Selective Substance Reactivity in Pancreatic Insufficiency

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Provocative Food Testing the Diabetic

Jim was an adult onset diabetic of five years' duration. In spite of strictly following his assigned diet and insulin, he had never been in good control. Because of being a brittle diabetic, he was under the care of a diabetes specialist. Perceptual distortions, obsessions, paranoia, and depression developed and justified the diagnosis of schizophrenia.

I first saw him when he tried to hang himself. His fasting blood sugar was 250 mg percent. The question was whether I should fast and food test him. Based on the evidence of Arthur Coca, M.D. (1972), allergist-immuno-logist, as reported in the book, **The Pulse Test,** in which he observed diabetics improving and even able to stop insulin when their allergies were managed, I decided to test him for allergies. By the fourth day of the fast his blood sugar was 100 mg percent and remained at 100 on the fifth day when food testing began. I tested his blood sugar before and one hour after each test meal and introduced no test meals until his blood sugar was normal before the test. There was an assortment of foods, proteins as well as carbohydrates, which he reacted to with hyperglycemia. There also were carbohydrate foods which did not evoke hyperglycemia. Whole wheat (Ralston) was especially striking. His blood sugar was 250 mg percent in one hour, and at the same time he wished to die. His diabetic diet included one-half slice of whole wheat bread three times a day. As long as he ate this wheat daily, he was an uncontrolled diabetic and paranoid schizophrenic.

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Hypoglycemic and Hyperglycemic Monitoring

Since this case I have monitored both hypoglycemic and hyperglycemic cases during deliberate food testing and have found convincing evidence that:

1. In either case the reaction is quite relative and may as well be a protein or a chemical as a carbohydrate.
2. Drawing conclusions about carbohydrate intolerance based on one food such as corn or grape sugar is not valid.
3. We could just as logically have diagnosed diabetes based on a reaction to whole wheat as to corn sugar since we would have arrived at approximately the same percentage.

4. Characteristically there are also symptoms other than hypoglycemia and hyperglycemia revealing evidence of an allergic or allergic-like reaction.

5. Characteristically a four-day fast reverts hypoglycemia to hyperglycemia revealing (a) a continuum between hypoglycemia and hyperglycemia, (b) hypoglycemia as the withdrawal stress phase of an addictive state, and (c) hyperglycemia to be a chronic reactive phase resulting from the metabolic failure to maintain the addictive adjustment.

Is Generalized Carbohydrate Intolerance Incorrect?

Could it be that John Tintera (1966) was wrong in using the single food glucose in drawing generalized judgment about carbohydrate intolerance? Could it be that medicine in general is operating under a fallacy, such as the generalization that one food (corn sugar) test reveals to us generalized carbohydrate intolerance and that based on this we can best manage diabetes mellitus?

With what I deemed a logical enthusiasm, I sent my patient back to his internist to report our findings of success. He reported, "I have no sugar in my urine, my blood sugar stays normal, I am not depressed, and I am mentally clear." The doctor's quick and confident reply was, "Food allergy has nothing to do with diabetes. He just did a better job of managing your diet than I did."

Then I took my findings about food and chemical selectivity in diabetes as demonstrated during food testing to an internist who frequently helped me with patients. He did not laugh. Instead he brought me evidence from the medical literature on diabetes that there are those who have observed that the carbohydrate intolerance is selective and not general and deplore that this fact is not used generally in the management of diabetes. He also brought me the evidence that diabetics are sometimes allergic to insulin.

Obscure Metabolic Dysfunction

It is recognized that the so-called complications of diabetes can and do exist separate from diagnosable disordered carbohydrate metabolism. "Indeed, there is accumulating evidence that the complications of diabetes mellitus can be found without any detectable disturbance in carbohydrate metabolism. This suggests that the syndromes of insulin insufficiency and the later complications are both related to a more fundamental, but at present obscure, metabolic disturbance" (Harvey et al., 1972). What is this as yet obscure metabolic disturbance in diabetes and other diseases with common symptoms and common tissue disorders? I submit that systematic ecologic testing of foods and chemicals reveals it to be hypersensitive (allergic, allergic-like, and addictive) reactions to foods, chemicals, and inhalants.

