APPENDIX I

SUGAR OXIDATION

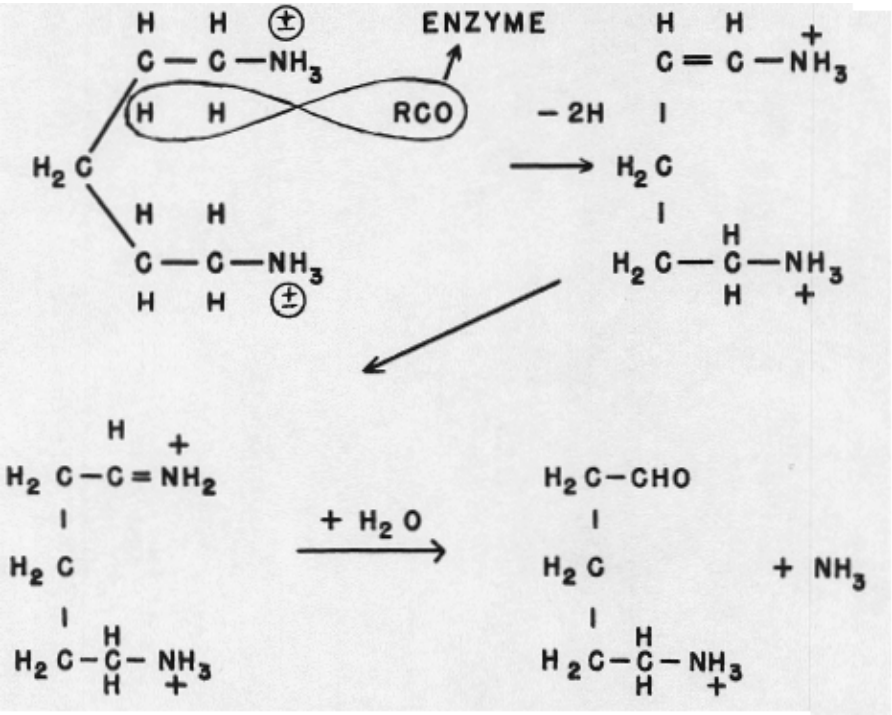
Although specific enzymes play a part in all metabolic reactions, it is also possible that non-enzymatic reactions play an equally important role in certain positions. There is no harm in visualizing a non-enzymatic factor in sugar oxidation. Fructose is much more easily oxidized than glucose so the enzymatic conversion of glucose to fructose is assumed. The non-enzymatic condensation of the Carbonyl group of fructose with the amine group of the cell's oxidation mechanism to form an azomethine double bond offers the advantage of holding the fuel and receiving the energy produced by the first oxidative steps. The diagrams picture the process. When cleavage takes place and the Amadori rearrangement is reversed, an acetyl free radical is formed which adds to the phosphoric acid residue of the cell and further oxidation steps pass their energy on to the cell via this union. This happens so long as molecular oxygen is at hand. When it is not, the end products are lactic acid under the reduction flux of the medium and the fermentation system takes over. The free radical and peroxide free radical intermediaries, in the presence of adequate oxygen, lead to such unstable oxidation-inviting fragments, that they can never be trapped.

It must also be recalled that though the carbon hydrogen bond strength equals 58.6 Kg-calories per molecule and the O-H bond- 110.2 Kg-calories, the set-up in sugar gives the hydroxyl hydrogen great freedom, so that when sugar is placed in heavy water, the deuterium replaces the hydrogen atoms at random and here the bond strength is so greatly reduced that dehydrogenation of the hydroxyl group virtually leaves an oxygen free radical. The very structure of fructose invites dehydrogenations of the carbon atoms and the hydroxyl groups, which gives specific and non-specific dehydrogenators the preference over any agencies involved in the Krebs's Cycle. If the progress would follow the scheme outlined above, no free degradation products would be at hand to be isolated. However, if the above process is crippled, the last steps would require known dehydrases and the latter products would be identifiable. The removal of hydroxyl hydrogen would take place very early, however, even before the carbon chain is broken and thus the process requires more facts for clarification. One therefore sees how futile it is to outline such mechanism as the Citric Acid Cycle or any other mechanism, in view of the facts stated above, and in view of the lack of further directive data.

APPENDIX II

DIAMINE OXIDASE ACTION

Zeller's idea of the action of diamine Oxidase, as published in "*The Enzymes*" edited by Sumner and Myrbaeck, 1951, p 554, may be condensed as follows, using putrescine as substrate.



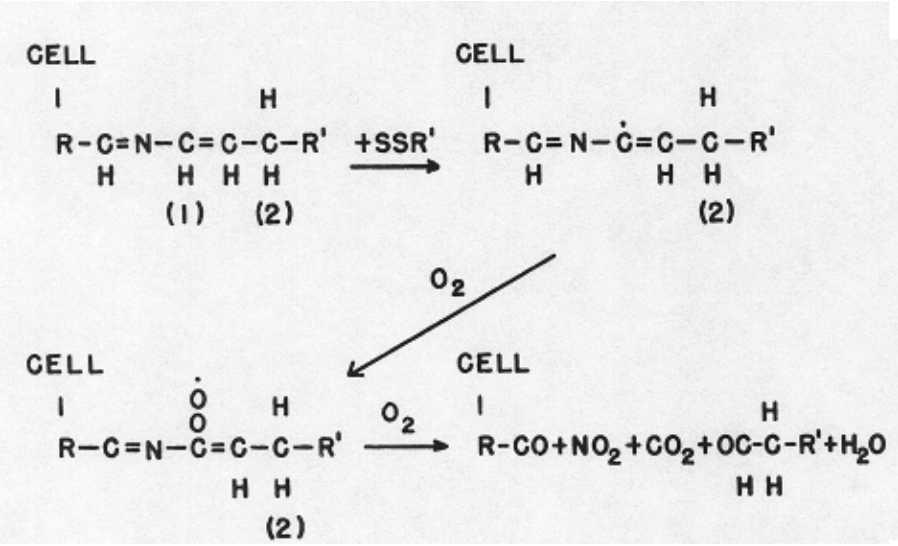
The products are hydrogen peroxide and ammonia, oxygen being the ultimate electron acceptor, and Flavin-Adenine dinucleotide, the electron carrier. The Carbonyl group of the Diamine Oxidase (DO) serves as a dehydrogenator removing the hydrogen atoms from the carbon atoms alpha and beta to the amine group that is to be removed. A double bond is thus produced. The other amine group is not attacked**.** When the hydrogen atoms so removed are taken up by the dinucleotide, the DO Carbonyl group is ready to start another cycle of detoxication. The steps are oxidation, a reversed Amadori shift, so to speak, and then hydrolysis removing the amine group as ammonia, as shown in the diagrams.

