boston, U.S.A., 1939, first printing 1924. Commentary at introduction by D. H. Arnott, M.D. London, Ontario, Canada.
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It is very clear that Dr. Koch's skills and insights eclipsed even some of those that were Nobel Laureates, mostly because the exact process by which he produced his survival factor compounds are not currently commonly known by chemists. Specifics have been obscured over time, again, a pity. This will be a resurrection project for the last phase of completing the puzzle.

Compilers of this anthology believe Dr. Koch's mistake was to attempt to elicit approval from his peers. Conclusions regarding what generated animus are not entirely certain, but it is known that economic interests were predominant. This issue is about economic sectors, entrenched belief structures, corporate interests, and less than laudable motives.

There were obviously some well ensconced in power and authority that appreciated his achievements. One of these appears to be Willard Dow, a founder of Dow Chemical, who referred to him as one of the world's greatest living chemists.[2] Of course, there were others whose knowledge permitted them to competently review the achievement his work represented. However, even prestigious persons were insufficient to derail what was essentially a pogrom directed toward all methods, and paradigm orientations, that challenged economic interest and the dominance of income streams.[3] Efforts on his behalf were also diminished, dismissed, and discarded over time. Koch's work is largely unknown to the public.

The answer is there is a cure for cancer, and many other conditions, people are being deceived, and great fortunes are amassed perpetrating this deception. Evidence is clear that suppressive measures are orchestrated and intentional, designed in venal form to shunt income streams regardless of effectiveness of treatments used or shunned. This is a basic theme from a sociopolitical-economic standpoint, and not ignored, but tertiary to presentation of the facts associated with Koch's work.

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[2] Dr. Koch dedicated this book to Dr.'s Dow and Hale, founders of Dow Chemical. [GO2] The GO2's are a feature of electronic systems frequently utilized in this work. The use value of this should be clear. Some references or specifics are itemized and linked in collected index zones. This zone will be continuously contributed to as materials become available and time permits. Again, the attributes of electronic media permit this over and above the frozen printed forms.

[3] Insert hyperlink to GNHA pages.
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The implications of Koch's method reach deeply into the treatment of many niches of healing, inclusive of psychiatric or emotional disorders. This, of course, renders further insight into paradigm considerations in that mind body issues are further elaborated for examination by readers and researchers. This renders the schisms and demarcations of specialties less definitive as presented in many mind body models of current consideration discussed in expansive studies such as the Chantilly Workshop on Nonconventional Medicine. [4]

Initially readers should recognize Frederick William Koch was not a charlatan, did not fabricate an elaborate hoax, nor was woefully misdirected. He was an earnest, dedicated, courageous, eminently skilled person. He did seminal work on physiologic function early in life that should have secured his renown and deserved continued recognition and respect from his peers.[5]

These words should not have to be written, but when a person is expunged from history, excommunicated, if they elucidate a truth, fight for a justice, or labor for liberty, and oppressive, castigating and minimizing intentional actions are taken against him, her, or them, an initial statement of reputation is sometimes impacting to readers. His efforts, interests, and desires were humanitarian, and designed to solve one of the oldest and most pernicious plagues humankind, and animals experienced throughout history. This is, of course, cancer. His work also impacted many other conditions almost to derive a pan-therapeutic agent, method, to address a plethora of acute and chronic conditions. This appears to apply to epidemiological clusters of diseases like tuberculosis, Hanson's disease,[6] and most probably inclusive of most forms of pandemic influenza, infantile paralysis, to itemize a few. This is quite a claim. Paradigm orientation is part of the spectrum of issues; money, power, and truth are part of what is at stake.[7]

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[4] Link to other work contained in Global Natural Health Advocates [GNHA] Internet pages. This workshop, and work product, however, gave poor representation of the Koch work.

[5] Hyperlink to GNHA Koch page. Koch determined the function of the parathyroids, 1914.

[6] Leprosy.

[7] Thomas Khun elaborated the term "Paradigm" as an identifier of worldviews and conducts in **The Structure of Scientific Revolutions**, 1962. To Kuhn, "paradigm" is a conceptual model explaining sets of scientific observations creating a framework to fit the observations. Philosophically this is not a novel, only an adaptation of positional attributes of the observer in relation to explanations of universal processes. There are teleologic and ontologic
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Although the contentious attributes of Koch's life derived from economic sources, his paradigm confrontations derived from his facile integration of what currently is presented as a holistic model of physiologic function. This is, however, non specific, and unclear in terms of what might be considered a philosophical orientation regarding the treatment of conditions. Koch does elaborate.

> …In other words, a mature statement can now be made that so far as malignancy is concerned, normal healthy animal tissues contain substances that prevent and abolished its basic pathology.
>
> As might be expected, no time was spared in the effort to learn the chemical structure and mode of action of the active substances concerned. These substances are all diffusible bodies of small molecular weight held in absorption by the lecithin cephalin fractions of tissue extracts, from which they can be removed by dialysis, and exist in exceedingly small quantity indeed. Their identification next led to the synthetic production and trial, not only in malignancy but in the various infections and allergic states commonly met with. I was also able to prepare synthetically the same bodies in somewhat more dynamic form than the tissue extracts yielded. From I these studies, the following conclusions are drawn:
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> 1. Natural immunity is a general property of tissues depending upon the presence of metabolites concerned in the oxidation process ordinarily belonging to production of energy for function. The metabolites seem to serve as photo-chemic sensitizers or catalysts.
> 2. The sufficiency in these tissue metabolites constitutes the resistance to disease and prevents mal- or dysfunction in general.
> 3. Deficiency in these materials eventuates in an interruption in the progress of oxidation and function. Consequently susceptibility to infection and symptoms and structural changes of disease are produced.

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characteristics to this, although some might prefer the terms empirical and rationalistic, each are merely complications or adaptations of the same universal cognitive functions ascribed to sentience, sapience and cognitive structuralization of reality. Kuhn described how paradigms alter and resist alteration particularly in science.
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4. Deficiency in these substances may result from exhaustive muscular effort, the exhaustion from too severe exposure to cold from the lytic action of poisons upon the tissue colloids that raises the surface tension and causes a washing out or leaking away of the important metabolites, and finally from the action of toxic molecules that inhibits or paralyzes the action of the metabolites through photochemic interference. We will deal with the theoretical and clinical aspects of each case as we go along.

Photochemic action has scarcely been investigated with reference to the part it plays in normal and pathological conditions, but in the industries it is receiving more and more employment, judging by issues from the patient offices, and it is in this field that theoretical to chemistry physics and mathematics have made the greatest strides of late. Inasmuch as the known carcinogenic substances are all fluorescent, the influence of photochemic action in neoplasia cannot be ignored. Indeed it offers a ready explanation not only of the action of the carcinogenic toxins but points out what should characterize the chemical structure of the recovery-producing and immunity-preserving substances. Correctly enough, the structures suggested are exactly those provided by the oxidation metabolites belonging to the natural immunity mechanism, as we shall soon see.[8]

Koch was a scientist and physician; this is a far more rare combination than lay people believe. He was a denizen of a particular time frame, one where so called biomedicine[9] had not yet devoured the health care sector. He became embroiled in battling an establishment not from libertarian or radical ideas or ideals, but because the establishment attacked him and his work. The work he pursued to all accounts because it was there, a problem to be solved, questions unanswered, and the conquest of disease the stage for intended victory. This is quite a pristine objective of life, work, and exploration for the sake of discovery and benefit.

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[8] **NATURAL IMMUNITY**, ITS CURATIVE CHEMISTRY IN NEOPLASIA — ALLERGY — INFECTION, A REPORT TO THE RESEARCH INVESTIGATION COMMITTEE, William Frederick Koch, Ph. D., M. D., 1936

[9] While there are various etymologies of the term biomedicine, some from the early twentieth century, circa 1914, significant usage of the term did not arrive until after the 1940's. At the time of Koch's early work, 1914-20 approximately, the economic units that opposed his innovations were in fact the conventional medical community, that is, his own profession. Of course, to typify this enclave in terms that are not entirely vogue today, it was allopathic medicine.
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Although, this alone did not make him righteous or honorable, just an extremely talented, insightful person, looking at things differently than norm, and deriving answers that transcended norm. The honor came from the courage it took to continue after narrow and selfish forces attempted to step on him, discredit what he found to be true, that which he saw work, and benefits to individuals he assisted to regain heath and productive lives. As an aside, it was not merely people benefiting. Veterinary sciences also benefit. For example, his researches and successes in treating bovine mastitis are clear examples of pan therapeutic application and non placebo[10] effects.[11]