Selective Response Versus Generalized Response

There are two firmly established concepts that are routinely accepted by diabeto-logists as gospel truths which are: (1) diabetes mellitus is due to generalized carbohydrate intolerance, and (2) diabetes mellitus reaction is dependent on total calorie intake and this especially applies to any and all carbohydrates, so much so that the insulin requirement is figured from the carbohydrate calories consumed.

When the validity of these two virtually sacred fundamentals is questioned, the diabetologist first feels he need consider the medical opinion no further since he automatically considers it is untrue. Yet questioning the truth of these two accepted fundamentals is precisely what an ecologic-metabolic examination of the diabetes mellitus disease process does.

The evidence of the incorrectness of (1) diabetes mellitus is due to general carbohydrate intolerance, and (2) the diabetes mellitus reaction bears a direct relationship to the amount of carbohydrates (from any source) is on this order:

1. Blood sugar monitoring of test meals of single foods after a four- to six-day avoidance period of these foods or chemicals reveals (a) carbohydrates to which there is no symptom and/or hyperglycemic reaction, (b) protein and fats to which there is a symptom and/ or hyperglycemic reaction, and (c) nonfood chemicals such as tobacco or petrochemical hydrocarbons to which there is a symptom and/or hyperglycemic reaction in the absence of food.

2. Random quantities of test meals of single foods such as "eat the amount you wish" reveals reactions to small as well as large amounts of foods. Indeed sublingual tests of food extracts reveal a high correlation of symptoms to these reactive foods and chemicals as well as intradermal serial dilution testing revealing whealing. The conclusions are that these are allergic and/or allergic-like reactions no matter how you examine for them.

3. Frier et al. (1976) give evidence that diabetes mellitus encompasses generalized pancreatic insufficiency and that the bicarbonate is the most deficient, followed by enzymes and last and least by insulin production. Rather than characterizing diabetes mellitus as carbohydrate intolerance, caused by insulin lack, it is more correct to describe the diabetes mellitus disease process in terms of generalized pancreatic insufficiency. On the other hand, it may be more valuable to describe chemical and clinical diabetes mellitus as simply stages in the generalized pancreatic insufficiency, in which pancreatic insufficiency is highlighted as the central disease process in which there emerge numerous types of degenerative diseases both mental and physical, with diabetes mellitus being only one of these variant disease syndromes. This explains the mystery of the varied so-called complications of diabetes which occur in the chemical as well as the clinical stage of the diabetes mellitus disease process. These really are simply the tissue deteriorations occurring due to the exocrine deficiency of the pancreas.

4. When the diabetes mellitus disease process is in its active form of maladaptive reactions to numerous foods and chemicals, then it is true that there is produced a generalized carbohydrate intolerance simply because inherent in these maladaptive reactions to whatever the reaction may be (proteins, fats, carbohydrates, chemicals, molds, bacteria or their toxins, arid so forth) the interference of carbohydrate metabolism occurs. No wonder the diabetologist thinks of diabetes as simply carbohydrate intolerance, since he never goes through the process of avoiding all the maladaptive reactive substances and therefore never discovers the selectivity of maladaptive reactive substances producing hyperglycemia.

As long as the subject is chronically reacting to maladaptive substances, it is true that the total calories count, and the carbohydrate calories especially count and can serve as a rough guide to the insulin need. However, as soon as the diabetic is withdrawn from the several selective substances he maladaptively reacts to, it is no longer true that the insulin needs can be judged by the carbohydrate calories or the total calories. But the secret is that the diabetologist draws his judgment from the subject in the chronic reactive state and the ecologist-metabologist draws his judgment from a recovered state of a chronic reactive state occurring after four to six days of avoidance. The ecologist discovers the diabetic's ability to recover from the maladaptive action by avoidance and discovers by single exposure testing what the specific maladaptive reactive substances are. The diabetologist, with his single glucose-tolerance test occurring in the chronic reactive state, discovers neither. Ecologists and diabetologists see diabetes mellitus in entirely separate frames of reference. Both are right in terms of their specific type of examination.

