



Principles behind Bacterial Pathogenicity

Eduardo Groisman^{*}

Department of Microbiology and Immunology, Tulane University Health Sciences Center, New Orleans, USA

DESCRIPTION

Pathogenic bacteria use a variety of ways to cause disease in human hosts. Bacterial pathogens produce a number of chemicals that bind to host cell targets, allowing them to support a variety of host responses. Bacterial molecular pathways for interacting with the host may be specific to some pathogens or conserved across species. The identification and characterization of all of these distinct techniques is crucial in the fight against bacterial illness. Bioinformatics and the availability of full genome sequences for some bacterial diseases will lead to significant progress toward this objective.

Globally, infectious diseases are the major cause of death. New infectious diseases are emerging, but the re-emergence of lethal infectious diseases, as well as the rising incidence of antibiotic resistance strains, poses a serious threat to human health and welfare. Significant data has recently emerged indicating that a wide range of microbial pathogens use similar mechanisms to generate infection and disease [1]. Many different bacterial pathogens, for example, have similar processes for adhering to, invading, and damaging host cells and tissues, as well as surviving host defenses and establishing infection. Many of these infectious similarities usually relate to the acquisition of huge blocks of virulence genes from a common microbial ancestor, which can then be horizontally transferred to other bacteria. The continual appearance of new strains of bacterial pathogens, many of which are resistant to various medications, is also directly responsible for the horizontal spread of vast blocks of virulence determinants. Antibiotic resistance in bacterial infections has become one of the most pressing challenges confronting critical care physicians. Understanding the molecular processes of microbial virulence, as well as the creation of innovative vaccines and other therapeutic agents for the treatment and prevention of infectious diseases, requires a better understanding of the common themes in microbial pathogenicity [2].

The genetic content of bacterial genomes is prone to rapid and dramatic change due to a variety of processes known as "horizontal gene transfer." Horizontal gene transfer appears to play a key role in the genetic evolution of emerging bacterial diseases, according to new research. Horizontal gene transfer refers to the transfer of genetic components from a donor organism directly into the genome of a recipient organism, where they create genomic islands blocks of DNA containing mobile genetic material. Pathogenicity islands are genomic islands that contain huge blocks of virulence determinants (adhesins, invasins, toxins, protein secretion systems, antibiotic resistance mechanisms, and so on).

Pathogenicity islands were first detected in the genomes of pathogenic E coli strains, but have since been discovered in the genomes of a variety of human, animal, and plant pathogens (Salmonella, Vibrio, Shigella, Yersinia, Listeria, S aureus, and others) [3,4]. Pathogenicity islands are extensive genomic DNA sections (about 10-200 kilo bases) seen in pathogenic bacterial strains but not in non-pathogenic members of the same or related species' genomes. Even though their G+C content differs significantly from that of the host microbe's genomes they are frequently flanked by direct repeats they are frequently associated with tRNA genes they are associated with integrate determinants and other mobility loci and they exhibit genetic instability, pathogenicity islands are thought to have been acquired as a block by horizontal gene transfer [5]. Plasmids and bacteriophages can also be transported horizontally, in addition to pathogenicity islands. Transformation, transduction, and conjugation all three methods for genetic exchange or transfer across bacteria appear to be critical for harmful species evolution. The full genome sequences of numerous key bacterial diseases were determined and analysed, revealing that horizontal gene transfer may be substantially more widespread than previously thought. As a result, a better understanding of bacterial pathogen evolution will be required to decipher the virulence mechanisms of emerging and re-emerging infectious diseases, as well as changes in virulence and drug resistance associated with these infections, in order to develop effective diagnostic and therapeutic strategies.

Antibiotics resistance which was discovered, more than 50 years ago, transformed medical treatment of infectious bacterial infections. However, the increasing use of antibiotics in recent

Correspondence to: Eduardo Groisman, Department of Microbiology and Immunology, Tulane University Health Sciences Center, New Orleans, USA, E-mail: groisman@18gmail.com

Received: 29-Mar-2022, Manuscript No. CMO-22-16577; Editor assigned: 01-Apr-2022, Pre QC No. CMO-22-16577 (PQ); Reviewed: 18-Apr-2022, QC No. CMO-22-16577; Revised: 22-Apr-2022, Manuscript No. CMO-22-16577 (R); Published: 29-Apr-2022, DOI: 10.35248/2327-5073.22.11.281.

Citation: Groisman E (2022) Principles behind Bacterial Pathogenicity. Clin Microbiol. 11:281.

Copyright: © 2022 Groisman E. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

decades has resulted in the creation of antibiotic-resistant variants of numerous bacteria, posing a severe global danger to modern medical practise. Antimicrobial resistance has developed in both Gram negative and Gram positive microorganisms. Diarrhoeal pathogens including Shigella, Salmonella, E coli, and Enterococcus faecium respiratory pathogens like Klebsiella pneumoniae and P aeruginosa urinary tract pathogens E coli, and M tuberculosis, which remains the leading like cause of death from a single infectious disease worldwide. Furthermore, methicillin-resistant S aureus, one of the most common nosocomial infection causative agents, and vancomycin resistance in Gram-positive organisms such as Enterococcus spp. and S aureus are posing significant challenges to modern clinicians in the effective treatment and management of infectious diseases. Evidence suggests that when Bacteroides bacteria with conjugative transposons (all producing tetracycline resistance) are exposed to low quantities of tetracycline, there is a 100-fold increase in gene transfer. Tetracycline resistance is also induced by sub inhibitory doses of the antibiotic in S pneumoniae organisms carrying the tet(M) gene.

Future Prospective Recent advancements in high-throughput polymerase chain reaction and DNA sequencing techniques, as well as microarray-based gene expression profiling, have enabled scientists to quickly determine the complete genomic sequences of both microbial pathogens and eukaryotic hosts, as well as measure gene expression levels and provide a molecular description of the events that occur after infection. The application of these methods to the genomes of microbial pathogens and their eukaryotic hosts, in combination with efficient analytical tools and genome-scale approaches to studying gene expression, is revolutionizing the development of new tools for infectious disease diagnosis, prognosis, and clinical management. The existence of a large number of full genome sequences of bacterial pathogens has significantly assisted our understanding of the infectious disease process, revealing that many of these species use similar processes to induce infection and disease.

REFERENCES

- 1. Anderson RM, May RM. Infectious diseases of humans: dynamics and control. Oxford university press. 1992.
- Finlay BB, Falkow S. Common themes in microbial pathogenicity revisited. Microbiol Mol Biol Rev. 1997; 61(2):136-69.
- Ziebuhr W, Ohlsen K, Karch H, Korhonen T, Hacker J. Evolution of bacterial pathogenesis. Cell Mol Life Sci. 1999; 56(9):719-28.
- Lindsay JA, Ruzin A, Ross HF, Kurepina N, Novick RP. The gene for toxic shock toxin is carried by a family of mobile pathogenicity islands in Staphylococcus aureus. Mol Microbiol. 1998; 29(2): 527-43.
- 5. Hacker J, Kaper JB. Pathogenicity islands and the evolution of microbes. Annu Rev Microbiol. 2000; 54(1):641-79.