Oxidative Separation of the Integrated Pathogen

Our Postulate proposes oxidation all the way through. Dehydrogenation alpha to a double bond of the pathogen that is integrated with the FCG, by an azomethine condensation. This may take place close to or far from the point of integration. The free radical produced adds molecular

oxygen, becomes a peroxide free radical, and splits producing two terminal Carbonyl groups. The double bonds of the Carbonyl groups activate the alpha placed hydrogen atoms facilitating further dehydrogenations by the SSR Carbonyl group. Thus, whether the azomethine double bond yields an oxide of nitrogen in the pathogen and a Carbonyl group, as was originally present in the host cell as its FCG right at the start, or as the end reaction of a stepwise oxidative progression starting at a distance, the results are the same; restoration of the host cell FCG and destruction of the pathogen with the production of energy. The diagrams illustrate.

If oxidation starts at a distant position, more energy is produced and this may be the way viruses are separated with return of the energy to the host cell, which was taken up by the viral colony during its vegetation. The same holds for the separation as in (D) to follow.

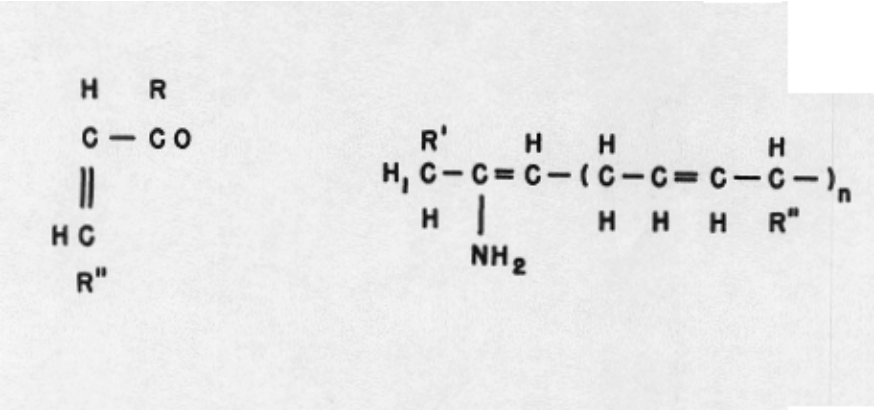


Integration of the Pathogen with the Host Cell Energy Producing Mechanism by a Free Radical Addition to the Double Bonds that Activate the F.C.G.

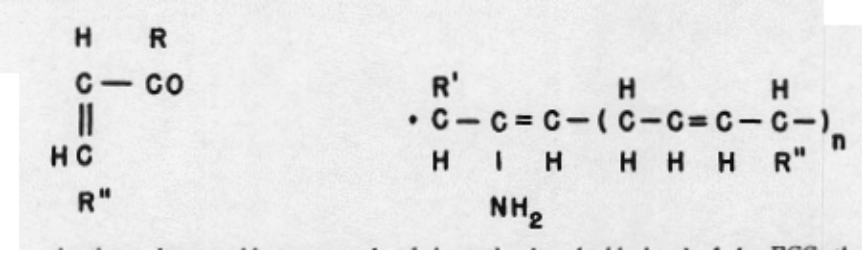
D

Host Cell Energy Producing System FCG and Double Bonds.

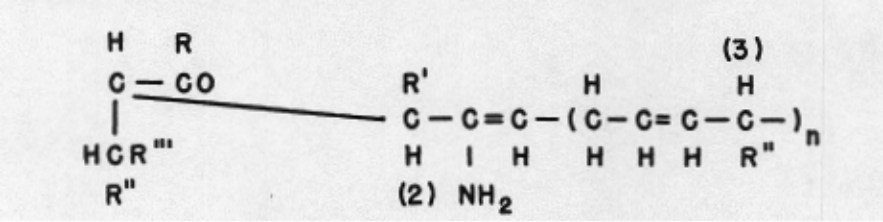
Pathogen (P)

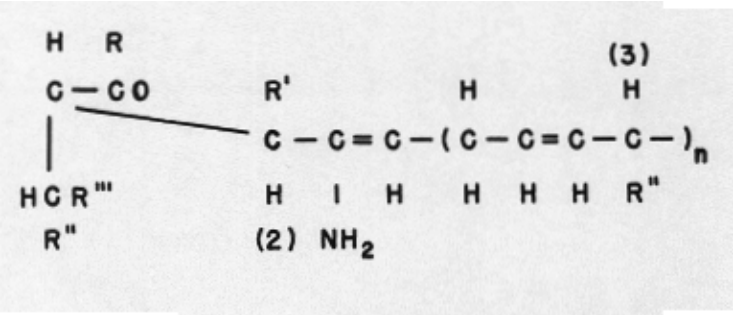


When the Pathogen (P) is dehydrogenated during anoxia a free radical is formed at position (H_1) which adds to one pole of the host cell's ethylene linkage that activates its FCG, thus, —

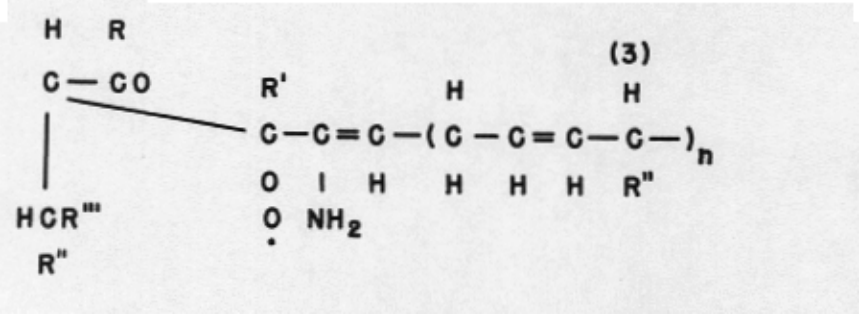


As the pathogen adds to one pole of the activating double bond of the FCG, the other pole of this double bond becomes a free radical which makes an addition to whatever group it has the most attraction for, is represented by R", thus, —

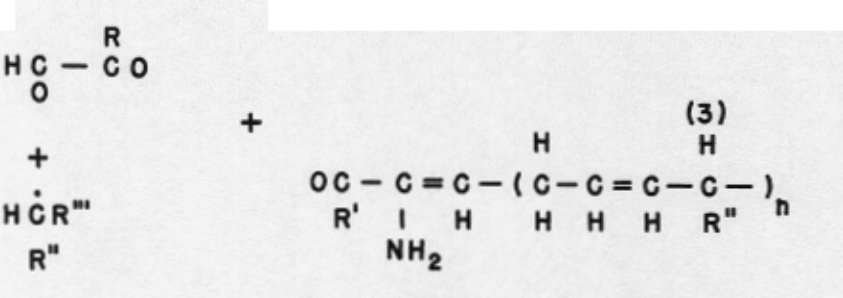




+SSR and OXYGEN to form a free radical and then a peroxide free radical at position (2)



Cleavage then takes place leaving the host cell FCG conjugated with a Carbonyl group replacing the former ethylene linkage that activated it.