The diabetologist says: (1) reduce calories if obese, and (2) reduce carbohydrates and if the blood sugar is still abnormal, cover the rest with insulin.

The ecologist-metabologist says:

1. Stop all addictions and other acute or chronic maladaptive reactions which are isolatable by single exposure tests with

symptom and blood sugar monitoring, cytotoxic tests, sublingual provocative tests, intradermal serial dilution tests, RAST, and so forth.

2. Start a four-day diversified rotation diet, leave out for three months reactive substances, then return these substances to the diet on a once-in-four-day basis only if no symptoms or hyperglycemia occur.

3. Treat all isolatable infections with autogenous and stock vaccines such as BCG and so forth. If needed, treat with antibiotics.

4. Provide adequate vitamin C to inhibit infections, which is about 10 to 12 g per day.

5. Provide adequate B6, B5, amino acids, and other supportive nutrients to make the building of antibodies to the autogenous vaccines and also to optimally support metabolic functions.

6. If obese, provide calorie reduction.
7. Optimum exercise.
8. Low cholesterol and low fat.
9. Routinely supplement pancreatic enzymes and bicarbonate.
10. Last and least, supplement insulin only if and when the above measures have not solved the problems of (a) hyperglycemia, (b) depression, (c) generalized aching or tension, (d) or other physiologic or psychologic symptoms if demonstrated to be amenable to insulin therapy and if associated with a demonstratable low serum insulin.

I submitted to several diabetic specialists a statement of case histories and laboratory findings about the selective maladaptive diabetic (hyperglycemic, acidosis, and symptom) reactions to carbohydrates, fats, proteins, and chemicals. Some welcomed the information as worthy of in depth study while others appeared to learn nothing from the evidence presented. For some the opinion rendered was, "diabetes is carbohydrate intolerance," and with this statement dismissed all other opinions and laboratory evidence, and considered the issue a closed book. For others, the effect was on the order of the sacred cow of "generalized carbohydrate intolerance is diabetes mellitus" has been slain, and righteous indignation is justified. One reviewer disqualified himself from giving an opinion based on what he considered his lack of experience and lack of information. One wonders if he disqualified himself based on an honest feeling of being disqualified by lack of information, or due to a condescending niceness to a fellow physician whom he considered to be in error.

Confirming Evidence of Selective Response to Ecologic Stimuli in Pancreatic Insufficiency

Will R. Ryan, M.D., associate editor of the 1975 Yearbook of Endocrinology, gives the following perspective note on diet and diabetes mellitus, "As things stand now, no one knows what the ideal diabetic diet is. I think there is more unsubstantiated dogma in diabetes than in any other area of endocrinology. My compliments to those brave souls who are finding the facts and not kow-towing to the 'religion' (1975)."

Albert Winegrad, M.D., and Douglas Greene, M.D., believe that environmental factors causative of diabetes mellitus should be isolated and that the marked variability in the development of overt diabetic polyneuropathy suggest the presence of both genetic and environmental factors (Winegrad and Greene, 1976).

My long-time friend, John Potts, M.D. (Potts and Lang, 1977), took my evidence seriously. He fasted seven adult onset, obese, insulin-dependent patients for four days and food tested them by means of single foods after which he monitored the blood sugar. The selectivity of hyperglycemic response was in evidence with proteins as well as carbohydrates. This selectivity of response confirms my observations. Four of the seven patients did not need insulin, and all lost weight on a diversified four-day rotation diet leaving out the foods which evoked hyperglycemia.

I have recorded evidence that, giving pancreatic enzymes ahead of test meals which are known to evoke both symptoms and hyperglycemia, both of these maladaptive responses can be prevented from

occurring, or materially reduced. The cytotoxic test revealed the same diminution of response after taking pancreatic enzymes.

