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UNESCO
Satellite Trace Element Center
(Ankara)

SECOND INTERNATIONAL ZINC SYMPOSIUM

OCTOBER 2-3, 1998
Ankara - TURKEY

ABSTRACTS

ZINC AND THYMIDINE KINASE IN ACUTE LEUKEMIA AND MALIGNANT LYMPHOMAS OF CHILDHOOD (PRELIMINARY REPORT)

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Zinc is known to be involved in cell proliferation and DNA synthesis. Among the mechanisms suggested by which zinc may regulate these processes is increased expression of a DNA synthesizing enzyme thymidine kinase (EC2.7.1.21) (TK). The aim of this study was to assess the status of zinc as well as copper and Cu/Zn ratio in relation to protein levels of TK inpatients with malignancy.

Twenty five children included in this study, eight patients were at the remission stage, and 7 were first diagnosed as acute leukemia or malignant lymphoma whereas 10 were controls. Serum TK levels were determined by I 125 based immune-radioenzyme assay. Zn and Cu levels were assayed by flame atomic absorption spectrophotometry.

The p values for difference between means were 0.19, 0.10, 0.28 and 0.14 for TK, Cu, Zn and Cu/Zn ratios, respectively ($p>0.05$) between the remission, first diagnosed and control groups. The overall correlation analysis combining all groups yielded the values $r=0.17$, 0.51, 0.32 between TK and Zn, Cu and Cu/Zn, respectively. Corresponding p values were 0.41, 0.21 and 0.16.

A number of earlier studies have suggested serum zinc and copper levels as well as copper/zinc ratio as an index of the activity of the disease and the efficacy of therapy in leukemias and lymphomas. There is also in vitro evidence supported by animal studies that transcription of TK is adversely affected by zinc deficiency. This is mainly thought to be associated with transcription factors with zinc finger domains binding to the promoter region of TK gene. However it should also be noted that not all zinc finger transcription factors are activators of transcription and in fact some newly identified (eg. RIZ1, RIZ2) gene products have been shown to repress HSV-TK transcription. In this preliminary study we have failed to show a strong relationship between serum Zn, Cu and Cu/Zn ratio and serum TK in a mixed group of patients. We suggest that these indices are crude to assess risk and studies with monoclonal origin malignancies concerning detection of individual transcription factors will reveal more about the complex mechanisms to regulate cell cycle.