Title: Role of efflux pumps in drug resistance in *M. tuberculosis*

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Genomic mutations in drug targets have been identified as the major cause of drug resistance in *M. tuberculosis*. However, a section of isolates do not possess these mutations despite being drug resistant. Approximately 20 to 30% of clinical isoniazid (INH) resistant, 5% rifampicin (RIF) resistant and 40% Ethambutol (EMB) resistant *M. tuberculosis* isolates do not harbor mutations. Therefore, it is evident that other, more undefined mechanisms play a role in drug resistance.

Analysis of genome sequences has shown that *M. tuberculosis* have multiple putative efflux pumps. However, the role of efflux pumps in intrinsic and acquired resistance has been neglected as a major cause for antibiotic resistance of *M. tuberculosis* and has only recently received attention.

We evaluated the role of putative efflux pumps in providing resistance to INH, RIF and EMB in clinical isolates of *M. tuberculosis*. Isolates were also analysed for canonical mutations in *katG, inhA, rpoB* and *embB306.* Our study suggested a possible association of efflux pumps and resistance to RIF, INH and EMB.

Although the mechanism behind efflux pumps overexpression and regulation is not clearly understood, it could be relevant to study mutations upstream or in the predicted promoter region of these genes to identify additional molecular markers for drug resistance.