

# Methods of Reversing the Stimuli- Evoked Pancreatic Insufficiencies of Chronic Degenerative Diseases

William H. Philpott, M.D. 1

## General Adaptation Syndrome

We are indebted to Hans Selye (1947, 1956, 1974) for the general adaptation syndrome. Hans Selye observed correctly that chronic stress leads to chronic disease.

1. Stage one is acute nonadapted reaction.
2. Stage two is adaptation reaction.
3. Stage three is chronic nonadapted reaction.

Hans Selye has spent much time in evoking inflammation and relieving and/or blocking inflammatory reactions. He correctly perceived the end point of stress breakdown as being adrenocortical. He specialized in using adrenocortical hormones in relieving and blocking inflammation. He gave recognition only in a small way to the role pancreatic enzymes play in evoking as well as relieving inflammation. This recognition was given in a conference held in his honor when Rocha Silva contributed a chapter to a book in 1968 (Silva and Kasmin, 1968). His 1976 updating of this

Ecology House Clinic and Laboratory, 820 N.E.  
63rd Street, Oklahoma City, Oklahoma 73105.  
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book, **The Stress, of Life**, focuses on the adrenocortical failure end point of the stress syndrome, and does not even mention the proteolytic enzyme failure as a point where the stress syndrome begins to fail.

## General Adaptation Syndrome Ecologic Stages

We are indebted to Theron Randolph, M.D., allergist (1956, 1976a, 1976b) for correlating Hans Selye's general adaptation syndrome with ecologic observations.

1. Stage one (acute nonadapted) is equated to acute allergic and allergic-like inflammatory reactions.
2. Stage two (adaptation) is equated to the stage of adaptive addiction. This was originally called masked food allergy. Later the stages of addictive withdrawal and relief on exposure were recognized which gave this the qualities of an addictive adaptation.
3. Stage three (chronic nonadapted) equated to the chronic disease.

Tissues are injured by the chronic inflammatory reactions. The diseases are named in accordance with the tissues injured, type of injury, organs disorder, autoimmune reac-

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tions evoked, secondary invading microbes, metabolic disorders evoked, nutritional deficiencies created by the stress overload, etc.

My observations and correlations of available information have been on this order.

1. A four- to six-day period of avoidance of the symptom-incriminated substances reverts the adaptive addiction (stage two reaction) to an acute nonadapted stage one reaction. This is the essence of provocative testing of foods and chemicals.
2. Acute maladaptive acidosis emerges in response to acute nonadapted reaction stage one, as well as withdrawal phase addictive adaptation stage two, and chronic nonadapted stage three.
3. Disordered carbohydrate metabolism begins to emerge during the late state adaptive addiction stage two reaction and strongly emerges in the chronic nonadapted stage three reaction.
4. Adaptive addiction stage two reaction leads invariably to chronic nonadapted stage three reaction which is named as a disease. The acute symptoms of provocative testing are observed to be the building blocks for chronic degenerative diseases either mental or physical. Thus acute arthralgia as a test reaction when chronic is named arthritis, acute test reactions of the colon when chronic and infected are named ulcerative colitis or diverticulosis, a stuffy nose and/or sinus test reaction when chronic and usually infected is named chronic rhinitis and/or chronic sinusitis, acute test reactions interfering with or distorting mental functions or emotions are when chronic named neuroses or psychoses depending on the type of symptoms. Acute test fluctuations of hypoglycemia and/or hyperglycemia, and the associated metabolic acidosis, can descriptively be identified as chemical diabetes mellitus, and chronic hyperglycemia and chronic metabolic acidosis emerging during the chronic nonadapted stage three reaction can descriptively be named as clinical diabetes mellitus.
5. Progression from stage one to stage two to stage three is tissue specific as well as substance specific. Thus a person may be stage two of