To be continued.

~~~~~~~~FOOTNOTES~~~~~~~~

[10] Link to placebo effects [GO2] Chantilly Workshop, Mind-Body Effects.

[11] Insert hyperlinks to A Least Common Denominator in Antibiotics by Albert L. Wahl, M.D., C.M. and GNHA Koch pages. [GO2] Use document map to acquire veterinary applications until full integration. Also GNHA Koch page, re photo with Larry Thatcher.
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NATURAL IMMUNITY SERIES

Cancer and Its Allied Diseases, 1926, 1929
Natural Immunity, 1934, 1936
The Chemistry of Natural Immunity, 1939
The Survival Factor in Cancer and Viral Infections, 1955, 1958
Portuguese Edition 1960

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TABLE OF CONTENTS

(Continued on next page)

TABLE OF CONTENTS (Continued)

DEDICATION

This book is dedicated to the memories o f two leaders in American Science and Industry, Dr. Willard H. Dow, and Dr. William J. Hale. Their humanitarian genius was great enough to build the vast Dow Chemical Company to its present, proportions and service, and also take interest in other humanitarian efforts, such as our own, which they investigated fully, evaluated carefully, and then supported effectively in our court battle.

ACKNOWLEDGEMENTS

Gratitude is due Professor Joseph Maisin, of Louvain University, for his many experiments in small animals with and without the writer, from which conclusions o f fact could be drawn.

Likewise gratitude is due Dr. Willard Dow, Dr. William Hale, Dr. Drake, Dr. Rubens and other Dow scientists for every help in every need, especially for their winning defense against United States Government attacks instigated by competitive drug interests.

FOREWORD

The world's leading surgical journal, the London Lancet, gave an editorial review of the present status of surgery in the treatment of cancer. It gave the same conclusions as did Sir James Paget a century ago, when he stated in his text on Cancer that this is not a surgical disease, that the condition was profoundly constitutional, and that operated cases did not live as long on an average as those that were left untouched. From 1910 to 1950, the American Cancer Control Society created an energetic propaganda that 85% of breast cancer could be cured surgically or by irradiation, and that early diagnosis was a prime advantage. Now after the statistics are analyzed the Lancet quotes the world's leading surgeons on the results of early operation with the same discouraging conclusions as Sir James Paget stated a hundred years ago. In the meantime life insurance statistics and others established the fact that operated cases, the early cases, lived less by two and a half months than the inoperable, far advanced cases that were not operated. Add to this two and a half months the year or so it took the early case to become inoperable and advanced, one sees that surgery done with all its courage, sacrifice and dexterity is not the attack that is required to win against this disease. The Lancet states, "The intensive campaigns to awaken the public to keep on the watch for tumors and report for the earliest possible diagnosis and treatment has met with good response, but the anticipated drop in the mortality rate did not follow." "Despite a long and intensive educational program for the early detection and treatment of cancer, the death rate from cancer of the breast shows no downward trend." In fact, "The comparative mortality index, which allows for changes in the age structure of the population, shows for men a rise of 6% in cancer mortality between 1938 and 1950." "The size of the primary tumor is no guide to curability; two-thirds of patients reporting with tumors of the breast which were smaller than a hazel-nut already showed metastases," and with regard to lung cancer, "If recent experience is typical, however, by the time definite abnormality appears in the radiograph, most cases of pulmonary cancer have progressed too far for successful resection." "Survival rates after simple excision, radical mastectomy, and irradiation, are depressingly uniform." "Our basic approach may be wrong; the attempt to treat cancer as a local disease rather than a general disease, may be as irrational as treating syphilis by excising the primary chancre." "In most if not all lethal breast cancer, remote spread takes place by the blood stream before interference is practicable." "The survival rates after different periods of delay before seeking medical advice often shows a curious paradox. Thus Swynnerton and Truelove reviewing 395 cases of gastric carcinoma, showed that the greater the delay and the longer the history of symptoms the greater was the survival rate." Here we find in the Lancet of April 3, 1954, p. 714, with other statements of similar import, the conclusions of the world's most advanced surgeons. A year later Dr. George Crile of the famous Crile Clinic in Cleveland gave thorough information to the profession and the public on this subject and was in exact agreement. Now comes the report of the 12th annual scientific

meeting of the Detroit Institute of Cancer Research. The consensus was the same, Dr. Harden B. Jones, professor of medical physics at the University of California, gave the ultimatum, The odds for or against the recovery from cancer are set long before the patient sees a physician." and "There is no evidence that treatment by surgery or radiation, the only recognized methods of therapy affect the course of the really malignant forms of cancer." and "Early treatment is a nice theory, but there is no evidence that it benefits the patient." "Some drastic cancer therapies not only do not help but are harmful."" "The tumor easily could have a billion cells before it is large enough to be recognized as cancer. Some of these cells are already in the blood stream."

Unfortunately, radiation does not answer the needs of the patient, but adds to the basic pathology. The convention of the American Roentgen Ray Society of September 1954, added to the report of the Roengtenologist of the University of Pennsylvania in 1925 when he stated that irradiation before and after surgery opened the vascular and lymph spaces and helped the spread of the disease instead of retarding it. His report was so unpopular that it was suppressed. But today the statistics are so disheartening that even the radiation therapists are bold in reporting that where deep therapy is poured into a neoplasm of one type, a more malignant form or a bone sarcoma is created underneath only too often. That the Survival factor is destroyed by irradiation is seen in the hereditary defects in the offspring of radiologists. These show in some 10,000 children, of radiologists, twice the incidence of cancer and more defects in eyes, heart and blood, than in children of physicians not exposed to irradiation. Eight to ten times more radiologists die of leukemia than general practitioners. When one recalls that viruses are thousands of times more resistant to irradiation than tissue or cancer cells, the situation is logical.

Fifty years ago nothing was known about cancer except the diagnosis, which was about all there was to become expert in. The gross and microscopic pathology was so well learned that the resort to the biopsy was regarded as a sign of poor training (Ewing) (Warthin). Our professor of pathology insisted that we make 100% correct diagnoses and give the microscopic description from the gross findings alone. Every surgeon on the University staff did it regularly.

Today, however, high specialization makes the biopsy an essential for many. For many years ahead of my day, all that was known beyond diagnosis was that cancer was caused by "irritation." But no one knew exactly what "irritation" meant, or how it operated to cause cancer. Further, there was no information to serve as a starter to investigate the problem. But still the walks through the hospital wards fervently cried for the solution. The surgeon was doing his untiring best and the radiologist hoped and hoped that his approach might some day prove fruitful. And yet no facts stepped forth to show how to even make a start, — nothing from within the cancer properties themselves.

So the writer decided it might be helpful to get the basic facts on any of the deepest injuries to the body chemistry that could be produced, observe their effects on

every tissue quality possible, and then figure out how any of these changes might take part in the pathogenesis of cancer. The effects of complete parathyroidectomy were chosen for this purpose, largely because the great experts of that day on this very subject seemed to have overlooked the main factors in parathyroid insufficiency, and because a subject as important as that should be at least reasonably explored.

As the writer's investigations progressed in accumulating more data it began to appear that he made the correct start. The findings were carefully evaluated, the conclusions drawn, and from these a postulate was formulated and tested out in the broad field of disease. It was hoped that if the venture would be propitious, a century of ignorance would be hurdled, and a basis for investigating the cancer problem itself would be reached. A landing in barren territory simply called for a fresh start and another trial. However, the first attempt proved fortunate. Our postulate had been drawn up with every effort at precision, and the conclusions were fruitful. Under the circumstances this was even more important than if our interpretations were correct or "true." For the aim was to reach a position of utility.

The utility has two leading aspects. One lies in the proof that the four primary cell functions — contraction, secretion, conduction, and cell division — are provided with energy that is produced and received by each functional unit in accord with one and the same pattern, and when interrupted so as to produce disease, tile fault is the same in pattern and subject to the same type of correction by one and the same atomic structure. The other phase of utility is the explanation of both viral and neoplastic parasitisms, the atomic bondings and electronic ionic displacements that constitute the integration of the pathogen with the host cell, which not only accomplish the pathogenesis, but actually provide for and invite the oxidative cleavage that leaves the host cell in normal functional perfectly reconstructed, and the virus no longer to be found. The text demonstrates this as well as the fate of the neoplastic cell and the process by which it is disposed of. These matters are based on firm chemical laws as the text will show.

So whether the cell contractile fibrillae as in asthma, or the secreting fibrillae as in hay fever, or the conductile fibrillae as in a compulsory neurosis, fir some other phase of insanity, or the mitotic fibrillar system as in neoplasia, happens i0 be attacked, the basic pathology is the same and its correction is necessarily the same, too. This is the subject we will demonstrate in this text. *We* have no thought that our presentation is the best that could be made. However, since we have opened the door and uncovered the mysteries it cm enclosed, it is our chore to make the disclosure. This door stands open for endless investigation and for collaboration as well. It should be inviting for *our* proofs of the cure of the many forms of cancer offered in this text stand firm, firstly in their diagnoses made by America's leading surgeons, with the patients housed in our proudest institutions where every facility for a firm diagnosis was at hand. Then, too, the clinical diagnoses were confirmed by our foremost pathologists. Secondly, the cures were demonstrated to be permanent with reconstruction of tissue so good function was

restored, and accomplished by a definite process without leaving even a microscopic trace of cancer cells.

It will be seen that whether the correction happens to be in far advanced cancer of the vital organs, widely metastasized, and the patient in extremis, or the correction happens to be in the terminal phase of rabies, hog cholera, or some other 100% fatal viral disease, the reversal of the pathogenesis follows the same definite order. his physiological aspect of the correction, we will attempt to show, depends upon well proven laws in chemistry that are basic to tissue cell energy production and energy use, and primarily basic to all vital processes. Thus a least common denominator in pathogenesis and its correction has been reached. It serves as a key to the interpretation of disease production and also to its correction in the whole field we have investigated so far.

CHAPTER I

THE POSTULATE

The initial work that spearheaded the Survival Factor Investigations was a research into the cause of the convulsions and deaths that always followed complete parathyroidectomy. The findings were published in "The Journal of Biological Chemistry" 12; 313, 1912, Koch, and 15; 43-63, 1913, Koch. This work was confirmed by Prof. Patton and his staff at the University of Glasgow, and published in the "Quarterly Journal of Physiology" in 1917 using two of the four numbers. numbers. For the care and excellency with which the confirmation was made, Patton was awarded the Triennial Prize in Medicine by Harvard University. This confirmation is of great importance because of the broad field of applicability of the facts brought forth and also the depth of their interpretations of disease processes.

The cardinal facts were just three:

(1) guanidin, methyl guanidin, and some other toxic bases were produced in the tissues and eliminated in the urines in fatal amounts that increased until the dog died in convulsions;

(2) calcium, lactate, and phosphate were eliminated in excess;

(3) the post-mortem findings showed antemortem coagulation of the blood in the large veins, and hemorrhagic degenerations of the liver and kidneys.

From these findings, several important conclusions were made, based upon:

(1) the chemistry of guanidin and its derivatives showing the activation of its amine group by its conjugation with an imide group, and also the tendency to deactivate this amine group by such substitutions as acetic acid as in creatine and amino-valeric acid in arginine;

(2) the fact that guanidin and methyl guanidin are highly toxic while creatine and arginine are not;

(3) The large elimination of lactic acid even while the lungs were well ventilated showing that fuel was not burned via an oxidation process, but was fermented hydrolytically to produce lactic acid, and, thus, the oxidation mechanism was blocked at its very inception;

(4) the block to oxygen transport in the blood and inter and intracellular fluids by the antemortem coagulation or gellation of tissue colloids that that depended on the lack of energy

production via oxidation to keep the colloidal particles charged on their surfaces and hence a failure in their dispersion; the resultant failure in oxygen transport further her blocked the oxidation mechanism in a vicious circle;

(5) the fact that the oxidations were blocked by an amine group of guanidin, that dehydrogenation is the first step in oxidation, that the carbonyl group is a good dehydrogenator, and that it can be inactivated by condensing firmly with an amine group as in guanidin, but functions normally by condensing with a weakly activated amine group as in creatine to form an azomethine double bond.

The conclusions are the following;

(1) after Parathyroidectomy, the activated amine group of guanidin and methyl guanidin condensed with the carbonyl group of the cell's energy producing mechanism for function (FCG) to form a firm azomethine double bond, and thus prev[illegible] it from initiating oxidations in fuels or toxins that came into the field;

(2) the failure to oxidize made it impossible to charge the tissue colloids, and so they precipitated as a gel and did not flow through the capillaries and tissue spaces or even through the large vessels to carry oxygen to the intracellular mechanisms, so an anoxia or hypoxia was secondary to the inability to start oxidation chains;

(3) that the oxidations of highest quality are chain reactions started by dehydrogenations, free radical production, addition of molecular oxygen to the free radicals to form peroxide free radicals, that carried the oxidation chain, or caused molecular cleavage into parts with terminal carbonyl groups that promoted further oxidation;

(4) that the Pasteur effect was a function of this tissue cell functional carbonyl group (FCG) and was suspended or destroyed by its condensation with tight binding amines as guanidin, but its function was supported by condensing with the weakly binding amine group of creatine after the hydrogen atom the FCG removed from fuel was passed on to an appropriate electron acceptor;

(5) that the Creatine-FCG azomethine bond is held until the energy developed as the oxidations progress caused phosphoric acid to enter this azomethine bond, combine the creatine and liberate it as a high energy carrying phosphate, and that the burning of fuel is regulated by the factors

concerned, — the FCG, creatine phosphoric acid and stored energy;

(6) that toxic amines of various metabolic, bacterial, viral or of fungal agents (present day antibiotics may be included) are able: to make the same crippling condensations with the FCG that no metabolic measure is able to break, and thus disturb the physiology in various ways;

(7) that to dislodge such toxins it is necessary to oxidize them away as no adequate hydrolytic provisions are available;

(8) that the double bond of the azomethine condensation activates the hydrogen atom of the carbon placed alpha thereto to provide for its easy dehydrogenation, and thus start an oxidation progression via free radical, peroxide free radical, and cleavage that burns away the amine group of the toxin or other pathogen, and restores the host cell functional carbonyl group;

(9) that the FCG is activated to be the preferred dehydrogenator through conjugation with the double bond of an ethylene linkage, that contributes electrons to it; additions to this linkage must destroy its activating powers.

We shall see how these conclusions fit into the pathogenesis of cancer and viral infections, and determine the controlling therapeutic measures. It will be seen also that they explain the long unsolved Pasteur Effect, which we hold to be a splendid demonstration of the presence and action of the Functional Carbonyl Group, the FCG, as we designate it for short, and a solid proof of the correctness of our working hypothesis and postulate.

CHAPTER II

VIRUS AND CANCER CELLS

Cancer cells and viruses are both parasites; that is they have to depend upon sources of energy and material that belong to other usages to conduct their characteristic activities. The virus cannot produce the energy it needs for its vital processes, so it gives signs of life only so long as it is integrated with a living source of energy and food which it diverts to its own ends. The cell it preys upon, the Host Cell, is killed thereby. The cancer cell is not able to perform the functions it was created to do for itself or any of the other cells of the body to which it belongs and is responsible. It has lost its capacity to conduct oxidations, and also the mechanisms that use the energy of oxidation for useful work. Instead, a low grade process, wasteful fermentation, is used to produce energy. This energy is transferred to the mitotic mechanism, where it forces cell division, as it has no functional mechanism to use it where its production is normally controlled by the demand for the function. The mitotic mechanism thus becomes parasitic upon the rest of the cell and the body as a whole.

Viruses may cause normal cells to go neoplastic: maybe all cancers require them. Several hundred synthetic substances are known to cause cancer. It has not yet been decided if or not viruses play a part here, too, but many cancerologists think so. It will be seen how the synthetic carcinogen may prepare the way for the virus to integrate with the mitotic mechanism and complete the neoplastic change.

