ABSTRACT

The current first-line drugs for NSCLC with *EGFR* mutation as the driver oncogene are tyrosine kinase inhibitor (TKI) which act specifically on blocking the mutated EGFR protein to stop the signal transduction and therefore inhibit the proliferation of the cancer. However, all patients are eventually developing acquired resistance to the first line of TKI therapies mostly after 6-12 months of treatment initiation and afterwards the resistance also occurs on the second line and later throughout the remedy. PD-1/PD-L1 blocker might be useful to be the alternative treatment. NSCLC patients with *EGFR* mutation subtype L858R mutation had the better overall response rate and overall survival after treatment with PD-1/PD-L1 blocker, compared those with subtype exon 19 deletion. To understand the mechanism which lies behind the better outcomes of the subtype L858R, this study measured the *PD-L1* DNA copy number and mRNA expression of *EGFR*-mutated NSCLC cell lines with L858R mutation (H1975), exon 19 deletion (H1650), and control wild-type *EGFR* (A549). These cells were cultured and treated with IFN-γ (30 minutes, 6 hours, and 24 hours of induction). The quantification of the PD-L1 transcription level (RNA) and the DNA copy number were done with quantitative Polymerase Chain Reaction (qPCR). The experiment also compared the expression with and without IFN-γ exposure. The result is that H1975 cell line had *PD-L1* DNA copy number reduction and that the better outcomes did not related to H1975 induced expression. The L858R subtype superior result might be related to tumor infiltrating lymphocyte existence and tumor mutation burden of the cancer.

Keywords: *NSCLC cell lines, PD-1/PD-L1 Blocker, EGFR subtypes, IFN-γ, qPCR.*