

# The FELIX Letter

NO. 17

A COMMENTARY ON NUTRITION

## A UNIFYING CONCEPT

To a child, everything on earth is easy to understand and simple to do . . . he has only to become a GROWN-UP! — at which time, of course, the great doors to wisdom and glowing deeds will swing open for him. Alas! by the time our child has become a certifiable adult, he will have noticed, with understandable grumpiness, that the magical doors have receded further and further in the distance and simplicity has given way to disquieting complication.

In the same way in science, the more doors to knowledge we open, the more labyrinthine are the corridors we find. The heralded accumulation of masses of data in molecular biology serves only to make the gleaning of significant truths from them a Sisyphean task. Doctors and scientists a few generations ago were afforded the luxury of being philosophers; today, any attempts to deliver comprehensive insights will land the deliverers smack in the middle of a controversial minefield, with a dozen specialists snapping pettishly at their heels! Neither specialization in medicine, nor fragmentation in research, is conducive to broad overviews.

## Pellagra Redefined

It thus takes courage or foolhardiness for a scientist to come up with an encompassing theory on the cause of modern illness totally at odds with conventional medicine and, further, to open to view a means of prevention/cure of singular simplicity. In the *Journal of Orthomolecular Psychiatry*, Donald O. Rudin, M.D.<sup>1</sup> reviews pellagra historically, not as a 'simple' illness caused by a deficiency of two essential nutrients (the B vitamin niacin and the amino acid tryptophan), but a complex disease embracing a large measure of human mental and physical suffering. Pellagra appeared in Europe shortly after corn planting was introduced from America around 1720, its medical name arising from the "rough skin" noted by afflicted Italian peasants. The disease occurred where corn was a

dietary mainstay and other protein sources negligible, and in the U.S. in 1915 alone, over 10,000 poorly fed people died of pellagra in the South, before belated recognition of its nutritional origins led to public-health measures and to its marked decline, finally, by the 1940's.

## "Three D's"

The "three D's" characterizing the disease were dermatitis, dementia, and diarrhea. "Dermatitis" consisted of a scarlet erythema resembling a severe wind-burn on hands, arms, face, legs, genitals, tongue and mouth; but just as commonly there was also a chronic drying and scaling dermatosis, often with an "aging spot" type of hyperpigmentation on areas most exposed to light. Dr. Rudin says this "strongly resembles our most common skin disease today, namely, drying and scaling skin syndromes, including dandruff; flaky paint dermatoses or 'dandruff' of shins and forearms . . .; follicular keratosis [permanent gooseflesh over upper arms, thighs, and buttock tips]; hand fissuring; aging pigmentation . . . and so on."



"Dementia" of classic pellagra could take the form of any of the major mental illnesses: schizophrenia, manic-depression, or severe neurosis, especially phobic. Rudin says: "These illnesses also account for the vast majority of cases seen today by psychiatrists and clinical psychologists."

The "diarrhea" of pellagra also included symptoms of alternating diarrhea/constipation, swelling, and gas, commonly diagnosed as "irritable bowel syndrome" or "spastic colon." Rudin notes: "Today, . . . irritable bowel syndrome is the single most common problem seen by gastroenterologists, accounting for fully one third of all cases entering their offices."

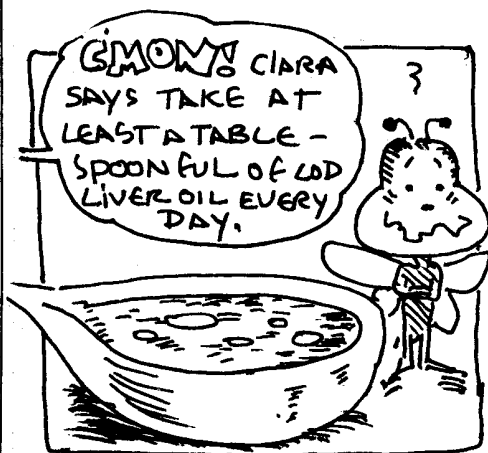
In addition to the "three D's", pellagra patients exhibited constant problems with fatigue, tinnitus, [head noises], and migraine-like headaches. The parallel to common modern symptoms is self-evident.

