The Nitrilosides (Vitamin B-17)-Their Nature, Occurrence and Metabolic Significance (Antineoplastic Vitamin B-17)

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Vitamin B-17 (nitriloside) is a designation proposed to include a large group of water-soluble, essentially non-toxic, sugary, compounds found in over 800 plants, many of which are edible. These factors are collectively known as Beta-cyanophoric glycosides. They comprise molecules made of sugar, hydrogen cyanide, a benzene ring or an acetone. Though the intact molecule is for all practical purposes completely non-toxic, it may be hydrolyzed by Beta-glucosidase to a sugar, free hydrogen cyanide, benzaldehyde or acetone.

We have proposed the collective generic term n-i-t-r-i-l-o-s-i-d-e for all such cyanophoric glycosides of dietary significance.

One of the most common nitrilosides is amygdalin. This nitriloside occurs in the kernels of seeds of practically all fruits. The seeds of apples, apricots, cherries, peaches, plums, nectarines, and the like carry this factor; often in the extraordinary concentration of 2 to 3 per cent. Since the seeds of fruits are possibly edible, it may be proper to designate the non-toxic water soluble accessory food factor or nitriloside that they contain as vitamin B-17. The presence of nitriloside in the diet produces specific physiologic effects and leaves as metabolites specific chemical compounds of a physiologically active nature. The production by a non-toxic, water-soluble accessory food factor of specific physiological effects as well as identifiable metabolites suggests the vitamin nature of the compound.

The ubiquity of the compound or its metabolites in plant and animal foods further corroborates its vitamin status. And the development of specific deficiency states as a result of its deficiency in or absence from the diet, and the correction of such pathologic deficiency states by supplying the factor confirm its vitamin status.

The diet of primitive man and most fruit-eating animals was very rich in nitrilosides. They regularly ate the seeds (and kernels) of all fruits, since these seeds are rich in protein, polyunsaturated fats, and other nutrients. Seeds also contain as much as 2 per cent or more nitriloside. There are scores of other major foods naturally, or normally, very rich in nitriloside. Let's consider now what happens when one eats the nitriloside-rich seeds of fruit.

In metabolism, nitriloside is hydrolyzed to free hydrogen cyanide, benzaldehyde or acetone and sugar. This occurs largely through the enzyme Beta-glucosidase produced by intestinal bacteria as well as by the body. The released HCN [hydrocyanide] is detoxified by the enzyme rhodanese to the relatively non-toxic thiocyanate molecule. The sugar is normally metabolized. The released benzaldehyde in the presence of
oxygen is immediately oxidized to benzoic acid which is non-toxic. Thus this newly designated vitamin B-17 (nitriloside) could account for:

1. The thiocyanates in the body fluids--blood, urine, saliva, sweat, and tears;
2. For part of the benzoic acid (and subsequently hippuric acid); salicylic acid isomers;
3. For the HCN that goes to the production of cyanocobalamin from hydrocobalamin, or production of vitamin B\textsubscript{12} from provitamin B\textsubscript{12}.

These are the physiological properties of the common nitriloside amygdalin. Before considering the possible antineoplastic activity of this vitamin B-17, let us recall that the benzoic acid arising from it has certain antirheumatic and antiseptic properties. It was rather widely used (in Germany and elsewhere) for rheumatic disease therapy prior to the advent of the ortho-hydroxy addition product of benzoic acid known as ortho-hydroxybenzoic acid or salicylic acid. It was originally obtained from beech-wood bark. As a matter of interest, the para- hydroxy isomer of benzoic acid occurs in the para hydroxybenzaldehyde aglycon (non-sugar) of the nitriloside found in the cereal millet. Millet was once more widely used in human nutrition than wheat. Wheat seed contains little or no nitriloside.

Recall now, that thiocyanate also was once widely used, in both Germany and American medicine, as an effective agent for hypertension. Used as such, as the simple chemical, the dosage was difficult to control. Obviously, this difficulty does not arise from the thiocyanate usually produced in the body through metabolizing vitamin B-17 (nitriloside). However, chronic hypotension has been reported in Nigerians who eat quantities of the nitriloside-containing manioc (cassava)--especially that of the bitter variety.

