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EDITORIAL

India's Latent Foe: Multi Drug Resistant Tuberculosis

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Tuberculosis (TB), an infectious bacterial disease, is caused by *Mycobacterium tuberculosis.* An effective drug for the control of tuberculosis is rifamycin which is clinically used in combination with ethambutol, isoniazid and pyrazinamide. Generally, a six month treatment regime is competent enough to cure tuberculosis, but due to inappropriate treatment and a non committal approach of the patients towards the treatment, there has been a drastic rise in the cases of Multi drug resistant (MDR) mycobacterial strains. Such cases, have increased in drastic numbers all across the globe especially in the developing and under developed countries, where severe lack of awareness has fuelled this ominous a phenomena. Besides this, the incidence of TB and HIV infection seem to go hand in hand, as a suppressed immune system makes the patient more amenable to other bacterial infections. India alone harbours around a million patients coinfected with HIV and *M. tuberculosis.* While several efforts are being made to tackle this problem, nothing of great significance has been brought out. Rifamycin, the classically used drug for curing TB is produced by an actinobacterium *Amycolatopsis mediterranei.* However, rifamycin and its semisynthetic derivatives are largely becoming ineffective against the drug resistant forms of mycobacteria. Eversince the commercial production of rifamycin in 1962, it has been a boon to the mankind in its ability to control TB. With immense efforts of the researchers, the yield of this wonder drug from *A. mediterranei* was increased from a meagre 500 mg/L in 1962 to up to 24 g/L through classical strain improvement methods. However, with the incidence of 73,000 cases of MDR-TB in India, and around a million across the world, there is an urgent need to develop rifamycin analogs or completely new drugs effective against MDR-TB strains. So far only six semisynthetic derivatives of rifamycin B could be produced by modifying the naphthoquinone ring at positions C3 and C4, beyond which, modifications are not possible. Alternative to this, could be the combinatorial approach of deleting, swapping or inactivating modules or domains of rifamycin biosynthetic gene cluster (also called polyketide synthase gene cluster). This approach has been successful with erythromycin polyketide gene cluster, in *Saccharospora erythraea,* but has not been applied to rifamycin polyketide synthase gene cluster, yet. Thus, in future, emphasis should be laid on this approach to tackle the problem of MDR-TB.

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