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.

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