Let us pause to reflect upon this question: Might not the rheumatic diseases as well as certain aspects of hypertension be in some cases partially related to a dietary deficiency in nitrilosides? One can hardly deny that the ingestion of a sufficient quantity of nitriloside-containing foods will metabolically yield sufficient benzoic acid and/or salicylic acid isomers to palliate rheumatic disease and certainly to decrease, however temporarily, hypertension as well as to foster the nitrilosation of provitamin B-12 to active vitamin B-12: cyanocobalamin.

Despite all this, are we justified in suggesting that cancer itself might be another chronic metabolic disease that arises from a specific vitamin deficiency--a deficiency specifically in vitamin B-17 (nitriloside)?

Again, let us reflect for a moment. There are many chronic or metabolic diseases that challenge medicine. Many of these diseases have already been conquered. What proved to be their solution? By solution we mean both prevention and cure. What really cures really prevents. Let us think of some of these diseases that have found total prevention and hence cure. We are speaking of metabolic or non-transmissible diseases. At one time the metabolic disease known as scurvy killed hundreds of thousands of people, sometimes entire populations. This disease found total prevention and cure in the ascorbic acid or vitamin C component of fruits and vegetables. Similarly, the once fatal diseases so aptly called pernicious anemia, pellagra, beri beri, countless neuropathies, and the like, found complete cure and prevention in specific dietary factors, that is, essential nutrients in an adequate diet.

I can hear an objection of course. But let me remind you that all the solved or conquered chronic or metabolic diseases were found to be simple specific dietary diseases. Remember this: before these diseases were understood, before the means of total prevention and cure were discovered, it was widely believed that these dietary deficiency diseases were due to viruses, bacteria, bad air, "infection," or some such cause.

Now I ask you to name a single chronic or metabolic disease that has ever found total prevention and cure except by specific dietary factors and/or factors normal to adequate animal economy. I have never found anyone who has been able to suggest a single chronic or metabolic disease that has ever been totally prevented and cured except through a factor essential to adequate diet and/or to the animal economy.
Let's go a step further, almost to the border of dogmatism, to advance an axiom in medicine and biology:

No chronic or metabolic disease has ever found cure or prevention, that is, real cure and real prevention--except through factors essential to an adequate diet and/or normal to animal economy.

I would welcome a contradiction to this principle; but even an exception would "prove the rule."

Does it seem likely, therefore, that cancer will be the first exception to this generalization that to date has not had a single known exception? In my humble opinion, certainly not. But does it follow from this that vitamin B-17 (nitriloside) is the specific antineoplastic vitamin? Logically, by itself, alone, this conclusion that nitriloside is the specific antineoplastic vitamin does not follow. However, examine the brilliant laboratory studies of Dr. Dean Burk of the Department of Cytochemistry of the National Cancer Institute in Washington. I believe that in light of the experimental evidence that he has produced, you might agree that vitamin B-17 (nitriloside) is indeed the antineoplastic vitamin.*

One might ask, then, whether we suggest that vitamin B-17 (nitriloside) or Laetrile is an effective cancer drug. Our reply must be: it is not a drug; it is a

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*Author's footnote: Dr. Dean Burk's paper was in the same program, also a report on the pharmacodynamics and clinical application of vitamin B-17 nitriloside (amygdalin) by Dr. Hans Nieper, a brilliant young man who combines an excellent ability in biochemistry with a genius in clinical medicine, in my opinion.

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vitamin. We feel certain that it will never be possible to speak of a true or effective "cancer drug," any more that it is possible to speak of a pellagra drug, a scurvy drug, a pernicious anemia drug, or the like. The U.S. Food & Drug Administration has just announced that the major drug (as contrasted to the normal animal product insulin) used in the palliation of diabetes--Orinase--is "no good." We know of no true drug that actually prevents or cures metabolic or chronic diseases--or really does any genuine good. We mean by "drug," of course, relatively toxic chemicals foreign to the body or foreign to the animal economy.

