# The Modernization Disease Syndrome as Substrate Pellagra-Beriberi:

A New Diagnostic Entity: Synergistic Malnutrition From Interacting Food Modifications

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#### Abstract

Today's multiple food manipulations may interact to produce a wide-spread difficult-toidentify synergistic malnutrition presenting as a highly idiosyncratic substrate-cat alystantinutrient vitamin-resistant Hoffer-type pellagra-beriberi mainly afflicting primates and accounting for the medically dominant modernization disease syndrome as a variant of the classical catalytic B vitamin-sensitive Goldberger-Eijkman-Takaki pellagra-beriberi.

#### Introduction

Authorities now link distortions of dietary fats to both the number 1 and 2 killers, namely heart disease and certain major cancers, especially breast, prostate and colon (1,2). But saturated and polyunsaturated essential fatty acids (EFA) have also long been implicated, particularly in primates, in arthritis (3), immune diseases (4diabetes (9-13), 8), eczemas (14),polyneuropathies (15), behavioral disorders including schizophrenia (16-21), cystic fibrosis (19) and other problems ranging from drying skin disorders to irritable bowel syndrome (20, 21).

Most of these newly prominent illnesses do not seem to result from diagnostic or therapeutic advances, for many are 1. A-307 Summit Drive, Bryn Mawr, PA 19010.

diagnostically striking, occur in the young or middle aged and have been tracked by medical officers as societies modernize, e.g. hypertension and schizophrenia (18,22) across the south sea islands. The conservative assumption is that they constitute highly idiosyncratic mav а modernization disease syndrome (MDS) in response multiple interacting to food manipulations adversely affecting co- and antinutrients which interact synergistically in the body to disrupt the body-wide lipid-based regulatory system, including prostaglandins, in ways depending on genetic susceptibility.

## The Reaction Coherence of Essential Nutrients

Most of the approximately 50 essential nutrients comprise a set of co-reactants: (1) substrates, including lipids (2) B vitamin containing enzymes, which process the lipids into products comprising the lipid regulatory system including prostaglandins and steroid derivatives, and (3) modulators, such as the EFA protecting anti-oxidants, vitamins A, C, E and selenium as well as dietary fiber which acts as the prime regulator of fat metabolism in the gut, thus, indirectly determining systemic EFA requirements. In addition,

Abbreviations: Linoleic acid, LA = 18:2w>6; y -linolenic acid, GLA = 18:3w6; dihomogamma-linolenic acid, DGLA = 20:3w6; arachidonic acid, AA - 20:4w6; a-linolenic acid, ALA = 18:3UJ3; eicosapentaenoic acid, EPA = 20:5u;3; docosahexaenoic acid, DHA = 22:6w3.

various dietary antinutrients, such as saturated fat, cholesterol and sugar, interfere with these co-nutrients in the Fundamental Reaction of Nutrition:

firming findings of the British fiber theorists (22). Selenium intake is low, of interest since veterinarians find that livestock suffers from a widespread selenium deficiency of uncertain

#### → Structural & regulatory proteins

EAA (10)-B vitamin enzymes co- & anti-modulators EFA(w6/w3)Structural & regulatory lipids: (PG 1,2/3,4; chol- & glyc-EFA esters)

Since the co- and anti-nutrients form a coherent reaction schema, there can be four limiting pellagra-beriberiform disease variants, namely, substrate, catalyst cofactor, modulator, anti-nutrient and their combinations, e.g., vitamin B deficiency schizophrenia, irritable bowel syndrome, arthritis, etc.; EFA substrate deficiency schizophrenia, irritable bowel syndrome, etc.; modulator schizophrenia, etc.; anti-nutrient schizophrenia, etc.; and the combined forms. Because the lipid products constitute a body-wide local tissue regulatory system — the tophormones — gene-dependent variations will produce a complex idiosyncratic or statistical illness structure — MDS will be identifiable not by examining the 'patient as a whole' but only by a statistical or epidemiological diagnosis.

## **Evidence for Dietary Deviations from the Traditional Standard**

Evidence for significant systematic damage to the modern diet is presented, first, in Table 1 which gives the nutrient values of a prototypical neomodern daily diet (post 1965, with relatively high w6 intake) compared with the same diet in its traditional indigenous and unprocessed form, corrected for changes in national dietary patterns over the past 100 years (e.g., 250% increase in sugar consumption) to approximate the prototypical temperate and cold climate diet of 1850 or earlier.

The Table shows that dietary availability of the more fluid cold climate w3-EFA in the neomodern diet is only 20% of the traditional diet (mainly as the result of increased consumption of w3 deficient warm climate oils, hydrogenation, decreased fish consumption and loss of cereal germ by machine milling) while more viscous warm climate w6 availability has changed little. Severe dietary fiber deficiency of about 75% exists, conorigin (21). Sugar consumption has risen 250%. Fatty acid isomer consumption has soared 2500% (hydrogenation) and from data on competitive enzyme inhibition (23) and isomer consumption levels, I calculate that 20-40% of EFA metabolic enzyme activity is blocked in the average person in the U.S., even as we reduce w3-EFA availability by 80% and adversely affect its processing through co-nutrient deficiencies of fiber, selenium and B vitamins as well as interfering antinutrient increases. The intake of cholesterol, saturated fat and B vitamins varies widely but the first two are significantly increased while vitamin B consumption may be reduced as much as 50% below the RDA in 20% of the population, mainly those consuming high levels of sugar (24).

Studies show that most of these nutritional deviations interfere synergistically with EFA utilization (25), thus effectively increasing EFA requirement, even as w3-EFA availability declines. Consequently, the effective w3-EFA equivalent deficiency is greatly in excess of the 80% dietary depletion. Evidence also indicates that lack of exercise in the face of an atherogenic diet in primates contributes significantly to atherosclerosis (26). In addition, the work of Selve (27) and others shows that stress also acts through the EFA-steroid system.