adaptive addiction to wheat, corn, or potatoes, and be stage one of acute nonadapted to strawberries, shrimp, etc. A person may be chemical diabetes stage two adaptive addiction to eggs and clinical diabetes stage three nonadapted to wheat. 6. Monitoring of blood sugar one hour after test exposures to foods and chemicals in several hundred tests gives convincing evidence of the development of pancreatic insufficiency as part and parcel of the reaction of addictive adaptation as well as occurring during the final nonadapted exhaustion stage three reaction. A small sampling of pH changes in the duodenum using the Heidelberg capsule reveals the characteristics of a normal alkali production of pancreatic bicarbonate when test foods do not evoke symptoms and low pancreatic bicarbonate production in response to symptom and/or hyperglycemia-evoking test food exposures. With this evidence at hand, we are better able to understand the emergence of acute metabolic acidosis as more than interference of carbohydrate, fat, and protein metabolism, but as strongly and immediately present due to pancreatic insufficiency as reflected in inhibited pancreatic bicarbonate production. My observations are that there is an observable continuum between stage two adaptive addiction in chemical diabetes and stage three nonadapted clinical diabetes in respect to exocrine pancreatic insufficiency. The observations of B. M. Frier that in the diabetes disease process bicarbonate is the most inhibited followed by the enzymes, and last and least the hormonal production of insulin holds true as a continuum of the entire spectrum from early to late in the disease process. Thus the pancreas emerges as the initial and most important stress shock organ. An overstressed pancreas adapts to this stress by inhibition, thus producing a selective stimulus-evoked pancreatic insufficiency. As far as food and chemicals are concerned, this pancreatic insufficiency adaptation can be identified as a state of addiction.

### **The Role of Inflammation and Symptom Production**

Among the several endogenous substances (histamines, kinins, serotonin, slow reactive substance, complement, epinephrine, norepinephrine, anaphylotoxin, acetylcholine, prostaglandins, and likely the hallucinogen trimethyltryptamine) (McGovern and Hayward, \*1970; Keller-meyer and Graham, 1962; Ostfield et al., 1957; Lee, 1974) described as capable of evoking inflammatory reactions the tissue hormone kinins are the most frequent, most severe, and often are the final common pathway producing inflammation evoked by the other inflammatory substances. Symptoms are evoked by the production of inflammation in specific tissues responding to contact with selective substances. The presence of kinins makes most of these inflammatory reactions occur. Several substances can raise and/or evoke the production of kinins, among which fibrinolysin from the blood and trypsin from the pancreas are the most frequent.

### **The Role of Proteolytic Enzyme Inflammatory Blocking and Resolution**

Fibrinolysin plays a double role in that it can raise the kinin level and also dissolve blood clots by lysing fibrin. The pancreatic enzymes chymotrypsin and carboxypep-tidase have a major role in resolving inflammation from any source and exercise control over the level of kinins available to make inflammations occur. The net result of pancreatic insufficiency is a lessened ability to resolve inflammatory reactions as well as loss of control over preventing inflammatory reactions from being evoked.

### **The Role of pH in Inflammatory Reactions**

An acid pH encourages production of kinins. An alkaline pH is necessary for adequate function of the proteolytic enzymes and speeds up the

destruction of kinins by chymotrypsin and carboxypep-tidase.

### **The Role of Amino Acids**

Proteolytic enzymes are built from amino acids, and if amino acids are deficient these inflammation-resolving and inflammation-blocking enzymes will be deficient. Of immediate import is the fact that certain amino acids evoke the duodenal and jejunal mucosa to produce cholecystokinin-pan-creazyme which in turn evokes proteolytic enzyme secretion from the pancreas. Thus dietary supplementation of free amino acids evokes pancreatic enzyme production. Of course this is dependent on the integrity of the duodenal and jejunal mucosa which unfortunately has been determined to be atrophied in some cases, especially in schizizophrenia (Meyer, 1935; Reiter, 1929; Buscaino, 1958; Beyerholm, 1929; Lehman, 1967).

### **Test Evidence of Proteolytic Enzyme Supplementation Value**

Evidence from Heidelberg pH studies: Male, age 52.

Provocative test meal for millet.

Symptoms: Stomach ache, headache, light-headed.

Hyperglycemic reaction: 200 mg percent at one hour post meal.

240 mg percent at two hours post-test meal.

Heidelberg pH test: Gastric pH 2.8.

After test meal gastric pH 1.8. Duodenal pH 2 at 50 minutes after meal began.

Note:

Small intestine pH remained acid in response to this symptom and hyperglycemic reactive food. Both hyperglycemia and lack of duodenal alkali are due to inhibition to pancreas as a response to food. Female, age 52.

Provocative test for soy.

