

# Matrix Metalloproteinase as Indicator of Dreadful Swelling Activity in the Pulmonary Tuberculosis

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## Introduction

The main factors of pathogenesis in the pulmonary tuberculosis aren't only the bacterial acidity and perceptivity of the host vulnerable system to the pathogen, but also the degree of destruction of the lung towel. similar destruction processes lead to the development of grottoes, in utmost cases taking surgical interventions besides the medicine remedy. Identification of special biochemical labels allowing to assess the necessity of surgery or remedy extension remains a challenge. We consider promising labels metalloproteinases assaying the data attained from cases with pulmonary tuberculosis infected by different strains of *Mycobacterium tuberculosis*. We argue that the presence of medicine-resistant strains in lungs leading to complicated clinical prognostic could be justified not only by the difference in middles of biomarkers attention (as determined by the Mann - Whitney test for small samples), but also by the qualitative difference in their probability distributions (as detected by the Kolmogorov - Smirnov test). Our results and the handed raw data could be used for farther development of precise biochemical data-grounded individual and prognostic tools for pulmonary tuberculosis.

Pulmonary tuberculosis (TB) has a long history as a major complaint in humans and creatures. In 2017, 1.7 million people failed from the complaint substantially in developing countries. A causative agent of TB *Mycobacterium tuberculosis* (MTB) - causes severe counteraccusations for a case generally associated with lung towel destruction. By now, there's a large group of cases with expansive medicine-resistant (XDR) and multi-drug resistant (MDR) tuberculosis taking not only a long-term treatment using the newest medicines, but also surgical intervention. Thus, unravelling mechanisms of lung towel destruction and the hunt for possibilities of early opinion and new approaches to the treatment of this pathology remain on the top of the docket for recent exploration [1].

One of the promising individual directions is a discovery of specific predictors or biomarkers allowing for assessing the necessity of surgical intervention or remedy extension. The "hunt for biomarkers" is an established approach in biomedicine especially for cancer and neurodegenerative conditions. It's aimed at chancing specific motes whose attention or exertion can either define the pathological process localization or prognosticate not only the remedy success, but also the pathology elaboration. At the same time, similar approach to tuberculosis is in its first stages in malignancy of a recent high demand. A promising seeker for possible biomarkers is a group of special enzymes - matrix metalloproteinases (MMP), involved in the destruction of the

lung towel. Under normal conditions, utmost of the MMPs aren't expressed; still, their overexpression is observed during inflammation [2].

The intensity of MMP- expression is regulated by anti-inflammatory cytokines and bacterial lipopolysaccharides. MMP- enzymes are synthesized as pro-enzymes, actuated at the post-translational position with participation of proteases and regulated by specific towel impediments (TIMPs,  $\alpha$ 2-macroglobulin). The part of MMPs in the destruction of the connective towel of the lungs substantially conforming of collagen - the main structural protein of the lung - caused by *Mycobacterium tuberculosis* (MTB) has not yet been completely delved. Still, several types of metalloproteinases (MMP-9) were linked, varying their attention situations with the development of the pulmonary tuberculosis. The main part in the inauguration of the destruction process of type I collagen is attributed to MMP- 1. MMP- 8 is an element of neutrophilic fragments modulating the exertion of chemokines. Its increase in pulmonary tuberculosis together with MMP-9 reflects the inflexibility of the destructive process. During effective treatment, MMP attention reduces [3].

## Description

The data were attained from 234 cases with pulmonary tuberculosis (TB) treated at the State Research Institute of Phthisiopulmonology (SRIP) in the time period of 2009 - 2017. The average age of the cases was  $35.6 \pm 0.8$  times. There were 145 men and 88 women. All eligible cases gave their concurrence to share. There are some missing values in the dataset due to the lack of clinical data saved as handwritten records in the Institute's sanitarium. The healthy group (20 persons) was chosen among scientists and clinicians of SRIP in such a way that the average age was harmonious with the case cohort. still, for the control group, only data on clinical labels (TIMP- 1, MMP- 1, MMP- 8, MMP- 9) were available. For farther analysis, TIMP- 1 will be substantiated as TIMP [4-6].

There were two groups of cases with diagnosed (by reckoned tomography, CT) forms of pulmonary tuberculosis infiltrative TB (ITB) and fibro-cavernous TB (FCTB). The first group includes cases examined at the Institute for the first time and who weren't treated yet. Cases with the FCTB form anticipated a surgical intervention and formerly took a course of the treatment to repress a bacteria excretion. The details of marker attention measures and CT procedures are given in the styles section below. The introduced dataset includes the information on the biomarkers' attention recorded and the characteristics of tuberculosis forms, similar as a number of inflammation foci, the towel destruction volume and the total lesion volume. Information about medicine-resistance (multi-drug, extra-drug and sensitive) of *M. tuberculosis* strains was also handed. As an appurtenant material, some fresh general characteristics of cases gender, body mass indicator, etc. are added when they were available in medical records [7-10].

