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Commentary

Applications on Antibiotic Therapy in Treatment of Bacterial Infections

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1. Description

An antibiotic is a particular class of antimicrobial agent that works against bacteria. Antibiotic drugs are frequently employed in the treatment and prevention of bacterial infections because they are the most effective type of antibacterial agent. Bacteria may be killed or have their growth suppressed. Only a few antibiotics also have antiprotozoal properties. Medications that block viruses are known as antiviral drugs or antivirals rather than antibiotics since viruses like the common cold or influenza cannot be treated with antibiotics. Since the discovery of penicillin in the 1940s, antibiotics have saved countless lives and are considered a miracle of modern medicine. Nearly all of the current antibiotic classes were discovered during the antibiotics' "golden age," which spanned until the 1970s. Since the 1970s, a method has been used for this aim which involves studying a drug's pharmacokinetics and pharmacodynamics in order to maximize antibiotic therapy. The conventional strategy looks into the relationship between an antibiotic's concentration in a biological fluid and its effect on a patient's response to treatment for clinically significant bacteria. Studies were able to identify the main factors influencing antibiotic exposure and treatment efficacy starting in the 1980s. Although the mortality rates for many different types of infections have been successfully decreased by the standard strategy to evaluating exposure response connections and directing antibiotic prescription, it still has significant drawbacks for some individuals.

Using conventional drug-exposure targets to organisms with developing resistance mechanisms or to particular patient populations presents additional difficulties. Traditional targets may not accurately predict patient outcomes in various situations for a variety of reasons. First, due to laboratory and biological variability, Minimum Inhibitory Concentrations (MICs) a measure of antibiotic action are largely unreliable in targets. Second, utilizing data from a single time point and a procedure that depends on the experimental design.

Finally typical targets are not optimized to prevent bacteria that cause infections from developing antibiotic resistance. *In-vitro* and animal infection models, which describe medication activity on the basis of the pace and extent of pathogen killing as assessed by viable colony counting are common in traditional ways to optimize antibiotic therapy. For instance, *in-vitro* dose-ranging experiments are used to determine an antibiotic's index the exposure metric that best represents an agent's killing activity. Although these conventional methods give information on antibacterial efficacy against different infections they are unable to identify the exact processes by which drugs work or how pathogen resistance develops and because of the brief duration they may overstate pathogen death. Understanding a medication's mechanism of action within the pathogen it targets and the pathogen's subsequent response to the drug is essential for providing the best possible antibiotic therapy.

Significant attempts have been undertaken to pinpoint the genes responsible for antibiotic resistance in order to understand how the pathogen reacts to antibiotic therapy. Currently genomic-based tests like a molecular fast diagnostic test can be used in clinics to find bacterial resistance genes. Furthermore little is known about how the antibiotic affects bacterial cells globally

including gene regulation expression and metabolic disruptions. In response to antibiotic treatments, it is now possible to simultaneously quantify the global gene expression profiles and thousands of proteins and metabolites in bacterial cells. This is made possible by the rapid development of high-throughput sequencing techniques and ultra-sensitive mass spectrometry technology. Finding these pathways can help in the selection of antibiotic doses that maximize bacterial killing and prevent resistance development. Antibiotic exposure an integrated analysis of bacterial genomes, proteomics and metabolomics data can clarify the dynamics of cellular responses to antibiotic exposure at the systems level. To illustrate the significance of dose optimization and combination therapy the integration of correlative and data after polymyxin exposure demonstrated rapid resistance development in several Gram-negative bacteria. The mechanisms of synergy can also be better understood by contrasting the cellular responses after exposure to immunotherapy and combination therapy.