The institution of parasitism within a cell, be it viral or neoplastic is a complex affair that depends upon a disposing cause besides the particular virtues of the pathogen to show its specific action. Of these anoxia or hypoxia is a leading factor. With plenty of oxygen available as normal structure determines, there would be no pathology if the oxidation catalysis were adequate, and logically enough, it happens that an adequate oxidation catalysis prevents oxygen deficiency in any tissue. This fact will become apparent as we go along. So the key to the correction will be seen to be the restoration or provision of an adequate oxidation catalysis. The initiating act in the oxidation process, namely, dehydrogenation is a main subject of this book. Then there is the subject of the environmental factors that have contributed to the block in the oxidation catalysis. These are discussed also in the hope that a good working picture of the matter is at hand.

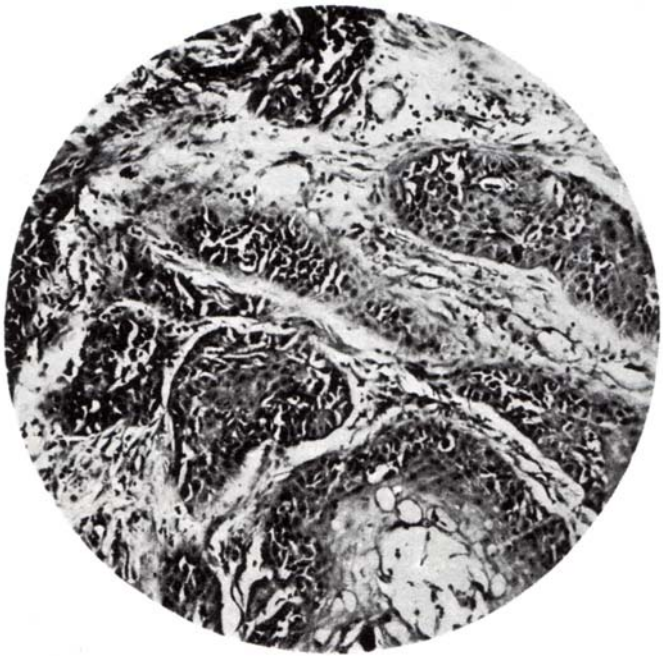
NATURE OF VIRUSES

The electronic microscope and bacteriophage studies have yielded rich information on this score. Their sizes vary from 8 millimicra, the size of a protein molecule, to 175 millimicra, the size of the smallest bacteria. Each type has its own shape and these vary from rod to spherule in form. The essential structure is a protein capsule within which lies a nucleoprotein mass. They are specifically cytotropic obligative parasites and always pathogenic. The protein capsule has specific antigenic powers that yield specific immunological responses, and serological reactions. The latter, for example, serve the differential diagnosis of non-paralytic Polio from La Grippe and other viral infections. This is the part that is convertible into a vaccine used to excite immunological reactions in the patient. There is no immunological response to the nucleoprotein part though this is the part that causes the pathology. The protein capsule protects the inner part and carries it to the cell where it attaches to its outer surface and injects the nucleoprotein content into its victim. An acquired immunity inactivates the capsule and prevents the attachment to and hence the penetration of the host cell. As soon as the material is injected, it breaks up  a myriad of similar units of nucleoprotein as by a depolymerization process, and each particle unites chemically with a nucleoprotein particle of the host cell, and thus integrates or becomes one with it t as by a co-polymerization act. The host cell particles with which they integrate are the “grana” that perform the vital functions of the cell and produce the necessary energy thereto by oxidation. As the virus shunts this energy into itself and uses it with host cell material for its vegetation, the host cell grana break down and are used up to form the viral colony which is soon a mass of provirus ready to mature and break forth from the dead host cell as infectious parasites.

It is the consensus among experts that once the nucleoprotein of the virus has penetrated, integration with the host cell is complete within a minute and a half, and no amount of vaccine, antitoxin or other serological effort can separate the virus and rescue the host cell. It is doomed. On the other hand, we will show how the bondings we postulate as forming the integration actually invite oxidative cleavage in a way that leaves the host cell in good functional status while the virus is no longer to be found. In this process the energy taken from the host cell to serve viral vegetation is returned to it to support its reconstruction, while the virus, undergoes a stepwise oxidation. This explanation is based on the repeatedly observed fact in the cure of Rabies, that when the reagent is given to promote the oxidative destruction of the virus, in an animal that has only maybe 12 to 24 hours to live, a period of 72 to 84 hours are required for the paralyzed victim to be free of paralysis and the affected nerves to be functioning normally. We have proved this restoration not only in terminal rabies, but in paralytic dog distemper, paralytic hog cholera, and paralytic Anterior Poliomyelitis. The same chemical. corrective measure was used in all and fit the pathology in all. This is taken to mean that the state of integration of the host cell and the virus presents the same atomic

bondings in all instances. The mine corrective attack was employed in the sensory nerve atrophies with loss of function, and in neoplastic disease with success, so it is concluded that the state of integration of the host cell and the pathogen is of the same order in all, and n least common denominator in pathogenesis and its correction has been established in a broad field.

PLATE I

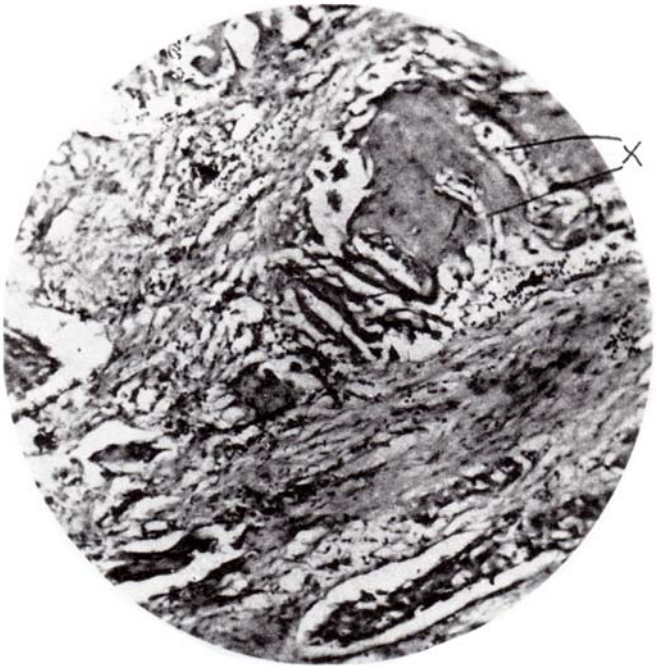


A. — Squamous cell carcinoma of the neck biopsy before treatment. (150X)

Though the atomic bondings that constitute the integrations are of the same order, the states of integration vary. Thus in "Polio" an acute lytic type causes the death of the host cell in hours or days as a rule, while a prolonged symbiotic type may cause extensive paralysis and atrophy that invalids the patient for many years or until death. And yet the host cell is not dead, but as its energy producing mechanism is paralyzed by the integration with the virus, it cannot function any more than a dead cell and the results appear the same. We have found by trial, however, that such cases can be freed of their offending virus and the host nerve cell returns to normal in function and so the atrophied muscles again get impulses to contract and rebuild themselves. Cases that were

extensively paralyzed and atrophied for three years have required three months to be restored to 95% or more of normal in function and muscle reconstruction, while a case extensively paralyzed and atrophied for over twenty years has required two years to be restored to 95% of normal, functionally and structurally. Since nerve cells do not reproduce, the cleavage of the integration is proven by these tests.

PLATE II



B. — Taken from the same tumor several weeks after treatment, showing the calcified coagulated hyalinized debris, into which angioblastic tissue is growing with an area of liquification preceding each ingrowing bud of angioblastic tissue.

Since the lytic type of infection only takes days or only hours to kill the host cell, the quicker the patient is treated the better the chance to have living cells freed from their viruses, and get complete recoveries. The case records illustrate these situations.

In neoplasia the integration of the virus or synthetic pathogen or bacterial toxin with the host cell is originally with the functional grana energy producing mechanism, until the latter is destroyed in building up the virus and nuclear material integrated with it and undergoing mitosis. This is the only mechanism left to accept energy produced by an uncontrolled process of fermentation, and so neoplasia is the expected result. The details will be discussed later, where it will be seen that the carbonyl groups that institute energy production and storage as ATP, and creatine phosphoric acid, are like those that mediate the transfer of this energy to the working mechanism and both are paralyzed by condensation with firmly binding amines, of virus or other carcinogens. The virus bound in the atrophic muscle of "Polio" we picture as similarly integrated. The integrations may exist long periods before actual destruction of the paralyzed functional mechanism is accomplished and, during this period, liberation of the cell from the pathogen can restore it to normal. For a period the pathogenesis is reversible therefore in viral infections and the same holds for cancer. This situation is exemplified in the microphotographs (A) and (B). (B) shows the destruction of the cancer cell after the pathogen is removed and the cell residues can function no longer. They then undergo calcification and digestion like a blood clot. Plate I (Medical Record of New York, Koch, October 30, 1920).

VACCINE PROBLEMS

From what was stated so far it is seen that vaccines for a specific virus do not immunize against the nucleoprotein that is the actual pathogen, especially after it has penetrated and integrated with the host cell, so to talk about curing cancer with vaccines or immune sera is a waste of time. Even the prevention of viral infection by vaccines is meeting the strongest statistical opposition since large scale small-pox and Salk vaccinations have been recorded. In line with what is known about vaccine structure, statistics appear logical when they show that paralytic "Polio" is increased both in incidence and fatality by use of the vaccine. One may compare various regions of different climatic conditions for the data. In all of these the Salk vaccine was enthusiastically applied, in greater number each year, and the incidence increase was tremendous each year, whereas, if the vaccine were effective there should have been at least a little statistical improvement. In Montreal, generally cool, they reported on August 27, 1959, 521 cases with 27 deaths, just while the "Polio" season was getting well under way, as compared with less than one hundred in 1958. In Ottawa, generally cool, 455 cases with 41 deaths were reported on August 22, 1959, as compared with 64 cases with 7 deaths in 1958. In all of Canada, even before the epidemic started to decline, there were 7 times more paralytic cases in 1959 than in 1958, with a greater death rate. In Detroit, much warmer, where vaccination was thorough, the number of cases in 1958 was 697, against 226 in 1957. In the District of Columbia, still warmer, the Health Department reported 7 times as many cases in 1958 as in 1957. In New Jersey, in 1958,

the Health Department reported twice as great an incidence as in 1957. The United States Public Health Service reported an increase of 15 ½ % of paralytic cases in 1958 over those in 1957 (49916 against 33.5%). In Hawaii (tropical) there were 65 victims including 32 paralytic cases in 1958; half of these paralyzed cases (16) had received three Salk shots, in an island where 60% had been vaccinated. In 1957 only 25 and 8/10^{ths} % were paralytic instead of 49 and 9/10^{ths} % in 1958. If the vaccine were effective there should have been a 60%, decrease in the incidence in the whole island of the paralytic infections, instead of an increase of nearly 100%.

Nationwide statistics issued January 4, 1960, by the United States Public Health Service, show that for the year 1959, up to December 26th (51 weeks), the increase in the incidence of Polio rose 85916 over that of the same period of 1958. There were 8,531 cases listed for 1959, of which 5,661 were paralytic, as compared to 5,987 in 1958, of which 3,090 were paralytic. We just showed m the great increase in 1958 over the incidence of the total and the paralytic cases of 1957. Where compulsory vaccination was practiced as in North Carolina and Tennessee, Bealle's investigations report a 400% increase in paralytic and non-paralytic Polio during 1959 over 1958. So it seems that the more vaccine that is used the more the actual infection that comes about. The statistical analysis teaches much about the nature of the virus.

Of course, this is comprehensible when one considers that the virus breaks up into its component units on penetrating the host cell, as if by a depolymerization process, and it grows by acquiring new units to add to each, as by a copolymerization process. Some investigators compare the viral structure to a deck of cards. The complete deck or complete virus with all its units is the parent pathogenic killer type. The vaccines may be regarded as incomplete decks, with not all the units required to make up the full killer type. Now, if a person carried vaccine units of, let us say, half or less than the killer type requires and another vaccination or infection by a crippled non-fatal virus comes along that presents the units missing in the protective infection or vaccination of a previous period either one of which alone can not produce the disease, the units all added up could constitute the complete killer type, and it has been shown that they are “shuffled” in at random to make up the full virus, vaccination may add to the incidence of serious or fatal infection, and the more the vaccination the more the chance for building fatal viruses.

This happened in the writer's early practice (1920). Two cases were vaccinated against small-pox from the same vaccine lot. One had no effect. The other came down with a rapidly fatal small-pox. There was no epidemic at hand in Detroit at the time, so it was concluded that the fatal case's inoculation carried units required by a previous silent infection to make it fatal.

SMALL-POX

Statistics on vaccination against Small-Pox in the Philippines when the United States took over are instructive. Reports run thus: In 1918, the Army forced the vaccination of 3,285,376 natives when no epidemic was brewing, only the sporadic cases of the usual mild nature. Of the vaccinated persons, 47,369 came down with small-pox, and of these 16,477 died. In 1919 the experiment was doubled. 7,670,252 natives were vaccinated. Of these 65,180 cases came down with small-pox, and 44,408 died. One sees here that the fatality rate increased in the twice vaccinated cases. In the first experiment, one-third died, and in the second, two-thirds of the infected ones. This speaks for the retention of viral units from the previous vaccinations, and indicates that, in the vaccine the shuffling in of units varies in different specimens of vaccine. It should be stated also that every epidemic of viral disease treated by the writer followed vaccination within a few months, when protection should have been had instead of an epidemic. This was so in Brazil, in Aftosa, Cinemosa, Hog Cholera and Rabies, and in Cuba in Hog Cholera.

The question arises then as to how one accounts for the decrease in the incidence of small-pox, since vaccination was instituted. The question is not easy to settle, since the hygienic improvements in sewage disposal has wiped out the means of spread of intestine carried viral infections. In the great small-pox days, excreta were thrown out of the window into the streets, then the outhouse was invented with its flies, etc. Today modern sewage is an obvious advantage, and soap and water are available even for washing the fingers of cooks and waiters in restaurants, and inspections by Public Health Officers help greatly in keeping down the spread of infection. It must be recalled that viruses integrate with bacteria and when these form spores, the integrated virus shares the protection of the spore against sterilization by chemicals and heat. They can thus survive for many months or years with full virulence. The intestinal tract is known to be a favorable habitat for such integrated viruses, so the hygienic measures of today would wipe out small-pox anyway without the benefit of vaccination, if there is any when carried on commercially. The present-day kitchen garbage disposal sink apparatus has cut down the incidence of the house fly so much that its universal adoption should become the greatest health booster of the century. In the writer's experience, vaccination is a laboratory success when the technique is correct all the way through. Commercially the statistics do not look so favorable when other variables are encountered.

CHAPTER III

CANCER

Glover showed in 1923 that the cancer virus existed in a pleomorphic germ that was bacillus in one phase and coccus in another, and virus in the third phase. He also showed it could exist in a fungus or micelium phase. The latter form has been identified lately by Irene Diller, and some others, and the whole chain of forms was independently proved by von Brehmer, in the last few decades as well. The work was thoroughly repeated and proved by my friend Jacob Engel and George Clark, at the U.S. P.H.S. laboratories, but, for reasons we will not discuss, they were not allowed to publish their findings. The infectious nature of natural cancer was thus proven beyond any doubt by carefully following the four laws of Robert Koch. Doctor Clark was able to get a paper read on this confirmation in 1953, at Rome, Italy, at the Sixth International Congress of Microbiology. So at last the facts are recorded in the archives of orthodox scientific literature.

In the usual viral infections, the host cell material and energy are used to build the viral colony with terrific multiplication of new viruses. In cancer, both nutrition and energy go into the building of new cancer cells and perhaps only an equal number of integrated viruses. For this reason it has been difficult to demonstrate the virus in certain cancer growths. Synthetic carcinogens numbering over two hundred have been tried out. One sees that the tame two atomic units required for viral integration with the host cell are to he found. These are:

(a) the activated amine group, and

(b) the highly mobile hydrogen atom alpha to a double bond in the most exposed area, the “K region”, as it is now named.

When dehydrogenated during anoxia it adds to the FCG activating unit. Carcinogens carrying the amine group that integrates with the host cell FCG to start the pathogenesis, as in acetyl amino-fluorene, and in Butter Yellow, and its analogues, hold the amine group in a protected state until the agent enters the body and hydrolysis or oxidation frees it of its protection, so it can make the azomethine condensation. Some experts think that the synthetic carcinogen prepares the cell for the virus carcinogen, but give no explanation of how this is done. Our postulate, on the other hand, shows that the amine group of synthetic carcinogens or of the fungus always found in cancer can play a part by inactivating the cell's FCG. The blocking of the oxidations that results brings about the colloidal gelling that causes the anoxia necessary for addition of the free radical (brought about in the virus by dehydrogenation) to the double bond that activates the FCG. Our thesis also shows how the carcinogen can produce cancer without the aid of a

virus by addition of either pathogenic atomic unit. Some animal experiments with neoplastic transplants, and some with carcinogenic agents are reported here for comparison to show that the n same reagent gave protection and cured in high percentage in each set. The parathyroidectomy experiments should be recalled in connection with the pathogenesis and the anoxia involved. This is a main pillar of our thesis as based upon our earliest findings which, of course, took considerable thought to be appreciated. They are not even appreciated today in the orthodox circles, although Warburg, the Nestor of the biochemical profession, has championed the fact that anoxia is the cause of cancer for decades. He is the pioneer who developed methods of study of the oxidations in tissues. However, he has not yet appreciated the place of the free radical in the process. It will be seen that it is this position of orthodoxy that has limited progress in the explanation of the mechanism of anoxia in causing cancer, and the true nature of the carcinogenic change. Nevertheless, his contributions that won for him the Nobel Prize in Medicine on this subject are a monument of support to our postulate. Hence we give some quotations from his most recent summary in "Naturwissenschaften," Vol. 42 - p. 401, 1955:

If one examines them in the light of the data we have presented in the preceding pages, it will be evident that they confirm our own thesis on the pathogenesis of cancer and disease in general. They point out the essential status of anoxia, which we have claimed is necessary for the pathogen to be changed by dehydrogenation (of the tissue metabolism) to a free radical which instead of being burned and disposed of as fuel in the presence of oxygen, is not burned m its absence but is able to add to the cell Grana and do so at the very point where the activation of the oxidations is generated, namely the double bonds that activate the carbonyl group which we credit with initiating the tissue oxidations by serving as a dehydrogenator of fuels and pathogens. Thus anoxia is essential to the pathogenesis as it disposes the pathogen, carcinogen, virus or what not, to be able to integrate with the host cell and block grana function. The following quotations support our thesis as far as they go.

> "One method for the destruction of the respiration of the body cells is removal of oxygen. If, for example. embryonal tissue is exposed to an oxygen deficiency for some hours and then is placed in oxygen again, 50 percent or more of the respiration is usually destroyed. The cause of this destruction of respiration is lack of energy. As a matter of fact, the cells need their respiration energy to preserve their structure, and if respiration is inhibited, both structure and respiration disappear."[12]