#### **Dietary Deviations and Disease Correlations**

The results of Table 1 are supported by analyses of national dietary consumption patterns and related studies shown in Table 2, which compare w3 and w6-EFA consumption in individuals, animal colonies and nations having a relatively low incidence of modernization diseases with others having reduced w3-EFA consumption and a high incidence of heart disease, schizophrenia, arthritis, phrynoderma, polyneuropathies,

Meniere's disease and other illnesses (see Table 2 references).

The general results of Table 2 also indicate that the intake of w3-EFA has been reduced about 80% in the unhealthy state compared to the healthy state while w6 consumption is unchanged. Therefore, the Table 1 data are entered into Table 2 as Study 1.

Study 2 of Table 2 compared the w3 and w6-EFA consumption in German occupied Norway. During this 2 year period the incidence of cancer, heart disease and schizophrenia all plummeted by a remarkable 40-50% and then rose again shortly afterwards, data collection being constant according to the authors of these studies. During this time, when the health improved, w6-EFA consumption was again unchanged while w3 consumption increased 5 fold. However, there was also a general reversion to indigenous unprocessed food during the occupation, implying that other dietary cofactors such as fiber, sugar and beef intake also normalized (see Table 1 and below).

Study 3 compared the EFA consumption in controls and children having phrynoderma (literally, 'frog skin') in *warm climate* India, where the differential requirement for *w*3 relative to *w*6-EFA is probably considerably less. In this case, which involved near starvation, there was a limitation on EFA intake of both types among the ill children while the healthy children consumed about twice as much of both families. The phrynoderma cleared over 4-6 months on linseed oil and other polyunsaturate supplements.

Study 4 gives the EFA consumption of Japanese in Japan versus the U.S. around 1960, before there was as much modernization of the diet in Japan as today. The incidence of bowel cancer and heart disease was found to be much higher in Japanese Americans and equaled that of U.S. citizens generally (1,2). The *w*6-EFA consumption was, again, about the same in the two Japanese groups but the *w*3 consumption of Japanese Americans was only 20% of the consumption in Japan. Meniere's and other diseases increasing in Japan following WWII are associated by Japanese investigators with increased fat consumption as dietary habits Westernize (20, 21).

Study 5 compared the EFA consumption of Eskimos and Danes, the former having a much lower incidence of heart disease and osteoarthritis and about twice the Omega-3 EFA intake. However, as a cross-racial study, interpretive caution is necessary.

Study 6 suggested that Omega-3 EFA consumption in Britain is low and recommended supplementation with fish oils.

Study 7 examined a child with an abdominal gunshot wound placed on total parenteral nutrition using low w3-EFA containing safflower oil (< 0.5% w3, 60% w6) as sole EFA source. Over 4 months she developed a variety of severe neuropathies which were rapidly corrected by substituting high w3- EFA containing soybean oil (10% w3, 60% w6) for the safflower oil. EFA serum profile studies established that her acute illnesses were the *specific* result of an w3-EFA deficiency which was *specifically* cured by w3-EFA supplementation with *non-hydrogenated* soybean oil.

Study 8 raised a colony of 6 Capuchins from infancy on a standard laboratory diet which would be regarded as healthy by all modern nutritionists using corn oil as sole EFA source (0.5% w3, 60% w6). By age 2, all the animals developed (1) drying and scaling Dermatitis and alopecia, (2) two developed intractable Diarrhea and (3) two developed a vicious genital selfmutilating Dementia, which would be called the "van Gogh" syndrome in psychiatry, where it is seen in both schizophrenia and mental retardation. While no single animal in this study showed all three of the classical 3 Ds of pellagra, namely Dermatitis, Diarrhea and Dementia, the colony as a whole did. This shows the importance of making what may be called a statistical, epidemiological or demographic diagnosis, which goes entirely beyond the medical adage to 'study the patient as a whole', since a true idiosyncratic illness can only be diagnosed by studying the group as a whole. In fact, the diagnosis was missed even by the authors of this study. Except for the selfmutilators, which were put out of their misery, all the animals recovered within a few months on adding linseed oil supplements (60% w3, 20% w6).

Study 9 raised rats over a full life-cycle on soybean oil as sole EFA source (10% w3, 60% w6). As adults, they showed significantly better maze performance than did life-time controls on safflower oil (< 0.5 % w3, 60% w6). Compared with the prominent, even catastrophic, illnesses and their relatively

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rapid development in the primates in Study 8, this long term result in rats plus other evidence (21) suggests that primates have a much greater dependence on *w*3-EFA than do sub-primates. In fact, *w*3 EFA can be the dominant EFA in brain. (RDA *w*3 and *w*6-EFA in temperate climates is about 1-2% and 6-8% of calories).

## Malnutritional Synergy

These findings suggest that a widespread fat-centered substrate and mixed pellagraberiberiform disease resulting from a synergistic malnutrition could account for the modernization diseases. According to the Fundamental Reaction of Nutrition, we can form a synergistic malnutritional index:

The synergistic malnutritional index,  $l_{sm} = (1 + I \ bjVj)$ , 7r(1 - aOxj = optimize; where x j a n d y j, i, j = 1, 2, 3, ..., are the sets of co-and anti-nutrient intake levels, respectively, and the 0 < a j < 1 and b; > 1 are their respective synergy*vectors*.

For example, evidence indicates that omega-3 EFA, dietary fiber and niacin supplements independently lower serum fats (28-30) while both human and animal studies (31) show that skin and musculoskeletal disorders can be ameliorated by supplements of either EFA or B vitamins, the best results being obtained from combined synergistic therapy.

