

RESEARCH

Wits node researchers uncover metabolic defect in clinical isolate of Mycobacterium tuberculosis

Researchers in the Wits node of the CBTBR have discovered that a deletion polymorphism present in the well characterized clinical isolate of *M. tuberculosis*, strain CDC1551, eliminated the function of the vitamin B₁₂-dependent form of the metabolic enzyme, methionine synthase (MetH). MetH is one of two forms of this enzyme, which is absolutely required for the production of the amino acid, methionine. Growth of the CDC155 strain was found to be severely inhibited by the addition of vitamin B₁₂ supplement to the culture medium. In contrast, growth of the laboratory strain, H37Rv, which contains an intact *metH* gene, was unaffected by the presence of this vitamin supplement. Using a genetic approach, the vitamin B₁₂ sensitivity of CDC1551 and of a mutant form of H37Rv in which the *metH* was deleted was shown to be due to repression by vitamin B₁₂ of the production of the alternate, vitamin B₁₂-independent methionine synthase, MetE. Further studies revealed that the vitamin B₁₂-dependent regulation of MetE involves the action of a RNA switch or “riboswitch” – a novel type of regulatory element that is the subject of considerable interest in many areas of bacteriology. This study, led by Digby Warner and Stephanie Dawes, was published in the May 2007 issue of the *Journal of Bacteriology*.

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