The residue left by the cleavage at the activating position is a free radical that adds molecular O (2) to undergo further oxidation.

If the H (3) is the most exposed and activated hydrogen atom, it will invite removal by the SSR, and the free radical formed there, in the presence of molecular oxygen, becomes a peroxide free radical and splits the molecule with the production of two terminal Carbonyl groups, as in the former instance. Each Carbonyl group double bond serves as activator of the hydrogen atom in the alpha position to it, and the process is repeated step-by-step until the FCG is reached where a

Carbonyl group is formed and the electrons, it can contribute, go to the FCG to activate it as the ethylenic linkage had done previously. The FCG is activated to a higher O/R potential than formerly, as the Carbonyl group is a better electron donor than the ethylene linkage. Also, the Carbonyl group does not add free radicals so readily as does the ethylenic linkage and hence, the FCG system is protected from inactivating additions, therefore, a higher degree of immunity is gained than what existed before. This Hypothesis seems to answer the fact that our cured patients are more resistant to disease than they formerly were and in fact more resistant than others usually are. Since viruses are built up of similar units, as a co-polymerization process, the last monomer added is most exposed and oxidation would start at this position. The energy liberated would then pass on to the host cell and support its reconstruction, so that when the paralysis as in Rabies and Polio has disappeared, one knows that not only the virus is separated out of the way, but the host cell's functional mechanism has been restored. Since so long as the virus is integrated with the host cell, the function is blocked and the functional mechanism is progressively destroyed to support viral vegetation; the reversal of the process, with reconstruction of the host cell so function has returned, means that the energy for this reconstruction must have come from the oxidative destruction of the virus, as there is no other source for such energy while the FCG is blocked. The facts have been demonstrated. The explanation, of course, is a matter of choice as limited by the facts.

IMPORTANCE OF DIVALENT AND MONOVALENT CATION BALANCE

The parathyroidectomy experiments emphasized another matter of the greatest clinical importance. It is the relation of the balance of divalent and monovalent cations to cell irritability. Ordinarily, the normal cell presents a disposition of the cations, adsorbed and un-adsorbed, by the cell colloids so that a water in lipoid phase is maintained. The lipoids thus diffuse to the periphery of the cell forming a concentration of lipoid there, which serves as a limiting membrane and tends to shut out water-soluble materials, while water and its soluble materials tend to concentrate in the center. The effect is reversed by an excess of monovalent cations and accentuated by divalent cations. The monovalent cations thus tend to cause a lipoid in water phase with the lipoids concentrated at the center and the water-soluble materials and water at the periphery. This tends to increase the entrance of water-soluble substances into the cell. Sodium and potassium thus tend to increase the cell's exchanges and the entrance of water-soluble toxins. Calcium, magnesium and strontium tend to reverse this situation. Calcium is important with magnesium in lessening the cell irritability, while sodium and potassium increase it. Variations play their roles in functional activities of all cells and will be apparent also in the functions of heart muscle.

After parathyroidectomy, the increase in irritability of the nervous system followed the loss of calcium from the tissues. The entrance of guanidine toxins gave convulsions of greater severity, the more the calcium was lost. Calcium transfusions reduced the convulsions for a time, that is so long as the kidney function was maintained. Other solutions as of potassium and sodium and

even distilled water did the same for a time; so diluting the blood and washing the toxic element out via the kidneys showed that calcium metabolism was not the whole problem after parathyroidectomy. However calcium, magnesium and strontium had a greater depressing effect than the monovalent cation solutions, and the suppression was greater in the order of the divalent cations named. Indeed strontium put the cells to "sleep" as it were by its excessive effect. It was such facts as these that convinced the writer that the function of the parathyroids was not just a matter of calcium metabolism as Carlson and his school claimed. As soon as the hemorrhagic glomerular nephritis after parathyroidectomy prevented the washing of the guanidines out of the blood, no amount of calcium or any other solution could prevent or hinder the convulsions and deaths of the animals. Thus it was evident that a toxic element existed, and the writer set out to find it.

A complicating factor is the normal place of calcium in activating ATP base for the transfer of energy into the working mechanism of the cell. Here is a chance for energy transfer also to the surfaces of the tissue colloids that aids their dispersion and oxygen transport, in order to get rid of toxic materials and preserve a better colloidal structure in the cell throughout its contents**.** Normal cell excitability and response to stimulus is thus maintained by this other function of calcium.

When one sees exaggerated reflexes or persistent excitability of a tissue, one therefore thinks of the dispersions of the lipoids to or from the cell surface and also the disposition of the monovalent and divalent cations. In cases of high irritability, one would not give transfusions of solutions that would increase the monovalent salt content. Isotonic salt solutions made with sodium chloride would not be used unless the sodium is balanced by calcium and some sort of a Ringer's solution should be used. Even the sera of the blood banks could be taken from individuals with high sodium chloride blood content. These matters deserve consideration.

Our diet calls for calcium in plentiful amount, as crude calcium carbonate. We also give the potentized calcium as carbonate to cancer patients. This is to diminish the nutrition of the sodium rich cancer cells and to reduce the pain caused by the ever present incompletely combusted metabolites that enter the nerve endings in the affected areas. Calcium and good intestinal lavage and a well-chosen diet go a long way in abolishing the pain in cancer. Likewise the Carbonyl Reagents tend to burn the incompletely combusted metabolites out of the way so they cause no more pain. The cancer cells also under good calcium supply do not tend to swell up so much and cause so much pressure, for the reasons just mentioned. All of these factors enter the treatment of the neuroses and psychotic states.

An illustration of the parallel run of toxins with failure of the use of calcium is seen in the treatment of dairy cattle that were badly diseased by hemolytic Staph Aureus infections of the mammary glands. After the Carbonyl (SSR) Therapy was used, both the hemolysins disappeared and the calcium content of the blood and milk increased. The lactiferous cells were able to use

the calcium for cell building and for milk production, and the germ no longer produced the hemolysins. This work was done at the University of British Columbia and by the scientists of the Ministry of Agriculture. Thus the toxic inhibition of the use of calcium must be removed to obtain its full biological effect.

STERIC ADVANTAGE AND HINDRANCE

There is a problem bound up in the activation of atomic groups by electron shifts as determined by the character of a substituent for hydrogen at a carbon terminal of a double bond. This is the steric arrangement of the groups concerned. It is observed that the groups taking part must lie in the same plane. We took data on this matter in our earliest observations. Comparison of the action of fumaric acid, maleic acid and maleic anhydride were made on the course of glandular tuberculosis where the enlargements were easily measurable — the cervical and supraclavicular glands. It was found that fumaric acid had no action, maleic acid showed some, and maleic anhydride gave a satisfactory response. This response was not continuous longer than a few months and the dose had to be repeated. This is not what we were looking for. We desired a continuous curative action that kept up until the patient was fully cured. Nevertheless the observations showed the effect of steric influence. Reference to these early experiments was made in our Court Trial in 1943, where comparison was made with Benzoquinone.