There are 11 published articles since 1925 informing us that diabetes mellitus involves generalized pancreatic insufficiency (Jones et al., 1925; Diamond and Siegel, 1940; Pollard et al., 1943; Chey et al., 1963; Vacca et al., 1964; Bock et al., 1967; Drewes, 1969; Yamagata et al., 1969; Baron and Nabarro, 1973; Domschke et al., 1975; Frier et al., 1976). The evidence of the diabetic's selective carbohydrate intolerance rather than generalized carbohydrate intolerance was published some time ago, and it was deplored that this fact is not employed in clinical practice. These established facts of

(1) generalized pancreatic insufficiency, and

(2) selective carbohydrate intolerance for some strange and unexplainable reason are not employed clinically by the diabetologist.

Proteolytic Enzyme Control of Kinin-Evoked Inflammation

The role and significance of generalized pancreatic insufficiency in inflammations evoked by exposures to foods, chemicals, inhalants, toxins, infections, etc., is understood when the necessity of pancreatic proteolytic enzymes (especially chymotrypsin and carboxypeptidase) in controlling and resolving kinin-evoked inflammation is understood (Wolf and Ransberger, 1972; Bell, 1974).

Absolute and/or Relative Pancreatic Insufficiency as Cause of Diabetes Mellitus

Saul N. Genuth, M.D. (1973) examined hourly glucose levels from 8:00 a.m. to midnight in normal, obese, juvenile and adult diabetic persons with and without obesity. Normal persons showed narrow glucose excursions and sharp postprandial insulin spikes. Obese persons showed threefold increase in fasting and postprandial insulin levels. In obese persons with mild carbohydrate intolerance, postprandial insulin release was delayed. Juvenile diabetics manifested complete insulin lack. Severely diabetic adults still showed definite although reduced postprandial insulin spurts, and the entire plasma insulin profile was still elevated by obesity. Daily peripheral insulin deliveries were calculated: normal, 31 units; obese persons, 114 units; juvenile diabetics, 4 units; adult diabetics, 14 units; adult obese diabetics, 46 units. Sixty percent of insulin delivery occurred postprandial in normal and nondiabetic obese persons while for frankly diabetic persons only 25 percent was delivered postprandial. The results clearly reinforce the belief that inadequate insulin secretion underlies diabetes even when obesity independently demands insulin hypersecretion. They also show that in severe diabetes postprandial insulin secretion is much more significantly impaired than basal insulin secretion in non-diabetics.

It appears evident from other studies (Friedman, 1973; Trowell, 1977; Leonard et al., 1974) that plasma fat and tissue fat beyond an optimum amount provide a state of insulin resistance which in turn metabolically demands more than a usual amount of insulin. The primary role of plasma lipid level over that of tissue fat in producing insulin resistance is likely an aspect of the observation I have made that obese, normal weight, or thin persons all are equally testable with deliberate food tests after a four- to six-day clearance fast. The test results in an obese person are equally reliable to that of a thin person. The evidence is strongly suggestive that the disease process reverts toward normal by clearance of plasma lipids even before weight reduction has occurred. In any event, diabetes mellitus represents « Jess than optimum insulin production by the pancreas even though in the case of obesity and a high-fat diet, the amount of insulin may be above normal for a normal weight person, but not adequate to meet the metabolic needs of an obese high-fat diet person. It should also, be considered that high plasma fat is an irritant to the vascular system and therefore also would demand more pancreatic proteolytic enzymes to control the inflammation evoked. Pancreatic insufficiency, producing

diabetes mellitus adult onset type, is a relative state and to some degree dependent on such metabolic factors as body fat, plasma lipids, toxins, irritants, infections, and reactions to foods, chemicals, inhalants, and other stress factors. One hopeful factor is the evidence of reversibility of the adult onset diabetes disease process with the removal of the varied stress factors. This is at least true of the early and intermediate stages. As the diabetic disease process continues, hyalinization of the pancreas can occur as well as repeated attacks of overt as well as silent pancreatitis.

Schizophrenics have epigastric distress as their most common complaint. The evidence I have documented, even with foods not producing symptoms, is suggestive of multiple, mild attacks of pancreatitis which damage the exocrine area of the pancreas, thus reducing pancreatic enzymes and bicarbonate production even more severely than insulin production. For this reason, supplementation of pancreatic enzymes and bicarbonate is in order in most chronically ill schizophrenics while only rarely is insulin indicated.

