

# Thiamin Content in the Organism during Acute Alcoholic Psychosis

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Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 125, No. 1, pp. 98-100, January, 1998  
Original article submitted January 17, 1997

Acute alcoholic psychosis is associated with a decrease in the vitamin B<sub>1</sub> content in the body and suppression of transketolase. After arrest of psychotic state, metabolic parameters reflecting thiamin content do not normalize for at least one week.

**Key Words:** thiamin; transketolase; vitamin content in organism; acute alcohol psychosis

Metabolic changes in chronic alcoholism (CA) and acute alcoholic psychosis (AAP) attract the attention of scientists and physicians due to their significance at the diagnostic, therapeutic, and rehabilitation stages of treatment. Vitamins B play an important role in the biochemistry of CA. These vitamins are essential for vital metabolic processes and regulation of specific functions of the organism, primarily those involved in the nervous system activity and energy processes. Some scientists believe that vitamin B deficiency is responsible for the majority of visceral diseases in CA [3].

Moreover, insufficient supply with vitamins is regarded as a factor promoting liability to narcological diseases, and CA is one of the major etiologic causes of secondary B<sub>1</sub> hypovitaminosis [2].

We decided to follow up the time course of thiamin in chronic alcoholics with the abstinence syndrome often aggravated by AAP.

## MATERIALS AND METHODS

Forty-three men aged 25-55 years with CA hospitalized at narcological hospital during abstinence ( $n=12$ ), AAP delirium ( $n=14$ ), and hallucinosis ( $n=17$ ) were examined. Control group consisted of 17 male donors without CA drinking no alcohol for 2 weeks before testing.

Thiamin-dependent transketolase (TK, CE 2.2.1.1) was assessed by measuring the rate of production of sedoheptulose-7-phosphate in erythrocyte hemolysate

and so-called TDP effect (increment of TK activity in the presence of thiamin) as a marker of sufficient supply of thiamin. Ribose-5-phosphate (Sigma) and thiamin (Fluka) were used. In accordance with common criteria, we considered that TDP effect less than 1.15 arb. units (15%) corresponds to adequate thiamin supply, 1.15-1.25 arb. units (15-25%) signals a moderate efficiency or risk of B<sub>1</sub> hypovitaminosis, and a value higher than 1.25 arb. units (>25%) indicates obvious vitamin efficiency [5]. Pyruvic acid in the serum was measured as described previously [1] with 2,4-dinitrophenylhydrazine using a Spectronic-700 (Baum-Lomb) spectrophotometer. Blood was collected after an overnight fast from the ulnar vein in January-February during the same season, in the morning. Studies were carried out on day 1 of hospitalization, on days 3-4 (when abstinent or psychotic status was arrested), and on days 6-7 (by the end of detoxifying treatment including, among other measures, vitamins B<sub>1</sub> and B<sub>6</sub> in doses of 10-15 mg intramuscularly every other day. Results were processed using Student's *t* test.

## RESULTS

Low activity of TK in comparison with the control was observed in all hospitalized subjects; this decrease was maximal (by 66%) in patients with delirium (Table 1). This indicates not only quantitative aspect of thiamin deficiency, but characterizes qualitative deficiency of energy production and biosynthesis of substances in a live cell.

TABLE 1. Time Course of TK Activity and TDP Effect in Narcologic Patients (MTm)

| Diagnosis           | Day | TK activity <sup>1</sup> | TDP effect, arb. units |
|---------------------|-----|--------------------------|------------------------|
| Control (n=17)      |     | 12.60±0.31               | 1.03±0.02              |
| Delirium (n=14)     | 1   | 4.26±0.85***             | 1.64±0.14***           |
|                     | 3-4 | 5.22±1.18***             | 1.80±0.26***           |
|                     | 6-7 | 5.05±1.57***             | 1.64±0.16***           |
| Hallucinoses (n=17) | 1   | 4.77±0.63***             | 1.48±0.13***           |
|                     | 3-4 | 7.03±0.83***             | 1.37±0.18*             |
|                     | 6-7 | 7.07±1.22***             | 1.37±0.12**            |
| Abstinence (n=12)   | 1   | 6.04±0.98***             | 1.25±0.07**            |
|                     | 3-4 | 6.83±1.34***             | 1.20±0.10              |
|                     | 6-7 | 9.18±1.64                | 1.18±0.08              |