Omega-3 EFA also suppress tumorogenesis in lower animals while Omega-6 EFA seem to be required for carcinogenic transformation. Dietary linseed oil supplements dramatically reduced the incidence of liver tumors in rats given oral carcinogens while marine dietary oil supplements suppress mammary tumorogenesis in rats (32). Other studies (33) suggest that mammary tumor enhancement is more likely to be related to Omega-6 prostaglandins than Omega-3 and may even require Omega-6 EFA. At the same time, mammary tumor suppression is reported with dietary supplements of the EFA-preserving antioxidants, vitamin E and selenium (34). Conversely, tumor enhancement occurs with selenium deficiency (35) while both selenium and EFA supplements enhance immunocompetence (36,37). Just as fats uncovered by antioxidants enhance mammary tumorogenesis, so fats uncovered by fiber enhance colon cancer (38). Of course, w3-EFA are now held to be important in normalizing cardiovascular physiology (1).

These findings suggest a resolution to the conflicting recommendation for increasing and decreasing polyunsaturate consumption by the Heart (1) and Cancer (2) Panels, respectively. By increasing w3 EFA and decreasing w-6 EFA consumption, the public can at once lower total polyunsaturates while increasing their efficacy for the prevention and treatment of *both* heart disease and the fat dependent cancers and, presumably, all the other symptomatic co-diseases comprising the MDS.

## **The Clinical Picture**

Hoffer and I have independently observed (20, 21, 39) that today's schizophrenia cannot be distinguished from the dementia of pellagra. Moreover, when today's diseases are viewed clinically *as a group*, the collective picture strongly resembles chronic mixtures of the classical B vitamin deficiency diseases, especially pellagra and beriberi, which often do not show pathognomonic signs such as Casal's collar or all 3D's in any one patient.

A review (20,21) of the classical literature of about 1900, shows that pellagra routinely produced symptomatic problems which are clinically indistinguishable from today's major illnesses (20,21). Thus, today's schizophrenia, manic depression and neuroses cannot be distinguished clinically from the pellagrous 'dementias' of 1900.

The diarrhea of classical pellagra actually consisted of (1) diarrhea *or* constipation *or* their alternation unaccompanied by other findings except for (2) distention and grumbling and (3) discomfort, i.e., it was 'functional'. Any two of these three problems now constitute the diagnostic criteria for what is now called 'irritable bowel syndrome', 'spastic colon' or 'mucous colitis', the single most prominent disease seen by gastroenterologists, occupying fully 30% of their practices today.

The chronic form of the 'dermatitis' of classical pellagra included dandruff and other drying and scaling xerodermatoses, which are so common today that physicians and dandruff advertisers alike dismiss them as part of the normal human condition, although any veterinarian encountering an animal colony with a similar incidence of problems would know he had a nutritional crisis.

Pellagra also commonly produces tinnitus, fatigue, immune and other idiosyncratic problems while beriberi produces everything from heart disease to its own variations of the psychoses and neuropathies.

## A Pilot Clinical Test of the Fatty Substrate Pellagra-Beriberi Hypothesis

I have reported elsewhere on the impressive response during a 3 year clinical pilot study of 44 chronic, steady-baseline, previously nonresponding nonplacebo reactors having a large variety of illnesses treated by w3-EFA supplementation using food grade linseed oil (LSO), which, unlike fish oils, influences all desaturases and, prior to WWII, was a traditional Nordic cooking oil (60% w3, 20% w6) (20,21). Conditions permitting, safflower oil (<0.5% w3, 70% 106) was used as a control, many cases being repeatedly cycled between these two oils over months.

There has been striking concomitant amelioration of a large variety of major mental and physical symptomatic ailments as the specific result of adding linseed oil to previously incomplete supplementation regimens including dandruff, arthritis, irritable bowel syndrome, tinnitus, sleeping disturbances, chronic infections, benign prostatic hypertrophy, allergies both food and airborne, discoid lupus, neuralgias, normalization of blood pressure in both directions and others (20, 21). Immune system correction has been particularly striking along with reduced cold sensitivity and easier weight control. Severe long term but remitting (brain competent) schizophrenia and manic-depressive cases have responded impressively. Evidently, therefore, these are all expressions of a highly pleomorphic and idiosyncratic statistical syndrome, even as with classical pellagra and beriberi.

## **Therapeutic Recommendations**

The risk costs of inaction make it imprudent to wait for the niceties of a multimillion dollar 10-year government sponsored study before making the following *no-risk* public recommendations:

(1) Consume more w3 and less w6-EFA by reducing beef (keep it lean) and increasing

consumption of northern beans (common. red, kidney, navy, pinto) and vegetables cooked al dente, chicken, (traditional) pork and 1/2 to 1 lb./week of fatty fish (blue, albacore tuna. haddock. rock, mullet, mussels, mackerel, salmon, trout, oysters). Try kippered herring or smoked salmon (lox) for breakfast (Japanese per capita fish consumption is 6 times ours). A teaspoon of flaxseed (30% linseed oil) spread over cereal is a dietary tradition in Northern Europe as well as classical Greece and Rome while wheat germ (as breakfast cereal) is also very high in w3-EFA. Keep total fats to 35%, w6 about 6-8% and w3-EFA about 1-2% of calories and control use of partially hydrogenated food oils and margarines. In the North, at least, use indigenous *nonhydrogenated* soybean, walnut. chestnut, hazelnut and linseed wheat germ, oils in place of southern cottonseed, sunflower, peanut, safflower, corn and olive oils or products containing them. Return to butter, used sparingly. Refractory cases may be overwhelmed by covert food antigens, requiring eliminationprovocation and chemical diets (Vivonex) properly supplemented per above. When using purified oils in tablespoon daily doses for therapeutic purposes, use supplements to replace stripped-out oil insoluble B and C selenium and possibly cysteinevitamins. methionine, all taken in divided doses with meals or, whenever possible, in time release form. The regimen is an updated form of the once common of cod liver oil supplements. For use prophylactic purposes take 1 tsp daily of linseed oil, cod liver oil or somatic fish oil. Because of vitamin A toxicity, avoid cod liver oil megadoses.