Research Considerations

Detailed broad-spectrum research will have to provide overwhelming convincing data before the diabetologist can be persuaded to take a serious look at ecologic stress-evoked diabetes mellitus in terms of generalized pancreatic insufficiency produced by multiple specific substances as foods in all categories, chemicals, inhalants, toxins, and infections. In the meantime, his therapy will essentially consist of manipulation of carbohydrate calories, total calories, and insulin supplementation.

The beginning of recognition by the diabetologists of ecologic factors in diabetes will likely be in terms of ecologic factors as nonspecific stress factors in the primary disease. Whereas the ecologist-metabolist believes his evidence obtained by monitoring blood glucose, acid base balance, and pancreatic function in a symptom-free and hyperglycemic-free state versus evoked symptoms and hyperglycemia by specific individualized foods, chemicals, and inhalants reveals that diabetes mellitus, especially adult onset type, is the natural biological consequence of these specific stressors. Genetics undoubtedly help decide which stressor will evoke this response.

Avoidance Period

(A) Fast on nonchemically treated water only for four to six days. Monitor the blood sugar daily during the fast. Provide supplemental nutrients if the blood sugar does not normalize within four days. The essential nutrients are B6 and vitamin C supported by other balanced nutrients.

Or (B) an alternative is four to six days of meals of single foods which are seldom used. This avoidance of commonly used foods achieves the same goal as fasting. With this procedure the testing starts immediately. This system has the disadvantage of easily confusing withdrawal symptoms from foods to which the subject is reactive with reactions to test foods. If a test is not conclusive, then test it over a few days later. By the fifth day, frequently used foods can be tested.

Test Period

Meals of single foods are given under the conditions of not reacting to any chemicals or inhalants in the environment.

The blood sugar and serum insulin are tested before and one hour after each test meal. If symptoms occur before one hour, then also do a blood sugar and insulin at that time.

Measured Meals

All test meals are weighed. Three amounts are tested in equal numbers of subjects: (a) a large amount, (b) a small amount, and (c) "the amount you wish to eat."

Cytotoxic Tests

Foods, chemicals, and inhalants are tested by cytotoxic blood tests.

RAST

Foods, chemicals, and inhalants are tested by RAST.

Testing Juvenile Diabetics

Juvenile diabetics and those who have had or threaten to have an acidosis crisis should not be fasted or single food tested in the routine manner. They should have the cytotoxic test, RAST, and intradermal serial dilution testing, after which they are placed on a four-day diversified rotation diet leaving out the positive cytotoxic, RAST, and intradermal foods. The insulin requirements are regulated on this rotation diet.

It is possible under these circumstances to test the first meal of each day,before the giving of insulin.

Heidelberg Capsule Test

Gastric and duodenal pH is tested with a symptom-free and normoglycemic food and also with a symptom evoking and/or hyperglycemic evoking food.

Return of Foods to Rotation Diet

In three months, retest all foods that were symptom and/or hyperglycemic evoking, and if the response is normal then return these to the diet on a once-in-four-day basis. Retest cytotoxic, RAST, and intradermals (if done).

Low-Fat Diet

Chronic use of high fats in the diet beyond an optimum amount interferes with several metabolic processes. Examples are in such as high insulin output failure-type diabetes mellitus in obese adult onset diabetes and the vascular response of atheromatous plaques in response to the irritation of high blood levels of fats. The general evidence of the metabolic interferences of high fats provides a rationale for believing that this chronic lipid irritation produces metabolic interferences extending itself into an increase of hypersensitive reactivity to foods, chemicals, and inhalants. This consideration is reinforced by recent evidence in which the optimum antistress general diet reverting chronic degenerative diseases is composed of 5 to 10 percent fat, 10 to 15 percent protein, and 75 to 80 percent entirely complex carbohydrates (Pritikan, 1976). There are a number of studies reinforcing the evidence that a high-fat diet is unduly stressful and therefore disease producing (Trowell, 1977). The ideal antistress diet appears to be low fat (up to 10 percent), protein, 10 to 15 percent, low free carbohydrate, and up to 80 percent complex carbohydrate, low cholesterol (5 to 100 mg per day), alcohol free, tobacco free, and caffeine free. It is also necessary that this be associated with daily brief vigorous exercise to stimulate metabolic functions.

