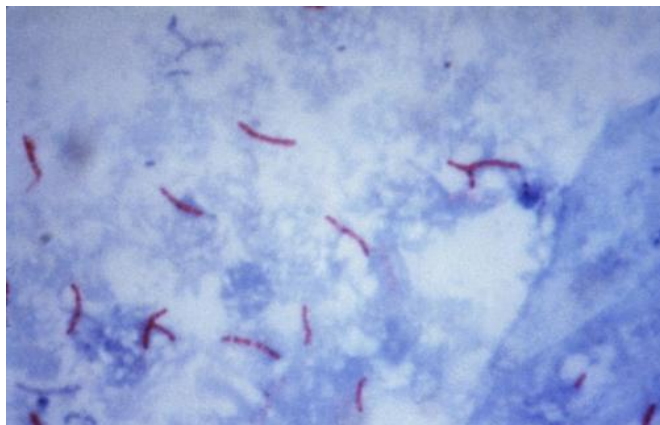


# Confronting TB resistance

12 June 2018, by Niyati Vachharajani



This photomicrograph reveals *Mycobacterium tuberculosis* bacteria using acid-fast Ziehl-Neelsen stain; Magnified 1000 X. The acid-fast stains depend on the ability of mycobacteria to retain dye when treated with mineral acid or an acid-alcohol solution such as the Ziehl-Neelsen, or the Kinyoun stains that are carbolfuchsin methods specific for *M. tuberculosis*. Credit: public domain

Tuberculosis, caused by *Mycobacterium tuberculosis*, is a highly infectious lung disease in humans. Unfortunately, resistance to anti-tubercular medicines such as fourth-generation fluoroquinolones is on the rise. In most cases, fluoroquinolone resistance is caused by mutations in a bacterial enzyme called gyrase.

Compounds called *M. tuberculosis* gyrase inhibitors (MGIs) display activity against tuberculosis in cellular and animal models, but little is known of their interaction with the [bacterial enzyme](#).

Neil Osheroff, Ph.D., and colleagues examined the mechanism of action of MGIs against purified *M. tuberculosis* gyrase. They report that MGIs effectively increase levels of gyrase-mediated single-stranded DNA breaks, which lead to chromosomal fragmentation, and maintain activity against commonly mutated fluoroquinolone-

resistant forms of the enzyme.

This mechanistic study, reported in the journal *ACS Infectious Diseases*, provides an important insight into anti-tubercular drug activity. Furthermore, it highlights the use of MGIs as potent anti-tubercular medicines and their potential in overcoming the serious threat of multi-drug resistant tuberculosis.

**More information:** Elizabeth G. Gibson et al. Mechanism of Action of *Mycobacterium tuberculosis* Gyrase Inhibitors: A Novel Class of Gyrase Poisons, *ACS Infectious Diseases* (2018). [DOI: 10.1021/acsinfecdis.8b00035](https://doi.org/10.1021/acsinfecdis.8b00035)

Provided by Vanderbilt University

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