(2) Use unprocessed foods high in fiber, e.g., stone ground cereals labelled more than 2% fiber. Lower cholesterol and sugar in take. Find the ratio of meat to vegetables that makes one feel best. Take a routine premeal in mixed fruitvegetable-cereal fiber cocktail: 1 part hydrophilic gel fiber (serum cholesterol lowering) and 4 parts miller's bran (better stool bulking) plus yogurt to suit (seeds the gut with favorable aerobes). Adjust amount (ca. 1 tbsp) at each routine meal to obtain the normal floating 'odorless coil' BM. Fiber also suppresses appetite and, with wheat germ, reconstitutes today's refined wheat products.

(3) Because of strong coupling of exercise to fat metabolism and atheroma formation

(32), in the absence of health problems, one should *work up over months* to mild continuous always *enjoyable* aerobic exercise, 1/2 to 1 hour every other day allowing talk over sustained breathlessness and never pushed to fatigue. Vigorous walking, walk-jogging, swimming, cycling, aerobic dancing etc. are most practical. Intermittent sports do not produce the cardiovascular training effect, which begins only after 10 minutes of continuous moderate breathlessness.

(4) Because of strong coupling of stress to steroid-fat metabolism, via Selye's adaptation syndrome, try to optimize this life-style factor to obtain the supersynergistic effect. Details of the supplement regimen for active illness have been given elsewhere (20,21).

(5) For illness and a nominal 150 lbs add to the above 1 tsp linseed oil at each meal, working up to as much as 1 tblsp tid or the toleration limit; a 1-a-day multivit/multimin (with selenium); 1-2 gm calcium. Continue for 4-6 months then taper off to zero or maintenance dose or try fish oil concentrate like Maxepa. MegaEFA may produce a beneficial fat, cholesterol and isomer flush as well as restore normal w3 EFA levels. Long term oil toxicity effects often resemble the original deficiency problems and also include sleepiness, general muscle aching or tendonitis, superficial peeling of finger tips or roughening of heel skin or knuckles which may be compensated by increased vitamin E, selenium or calcium intake. These problems can also be caused, synergistically, by excesses of the other essential nutrients. Corresponding to the symmetrical (bell-shaped) deficiency-toxicity picture, treatment often normalizes BP, serum fats, etc. from both directions. Travel versions are available as LSO capsules, bran-yogurt tablets and bars.

## **Discussion and Recommendations**

Medically oriented, orthodox nutritionists can be defined as those who say that we are the best fed people in history and that the function of nutrition is to support primary medical treatment. Reform nutritionists are those who say that while we are the best fed people in history we are also the most malnourished and that the function of nutrition is to provide the primary treatment for today's major illnesses, because they are mainly of nutritional origin, the function of medicine being to supply crisisintervention care in support of nutritional therapy.

Starting in the early 1950's, three lines of reform nutrition research each separately indicated that the modern diet is seriously distorted and may account for many or most of our major illnesses in modernized societies. C.L. Cleave and the British fiber theorists (22) demonstrated that dietary fiber is an essential nutrient, that it is severely deficient in the regular diet and that it is causally related to various bowel and bowelrelated systemic illnesses. A. Hoffer and his colleagues (39) showed that many of our major illnesses, including schizophrenia, can be viewed as vitamin-resistant forms of pellagra and beriberi related to dietary modification together with genetic susceptibility. H.M. Sinclair (40) provided evidence that w3-EFA deficiencies and excess saturated fat consumption are related to cardiovascular disease, a view now augmented by the Heart and Cancer panels, both of which find dietary fats to be the primary links to illnesses as disparate as heart disease and certain major cancers (1,2).

During this same time, there has been growing recognition by independent epidemiologists that a wide variety of the major illnesses of modernized societies are new or newly prominent and are related to dietary factors (41) rather than to increased diagnostic and therapeutic successes of modern medicine, uncovering heretofore obscure diseases.

The analysis of national food consumption data presented here shows that 70% of our food is now processed or exotic and that this has seriously distorted every essential nutrient family while at the same time significantly increasing the anti-nutrient load.

When analyzed biochemically, all these findings form a coherent biochemical and clinical picture, because all 50 of our essential conutrients, as well as the anti-nutrients, are part of a coherent biochemical reaction system - the fundamental reaction of nutrition - which constitutes the front end of our intermediary metabolism which has been delegated evolutionarily to lower sectors of the food chain. It follows that people in modernized societies today, living on a massively distorted dietary base, are at risk for a new kind of subtle but deadly unrecognized synergistic malnutrition ---the

modern malnutrition — now evidently constituting our primary public health hazard.

Biochemical analysis further indicates that these multiple synergistic dietary modifications can interact synergistically to produce a new diagnostic variant of the classical B vitamin deficiency diseases which once decimated entire populations in both the East and West during the 19th century. In particular, I propose that the traditional illnesses be redefined as catalytic B vitamin type pellagra and beriberi and that we now recognize, in addition, a fat-centered substrate and mixed — deficiency-toxicity pellagra-beriberi variant of the classical forms, these being the likely cause of what should be recognized as true Modernization Disease Syndrome, MDS, now accounting for the bulk of illness.

In fact, the synergy is well established in one small area by the fact that supplements from the three pioneering reform groups cited above ---fiber. w-3 EFA and niacin — can each alone reduce elevated cholesterol. serum Consequently, concurrent deficiencies, which are now common as the result of the modern dietary distortions, can account for the prevalence of this problem and, conversely, combined supplements of all three essential nutrients will provide the general solution to the problem provided we also correct the distorted modern diet causing the problem in the first place.

