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Role of the *CKIP1* gene in proliferation and apoptosis of the human lung cancer cell line H1299

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ABSTRACT. Casein kinase 2 interacting protein 1 (CKIP1) is a specific interacting protein of the casein kinase 2 (CK2) α subunit, and, by binding CK2 and other proteins, functions as an adaptor to regulate a series of cellular functions. Previous studies suggested that CKIP1 might play an important role in regulating oncogenic activities. However, few studies examining the function of CKIP1 in cancer cells have been performed. The present study aimed to investigate the role of CKIP1 in lung cancer. *CKIP1* mRNA expression was detected in 5 human lung cancer cell lines (H-125, H1299, LTEP-A-2, SPC-A-1, and NCL-H446) by semi-quantitative RT-PCR, and in 10 noncancerous lung tissues and 30 non-small lung cancer tissues by real-time quantitative PCR. A lentivirus-mediated small interfering RNA (siRNA) was used to knock down *CKIP1* expression in the H1299 cell line. To elucidate the impact of CKIP1 downregulation on H1299 cells, cell proliferation, DNA synthesis, and cell cycle distribution and apoptosis

were measured by high content screening assay, BrdU incorporation, and flow cytometric analyses, respectively. *CKIP1* mRNA was highly expressed both in H1299 cells and lung cancer tissues. We found that downregulation of *CKIP1* resulted in suppression of proliferation and colony-forming ability of H1299 cells, and led to S phase cell cycle arrest and G2 phase promotion, as well as a significant enhancement of H1299 cell apoptosis. Our study indicated that high expression levels of *CKIP1* were associated with the development of lung cancer, and that *CKIP1* knockdown may block tumor cell growth mainly by promoting cell apoptosis.

Key words: Casein kinase 2 interacting protein 1; Lung cancer; MicroRNA; Lentivirus

