

CHAPTER 9

The Antibiotic Problem

There is no evidence that a high-efficiency system of oxidation exists in bacteria, especially in disease germs. However, they do depend upon an unrecognized Carbonyl function that is activated by electrons received from the double bonds of an ethylene linkage, even in the performance of the Krebs Cycle. This is seen where it is necessary to keep the cycle going by supplying the unsaturated dicarboxylic acids, whereas the saturated dicarboxylic acids, as succinic, are of no help. Thus, even in the low-level Krebs Cycle, upon which bacteria depend, the activated Carbonyl function of this particular order is required. Here, antibiotic activated amine groups have a position in which to produce their toxic effects, and either kill, asphyxiate, or suppress vital activity. The modern antibiotics, in medical use, all contain the toxic amine group and their toxic effects are seen both on the germ and on the host, when used therapeutically. Suspensions of consciousness, injured liver function, and other metabolic injuries are frequently reported, and sometimes, instantaneous death.

Further, the injurious effects on bacteria excite well-proven mutations against the amine poisons. The survival factor in the germ has reached enough success in this combat to excite scientific envy. When the writer balanced up his observations on the reciprocal and antagonistic actions of Carbonyl and amine groups and the reactions of the tissues to them, one experiment that he published in the "Journal of Laboratory and Clinical Medicine" in 1916, exposed the existence of a basis for the mutation against toxic amines. The poison observed was trimethyl melamine. It is probably one of the most toxic substances in existence and produces instantaneous death when injected in fatal amounts into the blood stream. When sub-fatal doses are injected and the blood pressure recorded on the revolving drum, it is seen that a rapid and sustained fall in blood pressure occurs, but if more of the poison is injected during this period, in amounts that add up to more than the lethal dose, there is no fatal event, but only a minimal decrease in the blood pressure during this period of sustained depression. After the pressure returns to normal for ten minutes, a fatal dose will kill immediately. However, the occurrence of this refractory period indicated to the writer, that a basis for building a resistance to amine toxins and their derivatives existed, and to attack germs via toxic amine therapy would ultimately turn out unsuccessful. He, therefore, decided that for the correction of the basic fault in the host, the boosting of its Carbonyl function would be a preferable means of attack. To the surprise of all observers, it was found that after animals suffering with severe mastitis were given the Synthetic Survival Reagents, the most deadly *Staphylococcus Aureus* lost its toxic action, and its hemolysins were no longer formed.

Name of Cow	First Bacterial Count	Second Bacterial Count
Diane	472,000	165,000
Pearl	25,000,000	95,000
Edna	2,580,000	6,000
Lily	88,000	4,000
Molly	18,000,000	25,000
No. 11	110,000	1,000
No. 10	57,000	26,000
Nancy	10,000	1,000
Polly	215,000	172,000
6351	414,000	6,000
Beauty	24,000,000	18,600,000
Flossie	5,000	4,000
Mary	2,000,000	700,000
icl-2v	23,000	2,400
Star	483,000	82,800
7601	203,000	78,000
x33140	2,000	1,100
5051	228,000	5,500
Hole	45,000	10,000
Mona	3,300,000	16,700
Marjory	10,000	35,000
No. 13	3,000	13,000
No. 14	4,000	14,000

Later on, the same was found true for the Streptococcus causing dairy cattle mastitis. It is seen, in the following table, that where gangrenous conditions were caused by the infection after injury had taken place, the bacteria actually increased in numbers while the toxic symptoms disappeared, and rapid healing was going on. But, where no gangrenous condition was present, the infection and its changes subsided together, with rapid decrease in the number of bacteria. Thus, it is evident that the bacteria lost their pathogenicity after the host was treated with Survival Carbonyl groups, and became useful in clearing up the debris, they had formerly caused. Evidently they gained also from the Survival Carbonyl chemistry and were apparently enabled to serve constructively in the Great Biological Economy again. There were special studies by the University of British Columbia Veterinary Department on the loss of hemolytic properties of bacteria after the host was Treated, as well as restoration of the calcium balance showing that the tissue colloids were again in good dispersion, in other words, the tissue oxidations were restored. The following table of bacterial counts showings the reductions of bacterial counts and no change or the increase in these counts while healing and constitutional recovery is progressing is taken from the Annual Report (1944) of the Minister of Agriculture to the Parliament of British Columbia. *

* Copies of the Minister of Agriculture's Reports are available on this website.

ely-4p	20,000	12,500,000
Nigger	1,000	5,000
Jeannette	8,000	15,000
Vera	3,000	16,000

(Note — These counts were verified by a second laboratory).