~~~~~~~~~FOOTNOTES~~~~~~~~~

[12] Formatting is modified here. Quotes are offset, a printing characteristic not in the original text. This is to emphasize references, and to link other materials. No disrespect or misrepresentation intended. Another interesting aspect to this set of
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If one estimates the amount of normal function, one sees how the pathogenesis we have described will accomplish what Warburg reports here. Again,

> "If an injury to respiration is to produce cancer, this injury must, as already mentioned, be irreversible. We understand by this not only that the inhibition of respiration remains after the removal of the respiratory poison but, even more, that the inhibition of respiration also continues through all the following cell divisions, for measurements of metabolism in transplanted tumors have shown that cancer cells can not regain normal respiration, even in the course of many decades, once they have lost it."

> "But why are the body cells dedifferentiated when their respiration energy is replaced by fermentation energy? At first, one would think that it is immaterial to the cells whether they obtain their energy from respiration or from fermentation, since the energy of both reactions is transformed into the energy of adenosine triphosphate, and yet adenosine triphosphate = adenosine triphosphate. This equation is certainly correct chemically and energetically, but it is incorrect morphologically, because, although respiration takes place for the most part in the structure of the grana, the fermentation enzymes are found for a greater part in the fluid of the protoplasm. The adenosine triphosphate synthesized by respiration therefore involves more structure than the adenosine triphosphate synthesized by fermentation."

Since the enzymes and intermediaries of fermentation, which biochemists accept as playing a big part also in the cell respiration, bathe the grana and the grana do not use them, as the quotation shows, then the conventionally accepted process is not correct, and a different process and different set of enzymes and intermediaries are involved. This process must be one which inactivates the grana when oxygen is missing, hence the logical deduction is that the free radical is an essential part of an early intermediary as we have hypothesized for so long.