Symptoms: None.

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Blood sugar one hour post-test meal 90.

Cytotoxic test for soy was negative. Heidelberg pH test-Gastric pH 1.7.

Duodenal pH 1.5 at 50 minutes post-test meal.

Note: Normal intestinal pH with non-reactive food. A series of patients were tested on known reactive foods and chemicals by giving pancreatic enzymes one hour ahead and one-half hour ahead of each exposure. There was a consistent demonstration of a reduction and sometimes complete absence of symptoms. The same held true of hyperglycemic responses. In some cases it was found necessary to associate amino acids with the pancreatic enzymes in order to achieve a lessening or absence of symptoms. Tests were then performed to see if proteolytic enzymes and amino acids would reduce cytotoxic tests, and it was found that they did reduce the cytotoxic test reaction.

An example of the values of proteolytic enzyme and amino acid therapy are found in this example. A patient who was very sensitive to petrochemical hydrocarbons, and could not even drive to the office through car traffic without symptoms developing, had the following experience after one week of proteolytic enzyme therapy plus amino acids. This patient had complete relief from reactions to petrochemical hydrocarbon exposures, whereas before, even while on megavitamins and a diet of avoidance of incriminated foods on a four-day rotation diet, he was severely reacting to contact with auto exhaust, perfumes, gas stoves, etc. Before amino acid therapy began, he violated his diet by eating a meal of Mexican food and suffered numerous and severe symptoms. One week after enzyme therapy plus amino acids, he purposely ate a test meal again of Mexican food with the result of no symptoms occurring.

### Suggested Schedule for Enzyme and Amino Acid Therapy

Beginning of meal (only for those with achlorhydria or low gastric acid)

1. Glutamic acid hydrochloride, 5 grains.

a. Acidulin by Lilly or equivalent.

b. Alternative: Milcozyme by Miller.

Contents: glutamic acid hydrochloride, 5 grains

Betaine hydrochloride, 3 grains

pepsin, 1 grain pancreatic enzyme concentrate, 167 mg. End of meal

1. Two pancreas compound tablets or capsules, 325 mg or more (with or without duodenum).

2. Two 100 mg bromelain tablets with 10 mg papain.

3. One or two tablespoons ( $7^{1/2}$  to 15g) octamino powder or equivalent amino acid liquid, tablets, or capsules.

Thirty to 45 minutes post meal

1. Two pancreas compound tablets or capsules.

2. Bromelain with papain tablets.

3. One 10 grains sodium bicarbonate tablet or  $1/4$  teaspoon sodium bicarbonate and potassium bicarbonate mixture (two to one ratio).

Bedtime (optional)

1. Two pancreas compound tablets or capsules.

2. Two bromelain tablets with papain.

3. One 10 grains sodium bicarbonate or  $1/4$  teaspoon sodium bicarbonate, and potassium bicarbonate salts.

### Proposed Supernutrition Program

A proposed optimum supernutrition program is on this order:

Vitamin C -1-4 g three times a day.

Powder is the best tolerated. Pregnant women or potentially pregnant women should likely not receive more than 3 to 4 g a day due to a question of potential spontaneous abortion. However, spontaneous abortion from vitamin C in humans has not been demonstrated. The animals in which megadoses of vitamin C produced abortion were given this in addition to the vitamin C their livers make. Therefore a human equivalent of the animal experiments

producing abortion would be in the range of 20 to 30 or more grams of vitamin C per day. It is likely true that 12 g per day would not produce abortion in humans.

B6-100-500 mg three times a day.

B5-100-500 mg three times a day.

B2-100-500 mg three times a day.

B1-100-500 mg three times a day.

B3-500 to 1,000 mg three times a day as either niacin or niacinamide.

PABA-100-500 mg three times a day.

L-Glutamine—100-500 mg three times a day.

Vitamin E-200-800 units three times a day.

Vitamin D-400-800 units three times a day.

Vitamin D is optional due to the reports of increased arteriosclerosis with supplemented vitamin D.

Magnesium as a chelate-75-150 mg three times a day.

Manganese as a chelate-10-20 mg three times a day. Maintain these high doses for one or two months and then consider reduction. An initial hair test, which also should be repeated in six months to one year, should serve as a guide to supplementation of calcium, magnesium, potassium, manganese, zinc, and chromium as well as the possibility of toxic levels of lead, mercury, cadmium, or arsenic.