In Benzoquinone one finds the Carbonyl groups activated by conjugation with two ethylenic linkages where no hindering substitutions of the hydrogens are had. They all lie in the same plane. High-grade continuous curative action was had in a wide field. In fumaric acid, which is the trans-isomer of maleic acid, only one Carbonyl at a time is coplanar with the ethylenic linkage, while in maleic acid, which is the cis-isomer, all are double bonds; the Carbonyl and ethylenic both lie in the same plane. Here, however, the hydroxyl in the carboxyl group acts as a substituent of a dampening nature against Carbonyl activity. In maleic anhydride, the two hydroxyls are removed, and this offers an advantage, but not one that would equal the advantage given by the presence of hydrogen. Indeed in such an ideal molecule, the energy content is too high to let it exist as such. So as such it was not practical.

The energy content of maleic acid is 7 large calories higher than that of fumaric acid and hence, it is more reactive. The ionizing power of maleic acid is increased by electron shift from the ethylenic linkage and also from the other unsaturated (Carbonyl) group, tending to liberate one hydrogen as a proton. Other properties show this increase in energy content due to all double bonds lying in the same plane so far as the hydroxyl groups allow. This example of steric influence on reactivity must be helpful in understanding the change in reactivity of atomic groups concerned in the mitotic act — that is, the shift of energy that forces cell division. For example observations on fumaric acid by Friedman et al. show that fumaric acid has no influence on mitotic rates while maleic acid and its two methyl derivatives, all showed strong antimitotic effects. Friedman, Marrian, and Simon-Reuss, (*Brit. J. Pharmacol*., 3 (1948a) pg.263, attempted

to learn if the antimitotic action was due to sulphydryl addition, (Biesele p. 34. *Mitotic Poisons and the Cancer Problem,* Elsevier, 1958). But it was found that the addition products were not active. The chlormaleic acid and chlormaleimide added sulfhydryl in the same way as if non-chlorinated, but were inactive as mitotic inhibitors. Activation of Carbonyl by electron drift from the other double bonds was not considered by these gentlemen, even though the quality of the influence of the chlorine substituent on the distribution of electrons to the Carbonyl groups both in the cis-acid and anhydride forms was to withdraw them and decrease the all around electron density. This shows that the antimitotic effect is due to Carbonyl action, as this writer has interpreted it to depend upon electronic activation throughout his whole Postulate. Due to the presence of a conjugated ethylenic linkage, sulfhydryl can be added where such an ethylenic linkage can contribute electrons to the Carbonyl group, but that the addition of sulfhydryl has nothing to do with the antimitotic act, is right in line with this Postulate. This is just another of the antimitotic agent puzzles this Postulate has solved long before antimitotic work was ever undertaken. The unheeded data in the hands of the investigators showed that the antimitotic effect was due to Carbonyl activity enhanced by electron contributions from the other conjugated systems of double bonds lying in the same plane. Thus the steric effect of a virus or carcinogenic chemical that becomes integrated with the host cell must be viewed, in the light of its effect, on energy distribution and reactivity. This Thesis calls for an oxidative separation of the pathogen from the host cell where a hydrolytic effort could never bring its release, is in line with this Postulate. Indeed, it is a practical application of the idea.

APPENDIX III

A variety of cases with fuller discussion than **Case I**, the exophthalmic goitre case, showed how the basic pathology was the block in energy production and utilization in the functional mechanism. The Carbonyl group of function was combined with the pathogen or its activating double bonds; it could neither dehydrogenate nor form an azomethine bridge for energy passage in frank infection, allergy or neoplasia. Many conditions that were observed to support our Thesis, were not touched upon. So to make the report more complete we will show how the two obvious metabolic disturbances, epilepsy and sugar diabetes, also conform to the same pattern.

DIABETES

In this disorder, which is a symptom of a number of diseases, there is the suppression of function of the Islets of Langerhans, even though microscopically the cells may appear normal in number and structure. They may be diseases that destroy the islets and thus cause failure of insulin production. But in the vast majority of cases the trouble is functional and correctable in line with our Postulate. Enough clinical proofs were given before the Federal Court and the Federal Trade Commission to establish this fact. Two recent cases are presented here to illustrate two facts, which explain two different forms of response. In one, the high blood sugar was reduced to below normal indicating that new islets of Langerhans were formed to compensate for the loss of insulin. But even these were blocked by the inhibitor toxin, and when they were all liberated, there was an excess of insulin poured into the blood. The other extreme is the reduction of the high blood sugar slowly and gradually, showing that the islets were deficient in number and had to be repaired or increased to accommodate the demand for their product. In both, the general health was restored and thus the basic or constitutional cause was removed. In both the high protein diet was changed for an unrestricted carbohydrate diet of fruits, vegetables, cereals, bread, fats, honey and molasses. No proteins of animal origin were allowed and coffee, tea, alcohol and tobacco were forbidden. No insulin or other anti-diabetic drugs are given; the patient is left under the influence of a returning islet function as its cells are liberated from the paralyzing toxin. The blood sugar then comes toward normal as fast as the islet cells are relieved. Sometimes the blood sugar goes below normal as the compensatory hyperplasia of islet cells are also liberated from the toxin and go to work. Where the toxin has been integrated with the islet cells longer than the life cycle period of any of these cells, they naturally die off and are not replaced and so a deficit of islet tissue is observed in the blood sugar level that is above normal after all of the toxin is removed. To replace these islet cells takes time, as we will show by cases examined at this particular period. One must remember that the islet tissue produces insulin and hence no great deficit of this material is ever present in normal islet cells to

stimulate a compensatory hyperplasia as occurs in muscle cells that are exhausted by work and call for more tissue reconstruction.

Therefore compensatory hyperplasia in islet tissue is comparatively slow and the time to reach the usual normal level varies with the length of time the diabetogenic poison had been active. The recovery of the islets from the integrated toxin that had simply inhibited their function is rather rapid, and is accompanied by systemic reaction in which the tissues are generally cleared of toxic effects. The overweight and weakness is soon overcome and other disturbances leave, — allergies, etc. The recovery of the destroyed and deficient islets to a normal quota is slow as was just explained. However, the whole picture of diabetes changes with this exposition. These cases again show that the work of the SSR is to start an oxidation progression in the toxin that is integrated with the tissue cell and blocking its function. The difference between such a toxin integration and the viral integration will be seen by comparing the recoveries of diabetics with paralytic nerve infections in which paralytic dog distemper, Chapter 17, serves as a good example.

