ABSTRACT

Non-coding RNAs, which made up 98% of the human genome, have immense regulatory influence in cellular functionality that have not yet been fully explored. A study conducted by Prof. Vinay Tergaongkar's laboratory identified an important lncRNA, loc[redacted], which deregulates NFkB biology in cancer by sustaining covalent modification of p65, thereby regulating genome-wide occupancy of p65 on targets essential to inflammation, cancer progression, and CSC maintenance. HCC made up 90% of the primary liver cancer cases, and its progression and malignancy revolve around the CSC model. Being attributed to various inflammatory etiologies, dysregulation of NFkB is a major hallmark to its development. This study explores the breadth of loc[redacted] influence on NFkB signaling in HCC by analyzing its impact of NFkB's target gene expression, resulting stemness phenotype alterations, and drug resistance.