## A Missing Link?

In short, Rudin insists we are still a pellagra-ridden society, and the causes are still nutritional deficiencies, but the ratios of deficient nutrients have changed. Classic pellagra may have occurred in poor, corn-dependent cultures because corn lacks an essential vitamin (niacin) and amino acid (tryptophan). But corn also lacks one family of essential fatty acids, the Omega 3 [w3 EFAs], whose importance to human health has gone largely unrecognized. Today,

Rudin states, omnipresent "pellagraform" illnesses exist and stem primarily from a wide-spread deficiency of the w3 EFAs that have been systematically depleted from our diet and replaced by the other essential lipids, the w6 EFAs, incorrectly believed by a majority of researchers to be the ONLY unsaturated fats needed.\* [See FELIX LETTER #16.] Rudin defines the "Nutritional Missing Link" as a new awareness of the "special biomedical significance of the w3 EFAs for primates" (including man), possibly because of special needs for them in the highly developed primate/human brain. Since most experimental work on EFAs was performed on rats and mice, who usually didn't show gross abnormalities when w6 EFAs replaced w3 EFAs in their diets, the research picture has long been biased in this regard. A deficiency experiment, however, inadvertently performed on six infant Capuchin monkeys fed corn oil as the sole source of EFAs for two years (as part of a supposedly adequate diet), resulted in a whole gamut of pellagra-like symptoms, i.e., the three D's, including drying and scaling skin, intestinal inflammation, and severe psychotic-like behavior.

*Most of the monkeys recovered in a few months on supplements of w3-rich linseed oil.*



The preponderance in modern diets of w6 oils is mainly a 20th century phenomenon brought about by massive chemical refining and hydrogenation, and by selective breeding of plants to produce seeds with low alpha-linolenic acid content, in order to eliminate this w3 fatty acid which is apt to oxidize and produce "off flavors" in oils. For centuries, until World War II, linseed oil, a very rich source of alpha-linolenic, was in everyday use in Europe and Russia. In earlier days in the U.S., it also was commonly used in the home, but is now chiefly an industrial oil. The increased consumption of w6 oils and margarines in the home, in response to health pleas by the medical establishment and by advertisers, together with the hugely growing use of commercially packaged foods — baked or fried with w6 oils — has led to our present lop-sided situation. In addition, per person consumption of meat, which is low in w3 EFAs, rose in the U.S., while that of fish and seafood declined, thereby diminishing intake of the best source of the longer-chain w3s.



Rudin postulates three major types of pellagraform diseases, with many variations, depending on whether the dominant nutritional deficiency is of "substrate" (i.e., amino acid or essential fatty acid), B vitamins, or "modulators." Modulators are described as factors affecting the metabolizing of the amino acids and fatty acids into their ultimate cellular products, and include vitamin E, selenium, copper, zinc, and dietary fiber. "Non-dietary modulators" are exercise, climate, and psychological stress. Thus, the modern "w3 EFA-deficient" pellagra will present a different clinical picture in many respects from the classical "protein and vitamin-deficient" variety, yet the kinship will be discernible. The disease was always considered a complex one because its symptoms covered such a wide range of disorders. Rudin comments:

*As a disease, pellagra, along with beriberi, is the champion pleomorphic and idiosyncratic illness, as well as the prototypical "psychosomatic" disease. . . for pellagra can produce an awesome variety of different "unit" illnesses or co-diseases in the same or different people, depending on genetic and phenotypic factors. While psychiatrists like to say, "If one knows schizophrenia, one knows the whole of psychiatry," it is even more to the point to say, "If one knows the B vitamin deficiency diseases, one knows the whole of medicine, including psychiatry."*

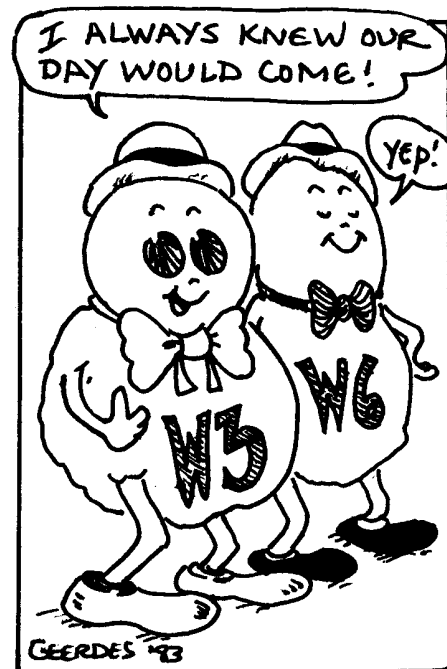
### Modern Beriberi?

