

# A potential link between tuberculosis and lung cancer through non-coding RNAs

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## Abstract

Pulmonary tuberculosis caused by *Mycobacterium* and lung cancer are two major causes of deaths worldwide and the former increases the risk of developing lung cancer. However, the precise molecular mechanism of *Mycobacterium* associated increased risk of lung cancer is not entirely understood. Here, using in silico approaches, we show that hsa-mir-21 and *M. tuberculosis* sRNA\_1096 and sRNA\_1414 could play important roles in the pathogenesis of both these diseases. Further, we postulated a “Genetic remittance” hypothesis where these sRNAs may play important roles. The sRNA\_1096 could be involved in tuberculosis through multiple infectious processes, and if transferred to the host, it may activate the TLR8 mediated pro-metastatic inflammatory pathway by acting as a ligand to TLR8 similar to the mir-21 leading to lung tumorigenesis and chemo-resistance. Analogous to SH3GL1, it may also regulate cell cycle. On the other hand, sRNA\_1414 is probably involved in survivability and drug response of the pathogen. However, it may be a metastatic factor for lung cancer providing EPS8L1 and SORBS1 like functions upon remittance. Further, all these three non-coding RNAs are predicted to act in rifampicin resistance in *Mycobacterium*. Currently, we are applying robust bioinformatics strategies and conducting experimental validations to confirm our in-silico findings and hypothesis.

Funding: TWAS-CNPq, CAPES