The cases submitted here are only to show that the SSR is not a substitute for insulin in the oxidation of sugar but is given to remove the pathogen that blocks the production of insulin.

I. Case of Sr. L. S., 53 years old
Dr. Jayme Treiger

He had a rich venereal past, malaria at 21 years of age, and was operated for varices in 1941. He complained of vertigo, edema of the lower extremities, grade 2, small varicosities. Aorta palpable, Fundus oculi, — veins with second grade manifestations (Wagner), Blood pressure 24/13, Pulse 96, Glycemia 112 mgm %, was placed on hyposodic diet, did not tolerate CIK, received Clortiazamide because of the edema. This reduced his blood pressure to 20/10, 18.5/11, and 2 1/11 with vertigo.

On January 19, 1960, there was dyspnoea and B.P. 20/11. On February 14, B.P. 22/12, pulse 84. Rauwolfia, Clortiazamide, Naturetin K, Mecanil, were given, April 10. On May 16, vertigo, tachycardia, and dyspnoea, after lying down, B.P. 25/13, pulse 90/m received more energetic hypertensive drugs and Alcachofre tincture, B.P. 22/11, pulse 84. On August 18, epistaxis, B.P. 26/12, dyspnoea, — Reserpine, Alcachofra, weight 85 kgms. Constrictive feeling in the neck, Blood sugar 320 mgms. %, Urea normal.

Treatment was given on September 24th, 1960, only a few drops intramuscularly of the serial system of Carbonyl groups, one-tenth of a microgram. He had a reaction on the next day; the edema and constrictive feeling in the neck disappeared quickly and three weeks after the Treatment he felt very well. Weight 82 kilos, blood pressure 17/10. Blood

sugar 75 mgms. %. He has remained in good health. The clearing up of the basic cause is evident in the normalization of the other evidences of disease, to which the diabetes was part and parcel. “All anti-diabetic drugs including insulin were stopped before the SSR was given.”

II. Case of Sra. M.P. 51 years of age, was given the same dose of the SSR within the same hour as the preceding case, so as to facilitate comparisons chronologically in their recovery processes — the depth of the pathogenesis with the recovery rate.

She had been diabetic since 1955, but was first seen by Dr. Treiger on August 24, 1959. The first complaints were articular pains, thirst and excess weight, 95.6 kgms., height 1.58 meters, blood pressure 17.5/9, edema grade 2 in both feet. Glycemia Aug. 1959, 240 mgms. %. Folin-Wu. Urine S.G.1.036, glucose 4x. As she did not accept very well the new anti-glycemiant, Diabenase, she started to receive insulin and Protamin during the whole of 1959 and 1960. But the blood sugar stayed always at higher than normal levels even though the insulin was taken always with 40 U PZI, running generally around 220 and 240. In June 1960, the blood sugar was 340 mgms. %, while receiving 60 U PZI, and by September 15, it rose to 398 mgms. % while receiving 60 U PZI. Before being given the SSR, she was taken off of insulin completely and all medication was stopped.

Treatment—On September 24th, 1960, her weight was 94.5 kilos. She was given a few drops of the same solution as the other case. In five days the sugar dropped to 210 mgms. %. In two months her weight dropped to 89.5 kilos even though she was taken off of proteins and given a liberal vegetable, fruit, cereal diet. The edema of the legs was leaving within a week of the Treatment, and she had a splendid reaction of a few days of fever and aching in her legs and bones generally. Her whole health aspect changed for the better. The blood sugar on November 30, 1960 was 160 mgms. %, and she is taking no insulin or other drugs. Her appetite is good and she feels the best she has ever felt. The blood sugar is not expected to reach normal until islet tissue is reconstructed to normal. It is expected that this will require from three to six months.

One would ordinarily think that the rich carbohydrate diet after the Treatment would precipitate a sugar crisis. However, the relief of the islet cells was fast enough to prevent that as these cases show. Further our diet regime that cuts out animal proteins also reduces the production of the pathogen in the bowel. It will be seen that the whole aspect of diabetes comes up in a new light, as these and so many other cases show. Reduction of the blood sugar as observed in these cases of diabetes while on a non-protein, high carbohydrate, liberal diet, without taking insulin has but one interpretation and that is the islets have again resumed their function. In other words, the inhibitant has been removed. How rapidly injured islets can be restored depends upon the situation at hand, and the management of the case is directed accordingly, including the withdrawal of insulin.

Epilepsy is definitely a metabolic disorder that may be subject to the same corrective Therapy as the following case shows.

It must be emphasized sufficiently that while severe diabetes can recover after the SSR is given, this recovery is part and parcel to the removal of the pathogen and its effects generally. Moreover, while in an early mild case the islet cells can be liberated from their integrated toxin so as to fully restore islet function, in the long standing heavily intoxicated cases, the islet cells that remain as functional structures may be reduced in number and the blood sugar will not return to normal until more islet cells are reconstructed. The amount of reconstruction required in such cases will be proportionate to the length and severity of the toxic period of injury. A few more cases should be scanned to observe these points in their varying aspects.

The toxic injury may be dominantly cardiovascular.

III. J.M., age 61 years in June 1960. She complained of pains in the limbs, dyspnoea after effort, constant cough, precordial pains intermittently, edema of the right ankle, and cholecystectomatized because of lithiasis. Physical examination revealed a blood pressure of 170/90; pulse 90; systolic murmur at A2; fundus oculi, hypertensive angiosclerotic retinopathy, grade 2; KWV, urine with traces of sugar. She was first given homeopathic remedies with improvement in the blood pressure. On 8/16/60 her B.P. was 140/80, but there was no improvement in the pains in the chest or limbs. Salicylates were given. On 9/14/60, glycemia was 190 mgms %. She was given a dose of the SSR and no other medication was given. She was given a rich carbohydrate diet and all animal foods denied. On 11/14/60, glycemia was 145 mgms %. On 12/2/60, glycemia 100 mgms % and she was entirely symptom free. Her blood pressure was normal. All the conditions cleared up at the same time on the unrestricted fruit, vegetable and cereal diet.

The toxic injury may be dominantly those of an old focal infection with allergic symptoms and lack of tolerance to anti-diabetic drugs and a failure of large doses of insulin to reduce the blood sugar notably with loss of resistance to infection.

In these cases the disappearance of all toxic effects comes quickly and the blood sugar drops to a level (without medication and on an unrestricted carbohydrate diet without animal foods) to a point that indicates how much of the islet tissue still remains undestroyed by the pathogen. Then the reconstruction of islet tissue goes on slowly as was shown above, and the rate will depend on the length of time the pathogen was active. In this way one illustrates that the SSR is a liberator of the functional mechanism, restoring it to normal action on a broad or maybe on all functional planes and not simply an agent to oxidize sugar. It demonstrates itself to be an oxidizer of all integrated toxins, we have investigated so far, with the widest possible symptomatology. The following two

cases are selected to illustrate a variety of tissue injuries that a diabetes-producing toxin can cause and the degree to which the islet tissue may be paralyzed and also destroyed.