## Conclusion

In this donation, we present clinical data on attention of metalloproteinases and their asset, which could be useful to reveal biomarkers for the inflexibility of an inflammation process in the pulmonary tuberculosis. Due to the limited sample size, still, it's rather delicate to surely assert which labels characterize

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the inflexibility of the complaint process. still, as saying the data using two different tests (Mann – Whitney U and Kolmogorov – Smirnov) made it possible to identify some trends. The large number of outliers for resistant TB might indicate the actuality of a heavy- tagged distribution. It implies that extreme events of redundant large MMPs attention might indicate a possible presence of the medicine- resistant strain in a case's organism. also, our approach provides an fresh sapience related to the statistical analysis of similar kind of clinical data.

While the conventional approach in medical statistics generally operates with the Mann - Whitney test alone, i.e., with the comparison of middles of data samples, the difference between the probability distributions might contain important information as well. It may serve as a diagnostics criterion, which distinguishes between clinical cases indeed when the Mann Whitney test demonstrates the coexistence of standard values. The typical illustration of similar situation is either presence or absence of extreme events in the data mentioned over. Therefore, we punctuate that an fresh test for similar kind of data are needed, e.g., the Kolmogorov - Smirnov test, which compares the accretive distribution of the two data sets and allows for distinguishing between them. These two tests, used together, can round each other and get more applicable information from clinical data with multiple missing values.

## References

- Goletti, Delia, Jann-Yuan Wang and Tom H.M. Ottenhoff, et al. "Update on tuberculosis biomarkers: from correlates of risk, to correlates of active disease and of cure from disease." *Respirology* 23 (2018): 455-466.
- Henry, M.T., K. McMahon, M.X. Fitzgerald and C.M. O'Connor, et al. "Matrix metalloproteinases and tissue inhibitor of metalloproteinase-1 in sarcoidosis and IPF." *Eur Clin Respir* 20 (2002): 1220-1227.
- Seddon, Jo, Victoria Kasproicz, Naomi F. Walker and Ho Ming Yuen, et al. "Procollagen III N-terminal propeptide and desmosine are released by matrix destruction in pulmonary tuberculosis." *J Infect Dis* 208 (2013): 1571-1579.
- Ugarte-Gil, Cesar A., Robert H. Gilman, Jorge Coronel and David AJ Moore, et al. "Induced sputum MMP-1,-3 &-8 concentrations during treatment of tuberculosis." *PLoS one* 8 (2013): e61333.
- Bhavanam, Sudha, Gina R. Rayat, Monika Keelan and Steven J. Drews, et al. "Understanding the pathophysiology of the human TB lung granuloma using *in vitro* granuloma models." *Future Microbiol* 11 (2016): 1073-1089.
- Walker, Naomi F., Simon O. Clark, Tolu Oni and Nuria Andreu, et al. "Doxycycline and HIV infection suppress tuberculosis-induced matrix metalloproteinases." *Am J Respir Crit Care Med* 185 (2012): 989-997.
- Singh, Shivani, Ajay Singh, Harriet Gardiner and Jon S. Friedland, et al. "Antimycobacterial drugs modulate immunopathogenic matrix metalloproteinases in a cellular model of pulmonary tuberculosis." *Antimicrob Agents Chemother* 58 (2014): 4657-4665.
- Esmedyayeva, D.S., N.P. Alexeyeva, N.V. Sapozhnikova and V.Y. Zhuravlev, et al. "The system of matrix metalloproteinases and their role in patients with pulmonary tuberculosis." *Biomeditsinskaya Khimiya* 62 (2016): 593-598.
- Kübler, André, Brian Luna, Bruno B. Andrade and Kevin W. Bock, et al. "Mycobacterium tuberculosis dysregulates MMP/TIMP balance to drive rapid cavitation and unrestrained bacterial proliferation." *J Pathol* 235 (2015): 431-444.
- Ong, Catherine W.M., Paul T. Elkington, Sara Brilha and Cesar Ugarte-Gil, et al. "Neutrophil-derived MMP-8 drives AMPK-dependent matrix destruction in human pulmonary tuberculosis." *PLOS Pathog* 11 (2015): e1004917.

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