As already mentioned, vitamin B-17 (Laetrile) is totally non-toxic. Its lethal dose in mice and rats, by injection, is about 25,000 milligrams per kilogram of body weight. It is so nearly non-toxic that in some studies the water, used as a diluent, presents a greater toxicity than the vitamin. This applies for acute, subacute and chronic toxicity. By mouth in test animals it is less than 1/20 as toxic as aspirin. Speaking of aspirin, let us recall that this great German discovery, the acetylation product of ortho-hydroxy benzoic acid, and some salicylic acid isomers, as well as benzoic acid itself, are the normal metabolites of dietary nitrilosides found in the seeds of nearly all fruits and some cereals. For example, millet, mentioned above, once more widely used than wheat, yields the salicylic acid isomer para-hydroxybenzoic acid, which arises as the metabolic product of its nitriloside: p-hydroxymandelonitrile-B-glucoside. In this you can discern, however dimly, the dietary-therapeutic profile of the salicylates as a means of satisfying a dietary deficiency in benzoic acid and the related salicylic acid isomers.

Returning to the non-toxicity of nitriloside; it is no more toxic than dextrose or ascorbic acid--and to the diabetic less toxic than the former.

I have noticed that newspapers are carrying wire dispatches reporting the studies of Professor Roger Williams of the University of Texas. He is quoted on the "toxicity" of commercial white bread as sold in the United States. You will recall that Doctor Williams is the discoverer of vitamin B-1 or thiamine; and the first to synthesize it. Doctor Williams, in effect, showed that commercial white bread as sold in the United States is
about 70 times more toxic than vitamin B-17. Doctor Williams fed four strains of white rats (noted for their vigor), nothing but commercial American white bread for three months. Seventy-five per cent of all the experimental animals so fed died of malnutrition before the experiment was complete. Those fed on whole wheat all survived. The commercial white bread was enriched by law with some crystalline vitamins, but not in a sufficient quantity and variety to prevent these rats being killed by the bread. So how about vitamin B-17 toxicity studies? White rats fed 70 times the normal human dose of vitamin B-17 (nitriloside) used in the palliation of human cancer were completely normal and healthy after 90 days. None of them died. There were some "physiological side reactions" to vitamin B-17--greater weight and appetite. After all they were receiving nourishment; a vitamin, not a vitamin-deficient ration or a drug.

The rats that died from eating commercial white bread--all 75 per cent of them--died as a direct result of a deficiency in vitamins found in the whole grain of wheat. There was the deficiency in vitamin E as a result of the missing germ or seed of the wheat, a deficiency of choline, vitamin B-15 (pangamic acid), vitamin B-6, biotin and other factors as a result of the missing bran taken from highly refined bleached white flour. Recall that the natural whole grain of wheat is composed of the starchy endosperm or bulk of the grain as well as the germ or the seed which carries the oils in which are dissolved the tocopherols or vitamin E; and the bran which contains an abundance of the B vitamins.

Those rats died from a vitamin deficiency produced by eating less than the whole grain, the whole food. When civilized man eats less than the whole fruit, for example, by discarding the seed or kernel he experiences a specific and total deficiency not only in oils and proteins but in minerals and such vitamins as vitamin B-17 (nitriloside) which is found only in the seed, not in the flesh of the fruit. By discarding the seed or kernel, man experiences a specific and total deficiency in vitamin B-17 so far as that fruit is concerned. Let me remind you that were man by circumstance limited to no source of food but apricots, peaches, plums, cherries and the like and ate only their fruit without their seeds he would in a short time develop a fatal deficiency in proteins and fats not to mention vitamins. He would die from this deficiency just as the white rats died from the deficiency produced by eating only the starch of wheat without the seed germ and bran. But if he ate the seeds or kernels with the fruit flesh, he would get proteins, fats and other nutrients essential to health.