With regard to the classic disease of beriberi, caused by loss of another B vitamin, thiamin or B<sub>1</sub>, Rudin proposes a similar set of modern-day variations depending on whether a deficiency of vitamin, modulators, or w3 lipids predominates. To the spectrum of illnesses produced by pellagra, a full-blown beriberi adds degeneration of nerves of the feet and legs, with pain and atrophy of the muscles;

Korsakoff's disease involving memory loss and psychosis; and severe congestive heart disease.

Rudin suggests that the *modern epidemic of cardiovascular illnesses might be a variant of classic beriberi*, one in which a deficiency of "lipid substrate," the w3 EFAs, dominates. Similarly, he suggests that the major disease of memory loss today, Alzheimer's psychosis, could be a "lipid substrate-dominant/vitamin B<sub>1</sub>-subordinate deficiency disease corresponding to Korsakoff's psychosis." He adds,

*In brief, we must ask if most of our specialists deal today mainly with lipid-substrate beriberi/pellagra, as they dealt one hundred years ago mainly with its inverse, B vitamin beriberi/pellagra. Of one thing we can be certain, that consideration of this idea will be as strongly resisted now as was the original idea that beriberi and pellagra were nutritional deficiency diseases.*



### The Great PGs

Why are the w3 and w6 fatty acids so important? For one thing, in about equal amounts they form the major unsaturated lipids of the human brain. For another, each EFA family is the precursor of its own multiple series of prostaglandins (PGs), the great regulatory molecules whose effects are so powerful that any nutrient deficiency short-circuiting their activity would have consequences at many different levels of functioning. For example, *thiamin and niacin are needed before the EFAs in food can be synthesized into PGs in our cells.* Thus, in their absence, the multiple breakdowns of normal functioning taking place in beriberi and pellagra may primarily reflect a failure of PG synthesis.

A different set of symptoms might be produced when there is a deficiency of the EFAs themselves, where no "substrate" is available for the vitamins to catalyze, but where, again, PG synthesis suffers.

Among the infinite functions known to be regulated by the PGs, which medical science is racing to explore, are those having to do with cardiovascular health (blood pressure, clotting time, vascular spasm, etc.), inflammation and repair, endocrine activity, and brain and neural activity. *The PGs are also fundamentally involved in regulation of the immune system*, whose correct functioning provides a major defense against cancer (as well as viral, bacterial, fungal, and parasitic infections), and also against auto-immune ("self-attacking") diseases such as rheumatoid arthritis.



The strength of Rudin's "nutritional missing link" concept becomes clearer as we perceive that (1) *normal PG biosynthesis is basic to mental and physical health*; (2) *PG biosynthesis cannot take place without vitamins, minerals, and BOTH families of essential fatty acids, all needing to be derived from the food we eat*; and (3) *widespread dietary depletion of w3 EFAs may be the unifying, underlying factor in modern diseases*.

### Food as Treatment

How can this newer awareness serve us in a practical way? Dr. Rudin has been working with a number of patients in a pilot study, using simple dietary changes to increase w3 EFA intake. Thirty-two (mentally normal) patients suffering from a variety of physical ailments of chronic duration have been taking 1 to 6 tablespoons daily of linseed oil, divided in two or three doses with meals. They also are asked to use only nonhydrogenated soy, wheat germ, walnut, chestnut, or linseed oil in cooking. Modest amounts of vitamin supplements are used, together with ample

vitamins C and E as anti-oxidants, and a premeal "cocktail" of either bran or psyllium seed with 1 to 3 tablespoons of plain yogurt in water.

Within less than a year on this program, presumably a rebalancing has taken place of w3/w6 ratios in tissues, and of the PGs derived from them, leading to impressive improvement in osteoarthritis (11 out of 11 patients!), migraine headaches, elevated ocular pressure, bursitis, chronic allergic conjunctivitis, tinnitus, irritable bowel, angina, cold sensitivity, and in every case, chronically dry, flaky skin and dandruff! At two years, the improvement continues.

