Correspondence

Selenium Deficiency and Schizophrenia

In his recent letter to the *Journal of Orthomolecular Medicine,* Berry (1994) hypothesized that there may be a subgroup of selenium deficient schizophrenics who respond well to niacin supplementation. He suggests such improvement may occur because the methyl acceptor niacin probably reduces selenium methylation and prolongs this essential trace element's action in the body.

There seems to be mounting evidence that selenium deficiency is, indeed, involved in schizophrenia. Comparisons of the 1964 prevalence of schizophrenia in the United States, with the spatial distributions of 219 environmental variables, established that the strongest positive correlation was with selenium deficient fodder crops ($r = 0.58497$, $p = 0.0001$). That is, where state schizophrenia prevalence rates were highest, the least selenium was entering the food chain (Foster, 1988; 1992).

Beyond this, Buckman and colleagues (1987) measured the activity of glutathione peroxidase, the selenoenzyme, in blood samples from a population of chronic schizophrenics and from a control group of age and sex matched non-schizophrenic mental patients. They found a strong correlation, in schizophrenics, between glutathione peroxidase activity in both isolated platelets and erythrocytes and computed tomography scan measures of brain atrophy and increased ventricle-brain ratios. These relationships did not occur in the control group and suggest a unique relationship of glutathione peroxidase, and hence selenium, to the mechanism of tissue damage found in the brains of schizophrenics (Buckman et al, 1987).

To summarize, in the United States, schizophrenia is more common in low selenium regions. Furthermore, schizophrenics tend to be deficient in the selenoenzyme glutathione peroxidase and show brain atrophy associated with reduced levels of this enzyme. Niacin, however, appears to mitigate against such selenium deficiency and has been associated with numerous cures amongst schizophrenics (Hoffer, 1994). It appears to me that all of these observations are consistent with the hypothesis that selenium Deficiency probably plays a key role in the etiology of schizophrenia.

References

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Harold D. Foster, Ph.D., Professor
University of Victoria
P.O. Box 3050
Victoria, B.C., Canada V8W 3P5

Follow-up reports on Chronic Schizophrenic patients described in this Journal, Vol. 9, Number 1, 7-37, 1994.

I assume that readers who have studied my report on the 27 chronic schizophrenic patients will be interested in what has happened to them since that report was prepared. In this and subsequent issues I will describe briefly their condition after I have seen them. I do not call these patients in routinely but do set up appointments when they are on medication which should be supervised closely. Otherwise it is their decision, in consultation with their referring physician, to arrange to be seen.

With each quarterly report I will include a very brief account of a chronic patient seen for at least ten years. The first is Ann, born in 1935. She was brought to me by her mother. A few days after I had come to Victoria in 1976 to start my practice I was in a drug store picking up something. I told the druggist who I was. An elderly woman standing behind me hesitantly spoke to me to ask

if I was the Dr. Hoffer who used vitamins. She then asked if I would see her daughter who was very sick and had not responded to treatment even after a two month treatment period in the psychiatric ward. I said I would see her if she wanted to come and was referred by her general practitioner. She came with her mother but she thought there was nothing wrong and had no complaints. It was difficult to obtain enough information for a proper mental examination. Her mother told me she was divorced after five years and had two children. Beginning in 1976 her personality had suddenly changed from a likeable, affectionate person to one who was hostile, depressed, very suspicious and totally disinterested in her children. She had run away from her home on two occasions. The police found her the second time and took her to the hospital. She did not respond to medication and was discharged no better.

I concluded she was probably schizo-affec-tive, i.e. showing both schizophrenic and depressive features. As she had not responded to antidepressant medication I admitted her to hospital and gave her seven electroconvulsive treatments (ECT) in combination with the Orthomolecular treatment program, chiefly vitamin B_3 and vitamin C. After discharge she began to improve and one year later she was normal. She then was able to cope with very severe stress generated by a teenaged schizophrenic child, also under my care, a difficult second child, and her mother, who also became very depressed. This patient carried an enormous load in caring for her family.

She remained well until 1988, on her vitamins and 25 mg of amitriptylene and 2. mg of perphenazine. She then developed severe low back pain, so severe she could not walk. She had heard about a grapefruit diet which she followed for 21 days losing 20 pounds. By the end of this period she was very depressed as well as suffering severe pain. I was asked to see her in the hospital and promptly admitted her to the psychiatric ward. The end of 1988 she was given five ECT. Early in 1989 she needed another seven ECT. She did not respond this time and was admitted two more times until December 1989. This time I kept her in for six months. For much of that time she was almost in a catatonic state, very depressed, unable to walk, suffering severe pain.
Toward the end she began to improve.

She has now been well for four years on vitamins and on the same two small doses of medication. Once more she has regained her normal personality and is again the main emotional support for her children. Her schizophrenic child is getting on well and the other is still under treatment.

This case is an illustration of the need to use every treatment modality which can be therapeutic and does not harm the patient. She needed a combination of megavitamins, medication, which was only useful in small doses, and ECT, combined with enough time in hospital to allow her to recover. Had she been treated like a first aid case in a brief treatment centre she would still be sick. Unfortunately the majority of patients are discharged much too soon, long before they have been able to stabilize on their treatment program.

The following patients have been seen since the first report on the 27 chronic patients was completed (JOM 9.1) Those who were not seen are probably unchanged since last evaluated.

J.L. - He was seen three times in 1993. On July 29 he was started on resperdal. One month later there was a slight improvement socially. He was seen three times in 1994, the last time May 25. He came with his friend, K.G. I reexamined his clinical state when I first saw him in 1976. The difference was amazing. He had not suffered hallucinations, nor delusions, and was free of depression for several years. I classed him as improved since he was not paying taxes (not working), and still remained hurt by his chronic illness. But he was cheerful and these two chronic men, who had never been able to have any relationship, had established a friendship. They would walk together, talk to each other. We were able to have a three-way conversation.

G.H. - She was seen once in July 1993 and remains well.

J.K. - She was seen twice in 1993 and once in 1994. She is well but feels more comfortable when she can see me about every six months. At age 73 her main concern was some arthritic pain in her back. She had decreased her niacin to 1 g daily. I suggested she increase it to 2 g.

L.K. - Was normal when seen Dec. 20, 1993.

J.P. - She remains much improved but needs to be seen regularly. She was seen eight times in 1993 and three times in 1994.

S.M. - She remains well. She was seen three times in 1993 and once in 1994.

R.B. - He was seen twice in 1993. After one month on an antidepressant he was slightly better but still remains only improved.

B.A. - He was in hospital for a couple of weeks in summer of 1993 but has been well since then.

R.W. - He is seen every two or three weeks and is continuing to make slow but substantial progress.

J.M. - He remains well.

K.G. - He was seen three times in 1993 and twice in 1994. He is improving slowly. He was seen May 25, 1994, with his new friend, Mr. J.L. I have described the relationship of these two men under J.L.

R.S. - R. dropped in May 2, 1993. He had not been following his vitamin program as carefully as he should have. A resurgence of symptoms such as paranoid ideas, changes in perception and difficulty in concentration made his realize he was relapsing. I went over with him what he should be taking, i.e. niacin 1.5 g tid, vitamin C 1 g tid, B-complex 50s od and added vitamin E 800 i.u. I increased his Haldol to 6 mg daily. One week later he was well again. He will remain on the Haldol for another month and then he will gradually decrease it again. He planned not to work over the summer. He had to give up his previous job when he began to relapse.