Antimicrobial Resistance: A Review on Progression and Future Changes

Sachleen Kaur
(ORC-ID: 0009-0002-1466-816X)

Abstract:

Antimicrobial resistance is a growing concern for the medical field due to mutations resulting in increased virility, transmission rates, and patient fatality rates. Researchers around the world are trying to combat this urgent issue with many different techniques. In order to do so, it is necessary to gain a thorough scientific understanding of how resistance occurs. This review explains how antimicrobial resistance is gained by pathogens such as *Staphylococcus aureus*, current day treatments, and future mechanisms to slow the rate of resistance.

Introduction to Antimicrobial Resistance:

Antimicrobial resistance occurs when a pathogen, such as a virus, bacteria, or fungus, gains a level of resistance to current treatments. This is a critical and pertinent medical issue as resistant pathogens cannot be treated using the same antibiotics, making them more dangerous. This results in an increased risk of disease propagation to larger and immuno-compromized populations. There are many reasons for this resistance and multiple ways it can be transferred, which are being analyzed in several ongoing research initiatives to limit antimicrobial resistance in the future.

How Resistance Occurs:

Antimicrobial resistance arises through natural selection. For example, in bacteria, when the selective pressure of antibiotics is applied, then the bacteria without the resistance gene die. Those with the gene for resistance, whether gained by a mutation or through a plasmid, survive.[1]

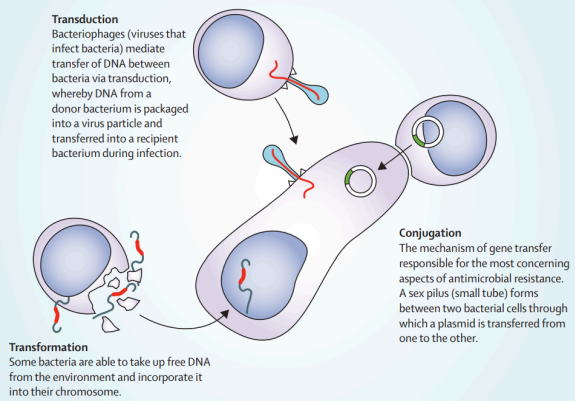

Figure 1: A description of how DNA is transferred between bacteria through plasmids[1]

These resistant strains of bacteria proliferate and become more common, explaining why resistance is so common now. One of the best examples of this occurrence is penicillin treatment against *Staphylococcus aureus*. Penicillin is a β-lactam antibiotic that works by inhibiting the cross-linking of peptidoglycan, which is the main component of bacterial cell walls. *S. aureus* gained resistance to penicillin as the bacteria with the *blaZ* gene survived treatment. The *blaZ* gene encodes for the β-lactamase enzyme which hydrolyzes the beta-lactam ring, a necessary part of penicillin-binding proteins (PBPs). Today, 90% of *S. aureus* is resistant to penicillin[2]. A similar course of events occurred with methicillin and the *mecA* gene, leading to the creation of methicillin-resistant *S. aureus* (MRSA).[2]

Therapeutics for Resistant Bacteria:

Resistant bacterias must be treated with novel antibiotics, and MRSA is now being treated with mupirocin which has a different operating technique.[3] The use of antibiotics can also be optimized by first using a loading dose which sets the baseline for future treatments and reduces the chance of overuse of antibiotics.[4] To slow the increasing amount of resistant strains of bacteria, the use of broad-band antibiotics should be controlled, and specific antibiotics targeted for specific bacteria should be used instead.

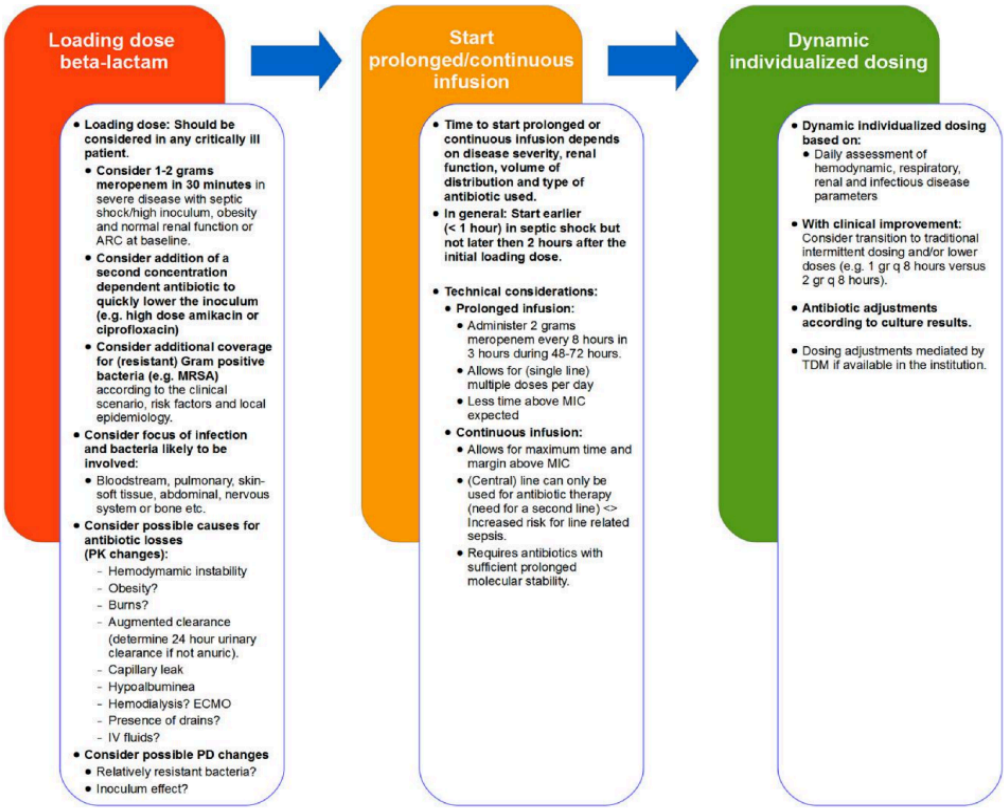

Figure 2: A detailed description of a new dosing technique to decrease resistance through a personalized treatment plan.