Vitamin B-17 (nitriloside) is also found in great abundance in a very wide variety of vegetable foods once eaten in great abundance by man, and the natural fodder of animals is similarly rich in the factor. In a paper which I hope to publish soon, I have listed over 62 plant foods eaten by man and over 70 common fodder plants that are very rich in vitamin B-17 (nitriloside). Their concentration of this vitamin compares favorably with that of vitamin C (ascorbic acid) so far as quantity and ubiquity are concerned. As in the case of many other vegetables, sprouts may contain 10 to 30 times as much vitamin B-17 as mature plants. It is not practicable to furnish here the several hundred references of the basic research on nitrilosides nor to list extensive tables showing the occurrence of this new vitamin in a wide range of foods. It would not be germane to explain the reasons why and how "modern diet" has been almost totally stripped of nitrilosides. Suffice it to say that the factors that made commercial white bread lethal to rats and gave the world the empty calories of refined white sugar also have served to produce a fulminating deficiency in vitamin B-17 (nitriloside) in the diet of so called civilized man.

So much for the specific nutritional aspect of vitamin B-17 (nitriloside). How can a compound that is totally non-toxic be relevant to a disease as serious as cancer, a disease perhaps as lethal as pernicious anemia once was? Would we not expect that very powerful cytotoxic compounds would be required to destroy cancer cells? Would these not be compounds like the nitrogen mustards, the antimetabolites, the cyclophosphoramides, methotrexate, 5-fluoruracil, 6-chloropurine, 6-mercaptopurine, azaserine, triethylenephosphramide, the nitrosoguanidines, and countless other compounds so toxic that some kill almost 25 per cent of the patients treated directly or indirectly through toxicity alone?

It is true that neoplastic cells are destroyed by cytotoxins. The cytotoxins used so far, the ones I have
mentioned, are more toxic to body or somatic cells than specifically to cancer cells. This is obvious. Otherwise we would be able to administer these cytotoxins until they killed all cancer cells and left the host alive. But they almost always, if not always, kill the host before killing the neoplastic cells. In the problem of neoplastic therapy we have in drugs an almost insoluble paradox. For an agent to be effective it must be both non-toxic to somatic cells and yet present powerful cytotoxins to neoplastic cells--cytotoxins like the cyanides and benzaldehyde.

Vitamin B-17 (nitriloside) releases a specific and powerful cytotoxin, probably the most powerful one known. This is hydrogen cyanide. Our formulation of Laetrile also releases an equimolar quantity of benzaldehyde which, before oxidation to benzoic acid, is a very powerful cytotoxin. We have here two very powerful cytotoxins. Doctor Dean Burk of the National Cancer Institute has brilliantly demonstrated, largely through utilization of the technics and manometer of Otto Warburg, that the benzaldehyde released by the hydrolysis of nitriloside or Laetrile is not only in itself a powerful cytotoxin but that it multiplies through a very powerful synergy the cytotoxic effects of both--cyanide and benzaldehyde--to an extent many, many times greater than the arithmetic sum of their separate effects.

These two compounds in synergy are more powerful cytotoxins than any of those that I have already mentioned above.

Why isn't the equimolecular quantity of benzaldehyde oxidized immediately by the cancer cells to harmless benzoic acid as occurs in body or somatic cells, and why isn't the equimolecular quantity of cyanide converted immediately to thiocyanate as it is in body or somatic cells? Recall that Otto Warburg himself received one Nobel Prize for proving the suboxidative activity of cancer cells. They ferment--fermentative metabolism rather than respiratory metabolism plays a large role in cancer. This metabolism utilizes less oxygen (in the free state); therefore, oxidation of benzaldehyde occurs much more slowly. Unoxidized benzaldehyde lags, as it were, in the neoplastic cell. This cell also lacks a very important enzyme possessed by body or somatic cells. This enzyme is rhodanese or thiosulfate transulfurase. It convert cyanide to the harmless thiocyanate. With the selective lag of both undetoxified cyanide as well as unoxidized benzaldehyde in the neoplastic cell, and the multiplication of cytotoxicity that the combination affords, the neoplastic cells suffer a lethal cytotoxicity while the hostal or somatic cells are totally unaffected--except possibly in a beneficial or physiological manner. We are dealing with a vitamin, remember.