Conclusions

Medical education bases much of its advice on generalizations. However, the conclusions drawn about reactivity to specific substances such as carbohydrates and so forth is not the same when drawn from the chronic reactive state as it is when an avoidance period of four to six days provides for a metabolic recovery from a chronic reactive state. Thus, gout is not simply disordered purine metabolism in which the need is to avoid high purine foods, and likewise diabetes mellitus is not simply carbohydrate intolerance in which carbohydrates are to be avoided, but in these cases as well as many other metabolic disorders, the culprit is to be found in specific foods, chemicals, and inhalants maladaptively reacted to which interfere with metabolic processes. These metabolic interferences cannot be determined during

a chronic reactive state in terms of reactions to specific substances, but can only be determined in the metabolic recovered state when exposed singly to the potential reactive substances as well as the metabolic shifts monitored and symptoms observed. The selectivity of responses thus observed is more believable than the customary generalizations, and therefore should be given preference in terms of treatment formulas.

Generalizations continue to be useful in clinical practice and should be used as far as they can be determined to be reliable. Some apparently reliable generalizations are such as, (1) daily brief exercise is necessary to maintain optimum metabolic functions, (2) tobacco inhibits pancreatic functions, (3) alcohol inhibits pancreatic functions, (4) addictive adaptation inhibits pancreatic functions, (5) pancreatic insufficiency leads to inflammatory reactions, (6) fat beyond 10 percent has multiple interferences of metabolic functions, (7) high free carbohydrate has multiple metabolic interferences of metabolic functions, (8) nutritional deficiencies of vitamins, minerals, and amino acids produce disordered metabolism, and (9) toxins from and immunologic reactions to chronic as well as acute infections lead to disordered metabolic functions.

REFERENCES

ANTAR, M.A., and OLSON, M.A.: J. Nutr. 85,329,1965.

BARON, J.H., and NABARRO, J.D.N.: Pancreatic exocrine function in maturity onset diabetes mellitus. British Medical Journal 4, 25-27,1973.

BASTA, L.L, M.D., WILLIAMS, C, M.D., KIOSCHOS, J.M., M.D., and SPECTOR, A.A., M.D.: Regression of atherosclerotic stenosing lesions of the renal arteries and spontaneous cure of systemic hypertension through control of hyperlipidemia. The American Journal of Medicine 61,420423,1976.

BELL, I.R.: The kinin peptide hormone theory of adaptation and mal-adaptation in psychobiological illness. Ph.D. dissertation, Stanford University, 1974.

BOCK, O.A.A., BANK, S., MARKS, I.N., and JACKSON, W.P.U.: Exocrine pancreatic function in diabetes mellitus. South African Medical Journal 41,756-758,1967.

CHEY, W.Y., SHAY, R, and SHUMAN, C.R.: External pancreatic secretion in diabetes mellitus. Annals of Internal Medicine 59, 812-821,1963.

COCA, A., M.D.: The Pulse Test. New York: Arc Books, 1972.

DIAMOND, J.S., and SIEGEL, S.A.: The secretion test in the diagnosis of pancreatic disease with a report of 130 tests. American Journal of Digestive Diseases 7,435-442,1940.

DOMSCHKE, W., TYMPNER, F., DOMSCHKE, S., and DEMLING, L: Exocrine pancreatic function in juvenile diabetics. American Journal of Digestive Diseases 20,309-312,1975.

DREWES, V.M.: Exocrine pancreatic function estimated after intra-duodenal and after oral stimulation, and exocrine pancreatic function in patients with newly discovered diabetes mellitus and in long-term diabetes. Modern Gastroenterology 1229-1231. Edited by O. Gregor and 0. Riedl. Schattauer: Stuttgart, 1969.