It may be noted that cow No. 13 shown above is reported on as drying up with mastitis and ely-4p is shown as improving in udder condition in spite of the high bacterial content. The last-mentioned cow, Vera, had a severe accident to the udder between Treatments which doubtless accounts for the increase in bacteria. However, she was making satisfactory recovery when last reported on.

Other experiments with cultures of the bacteria that caused the lesions, and their high toxicity in comparison to the loss of toxicity of cultures of the same germs planted at different periods after Treatment was given to the host, confirm the observation. This work needs further chemical and clinical investigation, aside from what is reported here. This is useful in showing how antibiotic resistant gonococci and pneumococci became nonpathogenic, after contact with the Synthetic Survival Reagent.

In line with our Postulate, the toxic amine groups of intestinal poisons, those present in antibiotics and those produced by fungi, tend to diminish the oxygen supply to the tissues and thus hinder the combat against viral diseases. The toxic amine group also inactivates the FCG and blocks its function so it fails to oxidize pathogens destructively. Altogether the toxic amine tends to favor the integration of the virus with the host cell's energy producing mechanism. This can be observed practically in the following case of measles.

MEASLES

CASE No. 13

Dr. Jayme Treiger

Baby R., 18 months of age, weight 11,500 grams, was first seen by Dr. J. Treiger 12/3/59. She took sick November 17, 1959, with a terribly itchy rash that was diagnosed an allergy, for which liver extract was given until November 22, when it was seen that it did no good and the condition became worse. Then until December 1, Aminophilin, Streptomycin, and Penicillin were given because of the high fever and aggravation of the

rash, acetyl salicylic acid was also given because the pulmonary congestion was increasing. There was no improvement. The rash became worse and so did the other symptoms during these nine days. Further, the child refused all food and drink but allowed sugar water to be put on the tongue. Prostration developed and constipation also as no food was taken.

At his examination the fever was 39.5°C., the rash of measles fully fulminated, there was a bad tonsillitis, pulmonary congestion, and great prostration. It was not unconscious, but noticed nothing, and refused food and drink. Homeopathic remedies were given that improved the chest and throat symptoms but the rash, fever and prostration persisted with no improvement in the mental state. The prognosis was very grave. Dr. Treiger gave one microgram of one of the Survival Factor Remedies, Parabenzoquinone, in one cc. of water, hypodermically, on December 5th to avoid Encephalitis.

Results. In a few hours there was improvement in the fever and rash. In 12 hours, the temperature was a normal 36°C. with the rash and itching about gone. The child asked for food and ate with appetite. During the rest of the day the recovery was completed, with good restoration of bowel function as well. Measles regularly recover under this Treatment in 12 hours. So even here where every toxic amine group available assaulted the body chemistry, the tissues were liberated by the oxidation potential and advantage of Benzoquinone, the weakest of the Survival Factor Reagents. One may contrast the therapeutic effects of toxic amines and constructive Carbonyl in this typical case.

ANTIMITOTIC AGENTS

The approach offered by antimitotic agents to hit the very center of the cancer problem has been most illuminating. This work was started in the past decade, and has been increasing world wide ever since. An excellent review is given by Biesele, of the Sloan Kettering Institution, in "*Mitotic Poisons and the Cancer Problem*," Biesele — 1958 — Elsevier. Very interestingly, it will be seen that the antimitotic agents present Carbonyl and amine groups of the same order that we have been studying for the last 49 years. Biesele, while paying no attention to these groups or their action individually, classifies all antimitotics as poisons. It must also be noted that since the antibiotic products of molds and soil bacteria have been studied, inspection of their structural formulae reveals similar active amine and carbonyl groups. In view of our experience, the classification as poisons should be reconsidered to group them as amine poisons and Carbonyl restorative or Survival Agents. Indeed the survival capacity is small because of the structures of the molecules, but for the service of the mold that would protect itself, these groups might have corrected the evil effects of the germ's toxins, or hindered them when being constantly produced by the mold in fair amounts. Certainly the mold did not attempt to