Further,

> "The first notable experimental induction of cancer by oxygen deficiency was described by Goldblatt and Cameron, who exposed heart fibroblasts in tissue culture to intermittent oxygen deficiency for long periods and finally obtained transplantable cancer cells, whereas in control cultures that were maintained without oxygen deficiency, no cancer cells resulted. Clinical experiences along these lines are innumerable."

~~~~~~~~~FOOTNOTES CONTINUED~~~~~~~~~

quotations is they appear as a dialogue though Warburg to benefit the reader and imply Koch's train of thought which draws conclusions beyond those of Warburg.
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Warburg emphasizes,

> "… but there is only one common cause into which all other causes of cancer merge, the irreversible injuring of respiration." He states, "In recent years it has been recognized that subnarcotic doses of urethane cause lung cancer in mice in 100 percent of treatments. Urethane is particularly suitable as a carcinogen, because, in contrast to alcohol, it is not itself burned up on the respiring surfaces and, unlike ether and chloroform, it does not cytolyze the cells. Any narcotic that has these properties may cause cancer upon chronic administration in small doses."

So Warburg recognizes that a carcinogen must be not destructible by the cell's oxidative mechanism or otherwise. It can then become integrated with the cell and become a part of it, and can become accumulative as the disposing anoxia provides occasion. Warburg states in this connection,

> "Any respiratory injury due to lack of energy, however, whether it is produced by oxygen deficiency or by respiratory poisons, must be cumulative since it is irreversible."

The essential nature of the process of fermentation subjects it only to control by the circumstances that control any enzyme action. These are temperature, the pH reaction of the medium, the concentration of the ferment and of the substrate. Besides such accessories as the magnesium ion or when needed, a co-enzyme will determine the speed and extent of the reaction. Fermentation progresses as well in a test tube as in a living cell. No physiological control of these qualities for the specific service of the cell economy are known, except one, and that is the presence of the oxidation process. Normally this is the presence or absence of oxygen. Pasteur was first to observe this relationship, and the great Warburg named it after this supreme observer, — the Pasteur Effect. This phenomenon was first described by him when observed in yeast cultures, but it is a common property to all cells that are obligatively aerobic. He reported that if a culture of yeast is deprived of oxygen, fermentation comes to the aid of the cell to supply the energy for vital processes, and if oxygen is again admitted to the culture, the fermentation ceases and oxidation takes its place. Fermentation is very wasteful and less than one-fifteenth as efficient as oxidation in the use of fuel material. The mechanism of the Pasteur Effect has never been explained by orthodox biochemistry. However, one will see that our postulate incorporates its explanation as a function of the Functional Carbonyl Group.

Oxidation has several positions of control in its process in line with our postulate. The first is the potency of the FCG which must start the process by dehydrogenating the fuel. When this carbonyl group is not free, as when the hydrogen it removes from the fuel is not taken away by some electron acceptor system, then oxidation is blocked. And for this, oxygen is essential as the ultimate electron acceptor

in aerobic organisms. So lack of oxygen has two steps in blocking oxidation or hindering it. Another position of control is the inactivation of the FCG by additions to the double bonds that activate it. This would happen as we explained when anoxia prevents the free radical formed by FCG action from becoming a peroxide free radical so it must add to some position as that of the activating double bond. When this happens the FCG is inactivated and the starting of another oxidation progression is blocked. Fuels so added may easily be burned away by the process we outlined for removal of pathogens, but normally it is not accomplished by FCG action when oxygen is again admitted unless the FCG belongs to a different unhindered structure. If a pathogen has been added the SSR is needed to free it.[13] However, when oxygen is lacking the FCG as a rule will not be relieved of its hydrogen atom and can not form a free radical in the fuel until oxygen is admitted, and this is a protection to the mechanism.

Physiologically, however, the control that is designed to serve the economy of the tissue itself and the organism as a whole is regulated by the need for energy production. Oxidation is regulated so quantatively but fermentation is not. The need for energy is determined by the work to be done by a functional unit, and this is regulated by nerve or some hormone action which releases the phosphate stored energy to become work energy. This creates a deficit of stored phosphate energy, and oxidation must get going to replenish the supply. We explained elsewhere how the FCG accomplishes this act. So physiologically oxidation is controlled by the need of energy for work; and the substances concerned in the process in addition to oxygen and the fuel are the FCG, creatine and phosphoric acid. Therefore free creatine and free phosphoric acid and possibly some calcium balancing factor will give the free FCG a chance to start oxidation progressions that will yield the energy to form energy carrying creatine-phosphoric acid, as we explained before. Dehydrogenation and FCG action are concerned in oxidation only, and not in fermentation. Therefore, fermentation is probably blocked by inactivating its initiating enzyme by the act of dehydrogenation to form an active free radical in it, so that it makes an inactivating addition that is split by phosphoric acid set free when it is liberated from an energy carrying ester. The ferment would then act through its restored hydroxyl group when energy fails, — a possibility only, as the need for energy production would be indicated by an accumulation of phosphoric acid, the first indicator for the need of fermentation, that is also able to liberate the ferment's bound hydroxyl group, that was inactivated by oxidation (dehydrogenation) during anoxia. Aside from such automatic control, there is the nerve and hormone control of the FCG and its azomethine double bond with the amine group of creatine where the nerve impulse determines its rupture to form the phosphate energy carrier, or its formation to discharge the energy load into the working mechanism. The phosphate energy bonds of

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[13] The SSR is a synthetically produced carbonyl compound of high Oxidation-Reduction Potential, used as a therapeutic agent.
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fermentation are not formed or split in that way. Their energy must enter the functional mechanism through a different door. Mass action and energy transferred by photosensitization may determine such activity. There is no data on which to base a decision. Fermentation need not be a general affair, but may be localized in some particular tissue where the FCG function is hindered. Function forced by fermentation energy beyond physiological control is the characteristic of all allergies including cancer. The type of allergy is determined by the functional unit involved, secreting fibrillae in hay fever, contractible fibrillae in asthma, conducting-synapse fibrillae in compulsion neuroses, mitotic fibrillae in cancer, etc. However, FCG block is necessary as described before. And the permanent block is done by a condensation with a firmly bonding amine as guanidin, or of some virus or an amine produced by decarboxylase action on some amino acid. When Victor C. Vaughan demonstrated in 1910 that the alkaline hydrolysis of various proteins gave rise to a toxic fraction that caused anaphylaxis, the writer was privileged to work in this kindly scientist's laboratory and isolated several toxic amines from his alkaline hydrolysate. They produced the allergic changes of fatal anaphylaxis. Later on it dawned on the writer that if his postulated FCG were blocked by toxic amines produced in a tissue by their decarboxylases that operate best in an alkaline medium, fermentation would be the result that could force the allergic responses that occur in anaphylaxis, and the type of response would depend upon the functional unit acted upon, while the amount of amine could be very small.

To check up on this a large number of allergy cases were treated with the SSR by the writer and several hundred collaborators. The results reported were about 85% recoveries obtained on one or two doses. These included the intractable asthmas and hay fever cases as well as the infantile eczemas that fall co respond to known methods. Some guinea pigs sensitized to egg white by the Vaughan method were also treated with success, but such experiments are not determinative unless all variables can be excluded, and they can not. At any rate, one sees here the practical meaning of the Pasteur Effect as modified by a pathogen. Physiologically, fermentation stops in an anoxic tissue when oxygen is admitted, but here, pathologically, the use of oxygen was blocked until the hindrance to the FCG was removed by use of a “super” carbonyl group, the SSR. Though in orthodox biochemistry the explanation of the Pasteur Effect has not yet been made, we see that the carbonyl group is the key to the situation both normally and in pathological processes. Our postulate is thus strengthened by its broad utility.

That the narcotic action promotes neoplastic behavior is only too well known to the clinician. Especially is this true of the oxides of nitrogen that present permanent free radicals that can block respiration and promote cancer growth most disastrously. Their essential action is to hinder cell respirations. Other confirmatory quotations in support of our postulate could be given, but this is enough.

While it was important to recognize the essential role of oxyge[illegible]ck in carcinogenesis, the observation is of no practical use until one understands the

mechanism whereby the anoxia disposes to neoplasia. It is too bad that Warburg was not aware of our findings after parathyroidectomy, the action of high-energy amino groups in attacking the oxidation initiating carbonyl group of the grana as we postulated, and that thereby the oxidation mechanism was blocked. It is too bad he gave no thought to the position of the free radical formed by these dehydrogenations and the absolute need of molecular oxygen to carry the oxidation progression forward, and that when the substrate acted upon could find no oxygen to combine, it must combine an appropriate double bond, and thus integrate with the cell's energy producing mechanism. It is too bad that he did not make these steps and then the third step to recognize that the very integration of the pathogen with the host cell invited the oxidative separation of both leaving the host cell in good functional status, while the pathogen was destroyed. He therefore missed the essence of the reversibility of carcinogenesis, and the means of bringing it about. We will give the details as we go along and the proofs for each step. However, what Warburg did establish was a great advance in cancerology and his prestige as a biochemist makes this support to our thesis, a most valuable one.

CHAPTER IV

PROOFS OF REVERSIBILITY

Official Test

The first official cures of cancer of the vital organs in the far advanced stages were had in 1919 under the auspices of the Wayne County Medical Society branch of the American Medical Association. Five cases were selected by an officially appointed committee of surgeons and one pathologist numbering five in all. The cases were officially selected as fit for the test, and the test was officially closed three weeks after the patients were treated and improvements began to show. Five years later it was seen that three of the cases were cured and possibly a fourth who lived too far away to be examined, but through the results in his case, new patients were sent to the writer, five years later. One of the test patients with cancer of the uterus, proven at laporotomy as extending throughout the abdomen and perforating the stomach so as to cause severe bleeding, lived fifteen years in good health after the treatment, died from an accident, and the coroner's autopsy showed no cancer was present, and the cause of death had been a brain injury. Yet the Official A.M.A.-W.C.M.S. committee reported no results, and the W.C.M.S. Cancer Committee that was appealed to five years later to change the false report, refused to do so and summarily denied all the diagnoses made by the official committee. The cures, however, could not be denied. They refused any further tests. No other official test was ever made. Every request for one has been refused. But the false report is still being circulated.

NATIONAL STATISTICS

Cancer mortality statistics during the decade, 1920 through 1929 inclusive, for the six largest cities of the United States, reported by Hoffman, in the “Spectator”, and elsewhere while we specialized in cancer treatment at Detroit, are given below. Since the only variable that entered the picture was our own therapy, we take the credit for the drop in the death rate in Detroit and its lesser increase in cities that sent us patients, as compared to the high increase in death rate in all largest cities that did not send us patients. While Philadelphia and Los Angeles showed a 30% increase in this decade, Detroit showed a drop of over 20%, and it was the only large city that showed any fall whatever in the death rate from cancer.

National Statistics

SIX LARGEST CITIES IN 1930	CANCER DEATH RATES (per 100,000 pop.) 1920	1923	1929
New York City	107.9	106.0	115.4
Chicago	104.9	103.7	107.7
Philadelphia	103.9	116.3	135.8
Detroit.	96.7	67.4	74.5
Los Angeles	96.8	130.4	128.1
Cleveland	93.8	89.9	104.2

Individual case studies that support this thesis are taken from the testimony of collaborating physicians in the U.S. Federal Court where they were proven factually incontestable. They demonstrate the different features of the reversibility.

UTILITY IN GENERAL PRACTICE

What is most important to the physician is the field of utility of synthetic Survival Factor therapy. The following table is contributed by Dr. Wendell Hendricks as part of a paper given before a convention of collaborators who were making observations with this therapy. The best showing is in the viral diseases which gave a 100% recovery rate, measles, mumps, infantile paralysis, and the acute infections as gonorrhea, rheumatic fever, sinusitis, etc., and influenza. The poorest showing was in Arthritis Deformans which gave only a 50% cure rate. This disease is 100% incurable otherwise, however, and perhaps the patients did not stay with the treatment long enough to give it a chance. In the 100% incurable hypertrophic type of arthritis a cure rate of 82% of 144 cases is certainly a good service with observations showing the permanency of the cures over a period of 4 years of check-up.

[DATA REGARDING CONDITIONS AND CURE RATES]

NAME OF SICKNESS	Number of cases	Age of patients	Average weeks between reactions	Time required for cure	% of cures	Years since cured
Allergies nasal ……	282	1-70 yrs	9	18 w	82	6
Acute Tonsillitis ……..	61	2m-54 yrs.	0	3 d.	100	4
Chronic Tonsillitis ……..	20	2-84 yrs.	3	6 w.	80	3
Vincent's Angina ……	35	2-57 yrs.	0	6 d.	89	4
Arthritis Deformans ……..	16	22-57 yrs.	9	36 w.	50	5
Arthritis Hypertrophic …..	144	24-82 yrs.	9	27 w.	82	4
Gonorrhea ……...	15	22-55 yrs.	0	3 w.	100	3
Bronchitis acute ……..	64	2m-56 yrs.	0	2 d.	94	4
Bronchitis asthmatic ……..	460	1-69 yrs.	9	27 w.	80	6
Bronchitis chronic ……..	35	3-67 yrs.	3	18 w.	80	4
Brucellosis ……...	35	29-58 yrs.	9	18 w.	93	3
Coccidioidomycosis ……	70	7-63 yrs.	0	3 w.	95	3
Cholecystitis ……	44	26-58 yrs.	3	18 w.	84	2
Coryza acute ……...	100	6m-74 yrs.	0	2 d.	100	5
Eczema ……	120	1-68 yrs.	9	27 w.	80	6
Rheumatic Fever ……	20	4-11 yrs.	3	6 w.	100	3
Gout ……	10	30-55 yrs.	0	12 w.	90	6
Influenza ……..	51	8-65 yrs.	0	8 d.	100	3
Nephritis acute ……	22	6-66 yrs.	3	6 w.	90	3
Nephritis Chronic ……...	20	22-68 yrs.	6	18 w.	85	3
Neuritis ……	67	17-84 yrs.	0	3 w.	85	4
Pneumonia ……...	22	1-69 yrs.	0	6 d.	82	3
Poliomyelitis acute ……..	10	3-16 yrs.	0	3 d.	100	4
Poliomyelitis chronic ……..	2	23-33 yrs.	9	18 m.	100	3
Syphillis chronic ……	10	24-47 yrs.	9	36 w.	80	4
Sinusitis acute ……	38	12-63 yrs.	0	3 d.	92	3
Sinusitis Chronic ……	27	2-66 yrs.	3	18 w.	78	3
Uticaria ……	75	1-72 yrs.	9	18 w.	88	4
Combined total of measles, whooping cough, mumps and scarlet fever ……	66	6m-36 yrs.	0	3 d.	100	3

CHAPTER V

ANIMAL EXPERIMENTS

When presenting a thesis such as this, it is good policy to eliminate superstitions and misinterpretations. For example, the idea that the cures we will report were secured by psychology or suggestion, or that after all the disease was not Cancer, Infantile Paralysis, Tuberculosis, or what it is represented as being. That the diagnoses were uncontradictable is established, and the pretreatment control period showing the downward course of the patient was also proven uncontradictable. The recoveries were established for many years, even several decades in some cases. It was also proven that no other remedy was used, and the sharp contrast between the pretreatment control period and the characteristic cyclic recovery that characterizes the recovery process after the Survival factor reagent was used settles the credit according to modern scientific procedures in drug testing. Because of the many shortcomings in the collateral control system when humans are under treatment, it has been discarded long ago, and the ACTH and Cortisone experiments show the clinical procedure of using an adequate pretreatment observation period to compare with the post-treatment period is the only reliable procedure known; and it is the procedure used by all highest rating clinicians for clinical tests. This is the system we use throughout, and each case should be studied with this in mind.

However, to remove any doubts about etiological and psychic factors we offer a few animal experiments, in which transplantable C 57 Breast Cancer was inoculated into mice, and also transplantable Sarcoma 37 was inoculated into mice, all at the Jackson Memorial Laboratory at Bar Harbor, Maine. The inoculated animals were then sent to Dr. Stanley Bandeen, of Louisville, Kentucky for treatment and observation. The C 57 inoculations were made on May 7th and the Sarcoma 37 on June 30, 1950. The experiment was terminated by a frost on November 13, 1950, that killed most of the cured animals and those undergoing recovery. The details are given below. Since the etiological factors in these experiments are uncontradictable, the diagnoses of the conditions treated are also uncontradictable. Some mice were killed by fighting. They are excluded from the statistics, even though they appeared cured. The treatment used was the 10-12 concentration of the serial system of carbonyl groups with free radical terminals, which we designate as the Synthetic Survival Reagent (SSR). We sometimes call it the Survival Factor reagent or remedy. The chemical formula is stated a little farther along. The dose is that equal to the smaller dosage of Vitamin B12 proven clinically active.

EXPERIMENT I

Twenty-five mice C 57 breast tumor transplantation, May 7, 1950. Five were held as controls, and the rest were divided into two sections: (a) 8 mice each receiving 4 minims of the reagent by injection, and (b) 12 mice each receiving 6 minims of the reagent. Treatment was given three days after tumor transplantation.

RESULTS

Controls: Five mice. All of the controls died of cancer from the 12th to the 24th day following tumor inoculation. Average length of life: 17 days after inoculation.

Section (a): Eight mice, 4 minims each, the third day after inoculation. All tumors had ruptured through on the 11th day and started to heal on the 12th day. They were completely healed on the 15th day. One of the mice had recurrence which proved fatal on the 44th day. On the 64th day one mouse gave birth to 3 young which lived until killed by frost on the 126th day after birth. Three mice died on the 64th and 66th days tumor free. One was killed fighting on the 32nd day. Three lived cured until killed by frost on the 190th day.

Death from cancer: 1

Death from fighting: 1

Recoveries: 6

Average length of life of those which recovered: 127 days.

Section (b): Twelve mice, 6 minims each, third day after inoculation. All tumors ruptured through from the 9th to the 10th day. All tumors were healed between the 13th and 14th days. Three mice died fighting, one on the 4th day, one on the 32nd day, and one on the 36th day. One died of cancer on the 38th day via recurrence. (On the 62nd day one mouse gave birth to 4 young. On the 112th day she gave birth to 3 young, her 2nd set). The rest, 8 cured mice lived to the 190th day when killed by frost, cancer free.

Death from cancer: 1 Death from fighting: 3 Recoveries : 8

Average length of life of those which recovered: 190 days.

EXPERIMENT II

Twenty-five mice were inoculated with C 57 Breast Cancer by transplantation on May 26, 1950. Five were used as controls. Four were treated with 2 minims, 8 were treated with 4 minims and 8 were treated with 6 minims of reagent.

RESULTS

Controls: One died fighting the third day. The other four died from cancer between the 12th and 18th days. Average length of life: 15½ days.

Section (a): Four mice treated with 2 minims of reagent. One died of cancer on the 24th day. Another died of cancer on the 26th day. On the 30th day and the 32nd day, the other two tumors healed. One died cancer free on the 104th day and the other died cancer free on the 128th day.

Death from cancer: 2

Death from fighting: 0

Recoveries: 2

Average length of life of those which recovered: 116 days.

Section (b): Eight mice were treated with 4 minims of reagent. Three tumors healed on tile 13th day, the others on the 11th, 12th, and 16th days. Three mice with healed tumors were killed fighting. One died on the 44th day, one on the 135th day, one on the 139th day, and two were killed by frost on the 177th day, cancer free.

Death from cancer: 0

Death from fighting: 3

Recoveries: 5

Average length of life of those which recovered: 135.4 days.

Section (c): Fight mice treated with 6 minims of reagent. On the 12th day 4 tumors were healed, and on the 15th day the other 4 tumors were he[illegible]. Two mice were killed fighting on the 16th day. One was killed on the 24th day, tumor recurrent. One died with cancer on the 34th day, tumor recurrent. One died on the 128th day and three were killed by frost on the 177th day, all four being cancer free.

Death from cancer: 2 (including the one killed fighting on the 24th day).

Death from fighting: 3

Recoveries: 4

Average length of life of those which recovered: 1643/4 days.

EXPERIMENT III

Sixteen mice received by transplantation Sarcoma 37 on June 30, 1950. Four were used as controls, and the rest were divided into three sections: (a) 4 mice received 4 minims each of the reagent, (b) 4 mice received 6 minims, and (c) 4 mice received 8 minims.

RESULTS

Controls: Four mice. All of the controls died of cancer between the 12th and the 20th day. Average length of life: 16 ½ days.

Section (a): Four mice, 4 minims each. Two died fighting on the 10th day and the 14th day before tumors were healed. The tumors healed on the other two on the 16th and 17th days; one died on the 110th day and the other on the 125th day, both cancer-free.

Death from cancer: 0

Death from fighting: 2

Recoveries: 2

Average length of life of those which recovered: 117 ½ days.

Section (b): Four mice 6 minims each. One died from cancer on the 18th day. Three tumors healed on the 35th day. They lived cured until killed by frost on the 136th day.

Death from cancer: 1

Death from fighting: 0

Recoveries: 3

Average length of life of those which recovered: 136 days.

Section (c): Four mice, 8 minims each. All 4 tumors healed on the 30th day. On the 84th day one mouse died fighting, this animal being cured. The other 3 remained cured until killed by frost on the 136th day.

Death from cancer: 0

Death from fighting: 1

Recoveries: 3 (4 if one includes the mouse killed fighting on the 84th day).

Average length of life of those which recovered: 136 days.[PPP26]

EXPERIMENT IV

Twenty-four mice were inoculated with Sarcoma 37 on July 28, 1950 and were treated with the reagent 5 days later. Four mice were held for controls, and the rest were divided into three sections: (a) 8 mice receiving 4 minims each, (b) 8 mice receiving 6 minims each, and (c) 4 mice receiving 8 minims each.

RESULTS

Controls: four mice. Two mice died from cancer on the 31st day, and two (lied from cancer on the 36th day. Average length of life: 33½ days.

Section (a): Eight mice, 4 minims each. Two died fighting on the 11th day and on tile 28th day after inoculation. Two died on the 84th day, cancer free. Two were killed by frost on the 108th day, cancer free.

Death from cancer: 0

Death from fighting: 4

Recoveries: 4

Average length of life of those which recovered: 96 days.

Section (b): Eight mice, 6 minims each. All tumors healed from the 18th day to the 23rd day. Two mice died cancer-free, one on the 83rd day, the other on the 101st day. Five of the others lived until the 108th day and were killed from frost. One survived the frost and lived to the 411th day.

Death from cancer: 0

Death from fighting: 0

Recoveries: 8

Average length of life of those which recovered: 142 days.

Section (c): Four mice, 8 minims each. Two tumors were healed on the 10th day, and two were healed on the 12th day. All continued in good health, cured, until the 108th day when 3 were killed by frost. One survived the frost and lived to the 412th day.

Death from cancer: 0

Death from fighting: 0

Recoveries: 4

Average length of life of those which recovered: 184 days.

It should be noted that the two mice that survived the frost, lived for an average of 411½ days and died free of cancer. This is equivalent to 41 years after cure on the human scale. One of the mice received 6 minims of the reagent and the other received 8 minims.

Discussion

The average length of life of the untreated controls was 20½ days, that of the treated animals that survived the frost was 411½ days, and those that lived up to the frost and were killed by it was 190, 177, 136 and 108 days for the different groups. We see that the frost reduced the possible life of the recoveries on an average from 411½ days to 153 days. When considering the average of Groups II, III, and IV, it must be remembered that the animals killed by freezing were all killed by the same freeze that killed those in Group I, and that those mice in the last three groups were treated 21, 56, and 84 days after those in Group I were treated. Therefore, the average length of life, while it appears to be shortened in the last 3 groups, actually was not shortened. However, the frost experience makes this experiment valuable in that it showed the effect of dosage, for the only frost survivors were those that received 6 and 8 minums, and those that lived co the frost were those that received the heavier dosage for the largest part.

Two minims showed very poor results as compared with the 6 and 8 minim dosages, but even the 2 minims gave cures, while the controls all died of cancer within three weeks. Hence a minim of a solution of one part to a trillion of water is a great deal of material when one considers the effects. It is just a few millions of molecules, that is all, and only one molecule should be able to start a chain reaction under ideal conditions.

As a comparison of cure rate, with the controls showing 100% de from cancer and 0 cures, in spite of the frost, the experiment is decisive, if any such experiments mean anything and it shows the effect of higher oxidation catalysis from the heavier dosage in fighting the cold.

In other experiments we took accounting on the 100th day or the 200th day instead of letting the frost sec the limit as in this experiment. The end result runs about the same as the others.

A matter of interest here is the recurrences of the tumor after it healed pre-eminently in 3 cases that received more than 4 minims each. There were two such that received 6 minims in one group, and one in another. The explanation can be found in the text. In these animal experiments the word “cure” is used to indicate the complete absorption of all tumors, visibly and palpably, the healing of the lesion, and the return of health to the animal.

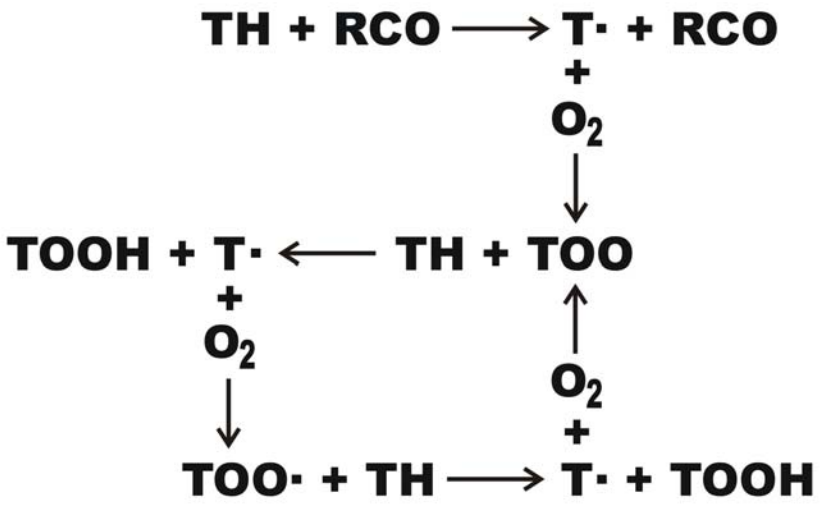
CHAPTER VI

ENERGY PRODUCTION

In order To explain the therapeutic procedure and reasons for certain features controlling the care of the patient, a consideration of the energy producing mechanism will be helpful.

We do not know what energy is, but we can differentiate several forms, and measure them in various ways. It is the consensus that all energy produced in die cell, whether by oxidation — namely the Krebs tricarboxylic acid process — or by fermentation, is the same and is stored as ATP (Adenosin triphosphate) high energy bonds, before it is transferred to the working elements of the cell co be transformed into the energy of work. No other mechanisms of energy production are recognized, simply because no intermediaries identifiable with any other processes have been encountered. However, there is plenty of room for the operation of a far more efficient process than the Krebs cycle, which indeed is a decarboxylation process nicely adapted to the lower forms of life. The clinical data show that the Krebs system does not fulfill the requirements of the oxidations that maintain health and that some other process of higher efficiency is present that provides the Survival Factor we have identified and reproduced for four decades for clinical use. The intermediaries of this High Efficiency Process are not to be trapped. They constitute the "smokeless flame" that supplies the energy as a preferred process. This is a postulate that will be supported by practical proofs later.

Ochoa and others have calculated that the combustion of a gram mol. of glucose in the tissues yields 450,000 calories of energy as the total from the various steps of the Krebs cycle. This gives 36 (38) high-energy phosphate bonds with a P/O ratio of three. The free energy Δ F of glucose is, however, — 691,000 calories, and the energy of combustion Δ H is -673,000 calories. Thus, 18,100 calories are consumed in the process. The energy calculated as — 450,000 calories from the Krebs process of oxidations is therefore — 220,000 calories shy of the — 673,000 calories available for work, and that are not accounted for in any way. Therefore, some other process of higher efficiency than the Krebs cycle has plenty of room to operate. Further, the highly inefficient Krebs process (65%) offers no protection against pathogens as it provides no O/R potentials high enough to start their combustions, but supports viral and neoplastic processes instead. It thus does not account for the survival oxidations that are clinically demonstrated. This process we identify with the FCG dehydrogenations that start a chain of oxidations via the free radical formed and its addition of Oxygen to produce a peroxide free radical as carrier of the process. In free circulating toxins the reaction may be pictured thus:



Where TH is the toxin, and RCO the Survival Reagent Carbonyl Group.

In combined or integrated toxins, the most mobile hydrogen atom is alpha to a double bond in the most activated region which would be adjacent to the position of integration with the FCG or its activating double bond. Free radical production where this hydrogen is removed and addition of molecular oxygen to form a peroxide free radical causes cleavage into two parts with carbonyl terminals. The azomethine double bond is broken, and the nitrogen oxidized away. The double bond of the new carbonyl groups formed by the cleavage activate alpha positioned hydrogen atoms where further dehydrogenations repeat the process forming more peroxide free radicals that are the best dehydrogenators and carriers of the oxidation chains to new molecules of fuel or toxin that enter the field. All of the peroxide free radicals serve destructively to toxins therefore and constitute what we referred to as the antitoxic state of structure of the toxin after the Synthetic antitoxic reagent worked on it. Koch, "Cancer Journal," October 1924, Philadelphia; Koch, "Investigations," 1924. Here the process was described as converting toxin into antitoxin.

Our postulate required that the FCG is activated by conjugation with the double bonds of an ethylene linkage from which it receives electrons. These two atomic groups are the center of romance in the maintenance of function and in the protection against toxins that enter the field. They are also the centers of attack of pathogens in viral, neoplastic and degenerative diseases. Whether or not a pathogenic response is to be had will depend upon:

(a) the presence or absence of a normal quota of molecular oxygen, and

(b) upon the firmness of condensation of an amine with the FCG, that guarantees or destroys its potency.

In case the normal range of energy production can not cleave the bond, FCG function is destroyed and disease results. Then a superiorly efficient dehydrogenator carbonyl group must be supplied to burn off the pathogen as previously stated. This restores the FCG for normal function. This super dehydrogenator is our therapeutic Synthetic Survival factor. (SSR).

The adequate oxygen supply plays two parts:

(1) It is the ultimate electron acceptor when the hydrogen atom removed from fuel or toxins by FCG is transferred to some oxidase system so the FCG is free to start new oxidations, and

(2) after the fuel or toxin is dehydrogenated to become a free radical, molecular oxygen must be at hand to convert it into a peroxide free radical to continue the oxidation process.

Otherwise the free radical would under hypoxia add to the closest reactive group that would accept it, and this is the double bond of the ethylene linkage that activates the FCG. The pathogen would thus integrate with the host cell energy producing mechanism where it would draw off energy for its own vegetation or to transfer ectopically, and produce disease, and at the same time block the activation of the FCG and stop further FCG dehydrogenations. Thus oxidation is blocked and the consequent colloidal degenerations would follow to produce further anoxia. Thus anoxia is essential to the integration of the pathogen with the grana when the FCG is still operating. However, the moment the pathogen is added to its ethylenic activating double bond, electrons are no longer contributed to it, so the FCG is no longer able to dehydrogenate, and the grana appears to be out of commission, destroyed and lost. Ectopic uncontrolled transfer of energy to various secreting and contractile or conducting functional systems referred to above we hold to be the cause of allergy.

Since creatine did not interfere with FCG function as did guanidin, and since it is the only amine possibly that forms high energy phosphate bonds, it was easy to assume that it played a role in the transfer of energy produced from the oxidation of fuel to high energy phosphate bonds as of ATP. It would accommodate this transfer by condensing with the FCG to form an azomethine bond until enough energy had been generated to admit phosphoric acid into the bond and unite it with the amine group of creatine sending the creatine phosphate off as a high-energy carrier. The FCG is thus free to start further dehydrogenations physiologically. However, if the amine condensed with the FCG forms a tight bond not separable under normal ranges of energy production as did guanidin in the parathyroid experiments, the whole train of pathological events must follow. For this

reason it is well to inquire into the sources of such pathogenic amines. One is the production of toxic amines in the acid colon by various bacteria that decarboxylate amino-acids. In many people, the intestinal flora are firmly entrenched and convert the food into one's poisons, that serve as the vanguard of disease. Animal proteins are the main sources of these toxic amines, and sulphides, while vegetables, cereals, and fruits supply plenty of protein and at the same time do not support decarboxylating germs. The intestine must be kept at a range of pH above 7, since the decarboxylations progress best at a pH of 3.5 to 6 when mediated by the streptococcus fecalis and so many others.

The fungus found always in cancer is an amine producer that could initiate the pathogenesis as explained above, with its whole train of symptoms. And the modern antibiotic amine poisons, especially those that attack the liver and cause suspensions of consciousness like the sulpha drugs, and any in fact, are to be scrutinized with great suspicion as the cancer death rate his increased so greatly since they have become so widely used. Sulphides and sulphydryl derived from food, add to the double bonds that activate the FCG and thus block its activation powers. The intestinal flora again are to be considered with the diet if one is to maintain a normal function of the FCG as an energy producer and protector against pathogens. Especially during the treatment period, when a dehydrogenator carbonyl group of highest efficiency has been administered, one must protect the oxidation progression that follows from being blocked, as can take place through permanent free radicals as the oxides of nitrogen. Gas anesthesia should never be used in connection with this therapy: Highly polar double bonds can also add to and quench the free radicals of the recovery process and block it, so certain terpenoids and even perfumes, and especially acrolein and polymerizing acrylic aldehydes from frying pans, are to be avoided in this regime. The proofs of our postulate are serious practical facts.

Some medications absorbed into the tissue colloids may alter the steric set-up so that the remedial carbonyl group which ordinarily could attack the hydrogen atom to be removed perpendicularly to the plane of the conjugation of its carrier carbon atom with the double bond that activates it, now finds a distortion that hinders this line of attack. Opiates and coal tar drugs, and especially aspirin, appear to interfere in this way.

THE SUPER-HIGH-EFFICIENCY CARBONYL GROUP:[14]

The atomic set-up of the reagent itself that carries the Super-high-efficiency carbonyl group must offer a steric advantage in each disease where it is applied. For example: in Hog Cholera, diphenoquinone proved 100% efficient in several epidemics, while it proved 100% worthless in Rabies, and the serial system of carbonyl groups used in Hog Cholera proved 100% worthless, while it was 86% efficient in Rabies in terminal