FRIER, B.M., M.D., et al.: Exocrine pancreatic function in juvenile-onset diabetes mellitus. Gut 17,6f&691,1976.

FRIEDMAN, G.J.: Diet in the treatment of diabetes mellitus. In: Modern nutrition in hearth and disease. Goodhart, R.S., M.D., and Shils, M.E., M.D., Ed., pp. 842-851. Philadelphia: Lea and Febiger, 1973.

GENUTH, S.M., M.D.: Plasma insulin and glucose profiles in normal, obese, and diabetic persons. Annals of Internal Medicine 79, 812-822,1973.

GRAHAM, D.Y., M.D.: Enzyme replacement therapy of exocrine pancreatic insufficiency in man. New England Journal of Medicine 296, 1314-1317,1977.

GRANDE, F., ANDERSON, J.F., and KEYS, A.: J. Nutr. 86,313,1965.

GRESHAM, G.A.: Atherosclerosis 23,379,1976.

HARVEY, A. McGehee, JOHNS, R.J., OWENS, A.H., and ROSS, R.S., Eds.: The principles and practice of medicine. New York: Appleton-Century Crofts 879,1972.

JONES, CM., CASTLE, W.B., MULHOLLAND, H.B., and BAILEY, F.: Pancreatic and hepatic activity in diabetes mellitus. Archives of Internal Medicine 35,315-336,1925.

KIEHM, T.G., M.D., ANDERSON, J.W., M.D., and WARD, K.R.D.: Beneficial effects of a high carbohydrate, high fiber diet on hyperglycemic diabetic men. Am. J. Clin. Nutr. 29,895-899,1976.

LEONARD, J.N., HOFER, J.L., and PRITIKIN, N.: Live longer now. New York: Grossett and Dunlap, 1974.

MATHUR, K.S., KHAN, M.A., and SHARMA, R.D.: Brit Med. J. 1, 30,1968.

McGANDY, R.B., HEGSTEAD, D.M., and STARE, F.J.: New Eng. J. Med., 277,186 and 242,1967.

POLLARD, H.M., MILLER, L, and BREWER, W.A.: The external secretion of the pancreas and diabetes mellitus. American Journal of Digestive Diseases 10,20-23,1943.

POTTS, J., M.D., and LANG, M.S.: Avoidance provocative food testing in assessing diabetes responsiveness. Diabetes 26,1977.

PRITIKAN, N.: High carbohydrate diets: maligned and misunderstood. Journal of Applied Nutrition 28,56-68,1976.

RYAN, W.G., M.D.: The yearbook of endocrinology. Theodore B. Schwartz, M.D., Ed. Chicago: Yearbook Medical Publishers, Inc., 1975.

TINTERA, J.W., M.D.: Stabilizing homeostasis in the recovered alcoholic through endocrine therapy: evaluations of the hypoglycemic factor. Journal of American Geriatric Society 14,126-148,1966.

TROWELL, H., M.D.: Diseases that may regress on longevity research (LRU regime of diet and exercise. Santa Barbara, California: Longevity Research Institute, 1977.

VACCA, J.B., HENKE, W.J., and KNIGHT, W.A., Jr.: The exocrine pancreas in diabetes mellitus. Annals of Internal Medicine 61, 242-247, 1964.

VESSEUNOVITCH, D., WISSLER, R.W., HUGHES, R., and BOREN-S2TAJN, J.: Atherosclerosis 23,155,1976.

WINEGRAD, A.I., M.D., and GREENE, DA, M.D.: Diabetic polyneuropathy: the importance of insulin deficiency, hyperglycemia and alterations in myoinositol metabolism in its pathogenesis. New England Journal of Medicine 295.1416-1421,1976.

WOLF, M,, M.D., and RANSBERGER, K. Ph.D.: Enzyme therapy. Los Angeles: Regent House, 1972.

YAMAGATA, S., GOTO, Y., TAKEBE, T., and KOEZUMI, H.: Exocrine and endocrine function of the pancreas in diabetes mellrtus and chronic pancreatitis. In: Modern Gastroenterology, 1298-1300. Schat-tauer: Stuttgart, 1969.