Pause again to reflect. Is it possible that this described cytotoxic synergy arising from the hydrolysis product of vitamin B-17 (nitriloside), is a coincidental or fortuitous phenomenon--a synergy totally ungrounded in any other biological experience, a pure accident? Or does this synergy represent the end product of the enduring effects of a process of natural selection between plants and animals through which a specific antineoplastic vitamin, vitamin B-17, has evolved in a natural environment once as abundantly rich in nitrilosides as ascorbic acid?

There is no controversy, of course, on the fact that equimolecular quantities of benzaldehyde and cyanide resulting from the hydrolysis of vitamin B-17 will selectively kill cancer cells. The cytotoxicity of these chemicals against neoplastic cells is known, but the margin of safety for these raw chemicals is very little greater than the most powerful cytotoxins--except that different from the latter there is no residual, cumulative or chronic toxicity from them. Contrast this to the utter non-toxicity of these same chemicals bound in the white sugary nitriloside molecule.

Wherein, then, is there a controversy over this vitamin in therapy? Though the major and practically sole controversy is and has always been a political one, if we were to try to pin-point a specific scientific criticism it would probably be this: what real or experimental proof is there that the nitriloside molecule is selectively hydrolysed or broken down to free cyanide, benzaldehyde and sugar at and by the neoplastic lesion? It is, of course, a commonplace--now almost a century old--that the nitriloside is split to its 3 major components by the enzyme Beta-glucosidase. It is also known that the malignant lesion contains a high concentration of certain
Beta-glycosidases (e.g., Beta glucuronidase). The proponents of vitamin B-17 for the prevention and palliation of cancer have long argued inferentially for the presence of specific Beta-glucosidase activity in the malignant lesion, which would account for or its selective lysis here with the release of the admittedly highly cytotoxic HCN and benzaldehyde in synergy.

The opponents of vitamin B-17 in cancer therapy have rather myopically, (I believe), argued that there is no proof that selective hydrolysis of the nitriloside occurs in the neoplastic cell. They reject all existing clinical evidence, however impressive, for this effect. Thus it is an extraordinarily important finding that Doctor Dean Burk reports on his observation of the effect of the incubation of C3H mouse mammary cancer with vitamin B-17 in the Warburg manometer. He reports that the malignant mammary tissue selectively hydrolyzes the added nitriloside to free cyanide, benzaldehyde and sugar with a highly effective cytotoxicity; and that this does not occur in benign or somatic control mammary tissue! This experimental observation means, of course, that the neoplastic tissue carries a specific Beta-glucosidase activity that normal or somatic tissue lacks, which lack here is obvious in view of the total non-toxicity of the material toward normal tissue. This very crucial experiment will, of course, be repeated and checked and rechecked in many laboratories.

Let us in summary simplify all this in terms of vitamin action. When vitamin B-17 enters the body (in foods, for example), it is hydrolyzed only to a very slight degree by body or somatic cells. This is obvious from the non-toxicity shown by B-17. But even if some of the B-17 is hydrolyzed by body or somatic cells, the very high concentration of the enzyme rhodanese in these cells converts the HCN immediately to relatively non-toxic thiocyanate. (This accounts largely for the thiocyanate that you find in blood, urine, saliva, etc., as stated above).

How different it is with the neoplastic cell! It contains great quantities of Beta-glycosidase. Fischman and many others in America have independently shown this in the case of Beta-glucuronidase. Sometimes there is over 1,000 times as much of this Beta-glycosidase as in the contiguous normal or body cell. The neoplastic cell is almost completely deficient in the enzyme rhodanese. Recall that when B-17 reaches the cancer cell the Beta-glycosidase there hydrolyzes it with the release of extremely large quantities of cyanide (relative to the situation in normal body cells). This selective effect occurs in a cell that is almost totally deficient in the enzyme rhodanese, which in normal body cells is present to detoxify cyanide to thiocyanate. Thus the end result of the presence of one enzyme that causes the selective release of hydrogen cyanide in cancer cells, plus an oxidative deficiency (fermentative metabolism) that causes a lag in benzaldehyde oxidation to benzoic acid, result in the selective persistence of free or undetoxified cyanide plus free or unoxidized benzaldehyde which synergistically exert their selective antineoplastic effect.