IV. Mrs. P. S., age 50, in August 1956, started with diabetic symptoms after a hysterectomy. Her blood sugar was found to be 430 mgms %. With insulin it came down to 173 mgms %. After August 1956, she did fairly well, using Insulin 20 U, and Nadisan, 3 tablets daily. After May 1958, she started taking Diabenase in varying amounts. On September 2, 1959, her blood sugar was 195 mgms %. In November 1959, it was 240 mgms %. In January 1960, the blood sugar was 300 mgms % with Rastonin, 2 daily. It stayed about the same until August when Rastonin had become intolerable due to toxic effects and was stopped on September 29, 1960. Her blood sugar was 325 mgms %. On October 4, 1960, she was given the SSR. She had a strong reaction on the third day that lasted a week with general achiness, fever, chills, etc. The reaction focalized as a heavy inflammation about an old tooth root, which opened and cleaned out with a good hemorrhage and then cleared up. Her weight dropped from 75 to 66 kilos by the middle of December, 1960, her blood sugar was 260 mgms % and she felt very well without any anti-diabetic medication, and on a diet of unlimited carbohydrates and fats and without animal proteins. This improvement is expected to follow the pattern of other cases with decrease in the blood sugar as fast as islet tissue is restored. At this stage the case differentiates between inactivation and destruction of islet cells.

V. Mrs. G. S., age 38, in October, 1958, showed a different type of toxicity in which she could not use insulin without toxic injury and she was unable to heal any wound or furuncle. This all cleared up concomitant with the correction of the injury to the pancreas. In this case as in the former case a strong systemic reaction ushered in the improvement. The insulin poisoning showed as an ecchymosis and general itchiness and rheumatic symptoms.

She was diagnosed as diabetic in 1957 since the wound from a Cholecystectomy done 2 years earlier would not heal and the blood sugar was 248 mgms %. She was given classical treatment and diet like the rest, first 40 U PZI, then 60 U, then 70 U, and then 80 U on November 6, 1959, as the blood sugar increased to 440 mgms %. On 80 U daily, it dropped to 300 mgms %, but ecchymosis and itching resulted. Two months later she presented an eruption similar to the leutic roseola, but the blood tests were all negative for syphilis. The insulin was reduced to 50 U, PZI, and was fortified with Rastonin, 2 tablets daily. The blood sugar was 380 mgms % on August 18, 1960, and she was put on Diabinese, 2 tablets and then 1 tablet per day when the blood sugar rose to 394 mgms %, September 17, 1960. She was given the SSR on October 19, 1960, with one Diabinese tablet and the blood sugar jumped to 360 mgms %, as part of a strong reaction. The Diabinese was stopped and a half dose of the SSR was given on November 18, 1960. On December 13, 1960, she was feeling very well, her furuncle healed without help, and the

blood sugar was 265 mgms % on an unrestricted carbohydrate diet without any medication or animal proteins. The break in the Survival chemistry showed in more than one direction in this case, and showed not only a paralysis of the islet cells, but their actual destruction which is not yet fully repaired. The job of repairing the islets is not a function of the SSR, but is made possible by the removal of the toxin that was integrated with the islet cells and destroyed them. With this toxin out of the way reconstruction can take place as in other cases.

That the pattern of integration of the inhibiting toxin with the FCG is the same in islet cell block, as it is in the hindrance of other cell functions, has been thoroughly demonstrated already. Moreover, no matter how symptomatology of nerve cell hindrance may vary, the architecture of the block is the same. The severity of the symptoms does not determine the length of time required for recovery, but the length of time the tissue has been occupied by the pathogen does determine the time required for restoration to normal.

Another case must be sketched in brief to illustrate this point and to show that sleep is an active function involving energy generation and use. This case could not use the energy because of FCG block. There was also an islet block of moderate degree, the blood sugar being only 161 mgms **%.**

VI. Mrs. M. S., age 42 years, came to Dr. Treiger in December 1960, in a highly nervous state. She was without thyroid symptoms, complained of utter inability to sleep for over six months and had been placed on the whole list of depressant drugs by different specialists. These drugs did not help her in the least, but even as their dosage was increased to the limit of her tolerance, she became steadily worse. She developed into a melancholia with a compulsion that she wished to die becoming ever constant. She was in terror and could stand it no longer. Dr. Treiger removed all drugs and the condition remained the same, fat and all, presenting a perfect homeopathic picture of treatment with Alum. He gave her Alum in carefully selected potencies, but it had no effect. He then gave her 2 millimicrograms of the SSR intramuscularly. At the time of Treatment, the blood sugar was 161 mgms %. The change for the better was rapid so that her whole physical status improved, the high excitement state left and she calmed down so that in less than a month she was sleeping soundly three nights a week, and the other nights were improving steadily. The desire to die faded away and the melancholia changed to a habitual cheerfulness. The blood sugar dropped to 100 mgms % within the month while on a meatless, high carbohydrate, honey, molasses diet. Here the clinical facts teach something that laboratory research has not fully settled about the physiology of sleep and its functional mechanism, namely that energy production and utilization are essential features and that the Carbonyl group is concerned in mediating both features of the function just as in any other tissue function, islet cells, thyroid cells and all the rest.

VII. It is pretty well demonstrated now that the SSR does not do the work of the insulin, but removes the disease. This is seen in Varicella, a viral disease with a natural collateral control. Two brothers are concerned. M. M. P., age 11 years, had fever and Varicella. The SSR was given to him on 12/3/60. The next day the fever increased to 39°C. On 12/5/60, the second day after Treatment, the pustules stopped coming and all of the lesions started to improve. He was feeling well and in a few days was all healed. His brother, age 6 years, refused the injection when he developed Varicella on Dec. 15, 1960. Four days later, 12/19/60, the Varicella showed the classical development with new pustules coming in waves. He ultimately got well, but the involution did not start within 48 hours as in the SSR treated case, but was still developing in the classical fashion as is usual for days afterwards.

To further define the position of the Functional Carbonyl Group and its activating double bonds of the Koch Postulate a review of a case of antibiotic resistant Gonococcus infection will help.