### Enzyme Sources

There are numerous good sources of pancreatic enzymes. The usual tablets or capsules contain 325 mg or more of pancreas compound. The test reactivity of these varied sources vary considerably in spite of having a comparable pancreas compound milligram value. Most are made from beef. A few are made from pork. If a pork source is needed, then Viokase brand is available as tablets or powder. Some contain pancreas compound plus duodenum such as, for example, General Research Laboratory's Pancreas with Duodenum. Bromelain and papain are useful vegetable sources of proteolytic enzymes, and can be obtained singly or together, for example, General Research Laboratory's Bromelain with Papain.

### Bicarbonate Sources

Ten-grain sodium bicarbonate compound tablets are made by Eli Lilly. The most physiological is a mixed powder of sodium bicarbonate 2/3 and potassium bicarbonate 1/3. This can easily be compounded by buying them separately. One-fourth teaspoon of this salt equals 10 grains.

### Amino Add Sources

Octamino Powder. This is made from predigested collagen and organ tissues of beef. This is a good source of amino acids. The recommended dose is one to two tablespoons three times a day with meals. This can be purchased from Dews Company, P.O. Box 147, Mineral Wells, Texas.

Liqu-A-Mone. This is a liquid beef amino acid source of collagen and organ tissues. Recommended dose is one to two tablespoons three times a day with meals. This is made by the Sivod Bioresearch Company, Madison Heights, Michigan 48071.

Liquid amino acid derived from beef collagen is made by several companies. This is not as complete as those also from organ tissues. Pork and beef sources are available.

There are amino acid tablets and capsules available from several companies made from whey, soy, or by bacteria.

### Ecologic Factors to Consider for Treatment

1. Avoidance of symptom-reactive inhalants and foods.
2. Four-day rotation of foods with foods kept in families. Reactive foods returned to the diet after three months if demonstrated not to be still symptom reactive. For details see the book, **Clinical Ecology**, pages 472 to 486.
3. Administration of autogenous bacterial vaccines and BCG vaccine, or other stock bacterial and viral vaccines.

### Values of Comprehensive Treatment

Factors such as economic factors or patient's preference force us on occasion to use proteolytic enzymes in a setting of less than desirable limited treatment goals. In proceeding with limited therapy goals we must bear in mind that the success rate diminishes as we leave out of our treatment such vital factors as addictions to foods, tobacco, alcohol, or lack of supportive nutrients, lack of treatment for opportunist organism, or lack of problem solving, etc. The physician should have firmly in mind the values necessary for an optimum treatment program, and an attempt be made to persuade each patient to strive for these optimum holistic treatment goals.

### How to Condition a Raise in the Threshold Level of Kinin-Evoked Inflammation

In addictions, toxic states, infections, and nutritional deficiency states the threshold level of kinin-evoked inflammation lowers. Restitution from the state of addiction, removal of the toxins, infections, and providing nutritional adequacy usually measurably raises the level of kinins at which level inflammation is evoked. However, there remains a group of "brittle reactors" in whom a low level of kinins continues to evoke inflammation. How can we correct this metabolic defect of inflammation evoked by a low level of kinins?

Brief intense stimuli that temporarily raise the kinin level are capable of normalizing the threshold of kinin-mediated inflammation. Herein lies the secret of the nonspecific beneficial effect of a wide assortment of brief intense stimuli. This common nonspecific value is found in such diverse areas as exercise, electrical stimuli, bacterial vaccines making use of either bacterial bodies or their toxins, intravenous EDTA, etc.

The evidence that EDTA raise kinins (Bell, 1975), thus increasing the tolerance for a higher threshold of kinins, likely in part explains why Peters et al. (1958,1961) reported a group of "porphyric schizophrenics" improved by EDTA chelation. My study of

porphyria in schizophrenia revealed it, when present, to be due to reactions to specific individualized foods and chemicals.

Exercise, bacterial vaccines, electrical stimuli, and/or other methods that raise the threshold of kinins at which inflammation is evoked should be used to increase tolerance to foods, chemicals, bacteria, toxins, etc. The nonspecific value of these restorative stimuli can optimally be achieved only in a framework of optimum nutrition. The metabolically restorative stimuli I find practical to use routinely are optimum exercise and autogenous bacterial vaccines.