~~~~~~~~FOOTNOTES~~~~~~~~

[14] INSERTED HEADING FOR IDENTIFICATION OF SUBJECT MATTER
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cases. Both diseases kill 100%, within 3 to 5 days. Rabies is neurotropic always and Hog Cholera rarely before the terminal hours. Our search has been for a molecule carrier of the Survival Efficient Carbonyl group that is equally applicable in all diseases where drug interference has not modified the steric set-up. This will be discussed later on. It will be seen, however, that to identify the high efficiency oxidation system, we consider normal to the cell, as of the same order as the therapeutic substitute used to rescue the FCG and restore normal function and structure, a few comparisons will have to be made. For example: that they are of the same order is seen in being blocked by the same agencies as anoxia, sulphydryl, and that their processes are blocked by permanent free radicals, highly polar double bonds, etc. Likewise, the restitution program, that follows the freeing of the FCG system of its pathogen by the Synthetic reagent, is the normal process. So the Synthetic reagent fits into the mechanism with equal grace as did the FCG before it was attacked by the pathogen. Likewise, since recoveries from viral and neoplastic diseases that could never be combated successfully before are accomplished by the natural resources of the body after the Synthetic Survival Reagent is used, they both fit the cell chemistry, but each in its respective capacity for survival. Since the recovery mechanism includes cyclic reactions at definite periods never seen in medicine before, just as the cure of the pathologies involved were never seen before, a deeper grasp on tissue physiology is made possible and a wider range of expertness can be acquired. Any successful clinician will recognize that expertness in this therapy depends upon study and experience, and some new viewpoints must be adopted. To illustrate let us review a few toxic cases.

THE REMEDY:

These cases show the characteristics of the recovery process, which itself gives evidence as to the nature of the etiological factor. Only the most pertinent data is used. It will be seen that each case presents a long pretreatment control period that definitely established the downward trend of health with the steady and often rapid advance of the diseases. Thus the best possible control for comparison of pretreatment and post-treatment progress was followed, and no confusing variables were permitted, as for example, other medications or treatment measures. Likewise factors that interfere with recovery were eliminated, so that the contest lay plainly between the therapy, the patient's cooperation, and physical advantage on the one hand, and the disease forces on the other. The remedy is named the Synthetic Survival Reagent (SSR). There are two forms, the Quinone form which when used is so named, and the carbonyl group chain form with free radical terminals. This is simply called the Synthetic Survival Reagent (SSR) or given a similar appellation. The quinone dose is two micrograms, and the SSR, two micrograms, millimicrograms and micro micrograms in water, given intramuscularly or under the skin.

With few exceptions, the case records are taken from Federal Court and Federal Trade Commission testimony, where they were proven factually uncontradictable. Some of the exhibits have been reproduced for use in this book. This policy was adopted to give the student full confidence in the proofs of a thesis as unusual as this one.

A case of toxic nodular goiter illustrates some of the main features of the oxidation mechanism of our postulate.

CHAPTER VII

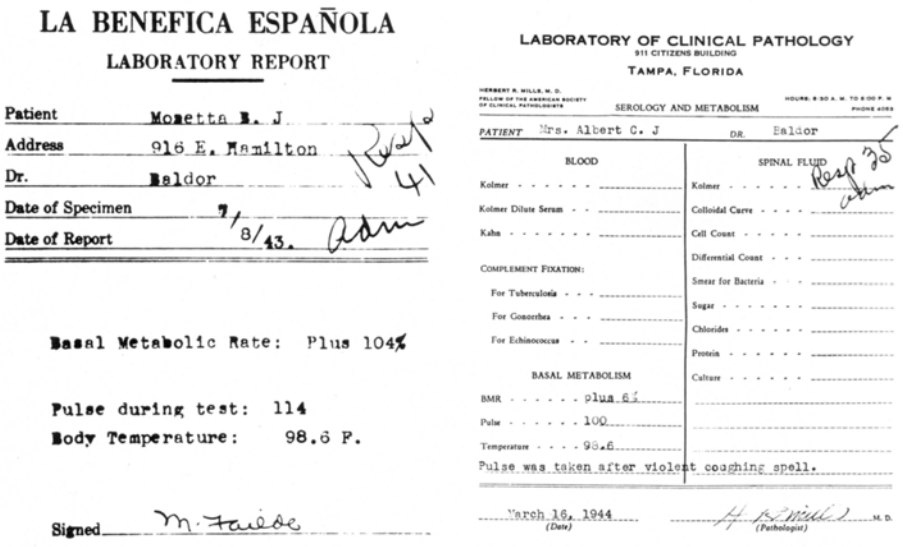
CLINICAL PROOFS OF HIGH EFFICIENCY AND SSR OXIDATIONS

TOXIC NODULAR GOITRE

CASE No. 1 **Dr. Baldor**

Mrs. M. J. was 35 years of age in July, 1943, when she came to Dr. Julian Baldor for treatment of a rapidly developing weakness, tremor, sweating, a big change in her appearance, loss of weight, twitching of her muscles, continual excitement, excessive nervousness, considerable loss of weight, pains in her legs, terrific heart palpitation, and shortness of breath. She noticed her eyes were "popping out", and that her neck had become enlarged by a number of hard nodular tumors. Although the situation had only started a few months previously it had advanced rapidly until she was almost helpless. Examination by Doctor Baldor showed a very rapid pulse, a blood pressure of 190/110 and the other symptoms mentioned, and the Basal Metabolism Rate was found to be plus 104, instead of plus 4 or 6 which would be normal. He noted the nodular development of the goitre which meant that iodine therapy would not help as it does in other cases without this pathognomonic change. The exophthalmia was excessive, and meant an advanced stage of toxicity. Operation could not be done under the circumstances, without first reducing the symptoms to the limit and, in the only way this is attempted, he gave her the iodine therapy with ice bags to the neck and quiet, on July 8, 1943. This treatment was continued until October 30th when it was discontinued as a failure. Indeed her whole condition had become so much worse, she was about wild with excitement and nervousness, she experienced things that made her think she was losing her mind, the muscle tremors increased and jerked uncontrollably besides. She had become so weak she had to be carried and had lost control of her hands and feet. The exophthalmus and the tumors of the neck had increased exceedingly, the heart showed signs of failing in the weakness of the ever rapid pulse, and the drop in the systolic pressure from 190 to 170, while the diastolic stayed at 110 showing that the toxic cause of the high blood pressure was still as bad as ever. Operation was out of the question, so Dr. Baldor decided to use the reagent discussed in this book. The iodine therapy was stopped two weeks, so she would be ready for the SSR treatment. In these two weeks, she became worse at the same speed as previously. Thus the pretreatment control period showed a steady advance of the disease.

