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Perspective

The Effects of Antibiotics on Bacterial DNA

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1. Description

Antibiotics target specific DNA gyrase have become a diagnostic roaring success for the last few years, and the occurrence of resistant bacteria has bolstered the hunt for new gyrase inhibitors. DNA gyrase is a type II topoisomerase that may create negative supercoils into DNA while consuming ATP. It is required by all bacteria but not by higher eukaryotes, making it a prom-ising target for antibacterial agents. The fluoroquinolones are a good example of gyrase-tar-geted medications, but with the rise in bacterial resistance to these drugs, we need to find new chemicals as well as novel ways to inhibit this enzyme. We examine existing gyrase-specific medicines and toxins, as well as the potential for generating new antibacterial drugs that target this enzyme.

The mechanism of supercoiling in gyrase makes it a good target for antibacterial medicines. The intricacies of this mechanism are still being researched, although biochemical and structural data largely support a concept known as the "two-gate mechanism." The N-terminal domain of GyrB (referred to as the N-gate), the GyrA–GyrB–DNA interface, where the DNA is cleaved (referred to as the DNA gate), and the C-terminal area of coiled coils, which forms the C or exit gate, are all open or closed interfaces in DNA gyrase. The principles consist reaction is expected to proceed as follows at the interface of the N terminus of the GyrA dimer and the domain of GyrB, the DNA G (or gate) segment connects with the enzyme, and DNA is wrapped around the enzyme in a right-handed supercoil of 130 base pairs. Wrapping DNA around the C-terminal domains of gyrase makes it easier for a second segment (the transported or T segment) from the same DNA molecule to reach the N gate, which is positioned over the G segment in preparation for strand transit.

The N gate is closed and the T segment is trapped when ATP is bound. The enzyme cleaves the G segment, establishing apart. DNA–phosphotyrosyl bonds, resulting in a double-strand break and GyrA's covalent attachment to the DNA. The T segment makes its way through the open DNA gate, the broken G segment, and finally the exit gate. The binding and hydrolysis of ATP drives the passage of the T segment through the G segment (strand passage). The N gate is opened when ATP is hydrolyzed and ADP is released, resetting the enzyme for the next super-coiling cycle. At the cost of two ATPs, one gyrase supercoiling cycle introduces two negative supercoils into the DNA molecule. Gyrase can relax negatively supercoiled DNA in the absence of ATP, effectively by the reverse method.

Fluoroquinolones are widely employed antibacterial medications that target DNA gyrase, but the wide range of adverse effects and developing bacterial resistance, combined with the lack of novel antibacterial treatments in the pipeline, has fueled intense research in this area. By screening chemical libraries, new chemical entities with various scaffolds containing DNA gyrase inhibitory capabilities have been discovered, which could serve as good leads for antibacterial drug development. As DNA gyrase inhibitors, a wide range of natural products and protein-based compounds have been found and researched, adding to the structural diversity that can be exploited and utilized in the development of new antibacterial medicines.

The creation of new chemical compounds having DNA gyrase inhibitory activity (from natural

sources, random screens, or rational design) will help to confirm the enzyme's utility as a target. This discussion provides an overview of DNA gyrase inhibitors derived from natural and synthetic sources, as well as the synthesis methods and biological activity range of various analogues. The most successful ones can be utilized as templates to develop new DNA gyrase-targeting drugs that are effective against resistant bacteria and biofilms.