# Better treatment for tuberculosis possible with biochemist's findings

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Recent discoveries by a Virginia Tech biochemist could lead to a more effective drug design to combat the bacteria responsible for tuberculosis infection. Spread through the air from one person to another, tuberculosis is responsible for approximately two million deaths per year, worldwide, and the emergence of drug resistant forms, specifically MDR- and XDR-TB, is an escalating challenge.

Once a person is infected with tuberculosis, he or she typically faces four to six months of treatment, if it is even available, using a combination of as many as six drugs. Symptoms include chest pain, coughing up blood, weakness, fever, and chills.

Marcy Hernick, an assistant professor of biochemistry and affiliated faculty member with the Fralin Life Science Institute, has discovered that the amino acid tyrosine plays several key roles in one enzyme involved in the pathogenesis of mycobacteria, the bacteria that causes tuberculosis. Tyrosine aids in the regulation of the binding and release of small molecules, as well as the chemistry carried out by the enzyme.

"When studying pathogenesis, we wanted to map out the active site of the enzyme to understand which amino acid chains were necessary for catalysis to occur," Hernick said. "We found a tyrosine residue on the structure that we wouldn't have thought to be important. But, after further analysis, we think tyrosine moves to carry out different steps in the catalytic cycle."

This information will be useful in the field of drug inhibitor design, Hernick explained, because scientists will want to develop a drug that can interact with tyrosine in order to alter catalysis. Hernicks findings were published in the Journal of Biological Chemistry this month.

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