### Corrective Measures for Stimuli-Induced Pancreatic Insufficiency

#### 1. Avoidance and Spacing

Of prime importance in correcting pancreatic insufficiency induced by maladaptive reactions to foods, chemicals, and inhalants is to avoid the stressful stimuli. A number of allergists, internists, and pediatricians have provided initial avoidance of the incriminated substance followed by spacing of the substances which has profitably been applied to large numbers of patients. An initial three-months' avoidance period of symptom-incriminated substances followed by a four-day rotation of these foods kept in families provides a 95 percent ability to return these foods to the diet. There remains an even larger number of "brittle reactors" requiring additional factors in their therapeutic program.

#### 2. Four-Day Rotation

Place all foods in a four-day rotation with the foods kept in families which usually prevents addiction to new foods from developing. For some the spacing needs to be more than four days. It is best not to eat between meals so as to reduce pancreatic stress.

#### 3. Pancreatic Enzymes and Bicarbonate Supplementation

Some recover pancreatic function by the process of initial avoidance followed by four or more days' spacing of foods, plus stopping all addictions to such as tobacco,

alcohol, coffee, drugs, as well as the removal of demonstrated individualized symptom producers such as petrochemical hydrocarbons, chlorine, cat, dog, and so forth. There are an even larger number, especially among the chronic psychiatric and neurologic cases, who have only partial recovery of the pancreatic function by the above process. For these, supplementation of pancreatic enzymes and bicarbonate are necessary for optimum health. Enzymes (325 mg or more of pancreatic substances) are best provided at the end of a meal as well as 30 minutes after the meal at which time bicarbonate (10 mg or more of sodium bicarbonate 2/3, plus potassium bicarbonate 1/3) is also provided. It is well to also give bromelain with papain with the pancreatic enzymes.

#### 4. Free Amino Acid Supplementation

Amino acids become deficient when pancreatic proteolytic enzymes are deficient. Of immediate importance is the fact that several of the amino acids when in contact with the duodenal mucosa evoked the hormones cholecystokinin-pancreozyne which activate the production of pancreatic enzymes. The recommended amount of free amino acids for an adult is up to 15 g three times a day, given at the end of the meal.

5. B-Complex Vitamin Supplementation B-complex vitamins, especially B6, support pancreatic function. Also B6 is used in transport of amino acids through the small intestine mucosa. 100 to 500 mg three times a day given with meals is the recommended dose. This is best supported by a balance of other B-complex vitamins. In some it is necessary to provide an equal amount of riboflavin in order to prevent riboflavin deficiency.

6. Ascorbic Acid Supplementation Vitamin C supports adrenocortical function. Vitamin C has a major role in preventing viral and bacterial infections. It is recommended that 4 to 12 g of vitamin C be given each day in three divided doses. It would be preferred if the vitamin C were taken an hour or so ahead of the meal.

#### 7. Autogenous Vaccines

A byproduct of pancreatic insufficiency is a

defective immunologic defense against bacterial and viral invasion. This deficiency can be corrected by administering autogenous vaccines which are made from all the bacteria cultured from each person. The building of antibodies against bacteria and viruses requires an optimum amount of amino acids and B-complex vitamins, especially pyridoxine and pantothenic acid (Axelrod, 1973; Axelrod, 1964). Vaccines also make a serious stress on adrenocortical function. Vitamin C is necessary for adequate protection of the adrenocortex when administering vaccines, either autogenous or stock. Vaccination with inadequate vitamin C for adrenocortical support can be dangerous to health or even life-threatening (Kalokerinos, 1974). 8. Protection Against Anticipated Exposure In order to shop or travel, the following is recommended for those hypersensitive persons who would have reactions to petrochemical hydrocarbons:

(a) Heparin, one drop (5,000 units/cc), solution sublingually placed. This can be used five or more times a day if need be. It is recommended to take one drop before the anticipated exposure and repeat this if symptoms develop. If one drop is not adequate, trials should be made up to five drops. The anti-inflammatory value of heparin has been documented (Dolowitz, 1971; Weaver et al., 1963; Fuller, 1958, 1960).