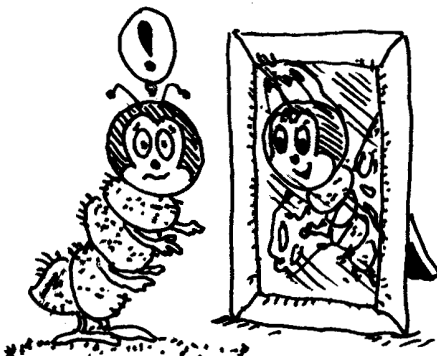
### Promise in Mental Illness

On a similar linseed oil program, eight of twelve mental patients suffering long-term schizophrenic, manic-depressive, or severe, incapacitating agoraphobia, have shown "moderate to very great improvement." (The four who did not respond were "non-remitting" cases, who, unlike the others, have never had even brief periods of remission once their illnesses began, suggesting possibly irreversible changes in brain tissue.<sup>2a, 2b, 2c</sup>)

As the program approaches two years, "the improvement continues, or even increases," Dr. Rudin writes. (He informed me over the telephone that a leading orthomolecular psychiatrist told him he has noted a "breakthrough", using linseed oil with patients, while their need for megavitamins appears to lessen.)

During their long illnesses, except for brief periods of remission, most of the 8 patients had not shown lasting amelioration of their symptoms on standard drug or megavitamin therapy. In light of this, the virtually continuous remission, for almost two years on linseed oil therapy, of one young woman, for example, who had suffered daily from violent self-mutilating, hallucinating, suicidal behavior for eight years, since she was 16, is indeed remarkable!

*Equally encouraging is the fact that the "pellagraform" physical symptoms of these patients, which included marked drying dermatoses, irritable bowel syndrome, tinnitus, migraine, and fatigue, have also ameliorated on linseed oil therapy, thus pointing to a unified basis for Rudin's hypothesis.* ■



## EVOLUTION

### & SCHIZOPHRENIA

Toiling in another vineyard, Dr. David Horrobin<sup>4</sup> has arrived at a theory on the etiology of schizophrenia that has an attractive tie-in to Rudin's "missing w3-EFA" hypothesis. He says, since schizophrenia occurs in all races without exception, it may have developed as an inheritable trait several million years ago, before separation of races took place. The trait persisted because it must have conferred an advantage on its carriers, at least at that time.

Sir Alister Hardy's "Aquatic Ape theory" of evolution suggests that, 4 to 8 million years ago, our primate ancestors were driven out of the interior of Africa to the shores of the sea by the intense heat and drought of that period, the survivors adopting a wholly or partially aquatic existence.\*\*

\*\* Based on this theory, certain puzzling human features become more logical in evolutionary terms, such as the absence of fur and the presence of subcutaneous fat, characteristics found in aquatic mammals but not in any primate but man; the presence of a physiological mechanism known as "bradycardia" (the diving reflex), found in diving mammals such as seals, whales and beavers, which slows the heart and reduces O<sub>2</sub> consumption during a dive, allowing a longer underwater stay — a trait absent in all land animals but ourselves; and the retention of head hair, and the development of large, fleshy, often pendulous breasts in lactating women — features which would have helped a baby to "hang on" while suckling its fur-less mother in the sea! S. S. Cunane<sup>5</sup> writes: "The only other non-human female with breasts comparable to the human female's is the aquatic group of sea cows."

### A Useful Mutation

Earlier, in terrestrial life, our primate ancestors had more abundant w6 EFAs than w3 EFAs in their foods; for this reason, their cellular enzyme systems preferentially metabolized the scarcer w3 EFAs to achieve a needed balance in PG production, which still holds true today. Their new aquatic/shore life would have drastically reduced their sources of w6 EFAs. (Marine plant and animal life is very high in w3 EFAs, particularly longer-chain highly unsaturated ones, but low in w6 EFAs.) Horrobin proposes *that a mutation, permitting more efficient utilization of the now scarce w6 EFAs, would confer survival value on its holders*. However, as the earth's climate slowly became more hospitable and our ancestors returned to a land-based life, carriers of this trait would be at a disadvantage once the rich w3 EFA sources were left behind.

