

# Letters

## **An Alternative Explanation of the Psychotropic Effect of Niacin in Schizophrenia**

Berry (1993, 1993) theorizes that a selenium transport protein might be defective in a sub-type of schizophrenia with consequent low levels of selenium in the body and the brain. If this is the case this might explain why niacin is an effective treatment in schizophrenia.

The metabolism of selenium involves its methylation (Palmeretal., 1969; Byard, 1969). The methylation of selenium is considered to be of toxicological significance (Chen and Whanger, 1993). A methyl acceptor such as niacin might very well reduce the methylation of selenium and hence prolong selenium's action in the body, and indeed research has shown that blood selenium levels are significantly and positively correlated with niacin levels — p less than or equal to 0.005 (Shultz and Leklem, 1993). If the theory put forward by Berry is correct niacin might have its therapeutic effect in schizophrenia because it prevents the metabolism of selenium and hence prolongs its action.

The theory is easily testable. If schizophrenics in the United States have whole blood selenium levels below .100 ug Se/ml then this would suggest that selenium metabolism in schizophrenia is abnormal.

The 1989 RDA for selenium was set to saturate glutathione peroxidase production (Levander 1991) and glutathione peroxidase production plateaus at .100 ug Se/ml (Thomson et al., 1977; Thomson et al., 1993). North America is a high selenium area and diets in North America will as a rule provide the selenium needed to saturate glutathione peroxidase production (Levander 1991). If schizophrenics present with whole blood selenium levels below .100 ug Se/ml then possibly these patients have a defect in a selenium transport protein and niacin by preventing the methylation of selenium might thereby have its antischizophrenic effect.

The selenium status of mineral and vitamin-free schizophrenics should be investigated.

## **References**

1. Berry: Possible involvement of selenium Transport Protein in a Subtype of Schizophrenia [letter]. *Biological Psychiatry* 34:422-423, 1993.
2. Berry: Selenium Deficiency and Clinical Findings in Schizophrenia: A Common Thread. *The J. of Orthomolecular Medicine*, Vol. 8, No. 1:21-24, 1993.
3. Byard JL: Trimethyl Selenide. A Urinary Metabolite of Selenite. *Archives of Biochemistry and Biophysics* 130:556-560, 1969.
4. Chen CL and Whanger PD: Effect of Vitamin B<sub>12</sub> Status on Selenium Methylation and Toxicity in Rats: *In Vivo* and *in vitro* Studies. *Toxicology and Applied Pharmacology* 118:P65-72, 1993.
5. Levander OA: Scientific Rationale for the 1989 Recommended Dietary Allowance for selenium. *J. Am. Diet. Assoc.* 91:1572-1576, 1991.
6. Palmer IS, Fischer DD, Halverson AW and Olson OE: Identification of a Major Selenium Excretory Product in Rat Urine. *Biochim. Biophys. Acta* 177:336-342, 1969.
7. Schultz TD and Leklem JA: Selenium status of vegetarians, non-vegetarians, and hormone-dependent cancer subjects. *Am. J. Clin. Nutr.* 37: 114-118, 1983.
8. Thomson CD, Robinson MF, Butler JA and Whanger PD: Long-term supplementation with selenate and selenomethionine: selenium and glutathione peroxidase (ED 1.11.1.9) in blood components of New Zealand women. *British J. of Nutrition* 69:577-588, 1993.
9. Thomson CD, Rea HM, Doesburg VM and Robinson MF: Selenium concentrations and glutathione peroxidase activities in whole blood of New Zealand residents. *British J. of Nutrition* 37:457-460, 1977.

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