(b) Pancreatic enzymes and bromelain can be taken ahead of the anticipated exposure with a measurable prevention of symptom production. One hour prior to anticipated exposure, or should symptoms start developing, the following amounts or more could be given: five pancreas compound tablets or capsules, five bromelain tablets with papain, and one 10 grains sodium bicarbonate tablet.

(c) 50 to 100 mg of B-15 can be taken ahead of an exposure with measurable reduction in symptom production.

(d) 4 to 8 g of vitamin C taken ahead of exposure can measurably reduce symptom production. B6, 1,000 mg, has an added value to the vitamin C.

(e) Preltron 334 mg or more.

### 9. Insulin Dependency

When the pancreas has been sufficiently structurally damaged, then insulin dependency develops. However, the indication is that there are large numbers of patients on insulin who are not truly dependent on insulin if their diet is regulated by selecting out the carbohydrates, proteins, and fats to which they are reactive, as well as stopping all addictions to alcohol, tobacco, coffee, as well as avoiding substances to which they are hypersensitive. John Potts, M.D. (Potts and Lang, 1977) found that four out of seven insulin-dependent patients did not need insulin when this program was followed.

However, there remains a group of insulin-dependent patients in which it is imperative that insulin be given for the maintenance of optimum health. The rule has been to use insulin to manage hyperglycemia. However, there is also another group of insulin-dependent patients in whom the controlled avoidance and spacing of food program handles the hyperglycemia, but not all the symptoms. These can be determined by measuring insulin response to a food which does not specifically evoke symptoms of hyperglycemia. If the insulin production is less than optimum, then a trial should be given of small doses of five to 10 or more units of insulin first, using regular for testing, and then proceeding to NPH or protamine zinc for long-term use. The insulin is titrated to the amount of insulin which gives maximum relief of symptoms without adversely reducing blood sugar. Some patients with pains, depression, tension, and general ill feeling will have these symptoms disappear on small doses of insulin. It is of interest to note that in pretranquilizer years psychiatrists recorded the relief of tension and other symptoms by the use of small doses of insulin. Indeed, it can now be demonstrated that there are patients whose blood sugar can be adequately managed by diet, but whose symptoms, both physical and mental, cannot be adequately managed without the use of small doses of insulin.

### Conclusions

The pancreatic proteolytic enzymes, chymotrypsin and carboxypeptidase, are nature's anti-kinin and anti-inflammatory agents, and can be characterized as nature's tranquilizers. Pancreatic trypsin and blood fibrinolysin are kinin evoking, and therefore are nature's inflammatory evoking enzymes. Nature has so arranged it that the pancreatic proteolytic enzymes have no observable feedback mechanism or disease-producing side effects as have been observed with adrenocortical hormones. Long-term use of pancreatic proteolytic enzymes carries no danger with the remote possible exception in hemophilia. However, these enzymes are not involved in blood clotting as such. In contrast to this "no harm" with chronic use of proteolytic enzymes, the major tranquilizers (phenothiazines) and anti-depressants (imipramine hydrochloride) and lithium inhibit kinins, but also have the disadvantages of frequently producing chronic diseases such as Parkinsonism, tardive dyskinesia, as well as the phenothiazine evoking a four- to five-fold increase in the incidence of overt clinical diabetes (Zumoff and Helman, 1977; Norman and Hiestrand, 1955; Hiles, 1956; Arneson, 1964; Waitzkin, 1966; Thonnard, 1968). Thus we see that with tranquilizers used to reduce the kinin-mediated inflammatory reactions, the basic disease process continues and may even increase, while using nature's own proteolytic enzymes and bicarbonate inhibition of kinin inflammation, the disease process is slowed down or even reversed.

The pancreas has the essential job of providing enzymes that control inflammation whether this be due to a cut, bruise, or food reaction, chemical reaction, heat, cold, anger-evoking norepinephrine which in turn evokes kinins, or anxiety-evoking epinephrine which in turn evokes kinins. The pancreas is the first body organ to have the job of handling inflammatory stress reactions or adaptation to these stresses, and is, therefore, the first organ to give way to stress. Hans Selye was right that chronic

stress, physical or mental, leads to chronic disease, physical or mental. He rightly understood the adrenal cortex as the organ that gives up the fight last and gave small recognition to the role of the pancreas, but now we can confidently add the role of the pancreas as having a primary role in handling stress-evoked inflammation.

