ABSTRACT

Gatifloxacin (GFLX) has been reported to be associated with both hypo and hyperglycaemia in non-diabetic primarily elderly patients. Several mechanisms have been proposed that explains Gatifloxacin induced hypoglycaemia. However, the mechanism leads to hyperglycaemia is not clearly understood. Results in this study, indicates GFLX induced hyperglycaemic and hypoglycaemic condition in RIN-5F cell is mediated by insulin mRNA degradation by activation of unfolded protein response, the degradation of insulin mRNA was glucose dependent which additionally evidenced by decreased synthesis of proinsulin and insulin content in RIN-5f cells. This study provides insight to understand, how GFLX mediated hypoglycaemic and hyperglycaemic mechanism occurred at elevated glucose condition and gives one of the several ways to understand an insight of GFLX and other fluoroquinolone mediated hyperglycaemic condition mechanism occurred at elevated glucose condition.

Function of microRNA (miRNA) has been painted in pathogen-host interactions recently. Right now, their role in active pulmonary tuberculosis dendritic cells is unknown. The aim of the study was to delineate miRNA expression in dendritic cells (DCs) of patients with active pulmonary tuberculosis. Differential expression of miRNAs were evaluated and validated by real time PCR array panel. Corresponding human monocytes derived dendritic cell model was also established in our lab as M. tuberculosis and DCs model infection. Secreted cytokines from dendritic cells was measured by multiplex ELISA to validate of DCs. We found that 51 miRNAs were differentially expressed between tuberculosis group and controls. More miRNAs (29 out of 51 miRNAs) were overexpressed than underexpressed during tuberculosis infection. For the first time, differential expressions of miRNAs in dendritic cells were found in active pulmonary tuberculosis. The study provides rationale for identifying the role of miRNAs in the pathogenesis of pulmonary tuberculosis and indicates potential for miRNA-based therapeutic strategies.