VIII. Mr. J. V., age 25 years, presented an annual venereal infection. Finally the antibiotics were not effective and he did not do well on homeopathic drugs either. On August 28, 1959, after an additional exposure, he presented an acute Blennorrhagia confirmed by bacteriological examinations. He was energetically treated with Penicillin, Terramycin and Tetracycline without the least effect. On October 23, 1959, he was given a dose of Benzoquinone, 6X dilution, when the bacteriological examination showed the infection was the same, a rich flow of intracellular and extracellular Gonococci laden pus. Examination one week later showed no improvement so he was given a dose of the SSR, 9X dilution on October 31, 1959. One week later, November 7th, he was feeling much better, including the prostate pain that had disturbed the passage of feces through the rectum. On the 9th day he had a reaction and on the 16th the urinary sediment still showed some intracellular diplococci. He was given a dose of the SSR, lOX, one-tenth as much as the former dose, on the 16th of December 1959. Three days later no more intracellular diplococci were found in the urinary sediment, but some extracellular germs were still present. Three weeks later he had a general systemic reaction of grippiness. He felt well thereafter, but there were still extracellular diplococci until the middle of March 1960, when the dose was again repeated. Ten days later a systemic reaction occurred and no more secretion was to be found. His health improved in every way. On May 30, 1960, the urinary sediment still showed some extracellular pleomorphic germs.

On June 7, 1960, he showed a gain of 10 kilos in weight, the best of health, no longer any signs or symptoms of the old or lasting infections, the prostate was normal and the urinary sediment entirely negative. He has so remained.

Here we see the hangover of the earlier Gonococci infection as extracellular diplococci that required three injections of the SSR and six months to be cleared away after the acute infection was gone. This shows also that the SSR is not an antibiotic affair at all, but works on an entirely different principle. This point must be emphasized here. This final review shows that viral infections, which do not respond to antibiotics, are correctable immediately by the high potency dehydrogenations secured by the SSR and the free radical and peroxide free radical carriers of the oxidations that continue the separation of the pathogen from the host cell's functional mechanism.

In the proceeding diabetes cases, it was shown that the longer the pathogen is integrated with the islet cells — not only is the inhibition of function proportional, but the amount of destruction of islet cells is also proportional. This is not a similar destruction to that which accompanies viral host cell integration, but one that blocks the formation of new islet cells after the integration is established a long time, longer than the life of the islet cell would be. Then no reproduction would take place in such cells and the amount of islet tissue would decrease. In viral integration there is the active destruction of the host cell as its energy is drawn off by the parasite and its structural material is also removed to support the viral vegetation. However, we have demonstrated in paralytic viral infections, that the toxin is destroyed when the virus undergoes a step-by-step removal by the oxidation process leaving a fully restored tissue cell once the virus is fully burned away. Reconstruction in diabetes recovery must be carried forward by the liberated cells and, as there is no utilization or depletion of insulin in the islet cells themselves, the shock for a compensatory hypertrophy is very weak as compared with that taking place in muscle when put under increased activity. Here the muscle fibrillae that do the hypertrophying are directly stimulated by work, while the stimulus to the islet cells is mostly a lowered partial pressure of the insulin at the cell's periphery and possibly some hormonal influence, if such exists. Therefore the restoration of islet cells is a slow process that succeeds only with time.

PLATE 1.

PLATE 2.

PLATE 3.

PLATES 1, 2, and 3 show the state of paralysis before treatment with SSR on Oct. 6, 1960.

PLATES 4 and 5 show animal after recovery.

PLATE 4.

PLATE 5.

The contrast can be seen in the case of paralytic Distemper in a dog. In such a case there is no chance to error by way of hysteria. In this case within two weeks a full restoration of nerve function was re-established, whereas in diabetes, the islet reconstruction goes on

for months before it is completed. Only the toxic inhibition of function is removed in weeks or rather a few months.

IX. Singo, a 10-year-old pointer dog, one month before receiving the SSR Treatment, became sad and trembling. He ran away and returned with bowel and urinary bladder paralysis. He was lavaged and received Nujol that helped the bowel movements. He also received Penicillin, Alcachofra, Terramycin, etc., which was prescribed by the veterinarian who made the diagnosis of distemper of the nervous system since there were chills, fever, then a lowering of the temperature with paralysis of the muscles of the left costal region, causing a curve of the spine and difficult breathing. These muscles also showed atrophy. There was also a paralysis of the left posterior quarter so that the leg would give no support and he could not use the paw, raise or lower it. The photographs show his condition before and after Treatment. Photographs 1-3 show this paralysis of the left posterior quarter when the dog is supported by his upper half and the right foot posterior is allowed to take some pressure for his support. The left posterior quarter was useless. The muscles of this limb were also atrophied. As none of the medication helped, he was given two ampoules of the Cinamose serum. It did not help him either. The integration of the virus with the nerve cells was thus established in a symbiotic way.

The SSR was given October 14, 1960, to save the dog, as the veterinarian decided he must be sacrificed. The recovery took two weeks to be completed. The atrophied muscles soon became redeveloped and full normalcy was established.

EPILEPSY

I. Under the heading of "Allergies Of The Nervous System", we gave a case of Psychomotor Epilepsy. This patient had taken thousands of dollars worth of suppressing drugs without suppression and steadily went on toward fatality. At the time that the experts agreed he could not live more than a few weeks, he was given two micrograms of Parabenzoquinone in two milliliters of water intramuscularly and was sent home as cured in five days. The experts who decided he was cured are the same men who gave the fatal prognosis after months of unsuccessful drugging. This was in 1941, at the famous government mental disease hospital of Rio de Janeiro. The Reagent that freed the tissues of their energy-producing and energy-utilizing impediment was a Carbonyl group activated by conjugation with the double bonds of an ethylene linkage. In the case to be given here, the Reagent carried a series of Carbonyl groups that contributed electrons to the Carbonyl group that did the dehydrogenating and thus started the oxidative removal of the pathogen. The following case also shows the constitutional nature of the disease.

II. E. A., 17 years old, first seen on August 13, 1958, had Diphtheria in childhood. Started periods of petit mal with loss of consciousness in crises of from 5 to 10 minutes,

getting more frequent, once or twice monthly, without convulsions. He suffered severe headaches since childhood. Blood pressure 14/5 cms. of mercury, Systolic murmur, grade 3 at apex, 2 cms., outside of the left mid-clavicular line 5th interspace also a diastolic murmur grade 1, at the same place. Pulse 86 per minute. There was a diagnosis also of persistence of the Ductus Arteriosus.

The Electro-encephalogram on August 22, 1958, showed:

"The basic sequence is several times disturbed by the interference of abrupt and irregular slow waves with 6-7, 5 c/s. of high voltage, diffuse and bilateral, predominating in the frontal-temporal areas. The hyperphase did not reveal any new fact."

"Conclusion: Abnormal electro-encephalogram, paroxistic cerebral Dysrythmia, diffuse and bilateral, predominating in the frontal-temporal areas."

"Signed, Dr. A. A. G."

Thereafter the boy was kept from work because of the losses of consciousness that occurred while under the regular treatment receiving constant doses of Trilafen and Mezantoine. The blood pressure came to normal levels, but recurred at times with the crises of loss of consciousness. Since December 1959, the epileptic attacks became more frequent in spite of regular doses of Mezantoine, Equanil and Promazionon.

In June 1960, this treatment was interrupted so that the SSR could be given.