She was so weak by Nov. 10th that she had to be carried into the car and into Dr. Baldor's office to be given the treatment, an injection of 2 micro micrograms of the Synthetic Survival Reagent (SSR). Every physician knows the value of the patient's own description of her symptoms and status, so we will let a few words from her personal report emphasize some of the points we wish to establish. She stated:



LA BENEFICA ESPAÑOLA

LABORATORY REPORT

Patient Moretta B. J.

Address 916 E. Hamilton

Dr. Baldor

Date of Specimen 7/

Date of Report 8/43.

Basal Metabolic Rate: Plus 104%

Pulse during test: 114

Body Temperature: 98.6 F.

Signed M. Faldo

LABORATORY OF CLINICAL PATHOLOGY

911 CITIZENS BUILDING

TAMPA, FLORIDA

HERBERT R. MILLS, M. D.
FELLOW OF THE AMERICAN SOCIETY OF CLINICAL PATHOLOGISTS

SEROLOGY AND METABOLISM

HOURS: 8:30 A. M. TO 5:00 P. M.
PHONE 4093

PATIENT Mrs. Albert C. J DR. Baldor

BLOOD

Kolmer

Kolmer Dilute Serum

Kahn

COMPLEMENT FIXATION:

For Tuberculosis

For Gonorrhea

For Echinococcus

BASAL METABOLISM

BMR plus 6%

Pulse 100

Temperature 98.6

SPINAL FLUID

Kolmer

Colloidal Curve

Cell Count

Differential Count

Smear for Bacteria

Sugar

Chlorides

Protein

Culture

Pulse was taken after violent coughing spell.

March 16, 1944 (Date)

H. R. Mills M. D. (Pathologist)

Author's Note: M. B. J. and Mrs. A. C. J are one and the same person.

> "First my trouble started in my finger tips with throbbing. It seemed as if the blood circulation was half stopped. My hands began to swell and I could not wear my rings any more. I had terrific pains and then I began having trouble with my legs. I began to have contraction of the muscles, my toes would draw up into knots. I went to Dr. Baldor about it. He gave me one thing after another, but I did not improve any. He sent me to the clinic for the metabolism test, and after that he started a different treatment. It was some drops. All the time I was taking on like crazy. I could not sleep at night. My husband had to lift me up in bed. My hands and legs got steadily worse. Finally I got so bad, my husband had to pick me up and put me in the car. I could not get in. My legs would just turn to water. Dr. Baldor gave me the Koch treatment. About two or three weeks later, I felt like a new woman. My strength came back, my legs and hands cleared up, and I can use them again. I now have a job demonstrating. I carry a suit case weighing fifty pounds in and out of homes."

The Basal Metabolism Rate was taken three months after the treatment, and found to be perfectly normal, namely plus 6, and physical examination showed her normal in all other respects, no sweating, no jerking, no tremor, no muscle twitches. The exophthalmus had completely disappeared, and so had the thyroid tumors. The thyroid gland was normal on palpation, inspection and function. The pulse was normal 80 to 90, and so was the blood pressure, 140/80. She was strong, slept well, and without any trace of the former disease.

43 CLINICAL PROOFS OF HIGH EFFICIENCY AND SSR OXIDATIONS

An analysis and interpretation of this case notes two toxic states — one that is the result of the forced secretion of the Thyroid cells, the *thyrotoxicosis* that nearly killed the patient. The other is the toxin that blocked the regulated energy production and the regulated energy acceptance and utilization in both the thyroid cells and the tissues in general. This is the *pathogen* toxin which the postulate identifies as an amine of higher 0/R potential than the functional carbonyl (FCG) group of the tissue cells could dehydrogenate and thus burn out of the way. The pathogen toxin therefore had the upper hand and as it was being increased in amount, its effects were also increasing as the block to energy production and energy acceptance by the tissue functional carbonyl groups. These normally initiated oxidations that produce energy efficiently, and received energy in a regulated way to perform work.

As a result the Krebs Cycle energy production took over, and had already largely replaced the high efficiency oxidations of the FCG-s, when the patient came under observation of Dr. Baldor. If the toxic amine pathogen had been subject to dehydrogenation at the hands of the FCG like the usual run of pathogens and fuel substrates, it would have been burned out of the way, and could not condense with the FCG of either the high efficiency energy producing system or the FCG of the energy accepting system of the cell and block their functions.

It was evident clinically that energy was not reaching the working mechanisms. This was seen in the steadily increasing weakness of the skeletal and heart muscles and nervous system. It was also clinically evident that energy production was going on at the highest rate shown by the BMR of 104%. It was also seen that the thyroid gland was forced to the limit in producing its secretion to push cell activity to evolve more energy. But no matter how much was produced even to the exhaustion of the patient, none was used by the energy starved cells. The patient lost weight rapidly and to the extreme to supply material for energy production, but it could not get into the working mechanism via the blocked carbonyl groups of the energy accepting system of the postulate. *The basic pathology then was the block to energy acceptance by vital working units.* Since one toxin, an amine of high activation was the pathogen, it is also evident that the FCG's of energy production and energy acceptance are similar atomic groups and since these are dehydrogenators, the postulate identifies them as highly activated carbonyl groups. This conclusion is supported by the type of response to oxidation the integrated toxin gives, after it is condensed with the tissue cell FCG's. That is, the type of cleavage observed is that of an azomethine double bond when its alpha positioned hydrogen atom actually invites dehydrogenation and is removed so a free radical can be formed and add molecular oxygen to become a peroxide free radical which accomplishes the oxidative separation with restoration of the functional carbonyl groups of the tissue cell, and the toxic amine group is s burned away. The facts of the case history support this explanation. (See A appendix) .

It is seen here that the pathologic state actually invites correction, and any clinician would suggest correctly how it could be done. He would say, since c the FCG can not dehydrogenate the toxin and start its combustion, because its O/R potential is too low, then the thing to do is to offer a carbonyl group of higher activation with a potential equal to the job. This is what was done in this case. A molecule of correct steric advantage carrying a carbonyl group of high O/R potential was used. The results were the rapid reversal of the Pathogenesis. As soon as the integrated toxin was burned out of the way, energy could enter the cell working units, and the urgent call for more energy stopped. The thyroid was not called upon to whip up the tissues to do more oxidizing, and the nodules it had developed to aid its work subsided and disappeared. The BMR dropped to a normal of plus 6%, and all of the symptoms of the thyrotoxicosis, and of the basic pathogen disappeared. The woman was normal in 3 months after one dose of a highly activated carbonyl compound.

NITROPHENOL SUBSTITUTION AND INHIBITION:[15]

This case proved a few things in the Koch postulate, and it also shows that the thyroid secretion takes no part in the oxidation process, any more than the poisonous nitrophenol series that some have classified as accelerators of the oxidations. As we pointed out here, thyroid function is to whip up the cells to put their oxidation apparatus to work to supply the energy needed for the occasion. It itself does not enter the oxidation process. Nitrophenol blocks various esterifications with phosphoric acid which normally form high energy carrying phosphate bonds. Thus it starves the cells of energy and the tissues are whipped up to produce more energy for survival, just as in the case at hand. Nitrophenol thus works as an “uncoupler" and is so classified. It prevents the energy accepting mechanisms from receiving the energy. In the case at hand the energy came to the doors of the energy accepting mechanism, the FCG of energy acceptance, but the door was closed, — blocked by the condensation with the amine compound. Thus the carbonyl group of energy acceptance was already occupied and could not condense with the amine of the ATP that carried the energy that would be liberated by ATP-ase with the help of calcium. Our postulate goes on to explain that with the liberation of energy by the hydrolysis of the ATP to ADP, the phosphoric acid set free can split the azomethine bond setting the ADP free to again do another cycle of energy transport with the acid.