kill the germ by the Carbonyl activity. It might even have tried to correct the germ's fault and thus showed some therapeutic discretion. Such agents could really be mold hormones designed to serve within its own level of oxidations and reductions — constructively. Representative of Carbonyl containing antibiotics are the substituted quinones, Phoenicin, Citrinin, Clavacin, and Spinulosin. They all offer Carbonyl activated by the double bonds of two ethylene linkages, but this activation is reduced to a low ebb, by the substitutes for the hydrogen atoms that would have allowed splendid antibiotic activity. The representatives of antibiotics carrying highly toxic amine groups are the Sulfa drugs, Streptomycin with its two guanidine groups, and all of the other synthetic products. Some of these, like Terramycin and Penicillin, carry Carbonyl activated by conjugation with an ethylene linkage, as well as the toxic amine groups, and in some, such groups are protected by a substituent as methyl, that can be readily removed leaving the nascent amine group. The amine groups are likewise activated by conjugation with double bonds.

The same active groups are seen in the antimitotic agents, amine and Carbonyl. Maleic acid and Benzoquinone carry the Carbonyl groups, and their activity is measured accurately even in such infinitesimal concentrations as one part to a million, and one part to a billion of water. It would be unreasonable to speak of such high dilutions as being toxic. So, from their own observations, these agents cannot be classified as poisons to the mitotic mechanism.

In 1943, before antimitotic agents were ever studied, we testified in the Federal Court in Detroit on the corrective action of high dilutions of both substances, which we had used therapeutically years earlier and with corrective results. Since no evidence could be found of their injuring any of the participants or any of the processes concerned with mitosis, Biesele gives them a pre-mitotic position, and loses the real action which is to reduce the necessity for mitosis by their protective action against injurious agents, and their rejuvenating vitality, or survival boosts that lengthen the life span and thus reduce the need for reproduction or mitosis to assure the cultured tissue cell's survival. The tissue culture effect is exactly what we demonstrated in animals and man even under the most adverse circumstances. That the action is attributable to the Carbonyl group as activated by double bonds in conjugation, one sees in the greater activity of Maleic anhydride over that of fumarate which we proved. This is a steric virtue, since in the anhydride both Carbonyl groups are in the same plane with the activating double bonds, whereas in the acid, only one Carbonyl group has that position. This action is, of course, curative and reconstructive in any reasonable dilution up to one part to a billion of water. As to the Carbonyl group working on a physiological and corrective basis that removes the functional and mitotic failure of survival, so well illustrated by cancer, we may refer to the same Court Testimony, which proves the complete permanent cure of far advanced terminal cancer of the liver, accomplished by one dose of two cc. of a dilution of one part

of Benzoquinone to a million parts of water. The diagnosis, which was made by a half dozen leading surgeons by laparotomy, and the cure in 1941, is still standing as perfect 18 years later.

A few quotations from Bieseles will be helpful, along with some explanatory discussion that we must give. This whole book of Bieseles supports our Thesis, in spite of its coming from outside and non-cooperative interests that see nothing in their findings, except cytolytic effects and destructive action. Their observations are well made and accurately recorded, but they lack the physiological viewpoint that gives these interpretations scientific and therapeutic values.

On page 31, it is reported, "The threshold of mitotic inhibition of chick fibroblast cultures by Benzoquinone was 10- μ (Meier and Schar, 1947), or 0.001 μ g/ml, the same as for Colchicine." "The mitosis inhibition caused by some quinones and by Maleic acid has paralleled their uptake of sulfhydryl." (P. 33). This shows, in line with our Thesis, that the action depends upon the activation of the Carbonyl group as recipient of electrons from its conjugated double bonds and that a physiological rather than a toxic action is to be expected, a dehydrogenating power that can initiate chain oxidation. Our diet has accordingly always eliminated sulfides and sulfhydryl. Lettre's suggestions, that "quinone blocked sulfhydryl compounds to inhibit mitosis," Bieseles finds "inadequate, as other antimetotics are effective without blocking sulphhydryl action." However, all students of the problem overlook the oxidation catalysis of activated Carbonyl.

On page 30, Meyerhof and Randall (1948) and Burrough (1955) are quoted on the inhibition of epidermal mitosis by adrenaline and adrenochrome in dilutions of one part to a million. They found that "this inhibition occurred not only with glucose as an energy source, but with fructose, lactate and pyruvate... Further experiments indicated that the critical influence on metabolism imposed by epinephrine or adrenochrome was not on glycolysis, the tricarboxylic acid cycle, or the cytochrome system." Thus, in line with our Thesis, the energy source that is cut off by toxic amines is the unidentified system which we named the "preferred smokeless process" initiated by activated Carbonyl groups, which burns all fuels and even the inhibitors that may be present, but which can be inactivated by highly potent amine groups as of phenylenediamine and epinephrine, which we point out, show reduction potentials of $E_0 = +0.38v.$ and $E_0 = +0.80v.$ Other supportive data deserve attention.