We should not reason that now that we have anti-inflammatory enzymes it doesn't matter anymore what a person eats, smokes, drinks, or what his nutritional state is. Indeed it matters very much, since giving anti-inflammatory enzymes under these non-physiological conditions does not prevent the progression of the disease process, and only minimally provides some degree of immediate lessening of symptoms and some degree of slowing down of the disease progression. The fact is that a person cannot be nutritionally deficient, toxic, infected, or addicted without suffering the consequences of progression of the disease process into a chronic degenerative disease of some type. Providing pancreatic proteolytic enzymes and bicarbonate simply provides physiological supplements in a stress-failing organism, and for a reasonable successful treatment needs all the other dynamics of the human organism honored, such as reduced physiological and psychological stresses, no addictions to anything, optimum nutrition, optimum exercise, optimum rest, optimum immunological defense against opportunist organisms and microbes, etc. Symptom-evoking foods, chemicals, and inhalants need to be isolated as well as avoided, and also spaced with a frequency below symptom production. This is the only way food addiction can be corrected or avoided. Acute infections are best treated with antibiotics and vitamin C, and other supportive nutrients. Chronic infections are best treated with autogenous vaccines while supported with supplements of vitamin C, B6, B5, and other nutrients.

In this dynamic physiological setting, supplemental pancreatic proteolytic enzymes and bicarbonate are provided. This program can evoke a kinin-inflammatory reaction by pancreatic trypsin-raising kinins, when necessary, as well as providing anti-inflam-

matory reaction ability and kinin-control ability when necessary through chymotryp-sin-and carboxypeptidase-inhibiting kinins and resolving inflammations. Humans would quickly die if there was no inflammatory ability to seal off wounds, wall off infections, etc. And equally humans would soon die if they had no control over inflammatory resolution or production. The dynamic availability of both inflammation and its prevention and resolution are necessary for the maintenance of life. With inflammatory and anti-inflammatory enzymes adequately available, it is other nonenzymes factors that decide whether it will be an inflammatory reaction or not. The maneuvering of these other nonenzyme factors such as reactions to foods, chemicals, inhalants, or the nutritional state, physical or psychic exposures to stress, etc., decide the use that will be made of the available inflammatory and anti-inflammatory factors. Thus, I view the use of pancreatic proteolytic enzymes in a setting of dynamic Orthomolecular medicine rather than a chemical patch on inflammation such as has been the practice with tranquilizers, antidepressants, sedatives, and hypnotics.

Pancreatic deficiency, whether produced by addiction or pancreatitis, affects most of all the bicarbonate production and last and least of all, insulin production. When bicarbonate and pancreatic enzymes are supplemented, the pancreatic insufficiency of insulin production is lessened. It is likely true that an assessment of individualized reaction to foods, chemicals, and inhalants followed by a diversified rotation diet plus enzyme and bicarbonate supplementation would reveal the majority of currently considered insulin-dependent diabetics as not being insulin dependent. There remains two types of insulin-dependent patients such as

- (1) for management of hyperglycemia, and
- (2) for management of symptoms produced by low level insulin interfering with general cellular function.

Viewed from an emotional reaction point of view, it can be observed that most patients with pancreatic insufficiency can profitably use in addition to medical management that of problem solving;

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training down phobias, obsessions, and compulsions, relaxation training, and so forth. Although correction of the abnormal physiology will reduce both the frequency and intensity of emotional reactions, we must rely on psychotherapeutic measures to train out unadaptive learned responses even though many of these were initially learned during the maladaptive reaction states to foods, chemicals, and inhalants.

For those with pancreatic insufficiency who also need psychotherapeutic intervention and are treated simultaneously for their disordered physiology and unadaptive learned responses, there exists a welcome breath of fresh air, such as (1) not being falsely accused of being responsible for organic-produced symptoms, (2) increased physiological stability in which state corrective learning is initially more successful and more stably maintained, (3) the increased awareness of the self and body function which is inherent in the subjective examination of monitoring physical symptoms and emotional feelings during the symptom-provocative tests, and (4) the realities of ecologic stimuli which observably evoke physical and mental symptoms do much to correct the trend of both patient and doctor to have mistakenly assigned interpersonal relationships, childhood experiences, and fateful life circumstances as causes of these symptoms. The therapist is rewarded by a marked improvement of his therapeutic successes under the state of improved physiological homeostasis.

