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Health Impacts and *Mycobacterium tuberculosis* Pathogenesis

Dr. Kashis Gour*

Department of Mycobacterium Pathogenesis, University of RRG, India

Corresponding author: Kashis Gour

kashisgour@gmail.com

Department of Mycobacterium Pathogenesis, University of RRG, India

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Abstract

One of the earliest known human diseases is tuberculosis. Despite the fact that it still accounts for two million deaths annually, it remains one of the leading causes of death. Although *Mycobacterium tuberculosis* is spread via aerosol droplets deposited on the alveolar surfaces of the lungs, the primary manifestation of TB is pulmonary disease, which affects bone, the nervous system, and many other organ systems. From this point on, the disease can progress in a number of different directions, most of which are determined by how the immune system of the host responds. Extrinsic factors, such as insults to the immune system and the host's nutritional and physiological state, as well as intrinsic factors, such as the immune system's genetics, influence this response's efficacy. Since some *M. tuberculosis* strains are said to be more virulent than others, as measured by increased transmissibility and a higher incidence of both morbidity and mortality in infected individuals, the pathogen may also play a role in disease progression. There is more TB than ever before, necessitating the development of new vaccines and drugs as well as more precise and speedy diagnostics, despite the widespread use of several antibiotics and an attenuated live vaccine. In order to identify *M. tuberculosis* targets that will aid in the development of these urgently required anti-tubercular agents, researchers are utilizing information gleaned from the complete sequence of the *M. tuberculosis* genome as well as new genetic and physiological techniques. Emerging pathogens known as environmental mycobacteria are responsible for opportunistic infections in both animals and humans. Human-mycobacterial interactions have a wide range of health effects that are likely much more complex than currently understood. Municipal water contains environmental mycobacteria that preferentially survive chlorination and use it as a vector to infect humans. Boundless chlorination of water has likely chosen more safe ecological mycobacteria species and possibly makes sense of the shift from *M. scrofulaceum* to *M. avium* as a reason for cervical lymphadenitis in kids. As a result, mycobacterial ecology has been impacted by human activity. Despite the fact that environmental mycobacteria's unique cell wall architecture confers high biocide and antibiotic resistance and their hydrophobicity facilitates nutrient acquisition, biofilm formation, and aerosolization, environmental mycobacteria's slow growth and hydrophobicity appear to be disadvantages. Environmental mycobacteria are major human pathogens due to their remarkable stress tolerance. Mycobacteria from the environment invade protozoa and exhibit parasitic and symbiotic relationships. Animals' molecular mechanisms for mycobacterial intracellular pathogenesis likely originated from protozoan survival mechanisms. Environmental mycobacteria may also play a role in allergies, chronic bowl diseases, immunity to other pulmonary infections, and the effectiveness of the Calmette-Guerin vaccine.

Keywords: *Mycobacterium tuberculosis*; Pathogens; Immune system; Organ systems; Vaccination

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Introduction

Despite the widespread use of a live attenuated vaccine and a number of antibiotics, tuberculosis (TB), one of the earliest human diseases ever recorded, continues to be one of the leading causes of death among infectious diseases. To stop the worldwide TB epidemic that kills two million people every year, we need new drugs and vaccines [1]. The genetics and physiology of *M. tuberculosis* and related mycobacteria must be studied in depth if new anti-tubercular agents are to be rationally developed. Understanding how *M. tuberculosis* and the host interact is just as important for understanding how these bacteria get around the defenses of the host and cause disease. *M. tuberculosis* genes that are or could be involved in virulence are identified using the methods described in this review [2]. In the not-too-distant future, some of these genes and the proteins they encode, as well as some that have recently been discovered, ought to provide brand-new bacterial targets that could be utilized in the development of vaccines, drugs, and more selective diagnostic reagents. Initially, a summary of various aspects of TB, including the disease's history, its clinical manifestations, and host and bacterial responses during infection, is presented to help the reader better comprehend the context for these strategies [3]. This initial discussion can only touch on a small portion of the many topics covered due to space constraints. Numerous excellent books and reviews provide additional background information.