Therefore, contrary to present teaching, Goldberger and Takaki-Eijkman may have the single solved only food factor nonsynergistic B vitamin-sensitive, *catalytic* form of pellagra and beriberi while leaving unrecognized and uncorrected the now dominant vitamin-resistant 'Hoffer pellagra-beriberi' variant resulting from a synergistic malnutrition caused by multiple interacting food modifications which presents as a fatty substrate or compound (substrate-catalystmodulator-anti-nutrient) pellagra-beriberiform illness, the well known idiosyn-cracy and pleomorphicity of such illnesses misleading the medical profession into thinking it is dealing with dozens of new unrelated illnesses requiring dozens of new specialities. Because some estimates make the modernization disease group the dominant health problem in modernized societies, these matters should be tested promptly by conducting, under national

auspices, an entirely new

kind of controlled multifactorial diagnostic and synergistic nutritional therapeutic *inter-disease concordance study* in *man*, which, for the first time, cuts across specialty and even extraclinical boundaries. There should also be conducted primate life-cycle tests of the *synergistic* safety and efficacy of the entire food base, placing one half of a colony of monkeys on the same *total supermarket diet and lifestyle* on which dietarily modernized man lives. Given the results of Table 2, we may freely predict that the experimental animals will develop all the modern diseases in a few years and recover under nutritional therapy, whenever the damage is not irreversible.

We have been through all this once before, when orthodox nutritionists and the medical profession tolerated massive food refining and other dietary modifications without competent test, the result of which was the original B vitamin nutritional plagues of 1800-1900. Because we have never outlawed the original refining practices and, indeed, have extended them in many new ways, we are evidently going through a related problem all over again and must conclude that these recurring problems are the result of deep structural problems in the organization of health care, research and regulation.

The origin of the problem lies in the fact that health care is monopolized by a high tech, high cost, high risk, crisis-intervention oriented allomedical profession which has an economic as well as a chauvinistic conflict of interest with orthomedical practice respect to using orthotherapy based on orthophar-maceuticals, i.e., our primary pharmacology consisting of natural products including especially essential nutrients. While orthodox allomedicine should have a monopoly technical on dangerous allopharmaceutical agents and other high tech methods, it should not monopolize, as it does, the entire domain of health practice, health regulation and health research, for this is rather like putting the fox in charge of the chicken coop in view of the fact that the bulk of illnesses today is not medical in nature at all, except by default, but in the first instance, involves lifestyle factors problems of the kitchen and the farm and not the clinic and laboratory.

To correct this unjustified monopoly, we should, as I have suggested elsewhere (42)

## TABLE 1 Food Damage Report For 1983: Ancestral and Neo-Modern Diets Compared for Fatty Acids and Other Components

	Cal. Serving	Ancestral w3-	E¥A w	6- EFA	NEFA	NeoModern u;	3-EFA	w6-E¥	NEF
	(grams)	Food	(gm)	(gm)	(gm)	Food	(gm)	(gm)	A (gm)
600	3 cups (200)	Stone gnd	0.2	2.5	2.0	1.5 cups refnd	0.0	0.0	<u>(gill)</u> 0.0
000	5 Cups (200)	wheat	0.2	2.0	2.0	wheat	0.0	0.0	0.0
250	3 serv	Fruit/grns	0.0	0.0	0.0	Fruit/grns	0.0	0.0	0.0
80	1 serv	1 egg	0.0	1.3	8.0	1 egg	0.0	1.3	8.0
100	1 tblsp (15)	Butter	0.2	0.7	14.0	Margarine	0.2	4.0	11.0
100	1 serv (80)	Tunafish	1.1	0.1	2.7	Hamburger (60	0.2	0.4	14.0
						gm)			
200	1 serv (80)	Pork/Chick	0.2	2.0	20.0	Beefsteak (100)	0.4	0.8	26.0
	. ,	(?hyd feed)				gm			
150	1 cup	Common				C			
	(115)	bean (navy,	0.6	0.3	0.3	Asparagus (etc.)	0.0	0.0	0.0
		kid.)							
150	1/4 cup (35)	Walnuts	1.5	9.0	5.0	Peanuts	0.1	3.0	8.0
	• · ·	(English)				(Cashews)			
150	2 tblsp (30)	Honey	0.0	0.0	0.0	Sugar 4tb (60	0.0	0.0	0.0
	-	-				gm)			
220	2 tblsp	Soybean oil	2.0	16.0	12.0	Cottonseed	0.1	15.0	15.0
	(30)	(Lard)	(0.4)	(3.5) (	25.0)	(hyd soy)	(0.7)	(11.4)	(18.0
							1.0	24.5	)
2000			5.8	31.9	64.0				82.0
	-								
Total		01.7 gm = $43$				107.5  gm =	46% of		
w3-E	FA	5.8  gm = 2.59		_		1.0  gm =	0.4%	of cal.	
			7% of oil)				(1.0%		
LU6-I	EFA =	31.9  gm = 14				24.5  gm =	10% of		
			1% of oil)				(23% c	of oil)	
w3I	6	0.2				0.04			
W Totol	EEA	27.7 am	_			25.5 cm	_		
Total		37.7 gm				25.5 gm		-	
	er Index =	0.4% of EFA	-			10.0% of EFA (h	<u>y</u> d	_soy)	
Vitan		60.0 mg				50.0 mg			
Selen		60.0 meg	_			75.0 meg	_		
NEFA		64.0 gm				82.0 gm			
Sat. F		25.0 gm				34.0 gm			
Chole		80.0 mg				520.0 mg			
	amins $=$ h	-				variable, low			
Fiber		25.0gm	-			8.0 gm			
Salt		2.0 gm			1.1	10.0 gm			C.
<b>Table I.</b> Prototypical n		modern diet on the right and its unadultera				-			
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pared for all food vah jes emphasizing EFA.