Note. <sup>1</sup>TK activity is measured in mmole sedoheptulose-7-phosphate/ml erythrocytes/h. \* $p<0.05$ , \*\* $p<0.01$ , \*\*\* $p<0.001$  vs. the control.

Although metabolic changes in different nosological groups were similarly directed, their degree was different. In delirium, there was a slight tendency toward an increase in the enzyme activity (by 18-20% of initial values), but this parameter was lower than in the control over the entire period of observation. In acute alcoholic hallucinosis, we observed a significant ( $p<0.05$ ) increase in TK activity on days 3-4: by 47% against its values at the peak of psychosis by the moment of hospitalization. Later, this value virtually did not change and remained significantly lower than in the control.

The time course of the activity of thiamin-dependent enzyme in abstinent syndrome was different. There were no changes on days 3-4, but by the end of the first week its activity decreased by more than 50% and was virtually the same as in the control.

The TDP effect as a quantitative measure of TK unsaturation with coenzyme, not depending on the method of investigation, is a more informative indicator of thiamin sufficiency. Assessment of this effect permits a more objective comparison of the results in dynamic studies [4]. Maximum TDP effect (164-180 arb. units) was observed in patients with alcoholic delirium. The absence of positive changes in this parameter even in the course of detoxifying therapy including vitamins B confirm somatoneurologic aggravation of alcoholic delirium impairing the metabolic processes. A tendency to reduction in the TDP effect was observed in the course of alcoholic hallucinosis, although by the end of follow-up the values were still increased.

Rapid arrest of the abstinent syndrome without psychotic complications was associated with a decrease of hypovitaminosis as early as on days 3-4 and virtually normalization of thiamin level.

The levels of vitamin B<sub>1</sub> supply and blood content of pyruvate correlate: thiamin deficiency involves an increase of pyruvic acid [6]. Examinations of patients with AAP confirmed this regularity (Table 2). This tendency in patients with AAP during treatment can be regarded as a reaction associated with more effective functioning of tissue enzymatic thiamin-dependent systems (pyruvate dehydrogenase complex, etc.). Interestingly, a decrease in the pyruvic acid concentration on days 3-4 coincided with cessation of acute psychotic state, although even by the end of the first week of hospitalization these values were higher than in the control, which, together with thiamin deficiency, indicated that metabolic processes did not normalize.

Thus, our studies showed a considerable thiamin deficiency at the peak of abstinent status and in AAP. This deficiency involves dysfunction of thiamin-dependent enzymatic systems. Probably, this is a pathogenetic factor responsible for the development of somatoneurologic states in narcologic patients and lingering psychotic states.

Despite subjective improvement and a course of intensive care which is generally completed by the end of the first week of hospitalization, thiamin content and metabolic processes in the patients are far from normal. The latter is neglected not only by the patients, which can be explained by their alco-

TABLE 2. Pyruvate Level in Serum of Patients with AAP (mmole/liter, M±m)

| Day | Control   | Delirium   | Hallucinoses |
|-----|-----------|------------|--------------|
| 1   |           | 2963±4.47  | 34.75±5.89   |
| 3-4 | 22.9±1.97 | 25.14±2.63 | 27.44±2.30   |
| 6-7 |           | 25.43±2.15 | 28.56±2.13   |

holic anosognosia, but even by physicians people. The possibility of thiamin deposition in human organism is very low, and early discontinuation of therapy can provoke vitamin-dependent somatoneurologic complications of CA.

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