It should be appreciated that the treatment of pancreatic insufficiency and the symptoms and chronic illnesses it evokes are not based on deductive reasoning stemming from speculative philosophy, but are rather based on objective induction evidence such as evoked hyperglycemia, a measured hypoproduction of bicarbonate, as well as observations of an objective observer associated with the subjective evidence of symptom production observed by the patient. This state of affairs is a welcome relief for both patient and physician who have so long approached this same set of symptoms with speculative philosophy only. Speculative deductions

cannot be completely dispensed with, but can be through the induction evidence and laboratory monitoring reduced by approximately 60 percent *over* that of the traditional deductive speculation about symptom causes.

It is a worthy goal of doctors in general and psychiatrists and neurologists in particular to rule out organic causes of symptoms before assuming individualized internal emotional causes. Experiences reveal that it is obvious that this goal can never be reached until ecologic-metabolic differential diagnosis is a serious part of the differential diagnosis of emotionally disordered persons, whether these be diagnosed as psychotic, autistic, learning disabled, hyperkinetic, neurotic, character disordered, or psychosomatic. This ecologic-metabolic differential diagnosis is as needed in these emotionally disordered cases as in chronic physical illnesses.

The evidence is that pancreatic insufficiency expressed as adult onset overt clinical diabetes, sufficiently serious to be insulin dependent under traditional treatment, will recover sufficiently in many cases to not be insulin dependent under the ecologic-metabolic program. The program is an initial three-months' avoidance of symptom-incriminated carbohydrates, fats, proteins, chemicals, inhalants, and hyperglycemia-producing substances associated with optimum supplementation of pancreatic enzymes, bicarbonate, amino acids, vitamins, and minerals. However, the management of hyperglycemia is not the only function of insulin. There is a general cellular need for insulin in the metabolic process. These chronic symptom insulin-dependent patients can be in either the chemical or clinical phases of the diabetes mellitus disease process and are diagnosed by (1) chronic physical and mental symptoms continuing in spite of adequate ecologic-metabolic management including the management of hyperglycemia and (2) by laboratory evidence of a less than adequate insulin response to a test meal of a food that does not evoke either symptoms or hyperglycemia. The insulin dosage can be titrated to a level that reduces or dispenses

with the chronic symptoms without adversely reducing blood sugar. It remains for a -large scale, definitive study to reveal the incidence of (1) insulin dependency reversible by ecologic-metabolic diagnosis and treatments, and (2) symptom insulin-dependent (not hyperglycemia insulin-dependent) subjects in the chemical as well as the clinical phases of the diabetes mellitus disease processes (generalized pancreatic insufficiency).

A valuable lesson for physicians to learn is that an ecologic-metabolic examination reveals that diabetics have far more than just hyperglycemic reactions to specific foods, chemicals, and inhalants, but that of equal importance is the readily observable fact of multiple physical and mental symptoms evoked during induction test exposures, and that these symptoms encompass the same symptomatology as the chronic degenerative diseases considered as complications of or associated with diseases of chronic diabetes mellitus. Thus the mystery of the many noncarbohydrate disorder diseases associated with diabetes (Harvey et al., 1972) is revealed to be the common denominator of maladaptive reactivity to foods, chemicals, and inhalants. Blood sugar monitoring during test exposures reveals the chemical stage of diabetes to be more frequent than the clinical stage of diabetes in these diseases. In any event these are simply stages of generalized pancreatic insufficiency resulting from the stress of maladaptive reactivity to foods, chemicals, and inhalants. Chronic stress leads to inflammation by the mechanism of reduced anti-inflammatory substances as well as increased inflammatory substances within the body. These inflammations are named as diseases. The name given the disease corresponds to the site and type of inflammation, the endocrine glands disordered, the autoimmune reactions evoked, the metabolic systems disordered, or the secondary invading opportunist organisms.

## Documentation Information Beyond the Scope of this Presentation

### 1. Textbook of Medical Physiology

Arthur Guyton, M.D. (1971) Gastric Function  
Small Intestine Function. Pancreatic Function.  
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