# **Diagnostic Value of Serum Adenosine Deaminase**

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**Resume:** A developed colorimetric method for the determination of serum adenosine deaminase (ADA) is presented. The test was applied to 87 patients - 65 adults and 22 children with the following lung diseases: pneumonia, tuberculosis, lung cancer, sarcoidosis, bronchial asthma. The high activity of the enzyme was found in tuberculosis - up to 52,438 U/I. The highest values were reported in children with primary pulmonary tuberculosis - up to 114, 232 U/I. It is believed that along with the main diagnostic methods used in pulmonology, the study of adenosine deaminase may provide some information about the etiology of some lung diseases

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## I. Introduction

Adenosine deaminase is an enzyme that represents the degradation by converting adenosine to inosine and deoxyadenosine to deoxyinosine. ADA is needed to differentiate lymphoid cells, especially T lymphocyte cells and plays a role in the conversion of monocytes into macrophages . The deficiency of ADA is impaired by cellular and humoral immunity. An increased serum concentration of ADA was found in some liver diseases, infectious mononucleosis and acute leukemia . The cerebrospinal fluid in tuberculous meningitis also shows high ADA activity compared to other diseases . The pleural fluid in tuberculosis is also of high ADA activity compared to para pneumonic and neoplastic effusions.

The aim of the present work is to develop a method for determination of serum ADA and its application in patients with various pulmonary diseases.

# II. Material and Methods:

A total of 87 patients are investigated - 65 adults and 22 children with various lung diseases. From the group of adults 25 are patients with lung cancer, 10 with pneumonia, 10 with tuberculosis, 5 with sarcoidosis and 5 healthy controls. From the child group 14 children are diagnosed with tuberculosis and 8 with non-specific pulmonary diseases. Diagnosis has been according to microbiological, radiograph, cytological and other methods.

Serum ADA activity was determined by the Karker method with some modifications. The substrate of the colorimetric reaction used adenosine from "Riedel de Haem" to prepare 26 mmol / L phosphate buffered saline (60 mmol / L) with pH 7.2. The colorimetric determination of the individual ammonia in the enzyme reaction was performed with phenol-piperochloride reagent, followed by reading at 628 nm. The activity of ADA was expressed as : "mmol of ammonia released from the substrate at 1 minute at 37C in 1 liter of test material "mmol / min / L = U / L .ADA normal values : 12-21 U/L.

## III. Results and discussion:

After checking the reproducibility of the ADA determination methodology, it was used for the study of 87 patients.

In adult patients, the following results are obtained:

Tuberculosis: 10 patients -  $32.0 \pm 3.3$  U/L

Sarcoidosis: 5 patients -  $30.0 \pm 1.2$  U/L

Carcinoma :25 patients - 20.7  $\pm$  1.8 U / L U/L

Pneumonia: 10 patients -  $19.5 \pm 2.1$  U/L

Bronchial asthma: 10 patients -  $18.3 \pm 1.4$  U/L

Healthy individuals: 5 patients -  $16.8 \pm 10.2$  U/L (fig. 1)



Fig.1 Distribution of the groups of adult patients

Patients with tuberculosis have the highest activity of the enzyme - an average of 32 U / L, almost twice as high as the healthy subjects. A higher concentration of the enzyme is observed in the groups of patients with carcinoma, pneumonia and bronchial asthma.

In the children group very high levels of the enzyme are detected in patients with tuberculosis with the highest value of 114.323 U / L. In children with non-specific pulmonary diseases, the highest reported concentration is 71, 382 U / L .

Interest in determining ADA increases after establishing the relationship between ADA deficiency and immunological dysfunction. The diagnostic significance of this enzyme in the cerebrospinal and pleural fluid is emphasized in a specific etiology of the basic process.

Conclusion: ADA is a predominant T lymphocytic enzyme and its serum activity is higher in diseases with stimulated cellular immunity. It is known that tuberculosis is one of these diseases. But not always the high percentage of T lymphocytes correlates with the elevated values of ADA / p>0.10. This indicates that the activity of this enzyme depends more on the stage of maturation of T cells than on their number. Despite the fact that the physiopathological mechanism for explanation of the increase of ADA is unclear, the clinical value of the results is highlighted in our study. In non-tuberculosis etiology, ADA is lower and has no diagnostic significance except the exclusion of a specific biochemical data process.

The importance of studying this enzyme in children, where diagnostic problems are difficult to solve with the use of research methods, should be highlighted.

The method for determining ADA is a relatively easy, inexpensive, sensitive and specific diagnostic procedure. It can be used as an adjuvant test for diagnosing tuberculosis, especially when other clinical and laboratory tests are negative and the diagnosis of tuberculosis is controversial.

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