References: *EFA & Isomers:* Exler, J, et al; J. Am. Dietetic Assoc. 71:518, 1977; Carpenter DL, et al; JAOCS. 53:713, 1976; Smith LM et al, JAOC. 55:257, 1978; Kirschman JD, Nutr. Almanac, McGraw-Hill, 1979. *Salt:* Goodhart RS and Shils ME, Mod. Nutr. In Health and Disease, Lea & Febiger, 1973 (1011-14). *Statistics:* Historical Statistics, U.S. Consumer expendit. patterns, 1949-1970, (829-31); Ann Rpt Nat Fd Survey Comm, Hsehold fd consumption, 1973-, Gov't Stationary Office, London.

Study		u;3-EFA	w6- $EF$ \ t		
	"Healthy" (% cal)	''Unhealthy'' (% cal)	''Healthy'' (% cal)	''Unhealthy'' (% cal)	
1. Traditional vs Neo-modern Diet (1) (Table 1)	2%	0.4%	14%	10%	
2. Norwegian Food Survey (2) (peacetime vs wartime)	>1%	0.4%	8%	5%	
Norwegian Linseed Study (3)	2%	0.4%	8%	5%	
3. Children-India (Health (vs phrynoderma)(4)	1%	0.5%	2%	1%	
<ul><li>4. Jap./Am. Food Survey (5)</li><li>5. Eskimo/Dane Food</li></ul>	2%*	0.4 %	6%	6%	
Survey (6) 6. British Food Survey (7)	2%*	1.0% < 1.0%	2%	5% <7%	
7. Parenteral child (8)	0.6%**	0.1%	4%	6%	
8. Capuchin Study (Linseed oil vs corn oil)(9)	2%	0.2%	10%	15%	
9. Rat Study (Brain func) (Soy vs safflower oil) (10)	1-2%	0.1 %	10%	10%	
Therefore: MDR (w3-EFA)	= 1% of cal	ories; RDA (w3	-EFA) = 2% of c		
MDR (w6-EFA)	= 2-3% of cal	lories; RDA <sub>(w6-E</sub>	EFA) = 5-10%  of	f calories	

#### **TABLE 2 Estimated Average MDRS and RDAs for the EFA**

Table 2. Various studies making estimates of national or individual consumption of Omega-3 and Omega-6 EFA for the healthy vs the unhealthy state.

These are upper limits, since w6 may be lost as isomers on hydrogenation. t

\* Japanese and Eskimo maritime diets are high in 5w3 and 6w3, which are, in some ways, more potent than 3u;3. Japanese were assumed to take 20% of calories as fat, otherwise 40% was assumed in all calculations when authors did not provide °?o of calories.

\*\* This is a parenteral value and does not allow for losses in the gut or intake of normal nutrients, which probably increase EFA requirements.

#### References

- 1. Rudin, DO, The Omega-3 Phenomenon: the Nutritional Breakthrough of the 80's. Rawson-Macmillan, July, 1987.
- 2. Natvig, H et al. A controlled study of the effect of linolenic acid on the incidence of coronary artery disease. Scand. J. Clin. Lab. Invest. 22(suppl 5): 1-20, 1968. Dohan, FC, Wartime changes in hospital admissions for schizophrenia. Acta Psychiat. Scand. 42:1-23, 1966.
- 3. Owren, PA, Coronary thrombosis: Its mechanism and possible prevention by linolenic acid. Ann. Int. Med. 65.167-84, 1965.
- 4. Bagchi, K, Haider, K and Chowdhury, SR, The etiology of phrynoderma. Am. J. Clin. Nutr. 7:251-8, 1959.
- 5. Insull, W Jr., et al. Studies of Japanese and American men: Comparisons of fatty acid compositions of adipose tissue. 7. Clin. Invest. 45:1313-27, 1969.
- 6. Bang, HO, et al, The composition of food consumed by Greenland Eskimos. Acta Med. Scand. 200: 69-73, 1976.
- 7. Reed, SA, Dietary source of u)3 eicosapentaenoic acid. Lancet 2:739-40, 1979. Annual Report of the National Food Survey Committee, Household food consumption and expenditure: 1973, etc., Her Majesty's Stationary Office. London.
- 8. Holman, RT, Johnson, S and Hatch, TF, A case of human linolenic acid deficiency involving neurological abnormalities. Am. J. Clin. Nutr. 35:617-23, 1982.
- 9. Fiennes, RN, Sinclair A J and Crawford, MA, Essential fatty acid studies in primates: Linolenic acid requirements of Capuchins. J. Med. Primal. 2:155-69, 1973.
- 10. Lamptey, MS and Walker, BL, A possible essential role for dietary linolenic acid in the development of the young rat. J. Nutr. 706:86-93, 1976.

create a new primary health care profession specializing in low cost, low tech, low risk orthomedical treatment and prevention, using agents and procedures under control of the patient. The motto of the primary health care professional — the modern hygienist — should be 'Primary health care keeps the doctor away'. For exactly the same reason, the FDA should be legislatively divided into the FA and DA, with reform and not orthodox nutritionists or physicians heading the Food Administration. Similarly, NIH should be divided into NIH-Medical and NIH-Nutrition, the enabling acts of these new institutions indicating that they have been separated to provide a competitive twocomponent health care industry, so as to end the current allomedical monopoly, which has failed to provide reasonable health care at reasonable cost for the many modern illnesses which are now putting our people and our nation at risk.

