Mycobacterial Diseases
ISSN: 2161-1068

Prasanthi and Murty, Mycobact Dis 2014, 4:6
DOI: 10.4172/2161-1068.1000172

Review Article Open Access

A Brief review on Ecology and Evolution of Mycobacteria

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Rec date: Aug 20, 2014, **Acc date:** Oct 13, 2014, **Pub date:** Oct 31, 2014

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Abstract

Tuberculosis is one of the grand old diseases and among the top burdened diseases of the world. Most Mycobacteria are environmental saprophytes except *Mycobacterium tuberculosis* and *Mycobacterium leprae*, which are obligate pathogens. However, several studies indicate that the selection of the pathogens in an ever changing environment do occur by a variety of deletion mutations over time. *Mycobacterium tuberculosis* might have originated from an environmental ancestor. Some studies even predict that some of the environmental saprophytic mycobacteria may become pathogens in near future because of the selection pressure of the environment. In this context, this article briefly outlines the ecology and evolution of the Mycobacteria.

Keywords: Mycobacterium; Ecology; Evolution

Tuberculosis, a grand old disease, maintains even today, it's rank as one among the top burdened diseases in the world, whether infectious or non-infectious. Others are Malaria, HIV, HBV, HCV, Leprosy, Diabetes, Hypertension, Cancer, Arboviral diseases etc.

Mycobacteria are basically environmental saprophytes and their pathogenic capabilities were selected by environmental challenges. They were thoroughly studied from sources like soil, animals, human beings, marshland, water, vegetation etc. Distribution of saprophytic Mycobacteria is dependent on environmental conditions including soil pH. Although rarely causing overt infection, these environmental organisms are able to elicit an immune response in man, as it certainly modifies the host response to the subsequent contact with pathogenic mycobacteria and also influences the protective effect of BCG [1].

Except *Mycobacterium tuberculosis* and *Mycobacterium leprae* all other species of Mycobacteria are environmental saprophytes. Mycobacterium tuberculosis is unique in being obligate pathogen contains no environmental saprophytic strains. Though Infectivity and pathogenicity have developed in this species a long time ago in the history of life on earth after which marked fluctuations in the earth's climate could have eliminated the environmental strains [2]. Gutierrez and her colleagues concluded that an early progenitor of *M. tuberculosis* was present in East Africa as early as 3 million years ago, and they suggested that it might have infected early hominids at that time [3]. According to a study, modern strains of *M. tuberculosis* have originated from a common ancestor about 20,000-15,000 years ago [4]. Tuberculosis in Egypt was documented more than 5000 years ago. Typical skeletal abnormalities of tuberculosis, including characteristic Pott's deformities, were identified in Egyptian mummies and were clearly depicted in early Egyptian art [5,6].

It is interesting to note that how the environmental Mycobacterial species have shifted from environment to take up an existence as an obligate pathogen. Evolution is defined as the adaptation of an organism to a new or changing environment. Evolution is therefore a dynamic process which generates diversity during the process of adaptation. For example, resistance to environmental toxins might also confer protection from the killing effects of antibiotics and host immune cells. Alternatively, the bacteria may routinely encounter and defend themselves against the phagocytic cells of animal hosts [1].

These variations occurred due to mutations, which lead to loss of various properties. Generally the process of random mutation is known to cause many more deletions than acquisitions of new features. Most of the environmental Mycobacteria are genetically complete forms and certain type of deletional mutations render the strains difficult to survive in the environment and more adaptable to parasitize a man when there is an opportunity [1].

Fortunately a very few saprophytic Mycobacteria are established as human pathogens. Virulence tends to increase as the bacterial strains adapt to the new environment. According to Darwinian Theory, development of virulence is a random event due to genetic variations. In this process, due to numerous deletional mutations, most of them lose the ecological battle for survival and disappear from the environment. This is due to the fact that *M.tuberculosis* is a strict mesophile, which has a narrow temperature range in which it will grow. Various genetic events involving deletion mutations over several years have resulted in the development of virulent strains of *M.tuberculosis* from a non-virulent progenitor [1]. It is well known that, *M. tuberculosis* belongs to the Mycobacterium tuberculosis complex (MTC), comprised of the agents responsible for TB or TB-like disease. The bacteria in this group share identical 16S rRNA sequence and greater than 99.9% nucleotide identity. *M. tuberculosis, M. africanum, M. microti,* and *M. bovis* are regarded as the four traditional species of the MTC. Recent advances in molecular biology made it possible to understand the genome-based phylogeny of the Mycobacteria, which is relevant for TB control. The use of DNA fingerprinting patterns where the samples are genotyped by restriction-fragment-length polymorphisms using genetic attributes specific to the MTC as markers, are valuable for tracking MTC disease [7,8]. With the availability of whole genome sequences of various

mycobacterial species, the comparative genomic analysis has shown that gene loss is a significant part of the ongoing evolution of the slow-growing mycobacterial pathogens [9].

Genetic variations like Single-nucleotide polymorphisms (SNPs) can result in silent amino acid substitutions that select for evolutionary benefit. The SNPs can be either synonymous (coding sequence remains unchanged) or non-synonymous (alteration in coding sequence) [4].Genomic comparison of multiple MTC strains has made possible to understand the role of SNP markers in the evolution, pathogenesis and epidemiology in *M. tuberculosis* and *M. bovis*. The high ratios of non-synonymous to synonymous mutations across coding sequences within MTC genomes suggest divergence of *M. bovis* and *M. tuberculosis* from a common progenitor. This genomic flexibility in the *Mycobacterium tuberculosis* complex permits an efficient host adaptation [10,11].

Another indication to show that *M.tuberculosis* might be a genetically depleted organism is its sensitivity to anti-tubercular drugs in contrast to environmental species which are mostly resistant to these agents. Antibiotic resistance enables organisms to survive in a highly competitive environment. Once removed from such an environment, there would be no selective pressure for the maintenance of such resistance. Thus the relative sensitivity of *M.tuberculosis* to antibacterial agents is the result of a long period of obligate parasitism. Antibiotic resistance may be lost or acquired by mutation. Other examples are *M.paratuberculosis*, which fail to synthesize mycobactin, a substance that is essential for iron uptake and *M.lepramurium* which can be cultivable with great difficulty, are genetic variants evolved from their natural ancestors [1].

Few studies predict an increasing incidence of interactions between humans and Mycobacteria in coming years. This will likely to result in more clinical cases of environmental mycobacteria. Three major factors driving this increase are (i) disinfection of drinking water with chlorine, selecting mycobacteria by reducing competition, (ii) disinfection attempts in medical and industrial settings may likewise select for mycobacteria and (iii) the increasing percentage of population with predisposing conditions, most notably AIDS, age and immunosuppressive regimens, e.g., after transplantation [12,13].

The environmental mycobacteria are slow growing relative to other microorganisms in water and soil which suggests that they are poor competitors. However, the compensating factors such as the hydrophobic, lipid-rich impermeable envelope, biofilm formation, acid resistance and metabolism of recalcitrant carbon compounds permit survival and growth of the environmental mycobacteria in a wide range of natural and human-engineered habitats. Understanding the dynamics of the ecology and evolution of various mycobacterial species greatly helps in devising the treatment or control strategies [14,15].

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