## Correspondência

## NEUROLOGICAL MANIFESTATIONS IN CELIACS AND VITAMIN E STATUS

To the Editor - I read with interest the article by Sigueira Neto et al.1 concerning neurological manifestations of celiac disease (CD). The authors had investigated serum vitamin E concentration in only one of the three presented patients, and it was reduced. Siqueira Neto et al. discussed pathogenic mechanisms of neurological dysfunction in celiacs and concluded "the immune rather than the nutritional changes of CD are given more credit in the literature". In my opinion, although the nutritional concept of neurological manifestations is "outmoded"<sup>2</sup>, we should keep in mind reports on possible associations between vitamin E deficiency in CD and neurological complications<sup>3,4</sup>. Twelve of 32 (37.5%) patients, presented in English literature, with CD and neurological complications with examined tocopherol had reduced plasma or serum concentrations of this vitamin<sup>5</sup>. The nervous tissue is particulary vulnerable to free radical damage. Several experimental observations have emphasized the role of oxidative stress and vitamin E deficiency in pathogenesis of neurological abnormalities<sup>6-9</sup>. Probably there is association between low alpha-tocopherol (the major vitamin E component) level in erythrocytes and multiple sclerosis in humans<sup>10</sup>.

Recently, several workers have suggested that the concentration of alpha-tocopherol in red blood cells is more useful as a measure of the vitamin status than its plasma/serum level<sup>11-13</sup>. I examined alpha-tocopherollevel both in plasma and in erythrocytes of 18 untreated patients with CD<sup>5</sup>. All of them had alpha-tocopherol concentrations in erythrocytes below the norm, but only six had reduced plasma concentrations of the vitamin. Two of the three celiacs with neurological manifestations had normal plasma alpha-tocopherol levels.

The possibility should not be excluded that a least some neurological manifestations in celiacs may have their origin in a deficiency of tocopherol<sup>3,14</sup> and correction of the vitamin status may offer some benefit for patients. Further more detailed investigation is required into this subject.

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**The Author's Response -** We thank Dr. Kamil K. Hozyasz for his interest in our study<sup>1</sup>. Dr Hozyasz suggests that some of the neurological manifestations in celiacs have their origin in deficiency of tocopherol and that correction of the vitamin status may offer some benefit for patients. We agree with him, and although not mentioned in our article all the patients had a slight reduction of serum vitamin E that was corrected after vitamin supple-

mentation. Despite the normalization of tocopherol levels, improvement was not observed. We did not measure alpha-tocopherol in red blood cells and maybe this measurement could be of help in our cases<sup>2</sup>. Although we think the nutritional concept of neurological manifestations in celiac disease (CD) can not be forgotten, we still believe that in our specific cases the immune changes are more important. The recommended daily allowance of tocopherol, based on the [alpha]-tocopherol form, is 22IU (14.7 mg) for adults and 28IU (18.7 mg) for lactating mothers<sup>3</sup>. Our patients are still receiving a 400 IU daily dose of tocopherol and neurologic status remains unaffected. We think that using this supplemental dose of tocopherol and with normalized serum tocopherol levels, it is not probable that our patients have low red blood cell levels of tocopherol, although it is still possible. We know that manifestations like dementia and ataxia have been described in patients with low tocopherol levels, but in CD neurological presentation can range from eplilepsy to polymyositis which are not conditions related to tocopherol deficiency<sup>4-6</sup>. The neuropathologic findings in gluten ataxia include

perivascular cuffing with inflammatory cells which also suggest an immune mediated event<sup>7</sup>. In conclusion, we think that vitamin supplementation should be administered to all patients with CD and neurological manifestations, although it is not possible to predict how many of this patients will recover because the immune origin of many of this symptoms will still be there.

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