A discussion of the clinical details of vitamin B-17, nitriloside in animal and human cancer is best left to our clinical students of the subject. They are faced with the fact that today more people per 100,000 of the population are developing cancer and dying from it at an earlier age than any other time in recorded history of the human race. At least one in three of the population develops clinical cancer and probably all develop subclinical neoplasms in the course of a lifetime. The situation, in our opinion, almost identifies itself in terms of a fulminating deficiency disease a priori. As our veterinary friends tell us, even our cats and dogs are showing an incidence of cancer parallel to that of their "civilized" owners. Observe how quickly these animals when released from an apartment or kennel will single out (and eat) such nitriloside-rich grasses as Johnson grass, Tunis grass or Sudan grass as a supplement to their diet. Some of these grasses contain as much as 17,000 mg of nitriloside per kilogram of dry weight!

In this presentation we have attempted to touch a vast and relatively unexplored area. But before closing let me introduce a little Yankee humor. It may be sick humor: judge for yourselves. We know of the white bread that will kill 75 percent of hearty rats in 90 days, of calorie-free white sugar, of cola drinks, of fulminating vitamin deficiencies, and the like. But in the United States there is one "school of nutritional thought" that, despite all this, sought to append the following statement to the labels of all bottles of vitamins:
"Vitamins and minerals are supplied in abundant amounts by the foods we eat. The Food and Nutrition Board of the National Research Council recommends that dietary needs be satisfied by foods. Except for persons with special needs, there is no scientific basis for recommending routine use of dietary supplements."

The lethal commercial white bread is by law supplemented, but not supplemented enough not to kill the rats. It is argued, of course, that this won't hurt man too much unless he relies almost solely on this staff of life and is no tougher than the rats!

Lest this new vitamin B-17 or nitriloside still be a less concrete reality in your mind than ascorbic acid, thiamine, niacin or the like, let me leave you with an example of a daily ration or diet remarkably rich in nitriloside or vitamin B-17. For breakfast we start with buckwheat, millet and flax-seed gruel; all three cereals are very rich in nitriloside. On our millet bread toast we put some nitriloside rich elderberry jelly. The stewed apricots we eat carry the nitriloside-rich seeds, which we detect through their delicious almond-like flavor. At lunch we have nitriloside-rich lima beans or possibly a succotash containing nitriloside-rich chick peas. Our millet rolls may be spread with plum jam carrying the nitriloside-rich seeds that add so much to the flavor of the jam. We may choose some nitriloside-rich elderberry wine. For dinner we may have a salad with some nitriloside-rich bean sprouts and nitriloside-rich millet sprouts. Our dinner rolls may be made of nitriloside-rich buckwheat and nitriloside-rich millet and sweetened with nitriloside-rich sorghum molasses extracted from sorghum cane--almost all of the foregoing are very rich in nitrilosides. For our meat course we may have rabbit that fed on nitriloside-rich clover and as a result carries 5 to 10 times more thiocyanate and nitriloside than animals not so fed. If the milk we drink came from cows that ate fodder rich in nitrilosides this milk will contain as much as 7 times more nitriloside than a cow living on nitriloside-deficient fodder. At the end of the dinner we may choose a nitriloside-rich apricot, peach, cherry, or plum brandy originally prepared from crushing the entire or whole fruit. We may also choose a number of wild berries very rich in nitrilosides--all members of the raspberry family. We may nibble on some nitriloside-rich macadamia nuts or chew nitriloside-rich bamboo sprouts.