Nontuberculous mycobacteria's clinical manifestations and treatment have been the subject of numerous excellent reviews. The purpose of this review is to provide a broader perspective on the health effects of human-environmental mycobacteria interactions and to discuss a number of factors related to those interactions. A fascinating group of pathogens that can affect humans, animals, and birds are the environmental mycobacteria [4]. They have a significant impact on human morbidity and mortality as well as agriculture's economic viability.

TB can manifest in a variety of ways, as will be discussed in the following section, including a form that attacks bone and causes skeletal deformities. Because hard tissues like bone can be preserved for thousands of years, it is almost possible to identify people who died of bone TB more than 4,000 years ago. The prevalence of tubercular deformities in ancient Egyptian skeletons suggests that the disease was widespread in that population [5]. Similarly deformed bones have also been found in Neolithic sites in Italy, Denmark, and Middle Eastern nations, suggesting that TB existed worldwide up to 4,000 years ago. Much recent research has focused on the origin of *M. tuberculosis*, the TB virus. It is believed that the bacteria in the genus Mycobacterium, like other actimomycetes, originated in soil and that some species evolved to live in mammals [6]. The domestication of cattle, which is thought to have occurred between 10,000 and 25,000 years ago, would have allowed a mycobacterial pathogen to spread from domesticated livestock to humans. As the bacterium adapted to a new host, it would have evolved into *M. tuberculosis*, which is closely related to M. pylori. In particular, it has been hypothesized that *M. bovis*, which in cattle causes a disease similar to tuberculosis, was *M. tuberculosis's* hypothetical evolutionary predecessor. Due to the fact that it was developed prior to the genomes of the human and animal pathogens M. africanum, *M. microti*, and *M. canetti*, as well as *M. tuberculosis* and *M. bovis*, being characterized by DNA sequencing and related methods, this hypothesis is now considered to be doubtful in light of new data [7]. The DNA sequences of the members of the *M. tuberculosis* complex have been found to be more than 99.9% identical in these studies. However, the presence of rare synonymous single-nucleotide polymorphisms (sSNPs) makes it possible to differentiate between these closely related bacteria. A study of the distribution of deletions and insertions in the genomes of the *M. tuberculosis* complex provides strong evidence for the independent evolution of both *M. tuberculosis* and *M. bovis* from another precursor species, possibly related to *M. Canetti*. SSNP analyses also suggest that *M. bovis* evolved at the same time as *M. tuberculosis* [8].

Hippocrates describes patients with consumption (the Greek term is phthisis), i.e., wasting away associated with chest pain and coughing, frequently with blood in the sputum, in the seventh century B.C. Assyrian clay tablets describe patients coughing blood [9]. At this point, the recurrence of portrayals of patients with TB-like side effects demonstrates that the infection was at that point very much settled in. It is possible that Indo-European cattle herders who were infected with tuberculosis brought the disease with them when they moved to these areas and came into contact with infected cattle. The hypothesis that Indo-Europeans brought *M. tuberculosis* to Europe and Asia during their migrations has also been supported by analysis of various human phenotypic traits, such as lactose tolerance, which are linked to the raising of cattle and selection for the ability to utilize milk [10].

Conclusion

In the coming years, we anticipate an increase in interactions between humans and mycobacteria. There will probably be more clinical cases of environmental mycobacteria as a result of this. Chlorination of drinking water, selecting mycobacteria by reducing competition, disinfection efforts in medical and industrial settings may also select for mycobacteria, and an increasing proportion of our population with predisposing conditions, most notably AIDS, age, and immunosuppressive regimens, such as transplantation, are the three primary drivers of this rise. As the drug-resistant human immunodeficiency virus unavoidably spreads, we also anticipate a rebound in the number of *M. avium* complex infections among AIDS patients. It is unknown whether

environmental mycobacteria are also to blame for the rise in autoimmune diseases.

Second, new species of opportunistic mycobacteria that thrive in the environment will continue to be discovered. This is partly due to the growing use of disinfectants to "sterilize" habitats, the rising number of people who are predisposed to environmental mycobacterial infection, and more rapid and sophisticated identification methods like 16S rRNA gene sequencing. Humans, on the other hand, have a significant impact on the ecology of mycobacteria. *M. avium* appears to have replaced *M. scrofulaceum* in the environment, possibly as a result of widespread chlorination of drinking water.

Acknowledgement

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Conflict of Interest

None

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