OPEN ACCESS Freely available online

Journal of Infectious Diseases & Preventive Medicine

Opinion Article

A Short Note on the Immunopathology of *Mycobacterium*

Ervin Khazi*

Department of Infectious Disease, Shahid Beheshti University, Tehran, Iran

DESCRIPTION

A fundamental question in bacterial speciation is how sequence clusters of closely related strains emerge and persist. These clusters arise despite significant variation in colonization, virulence, transmissibility and other clinically important phenotypes. For relatively clonal species, sequence clusters are characterized by sharp and unambiguous boundaries. Some of the most epidemiologically important pathogens such as *Mycobacterium tuberculosis* and *Yersinia pestis* evolve clonally, which means there is little or no recombination occurring between strains. The paradigm of the systemized search for the microbial basis of disease, followed by the development of antimicrobial and other therapies to eradicate these disease-causing agents is not firmly established in human and veterinary clinical practice.

In highly recombining species, the mechanisms of how sequence clusters pull away from the force imposed by gene flow between clusters and subsequently remain stable is not so straightforward. If the recombination rate, measured as the rate at which polymorphisms accumulate through recombination relative to mutation, varies over time or under spatially and temporally fluctuating selective pressures, this might allow some lineages to form genetically cohesive clusters. Hybrid or mosaic genotypes that are those that are intermediate forms as a result of recombination between two species are likely to create clusters that are not neatly partitioned into consistent identities.

For many bacteria, the spatial isolation of lineages or allopatry can give rise to local variants. In ST329-MRSA, global patterns of diversification and dispersal reveal a strong geographical clustering at continental, national and city scales. At the global scale, genomic comparisons indicate that three monophyletic clades, which mostly represent European, Asian and South American populations, reflect multiple independent exports from Europe over only a few decades. On the other hand, country-wide geographical patterns are consistent with patterns of human movement between cities. Many infectious diseases including those caused by bacterial pathogens are constrained in their geographical distributions by ecological barriers to the spread or establishment of populations, even in the face of the homogenizing forces of human migration. In some pathogens, such ecological barriers may be due to differences in host association and vector dispersal. The phylogeographical structure of bacterial pathogens is expected to mirror the structure observed in their reservoir hosts, but the association may not always be as straightforward as expected. An excellent example is the spirochete *Borrelia burgdorferi* sensu lato species complex, the causal agent of Lyme disease, which consists of >20 species. In multiple *Borrelia* species, the distribution and migration of both host and vector appear to greatly determine the geographical distribution of the bacterium.

In recent studies, limited geographical structuring between countries in populations of *Borrelia* spp. associated with birds was observed, likely as a result of higher rates of migration of the host, but there exists a strong signal in geographical structure in the bacterium associated with the small mammals. In another study, B. *burgdorferi* populations from Europe and the United States isolated from human patient's exhibit an overlap of sequence types, which is in contrast to populations found in tick vectors.

In some cases, strains coexisting within the same host species remain isolated despite having ample opportunity for genetic exchange. This has been observed in *Campylobacter jejuni*, a gut colonizer of many animal species and a causal agent of gastroenteritis in humans. Despite having a high degree of niche overlap and the ability to readily combine with each other in vitro, two generalist lineages possess separate gene pools. A cryptic ecological barrier within the host appears to exist between the two lineages, which likely explain the lack of gene flow between them. The exact nature of this barrier however remains unclear. In other cases, genetic barrier can arise that prevent recombination between strain lineages; this has been reported for B. *pseudomallei* where lineage-specific restriction-modification systems carried on mobile genetic elements, allow recombination within but not between clades.

The majority of the 60 species of the genus *Mycobacterium* are harmless, although a few cause serious disease in humans and other animals. Studies with M. *tuberculosis* reveal it survives in the normally hostile environment of the phagosome by reducing fusion between the phagosome and lysosomes. It also prevents the full acidification of the phagosome by reducing the fusion of

Correspondence to: Ervin Khazi, Department of Infectious Disease, Shahid Beheshti University, Tehran, Iran, E-mail: ekhazi@gmail.com

Received: 10-Feb-2021, Manuscript No. JADPR-22-16210; **Editor assigned:** 15-Feb-2021, PreQC No. JADPR-22-16210 (PQ); **Reviewed:** 03-Mar-2022, QC No. JADPR-22-16210; **Revised:** 09-Mar-2022, Manuscript No. JADPR-22-16210 (R); **Published:** 16-Mar-2022, DOI:10.35841/2329-8731.22.10.261.

Citation: Khazi E (2022) A Short Note on the Immunopathology of Mycobacterium. J Infect Dis Preve Med. 10:261.

vesicles containing the proton pump. Precisely how the bacterium controls the fusogenicity of the phagosome in which it resides remains an interesting puzzle and there is considerable interest in identifying the genes that underline this property. Prior to the emergence of MDR strains, the success rate of drug treatment was over 90%. Similarly, the vaccine varies widely in efficacy among different geographical locations and no improvements have been made to the vaccine since it was first developed. The mixture of complacency and poor management has allowed tuberculosis to escalate into a global pandemic that we will probably now never be able to eradicate. Sequence clusters can be observed at different scales, from geographical distributions to within host and vector species. These sequence clusters may fuse through recombination or remain distinct from each other because of ecological or genetic barriers to recombination. The question as to whether these clusters are biologically meaningful must be addressed on a case-by-case basis.