Resistance in Viruses and Bispecific Antibody Treatment:

Viruses also have antimicrobial resistance, caused by the similar natural selection experienced by bacteria. Viruses are DNA in a protein case, and being non-living, they mutate at a much faster rate, explaining why there are so many new strains of viruses every year. To combat viruses, the immune system 'flags' these pathogens using antibodies which call leukocytes to destroy them. A new class of antibody known as bispecific antibodies can be used against resistant virus strains such as HIV. Bispecific antibodies have two binding sites, one which binds to the pathogen and the other which binds to an immune system cell. This leads to more efficient targeting and a higher survival rate.[5]

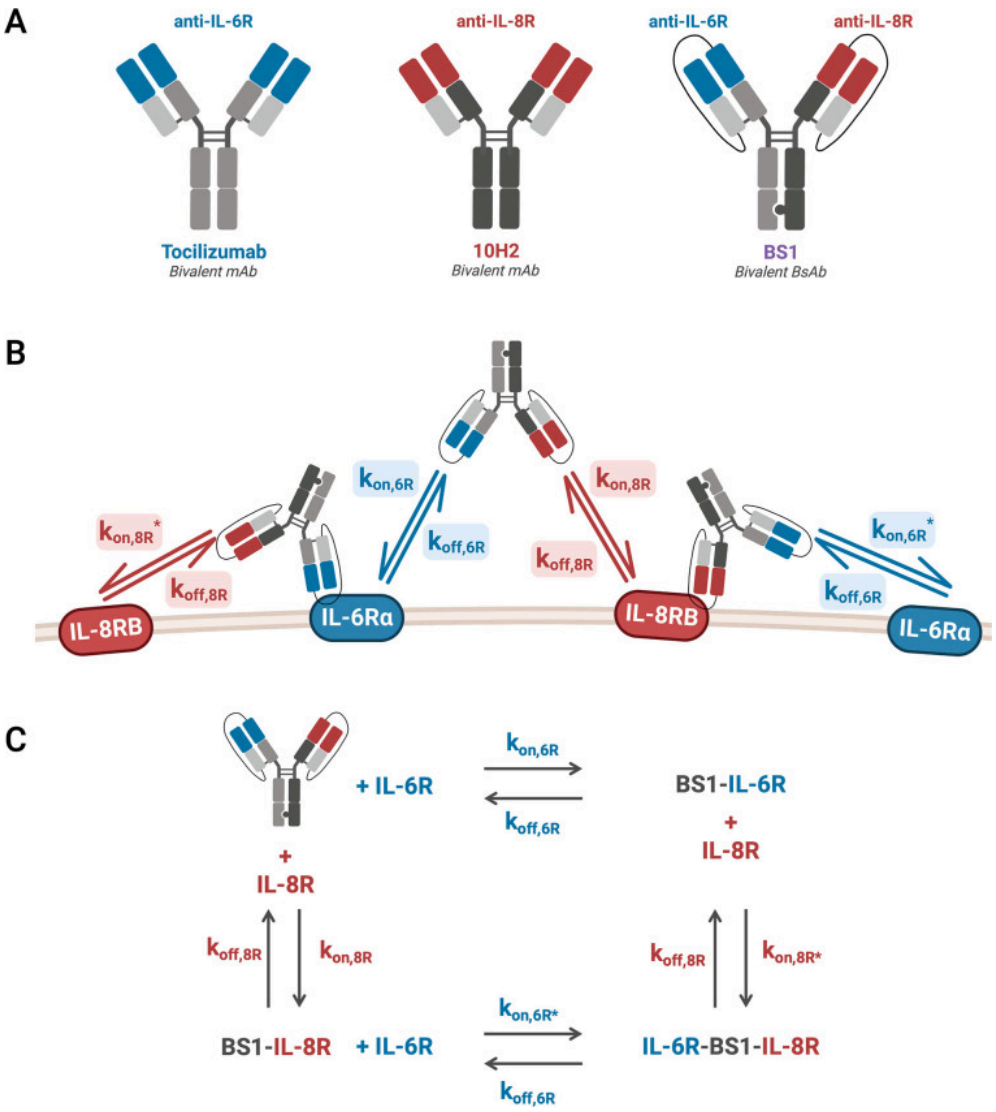

Figure 3: A comparison of monoclonal (mAb) and bispecific (BsAb) antibodies.[6]

Computer models have revealed that using one bispecific antibody is much more effective than using two monoclonal antibodies, and future tests can create bispecific antibodies for viruses that currently have no cure.[6] Additionally, since bacteria and fungi have different surface receptors from human cells, bispecific antibodies can be extended for their use.

Conclusion:

Antimicrobial resistance is a critical issue for current medical research, and it has many possible treatments that can be used in the future. Resistance occurs through natural selection and is prevalent in bacteria, viruses, and other pathogens. Through using new antibiotic treatment plans and bispecific antibodies, the production of resistant strains of pathogens may be controlled. Hence, the prevalence of highly contagious pathogens will decrease, leading to better patient outcomes.

References:

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