#### **Text References**

- Grobstein, C, NAS/NRC, Diet, Nutrition and Cancer. *National Academy Press* (2101 Constitution Avenue, NW, Washington, D.C. 20418), 1982.
- 2. Grundy, SM, et al., Rationale of the diet-heart statement of the American Heart Association. Report of the Nutrition Committee, American Heart Assoc. *Circulation*, 55/839A-54A, April, 1982.
- 3. Lucas, CP and Power, L, Dietary fat aggravates active rheumatoid arthritis. *Clinical Res.* 29:1'54A, 1981.
- 4. Goldyne, ME and Stobo, JD, Im-munoregulatory role of prostaglandins and related lipids. *CRC Critical Reviews in Immunology*, 2:189-223, 1981.
- 5. Editorial, Prostaglandins and immunity. *Lancet* 1:24-5, 1981.
- 6. Horrobin, DF, *The Prostaglandins:* Physiology, Pharmacology and Clinical Aspects. Eden, 1978 (p. 242).
- 7. Weissmann, G, Release of mediators of inflammation. *Prost. & Thera.6*{2):\-6, 1981.
- 8. Meade, CJ and Mertin, J, Fatty acids & immunity. Adv. Lipid Res. 16:127-65, 1978.
- 9. Ebihara, K et al. Effect of Konjac mannan on plasma glucose and insulin responses in young men. *Nutr. Rep. Int'l.* 23:517-83, 1981.
- Honigman, G, et al., Influence of diet rich in linolenic acid in diabetics. *Diabetol.* 23:175, 1982.
- 11. Houtsmuller, AJ, et al., Favorable influence of

linoleic acid on the progression of diabetic microand macroangiopathy. *Nutr. Metab. 1* (Supp24):105-18, 1980.

- 12. Dodson, PM, et al., High-fibre and low-fat diets in diabetes mellitus. Br. J. Nutr. 46:289-94, 1981.
- Singer, P, et al., Decrease of eicosapentaenoic acid in fatty liver of diabetic subjects. *Prost. Med.* 5:183-200, 1980.
- 14. Hansen, AE, et al., Eczema & essential fatty acid. *Am. J. Dis. Child.* 73:1-18, 1947.
- 15. Bower, BO and Newsholme, EA, Treatment of idiopathic polyneuritis by a poly-unsaturated fatty acid *diet.Lancet* 7:583-5, 1978.
- 16. Lamptey, MS and Walker, BL, A possible essential role for dietary linolenic acid in the development of the young rat. *J. Nutr.* 106:86-93, 1976.
- Fiennes, RN, Sinclair, AJ and Crawford, MA, Essential fatty acid studies in primates: Linolenic acid requirement of Capuchins. /. *Med. Primat.* 2:155-69, 1973.
- 18. Obi, FO and Nwanze, EAC, Fatty acid profiles in mental disease. /. Neurol. Sci. 43:447-54, 1979. Dohan, FC, et al., Schizophrenia: Variations in prevalence rates vs differences in cereal grain consumption. Soc. Biol. Psychiat. Meeting. Sept. 1980.Dohan, FC, Wartime changes in hospital admissions for schizophrenia. Acta Psychiatr. Scand. 42:1-23, 1966. Torrey, EF, Schizophrenia and Civilization, Aronson,1980. Page, LB, Damon, A and Moellering, RC, Antecedents of cardiovascular disease in six Solomon Islands societies. Circulation 49:1132-46, 1974.
- Chase, HP, et al., Intravenous linoleic acid supplementation in children with cystic fibrosis. *Pediat.* 64:207-13, 1979. Harper, TB, et al., Essential fatty acid deficiency in rabbit as a model of nutritional impairment in cystic fibrosis. *Am. Rev. Respir. Dis.* 126:540-7, 1982.
- Rudin, DO, The dominant diseases of moder nized societies as omega-3 essential fatty acid deficiency syndrome: Substrate beriberi. *Med. Hypotheses* 8:17-47, 1982.Rudin, DO, The major psychoses and neuroses as omega-3 esential fatty acid deficiency syndrome: Substrate pellagra. *Biol Psychiat.* 75(9):837-50, 1981.Rudin, DO, The three pellagras. *J. Orthomol. Psychiatry* /2(2):91-110, 1983.
- 21. Rudin DO, The Omega-3 Phenomenon: the Nutritional Breakthrough ofthe80's. Rawson-Macmillan, Summer, 1987.
- Cleave TL, The Saccharine Disease: The Master Disease of Our Time. Keats, New Canaan, CT, 1975.Heaton, KW, Bile Salts in Health and Disease. Churchill Livingstone, 1972. Reddy, BS, Dietary fiber and colon cancer.

Can. Med. Assoc. J. 123:850-6, 1980.

- 23. Hill, EG, et al., Perturbation of the metabolism of EFA by dietary partially hydrogenated vegetable oil. *PNAS* 79:953-7, 1982.Rosenthal, MD and Doloresco, MA, Effects of trans fatty acids on fatty acyl delta-5 desaturation by human skin fibroblasts. *Lipids* 19:869-74, 1984 Deschrijver, R and Privett OS, Energetic efficiency and mitochondrial function in rats fed trans fatty acids. *J. Nutrition* 114:1183-91, 1984
- 24. King, JC, et al., Evaluation and modification of the Basic Four Food Guide. J. Nutr. Ed. 10:21-9, 1978.Guthrie, HA and Scheer, JC, Nutritional adequacy of self-selected diets that satisfy the four food groups guide. J.Nutr.Ed. 13:46-9, 1981.
- 25. Ayala, S, Brenner, RR and Dumm, CG, Effect of polyunsaturated fatty acids of the alphalinolenic series on rat testicle development. Lipids 72:1017-24, 1977.Dumm, INT, et al, Effect of catecholamines and beta blockers on linoleic acid desaturation activity. Lipids 13:649-52, 1978. Dumm, INT et al, Comparative effect of glucagon, cAMP and epinephrine on desaturation and elongation of linoleic acid by rat liver microsomes. Lipids 77:833-6, 1976. Dumm, INT, Effect of glucocorticoids on the oxidative desaturation of fatty acids by rat liver microsomes. J. Lipid Res. 20:834-9. 1979.
- 26. Kramsch, DM, et al., Reduction of coronary artery atherosclerosis by moderate exercise in monkeys on an atherogenic diet. *NEJM* 305:1483-9, 1981.
- 27. Selye, H, *Stress in Health and Disease*. Butterworth, 1976.
- 28. Saynor R, Effects of w3 fatty acids on serum lipids. Lancet 2:696-1, 1984. Bronte-Stewart, B, et al., Effects of feeding different fats on serum cholesterol level. Lancet 7:521-7, 1956.Owren, PA, Coronary thrombosis: Its mechanism and possible prevention by linolenic acid. Ann. Int. Med. 65:167-84, 1965. Sinclair, HM, Valedictory address. Prog. Fd. Nutr. Sci. 4:131-4, 1980. Phillipson, BE, et al., Reduction of plasma lipids, lipoproteins and apoproteins by dietary fish oils in patients with hypertriglyceridemia. NEJM 312:1210-16, 1985. Kromhout, D, et al., The inverse relation between fish consumption and 20 year mortality from coronary heart diseases. NEJM 312:1205-9, 1985.
- 29. 29. Kritchevsky, D, Fiber, lipids and