One sees that there is no similarity in the actions of the thyroid secretion, the nitrophenols and the highly activated dehydrogenator carbonyl compound (SSR) used to

~~~~~~~~FOOTNOTES~~~~~~~~

[15] ADDED SUBHEADING TO EMPHASIZE SUBJECT MATTER. This constitutes an elaborate and somewhat complicated analysis as to how and why conventional thyroid treatments do not correct underlying causal mechanisms contributory to thyroid dysfunction and subsequent pathological chains of symptom pictures as the patient degrades physiologically, undergoes additional stressors or traumatizing events.
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oxidize the pathogen out of the way. The SSR actually took the leading part in the oxidation mechanism and did the work the normal oxidation initiator would have done if it had an adequate O/R potential. It is not possible to compare a reagent that prevents energy storage for use in work, with an agent that produces energy for use in work, and besides, actually starts the oxidation process in the cell by burning away the pathogen that was blocking energy production.

Further, the nitrophenols are pathogens whose action can wear out if not forced too long. But if f they are forced too long, they are subject to reduction to aminophenols, which would then act much like the pathogen in this case, and block the initiation of the oxidation progression, and bring about a dangerous situation much like in the case at hand. The nitrophenols proved to be pathogens in the attempt to beautify obese women. The reduction in weight took place but in too many the destructive action continued because of the situation that existed in the case we are discussing, and these victims went on to their deaths. They need their FCG's freed from the obstructive amine as was accomplished in this case. However, the experts are still at sea with regard to the true action that caused the fatalities.

The block in the use of the energy of oxidation by dinitrophenol is seen also in its inhibition of mitosis in Sea Urchin eggs reported by Clowos (1951 Ann. N. Y. Acad. of Science). Even though the dinitrophenol in doses of .01 mM concentration caused a fourfold increase in the consumption of oxygen, the mitosis and phosphorylation was cut in half, and further increase in the concentration of the poison completely blocked mitosis and phosphorylation. So whether the oxidation process is blocked in producing energy, m transferring and carrying energy in phosphate bonds, or in receiving this energy, the reactive response is to produce more energy to make up for the energy starvation in the tissues whose working mechanisms do not receive the energy. Thus an analysis of effects of toxic amines and nitrophenols shows they do not give impetus to the oxidation mechanism, but block its ultimate purpose — the supply of energy to the vital mechanisms of the tissues. Here we find in 1951, a nice confirmation of our postulate measured with microscopic accuracy.

The thyroid secretion is a hormone whose intimate action is still unknown. However, it does not take any part in the oxidation process itself. Comparing its action with that of the SSR, one sees that the latter took the leading part in the oxidation mechanism. Further the action of nitrophenol and of thyroid extract are of different orders and challenge comparison. The form always has a toxic action, the latter is physiological, but the action of both, as explained before, is very different from that of the SSR. The high BMR in the case at hand has a pathologic cause depending on the pathogen that blocked energy acceptance by the cell's working mechanism.

The statement of some biochemists that the oxidation process has no immunological significance is based on the fact that the Krebs Cycle has none. We gave the reasons before. The 0/R potentials of the participants are too low. Then these

biochemists also hold that the Krebs Cycle is the only mechanism concerned in the tissue oxidations, and is all sufficient. They do not consider that the Krebs Cycle is a hang-over from the process used by primitive forms as bacteria and though it is retained by the higher forms as animals and man, it is only used by such as an alternative pathway when the High Efficiency System already explained is inactivated for a time. That it offers no protection is seen. Moreover, it gives no clues to the explanation of the Pasteur Effect. The early chapters of "The Survival Factor in Neoplastic and Viral Diseases" show how both depend on the action of the FCG. While the carbonyl group that initiates the oxidations of the High Efficiency Smokeless System, lacks the high 0/R potential carbonyl dehydrogenator that some pathogens require for their destruction, yet its range of 0/R potentials is twice as high (0.7 v) as that of the Krebs Cycle participants (0.3 v). So the opportunity to give protection by the High Efficiency System is considerable, — enough to maintain good health under the usual circumstances. The use of a Super-high carbonyl dehydrogenator of correct steric advantage is proven in this case to offer protection by way of an oxidation process that imitates that postulated for the High Efficiency System, and a close analysis of this case is all that is needed to prove the existence of the High Efficiency System. However, two more cases will be submitted to show that the toxic basis for malfunction can be removed, and the pathology corrected by the processes of adequate dehydrogenating efficiency, started by the Super-high dehydrogenator, and continued by the natural dehydrogenator (FCG) system.

POST-PNEUMONIA NEPHRITIS

CASE No. 2 **Dr. Evans**

Tom F., 4 years old, was recovering from bilateral bronchopneumonia, when he suddenly took a convulsion of considerable severity. Oedema rapidly developed with blurred vision, headache, dizziness, delirium, etc. The urine secretion diminished as the oedema rapidly increased. The blood pressure was found to be 146/68, and the blood non-protein nitrogen 74.6 mgms. % . Twelve hours later the pressure rose to 160/100, and two days later it was 180/130 showing a rapid development of the pressor substance that blocked the kidney elimination. The oedema had developed by then to the point where the contours of the chin and neck were obliterated, and very little urine was passed. Then the second convulsion took place. It was severe and the boy passed into coma. It was in this condition that he received the Synthetic Survival Reagent. A few hours later, the mental symptoms had improved, he came out of the coma; soon the headache, blurred vision, delirium, etc., gave way to rational me[illegible]comfort, the blood pressure steadily dropped and the urine increased as the oedema disappeared. The blood pressure was found normal in a few days with a normal non-protein blood nitrogen of 25 mgms. % . The correction was completed by rescuing the FCG so it would go back to work again. The pressor substance is well known now to be a toxic amine, so our thesis is supported nicely by this case also.

ECLAMPSIA

CASE No. 3 **Dr. Baldor**

Mrs. D. was married seven years and could never carry a baby to term. Abortion was required before the end of the second month of pregnancy each time, and the period was shorter each time. This was the 4th pregnancy, and they all followed the same course and symptoms but with increasing severity. In each instance she vomited profusely, with much salivation constantly; the urine was progressively decreased until only blood came, just as in the last hours of the parathyroidectomy intoxication. Convulsions followed by coma called for immediate abortion, if life was to be saved. This time, however, Dr. Baldor tried the Synthetic Survival Reagent as her big ambition in life was to have a baby. Twenty hours after the injection was given, vomiting had decreased from 20 times a day to twice per day. The urine increased and, in 72 hours, she was passing half a liter a day. This urine still carried blood and albumen. In four days, she passed a full quart of urine per day. The vomiting disappeared entirely within two weeks, but the salivation had continued and, during the third week, vomiting started again. She was given another dose of the Survival Reagent, and all cleared up quickly thereafter. No more symptoms of eclampsia returned. She carried her baby comfortably into the seventh month, when she had an automobile accident, and spontaneous abortion threatened, so she was delivered of a 5½ pound baby that thrived well. She had no return of eclampsia symptoms and gained full health quickly.

Here we see again that the toxin that blocked the oxidations of function and the regulated energy acceptance by the working mechanisms, could be removed by an atomic group similar in kind but of higher 0/R potential. The allergic uncontrolled spasms of the small blood vessels, and the anoxia caused by colloidal gellation, had to yield to restored efficient FCG function. The basic pathology was met and corrected, at its very inception. Still, some of America's greatest biochemists and clinical experts claim that "the oxidation mechanism has no significant action or position in the maintenance of health or in the combat against disease." They are limited, of course, by the performances of the Krebs cycle, which to them is the whole oxidation mechanism. But, if one were to accept such a dictum, one would have to add "it is *impossible to die of* asphyxia." The predicament is rather contrary to progress.

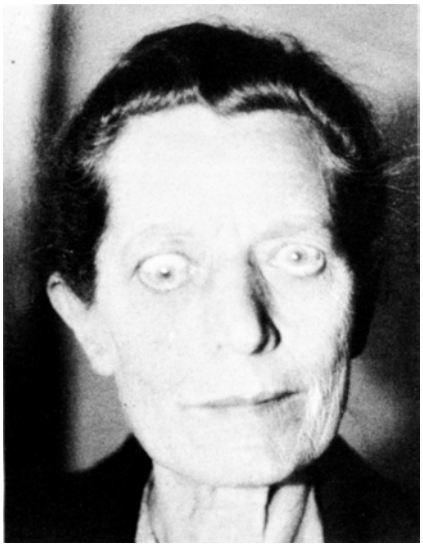
TOXIC GOITRE AND CANCER OF THE STOMACH

CASE No. 4

To show that one toxic agent (removable by one corrective attack) can cause a toxic hyperfunction as in Case I, and also cause a very high grade malignant neoplasm of the stomach in the same person and at the same time, the case of Mrs. W. is offered.

At the time this patient was treated, the Geiger Counter had not yet been invented, so it was impossible to estimate the earth's irradiations in her environment. However, it is noted that she lived in what is known as the goitre belt, a region of iodine deficiency and also of high cancer mortality rate. Her daughter had been treated for a rapidly developing brain tumor. Many other patients came from this region for treatment. However, one thing this study lacks is a systematic correlation of the terrestrial radiations with cancer incidence and also the number of conditions allied to cancer to be met; and most of all, how the terrestrial rays affect the recovery rates both of the neoplasms and of the allied diseases.

There was no history of cancer in the ancestry, but her husband died of cancer 8 years previously, and her daughter, with a very malignant tumor, was only 28 years of age as compared with the patient's age of 58 years, at her first visit. One recognizes here the vigor of the carcinogenic flux of this region. Both the mother and daughter made typical recoveries under the treatment. There was nothing in the geophysical environment that interfered with the cyclic reactions and the steady progress of the recovery process. One feature to be noted is that as cancer is associated with aging processes, this patient, at only 58 years of age, looked like a person twenty to thirty years older. The skin and tissues in general were senile, though the hair was not gray. During the recovery process the senility changes disappeared. The main features were AS follows:



Mrs. W. before treatment showing the exophthalmus from toxic goitre excited by the carcinogenic toxin.

Mrs. W. after treatment and recovery from cancer of the stomach, and toxic goitre as secured from one chemical reagent. The exophthalmus is gone for good.

The disease started two and one-half years previously as a steadily-increasing nervousness, progressive cardiac weakness, tachycardia, increasing ease of perspiration, loose bowels, and tremor of characteristic hyperthyroid type. Radiographs showed considerable enlargement of the heart and mediastinal shadows early in 1927. There was dyspnoea on slight exertion or lying down. Exophthalmus developed rapidly, the skin was bronzed, and gastric distress and inefficiency set in. The feet and ankles swelled considerably, yet she lost weight, falling from 150 pounds to 108 pounds in less than nine months. The physical examination revealed the exophthalmus as shown in the photograph before treatment. There was also a greatly enlarged lymph gland (walnut size) in the left supraclavicular space; the veins of the head and neck engorged with blood when she laid down, and percussion showed a marked increase in the mediastinal dullness. Examination showed the epigastrium and the whole area below the costal border down to two centimeters below the umbilicus on the right side to be occupied by a huge, bulging, solid, fixed, irregular tumor. The stools showed decomposed and occult blood. There was vomiting and great weakness and considerable pain throughout the abdomen. Thus the stomach, the liver, and probably the suprarenal glands were involved by the neoplasm. At the time of this examination she was very weak.

One dose of two cc. of a 10^{-12} solution of the (SSR) serial carbonyl system was used on September 28, 1929. The recovery process exhibited the usual cyclic three week reactions, with chills, fever, and general aching; and with improvement following each reaction until the recovery became complete. At last report, ten years later, she was in good health. We lost track of her thereafter.

Regular FCG function, both for producing and using ATP energy, was blocked and this showed for the thyroid cells, the stomach growth mitotic mechanisms and the general tissue oxidations as demonstrated by the senility changes. Still the Krebs Cycle oxidations went on, and fermentation supported the neoplastic cells. Had we supplied a carbonyl group of FCG oxidation potential, we probably would have gained nothing. However, a carbonyl group of boosted 0/R potential cleared the inactivator of FCG functions away so normal FCG metabolism (in contrast with the Krebs metabolism) was restored, and senility, toxic goitre, and cancer, all faded away permanently.

TOXIC GOITRE AND ETIOLOGICAL TOXIC FOCI

CASE No. 5 **Dr. Jayme Treiger**

In this case the pretreatment control or observation period lasted from September 12, 1953 until March 13, 1958. The development of the etiological factors with the progress of the disease itself was well noted.

Mrs. D. S., F. 27 years old, married, a thin brunette woman, very nervous, complained of dyspnoea, cold sweating, pharyngial spasm (sensation of an egg in her

throat), able to bear heavy duties but not simple ones, urine sometimes fetid and strongly colored, acne, leucorrhea, sometimes bloody, and painful nodules in the right breast. These breast symptoms arrived after a second electro-coagulation of an ulcer on the cervix uteri, produced after the second childbirth. These nodules were helped by hormone treatment for a while but had returned, with further toxic symptoms as a tachycardia of 106 per minute, and slight thyroid enlargement, that started nine years previously. She had pertussis, measles and vericella during childhood.

During and since childhood, she had periodic crises of angina with high temperature and pus from the tonsils. Homeopathic treatment helped the tachycardia and the throat spasms and made her feel much better, but the basic pathology was not retarded, and she went to a gland specialist who treated her from November 1954 to January 1956. From him she received Dexamyl, Somniphene, Prometron, Ovocycline, Diiodotyrosine, Apliotil, Thiouracil, and Nodular on different occasions. She did not improve on this series of modified benzene rings, though enough were tried. This shows that the therapeutic conception was not based on physiological considerations, but was the fruit of modern pharmacology.

Feeling worse, she returned to Petropolis. The B.M.R. by Dr. T. showed a plus 45 and a Cholesterol of 122 mgms. percent on 3/12/S8. She was now exhausted, extraordinarily excited, always tired, difficult to sleep, with frequent nightmares, pulse 106 per minute, and her blood pressure in a low range. She was given 2 millimicrograms of the SSR intramuscularly on March 13, 1958, and the reactions that followed are indicative of the sources of her toxins.

Reactions:[16]

> Tonsillitis that was suppressed from activity while under the phenolic treatments mentioned above, started to be active with high fever, pus discharge and pain in violent periodic crises. The bloody drainage from the cervix uteri that was suppressed by the cautery started up again. However, one week after the treatment in spite of the strong angina crises, she was feeling quite well, as if with renewed vitality. A few weeks later she reported again. The pulse was normal, 82 per minute, the blood pressure normal 120/90, and as[PPP43] her good health was being restored, old symptoms of years of little difficulties returned briefly and

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[16] Koch uses the term reactions to describe physiologic events which are parallel to what other professions and persons have termed retracing or recurrence of clinical symptoms either suppressed, submerged, masked or rendered Subclinical by the effects of what are usually pharmaceutical agents that incompletely impact disease etiology. This is a paradigm icon, or characteristic of systems that argue homeostatic, ecological, or biological terrain principles as preeminent over manifestations of symptoms as true identifiers of pathology and pathologic conditions extending over time frames which may last a considerable portion of a patient's life.


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disappeared. She felt good enough to not need a doctor. The throat had normalized and the cervix uteri had healed, and she did not return for more observation. She had received two injections of the SSR, the second one a year after the first, for while the cervix showed no abnormality on examination, there were symptoms suggesting reaction in the deep scars within. The BMR in February, 1960 showed 6% over normal, the breasts, tonsils, uterus, nerve responses were normal, temperature 36.7°C, pulse 60, the B.P. 110/70, and she enjoying the best health she had ever experienced.

In this case the etiological lesions that brewed the toxins that attacked the breast tissues and the thyroid gland were respectively the cervix infection and the tonsil infection. The cautery sealed up the drainage facilities, and made the scar tissue that was infected more anoxic. The reactivity of the reticuloendothelial cells of the tonsils to their contained infections was suppressed by the phenolic derivatives, so the thyroid was poisoned all the more. Further, the poisons from the cervix and those from the tonsils while showing some specificity to the thyroid and breast tissues, were also general poisons and affected all of the tissues making her nervous and weak aside from a special thyroxin effect. Here the relation of the reactions (following the treatment with the SSR reagent, which were severe) to her improvement in tissue function showed that these reactions were not of a vaccination nature, but were actual reticuloendothelial battles against the disease agents going on in conjunction with the chain oxidation of these agents. Then, too, as the various FCG units were liberated from combined toxins and went back to work, she started to feel normal and her various functions behaved normally again. It is to be recalled that after the SSR was given the tonsils became acutely inflamed, and the cervix lesion broke loose with a strong inflammatory process. Thereby both lesions were cleared of their imprisoned germs and fibrosis integrated pathogens, and as the induced oxidations burned the pathogens away, the fibrosis disappeared also. The anoxic centers were wiped out, so the disease was cured right at its very inception. She has no more sore throats nor cervix troubles, and no more secondary effects, as thyroid enlargement or abnormal function. The breasts have no more nodules either. Her general health is normal. Her nervous system is steady. She sleeps normally and does not sweat as she formerly did. In other words, the pathology was fully reversed and discarded. This same course will be seen in the other cases reported here, and in all others when one takes the trouble to thoroughly check the recovery course.

APPENDIX[17]

SPECIFIC EFFECTS OF THE CARCINOGEN, VIRAL AND CHEMICAL

To account for the Pregrowth Symptoms and Changes would take much experimental work. However, we have a few clinical facts that are so evident that there need be no doubt. One is that the reticuloendothelial system weakens and trophies just previous to the appearance of the tumor, and before it goes truly malignant. This happens in natural and in chemically induced cancer as well as in virus produced cancer. The fatigue and the final atrophy of the protective cells of the liver, spleen and lymph glands must result in abnormal products which are atrophy producing. The carcinogen, be it chemical or viral, must be ligated with these products as a chemically integrated complex, and wherever such products are carried they Must have their effects. Embryos developing under such circumstances must be defective, and adult tissues will undergo the changes that give the picture of cachexia as an end result. But preliminary thereto, there must be functional injuries such as were described in part in the text, the bone marrow function, the nervous system function, the skin function and what not. In fact every tissue is subject to the action of such poison in varying degrees. Thus a differential diagnosis may be based on the chemical status of the lymph glands even before a tumor is recognized.

Our postulate provides for the polymerization of the carcinogenic toxin as it develops to the cancer producing stage, and this provision is based upon the chemical and clinical circumstances that stare one straight in the face. Atrophy precedes neoplasia. If one answers that the neoplasia is a reaction to the atrophy stimulus as hay fever is to the pollen stimulus, one must still offer a mechanism for the reaction. The simplest mechanism that could be involved is that the toxin produces both changes, and this mechanism we have already explained as due to block in energy production and transfer. Recovery from the states caused by the carcinogenic agent, be it virus or chemical, is therefore a satisfactory support to the contention, since the same agency accomplishes the corrections of all such states, atrophy, pregrowth toxic state, cachexia, and the tumifactions.

~~~~~~~~FOOTNOTES~~~~~~~~

[17] THIS APPENDIX APPLIES TO AN INTRODUCTION TO FREE RADICAL THERAPY SECTION. This material was originally at the end of the book, here inserted to quickly reference to the relevant section.
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The best proof of the correctness or practicability of any postulate in medicine is doubtless the curative value of its application. So to check up on the correctness of our thesis on the atomic bondings and electronic dispositions responsible for the integration of the pathogen with the host cell's functional mechanism, we developed an additional step for demonstrating the presence of the double bond that activates the production of the free radical in the pathogen which makes the pathogenic addition. Likewise the double bond that activates the condensation of the pathogen's amine group with the carbonyl of the host cell can be demonstrated. As we showed, these additions depend upon anoxia.

By starting out with the contribution of an atom of hydrogen from the reagent to one pole of this double bond or of any other double bond in the pathogen whose exposure and high polarity invites the addition, a free radical MM is produced at the other pole of the bond which simultaneously adds to the free radical formed in the reagent when its hydrogen atom was lost to the pathogen. By synthesis, the hydrogen atom concerned was made most active by receiving electrons from the double bond system with which its position was conjugated, and the free radical left in the reagent that adds to the other pole, brings to the pathogen a moiety that is a great electron donor and hence invites ready oxidation (dehydrogenation) even by the weaker of the tissues' oxidation agents. Thus the integrated pathogen is left at the mercy of the ordinary tissue reagents that can accomplish its progressive oxidation away from the host cell's functional mechanism leaving its FCG activated by conjugation with a carbonyl group, the advantage of which we explained earlier.

The first step then is a reduction which opens the way for the burning of the pathogen off from the host cell by ordinary cellular dehydrogenators. The advantage of this means of attack is quite evident, and our next edition will report the clinical results. These have been most gratifying so far. In fact, hog cholera and dog distemper, which required different reagents, are both quickly cured in the highest percentage on the one reagent discussed here.