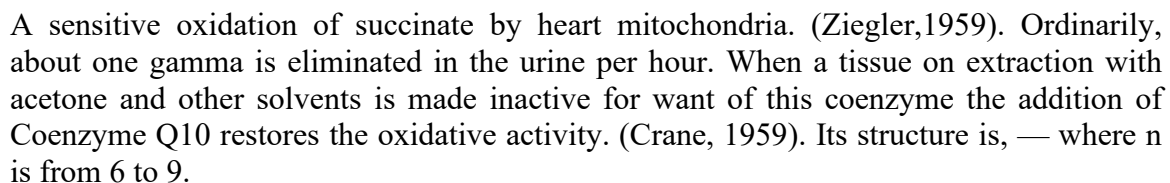
Thus on page 50, Bieseles reviews the contributions of Gellhorn, Hirschberg and Kream (1952) demonstrating that "the differential susceptibility of various tumors to inhibition by 8-azaguanine was inversely related to their ability to deaminate 8-azaguanine to the non-inhibitory 8-azaxanthine. In tissue culture inhibition by 8-azaguanine and the non-

inhibitory effect of 8-azaxanthine on Brown-Pearce tumor were confirmed by Flint, Hirschberg and Murray (1953). Here, again, the inhibitory effect is due to an activated amine group while the protection against the action is a matter of oxidation that removes the amine group and substitutes a Carbonyl group in its place, as we claim happens to viral and other amine poisons during the reversal of carcinogenesis. A further example of this inactivation of toxic amines is found in the observation of Woodside (1953) in "tissues of the mouse in which 8-azaguanine restricted mitotic rates in carcinomas 755 and E0771 but not in tumors C954, C1300, S180 and S91, nor in ileum, jejunum or testis." It must be recalled that the ileum, jejunum and testis are rich in diamine oxidase, and here a Carbonyl action is also involved in removing the toxic amine group and replacing it with a Carbonyl group in line with our Thesis. Here is another indication that free radicals and peroxide free radicals are involved.

On Page 50, Biesele suggests that the "inhibitory effect of 8-azaguanine may be related to its incorporation into ribonucleic acid, as suggested by Kidder, Dewey, Parks, and Woodside (1949) and demonstrated by Mitchell, Skipper, and Bennett (1950). The latter authors found, however, that the amount of 8-azaguanine incorporated into visceral RNA exceeded the amount incorporated into tumor RNA. This posed a dilemma for which Parks (1955) saw no solution.

However, 8-azaquinine-2-¹⁴C was incorporated into RNA of the susceptible mouse leukemia L1210 at a level 100 times that of its incorporation into the derivative azaguanine-dependent leukemia (Bennett, Skipper and Law, 1953). "The incorporation of 8-azaguanine into tumor RNA probably produces defective RNA. It is at this polynucleotide stage that Mandel (1955) envisioned the action of 8-azaguanine as an antimetabolite, rather than as an antagonist of free purines." Here we see in line with our thesis that normal tissues are armed with many more active Carbonyl groups than are the susceptible tumor tissues and these Carbonyl groups can condense with the toxic amine structures and hold them in combination instead of their oxidative destruction. So our interpretation of the induced antimitotic activities and their prevention is that the same survival factor dealt with in this book is concerned and that the antimitotic data gathered so far, gives further support to our Thesis with measurable proof of the efficacy of high dilutions. Though not recognized by the investigators, their data again show one phase of the interaction of amine and Carbonyl groups dealt with in this book. Their data proves that the protective action is an oxidation, though they failed to investigate the whole process.

THE QUINONE STRUCTURE AS AN ELECTRON ACCEPTOR IN NORMAL TISSUE METABOLISM

CC(C)=CC(C)(C)C1=C(C(=O)OC)C(=O)C(C)=C1C(=O)OC

The kinetics of Coenzyme Q have been worked out by Britton Chance, and the mechanism of its reduction in mitochondria has been investigated by Ziegler, while the specificity of Coenzyme Q homologues in electron transport restorations has been studied by Crane. Thus the physiological position of the quinone structure in metabolism is amply assured, and measured during the past year. This recent work confirms our Postulate, the chemistry of which we put into practical action some decades ago.