


Chapter 5


Bioinformatics of Tuberculosis and Post-TB Lung Damage

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ABSTRACT

*The global burden of tuberculosis stems not only from its active infectious phase but also from the enduring pulmonary sequelae collectively termed post-tuberculosis lung disease (PTLD) that can persist long after the completion of treatment. This makes tuberculosis (TB) still a worldwide health burden. The important contribution of bioinformatics to grasp the pathophysiology, diagnosis, and long-term effects of TB is investigated in this chapter. This chapter discusses computational approaches and high-throughput technologies genomics, transcriptomics, proteomics, and systems biology that have expanded our understanding of *Mycobacterium tuberculosis* (*M.tb*) biology and host-pathogen interactions. The ways in which bioinformatics techniques help to simulate immune responses that lead to PTLT, predict treatment results, and uncover biomarkers for early identification are especially underlined.*

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Additionally discussed are the possibilities of multi-omics data, machine learning algorithms, and clinical datasets to direct individualised therapy plans and enhance post-treatment monitoring.

INTRODUCTION

Mycobacterium tuberculosis (M. tb) remains the foremost infectious agent contributing to global morbidity and mortality, with an estimated 10 million new cases and approximately 1.5 million associated deaths recorded worldwide (Tomori et al., 2018; Alyerten et al., 2020). Bioinformatics has been applied to high-throughput datasets, including gene expression, protein-protein interactions, and microbiome sequencing, and has been used not only to improve diagnosis accuracy but also to clarify mechanisms underlying TB progression and resolution (Zhao et al., 2020; Li et al., 2020). By integrating computational biology and clinical data, strong biomarkers and treatment targets can be identified, thereby providing the necessary tools against both active pulmonary TB and its long-term consequences, sometimes known as post-TB lung damage (PTLD) (Ogidi et al., 2024).

The discovery of differently expressed genes (DEGs) with prognostic value is a major progress made possible by bioinformatics. Zhao et al. (2020), for instance, showed their link with treatment outcomes by aggregating several transcriptome datasets and validating peripheral blood biomarkers such as SLAMF8 and LILRB4. Li et al. (2020) also highlighted hub genes, including ICAM1, MMP9, and IL6, which are major modulators of TB pathogenesis, using gene ontology and protein-protein interaction network analysis (Li et al., 2020). Early diagnosis and customized treatment strategies are based on these computational discoveries.

Bioinformatics has also shown molecular causes of post-TB lung damage such as persistent fibrosis, bronchiectasis, and airflow resistance, they are the conditions that impact a notable fraction of TB survivors (Allwood et al., 2016; Adeloje et al., 2018). Lung tissue remodelling and chronic dysfunction following TB treatment have been associated with genetic variations in matrix metalloproteinases (MMPs) and their inhibitors (TIMPs), including MMP-1, MMP-9, and TIMP-2 (Allwood et al., 2016). An important first step towards tailored post-TB treatment, computational genomics, and host-genotype databases are being used to identify susceptibility profiles predicting who are at risk for long-term sequelae. The microbiome of lung tissues is also important in PTLD studies. Projects combining bioinformatics pipelines with metagenomic next-generation sequencing have revealed changes in microbial networks in lung tissue affected by tuberculosis (Li et al., 2023). A comparative study made possible by computational technologies, including Bowtie2, Kraken, and microbiome-specific R packages, increases our knowledge of opportunistic

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