Results—After three weeks there were no more attacks, sleeps well, eats well, is no longer tired and without headaches. The Electro-encephalogram was repeated by the same expert. The report was dated, September 30, 1960:

"Electro-encephalogram presents no evidence of abnormality."

The patient is ready to go back to work but this will be deferred until the EEG is repeated after a couple more months. However, it is established that normal function has returned, and the energy production and use is not interfered with any more. In these cases two different means of activating the dehydrogenator Carbonyl group were used. Hence the correction of the pathology depends upon adequate dehydrogenation in the presence of molecular oxygen. The correction is constitutional evidently here too.

LATE REPORT ON MRS. M. H.,
Case No. 49

To demonstrate the permanency of the cure of Tuberculosis by restoring the cell functional mechanism, we give Dr. Paul V. O'Rourke's description of the status of both lungs which was made on August 1, 1960 from the latest X-Rays. This leading Roentgenologist states:

"Mrs. M. H.---- was seen by me on July 1, 1960. Her chest X-Ray at that time showed the bony thorax to be intact, with the heart in normal position and contour. The left hemidiaphragm is normal in position and contour. The right hemidiaphragm is elevated because of a Phrenic nerve paralysis done some 30 years ago. The left lung showed some increased markings, most of which are vascular, and a small amount of calcific deposits. The right lung is greatly reduced in volume because of the extreme elevation of the right hemidiaphragm to the level of the third anterior rib. At the extreme apex, there are numerous small-calcified areas, which are the result, undoubtedly, of her previous tuberculosis of some thirty years ago. No active lesion of tuberculosis or rarefactions resembling cavities can be seen."

Mrs. M. H. was treated in the terminal stage of this disease and at a time when her state of exhaustion was so desperate that even taking a radiograph would not be risked.

This recovery was obtained while at work after the active attack was subdued by the SSR and a few weeks of bed rest until the fever abated. It speaks for the curing of the tuberculosis germ and the ability of her metabolism to keep tuberculosis germs cured, and harmless, even perhaps useful.

EXERCISE AND REST

Enough exercise to fatigue the muscles a little each day is wise, but the heart must not be pushed too hard during such diversion. Walking on level ground serves well and there are plenty of games that stimulate the spirits as well as the muscles that are helpful. Common sense is the guide. As for quality sleeping, a neat bed with the patient laid out like a mummy is an atrocity. There must be freedom so that relaxation is possible. Stupid bed provisions call for narcotics, and narcotics defeat recovery. Such care must be avoided with our patients.

APPENDIX IV

As the work progressed it was deemed advisable to obtain data on the curative action of molar peroxides and their unsaturated precursors. For an investigation of this importance the best talent available was necessary. The American research institutions were examined carefully and found wanting. Europe was likewise scanned until Professor Joseph Maisin was selected as the best prepared for a task of this type. Cancer was induced in many hundreds of mice and rats by the usual carcinogens and tumor transplants and treated with the peroxides of formaldehyde and formic acid and with unsaturated substances prepared at the time of their use. These latter substances were pre-peroxides by virtue of their high double bond content. The tables appended show the results of their use. These observations were made at Louvain in between 1934-35.

The protective action was found on further investigation to be due to the free radicals formed by the dehydrogenating action of the Carbonyl groups present in the dehydrated unsaturated bodies and the free radicals formed in the peroxides which gave rise to peroxide free radicals. A comparison with the highly efficient curative action of the Reagent used in the Bandeen Experiments, (pages 30-35) bears this conclusion out since in the latter experiments the very highest Carbonyl ability to dehydrogenate was provided whereas in the Louvain Experiments no attempt was made to boost any activity at all. Nevertheless the latter experiments show that it is impossible to prepare molar peroxides sufficiently clear of free radicals to serve for pure biological tests. The speed with which free radicals are formed by light and by simple rubbing of the dry crystals also shows this trend. This is one of the basic provisions of nature for the continuance of life on our planet.

Protective Action of Diformaldehyde Peroxide

The mice received applications of Benzene solution; 1/200 Benzopyrene three times each week. The peroxide was injected, 1 cc. solution 1/20,000, on the 35th and 65th days.

Groups	Mice At Start	Mice Surviving To First Cancer	To 147th day	Day Of Observation	Relative Percent Tumors	Relative Percent Cancers	Absolute Percent Tumors	Absolute Percent Cancers
				CONTROLS				
1	40	28	21	147th day	53	48	55	48
				INJECTED				
2	50	40	27	147th day	4	—	5	2.5

Peroxide of Diformaldehyde

1/20th of a mgm. as one dose was given at the end of the first month of benzopyrene application.

Groups	Mice Surviving To First Cancer	To	Percentage Absolute Of Cancer
Controls	28	120th day	18
		210th day	57
Injected	40	120th day	5
		210th day	7.5

1/10 mgm. of the peroxide of diformaldehyde was given as one dose intraperitoneally before the first benzopyrene application.

Controls	78	120th day	30
Injected	75	120th day	10

Mice receiving three drops a week for 6 to 7 weeks of a benzene solution of benzopyrene, of a strength of one to two hundred. For treatment each animal received one dose of ½ cc. of a 1/5000 solution of the peroxide of formic acid.

Groups	Mice Surviving To First Cancer	To	Percentage Absolute Of Cancer
Controls	68	120th day	45
		210th day	69
Injected	52	120th day	23
		210th day	31

For treatment each mouse received 1/10 mgm. as one dose of diformaldehyde peroxide every 14 days after the first application of benzopyrene.

Controls	78	120th day	30
Injected	30	120th day	9

Unsaturated Substances

All mice received twenty applications of a benzene solution of benzopyrene 1/200 three times a week for twenty doses, and then one dose of the unsaturated substances.

EXPERIMENT A

	Controls			*Injected*		
	Number Of Mice Alive	Percent Of Tumors	Percent Of Cancer	Number Of Mice Alive	Percent Of Tumors	Percent Of Cancer
Day 60	75	36	1	62	10	0
Day 80	72	50	8	61	30	1.5
Day 100	71	65	16	60	38	4
Day 120	66	67	22	56	38	10

EXPERIMENT B

Day 60	71	28	0	75	22	0
Day 70	70	40	5	69	27	1

One finds that the phenomena that control polymerization are active here in the molar peroxides and the unsaturated substances in bringing about free radical activities.

SPECIFIC EFFECTS OF THE CARCINOGEN, VIRAL AND CHEMICAL

To account for the Pre-growth 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 reticulo-endothelial system weakens and atrophies 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 legated 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 a block in energy production and transfer. Recovery from the states caused by the carcinogenic agent, be it viral or chemical, is therefore a satisfactory support to our contention, since this same agency accomplishes the corrections in all such states as atrophy, pre-growth toxic state, cachexia, and tumefactions.

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 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.