In such a menu of three meals in the course of a day we should ingest over 300 mg of nitriloside or vitamin B-17 in our foods--every one of which contained nitriloside. The quantities of the vitamin B-17 in the described foods have been very carefully determined by independent workers over the years. Because of our cultural antipathy to cyanide, our food technology has made every conceivable effort through processing, hybridizing, distilling, etc., to remove every trace of derivable cyanide from foods for man and animals. It is good that this irrationality has not to date, at least, completely removed the cyanide-containing vitamin B-12 or cyanocobalamin.

Finally, let me conclude with this. In nitriloside or vitamin B-17 we have a new vitamin in which all of us are severely deficient. This fact is beyond question. As to the clinical application of vitamin B-17 (nitriloside) in human and animal cancer, we feel that every case is morally entitled to whatever vitamin B-17 can offer, just as everyone being stricken with scurvy, pellagra, or pernicious anemia is morally entitled, respectively, to vitamin C, niacin, vitamin B-12 and folic acid. Indeed, the matter goes far beyond clinical cancer itself. Mankind cannot afford any longer a human and animal population deficient in vitamin C, vitamin B-12, vitamin B-15, vitamin B-17 or any other vitamin essential to animal or human nutrition.

However, the capacity of political power for stupidity is truly infinite. We can not predict how long the orderly clinical study of crystalline vitamin B-17 will be delayed. But take some comfort in this. Were vitamin B-12 and folic acid completely proscribed tomorrow, liver would still offer complete salvation in pernicious anemia. Similarly, one gram of defatted apricot seed or kernel carries about 30 milligrams of nitriloside. Six or seven teaspoonful will supply what our clinical investigators consider an adequate oral dose--one gram. It is best that the B-glucosidase enzyme be completely heat inactivated in such material.

So far as other parts of the world may be concerned, I fear no such described obstruction. In Germany I was
very happy to find from four to five proprietary and ethical brands of vitamin B-15 (pangamic acid), or its DIPA analogue, and I look forward to seeing a similar distribution of vitamin B-17 (nitriloside) very soon. In visiting the great museum in Hanover I was pleased to find in a display of food-stuffs recovered from Stone Age digging in Europe that of eight food plants shown, three of them are heavy nitriloside-producers. One was Himbeere (Rubus idaeus), another Brombeere (Rubus fruiticosus) and Schwarzer Hollunder (Sambucus niger) or the common elderberry (from which the nitriloside sambunigrin was originally isolated). In the United States the Lovelock Caves in Nevada have yielded petrified animal and human faeces (fecoliths) that through carbon-dating have been found to go back many years. They showed numerous remnants of nitriloside-bearing plants.

Just as the German chemists Huber and Weidel in 1873 first synthesized niacin through the oxidation of nicotine about forty years after Wohler and Liebig in your country first isolated and identified the first nitriloside, amygdalin, and just as niacin was destined half a century later to be identified and defined as the factor that prevents and cures pellagra in man, so we find that the nitriloside isolated and identified over a century ago in Germany likewise is now achieving the status of a vitamin--vitamin B-17. Let us hope that like niacin it has at least left the chemical museum to serve the impelling needs of improved nutrition.

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A noted biochemist, Ernst Krebs, Jr. took his student work at Hahnemann Medical College in Philadelphia 1938-41. He received his AB at the University of Illinois in 1942; he did graduate work at the University of California during 1943-45, researching in pharmacology during the periods of 1942-45. He is science director of the John Beard Memorial Foundation, having held this position since 1946. He is the author of "Unitarian or Trophoblastic Thesis of Cancer" (1950); co-discoverer of pangamic acid (1948), the role of pancreatic enzymes in human cancer (1948-50), and the relevance of the nitrilosides (Vitamin B-17) to animal and human nutrition.

This paper is a summary of remarks presented in German before a congress of the International Medical Society for Blood and Tumor Disease, Nov. 7, 1970, in Baden-Baden, West Germany. On this occasion, the author received an award honoring his discovery and research on vitamin B-15 (pangamic acid) and vitamin B-17 (nitriloside).

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A partial bibliography is printed here. A complete listing of references will follow in a subsequent issue.

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