arteriosclerosis. *Am J Clin Nutr.* 57:S65-S74, 1978. Terasawa, F, et al., Effects of Konjac flour on blood lipids in elderly subjects. *Eiyogaku Zasshi 37:23-8*, 1979.Kiriyama, S, et al., Hypocholesterolemic activity and molecular weight of konjac mannan. *Nutrition Reports International.* (5:231-6, 1972.

- Hunninghake, DB, Pharmacologic therapy for the hyperlipidemic patient. *Am. J. Med.* 74(5A):19-22, 1983.Hotz, W, Nicotinic acid and its derivatives: A short survey. *Adv. in Lipid Res.* 20:195-211, 1983.
- 31. Birch, TW, The relation between vitamin Bg and the unsaturated fatty acid factor. JBC 124:115-93, 1938.Quackenbush, FW, et al., Linoleic acid, Pyridoxine and pantothenic acid in rat dermatitis. J. Nutr. 24:225-34, 1942. Evans, HM and Lepkovsky, S, The sparing action of fat on vitamin B. JBC. 96:165-11, 1932.Bhat, KS and Belavady, B. Biochemical studies in phrvnoderma. Am. J. Clin. Nutr. 20:386-92, 1967. Ellis, J, et al., Clinical results of a crossover treatment with Pyridoxine and placebo of the carpal tunnel syndrome. Am. J. Clin. Nutr. 32:2040-6, 1979.Folkers, K, et al, Biochemical evidence for a deficiency of vitamin Bg in carpal tunnel syndrome. PNAS. 75:3410-12, 1978. Folkers, K, et al, Enzymology of the response of the carpal tunnel syndrome to riboflavin and to combined riboflavin and Pyridoxine. PNAS 81:1016-18, 1984.
- 32. Schramm, T, Effect of fatty acids on car cinogenesis. Wiss. Z. Humboldt. 11:184-5, 1962. Karmali, RA, et al., Effect of omega-3 fatty acids on growth of a rat mammary tumor. JNCI(U.S.) 73:451-61, 1984.
- Abraham, S and Hillyard, LA, Effect of dietary 18-carbon fatty acids on growth of transplantable mammary adenocarcinoma in mice. *JNCI* (U.S.; 77:601-5, 1983.Chan, PC, et al., Effects of different dietary fats on mammary carcinogenesis. *Cancer Res.*43:1019-83, 1983.
- 34. Horvath, PM and Ip, C, Synergistic effect of vitamin E and selenium in the chemoprevention of mammary carcinogenesis in rats. *Cancer Res.* 45:5335-41, 1983.Witting, CH, et al, The tumor-protective effect of selenium in an experimental model. J. Cancer Res. Clin. Oncol. 104:109-13, 1982. Young, EO and Milner JA, Influence of zinc and selenium intake on DMBA induced mammary tumors in rats. J. Nutr. 112:R30, 1982.
- 35. Ip, C and Sinha DK, Enhancement of mammary tumors by selenium deficiency in rats

with high polyunsaturated fat intake. Cancer Res.

- 36. Goldyne, ME and Stobo, JD, Immunoregulatory role of prostaglandins and related lipids. *CRC Critical Reviews in Immunology* 2:189-223, 1981.Hansen, HS, EFA supplemented diet increases renal excretion of PGE2 and water in EFA deficient rats. *Lipids* 7(5:849-54, 1981. Prickett, JD, et al., Dietary enrichment with polyunsaturated fatty acid eicosapentaenoic acid prevents proteinuria and prolongs survival in NZB x NZW Fl mice. *J. Clin. Invest.68:556-9*, 1981.
- 37. Spallholz, JE, Anti-inflammatory, immunologic and carcinostatic attributes of selenium in experimental animals. *Adv. Exp. Med. Biol.* 755:43-62, 1981.
- Trudel, JL, The fat/fiber antagonism in experimental colon carcinogenesis. *Surgery* (U.S.) 94:691-6, 1983.
- 39. Hoffer, A and Walker, M, Orthomolecular Nutrition.Kt<sup>^</sup>ts, New Canaan, CT, 1978.
- 40. Sinclair, HM, Valedictory address. Prog. Fd. Nutr. Sci. 4:131-4, 1980.
- 41. Hegstedt, DM, Testimony. Hearing, Senate Select Committee on the Relation of Nutrition to Disease. Congressional Record, 1976.
- 42. Rudin, DO, Call for a national conference to form the American Federation of Primary Health Care Societies. J. Orthomol. Psychiatry 13:(1): 25-26, 1984.

<sup>47:31-4,</sup> 1981.

