

Abstract

NFkappaB (NFkB) proteins are a family of related, evolutionarily conserved "rapid response" transcription factors that have low basal activity and that after induction, return quickly to latency. They are involved in the control of many cellular and physiological processes, such as inflammatory and immune responses, embryonic development, cellular growth and apoptosis. In turn, abnormal NFkB signaling activity results in a number of diseases, including cancer, arthritis, chronic inflammation, asthma, neurodegenerative and cardiovascular diseases, and defects in embryonic development.

In this study, a novel lentiviral vector encoding destabilized luciferase Luc2P under the transcriptional control of NFkB was constructed, which can be used to study regulation of NFkB by simple bioluminescence assay in high-throughput screening (HTS). This vector was used to create stable hepatic cancer cell lines termed HepG2-NFkB-Light and Huh7-NFkB-Light. These cell lines were characterized with a set of chemicals known to affect NFkB signalling activity, and results are presented in this paper. Briefly, *E.coli* lipopolysaccharide (LPS) induced NFkB activity in Huh7 cells, but not in HepG2 cells under conditions studied. Several known inhibitors (Bay 11-7082, andrographolide, QNZ) were found to reduce significantly NFkB signalling. These cell lines will be next used in a collaborative project to study regulation of NFkB signalling pathway in hepatocellular carcinoma.