

Lung cancer is statistically one of the most common diseases worldwide, and presents a serious threat to human health and life^{1,2}. In this study, we explored the effect of curcumin on proliferation in NSCLC A549 cells and investigated its associated mechanism. Curcumin is a yellow pigment found in turmeric rhizomes, reported to exhibit various anti-inflammatory, anti-angiogenic, anti-proliferative, and antioxidant properties. Oxidative stress is one of the most common causes of cellular damage, mostly due to the build-up of reactive oxygen species (ROS) free radicals through impairments to antioxidative enzymes, such as superoxide dismutase (SOD). In the absence of Wnt binding (off-state), β -catenin is downregulated via a degradation complex including GSK-3, CK1, Axin, APC, and PP2A^{14, 15}. Results indicated that curcumin could inhibit NSCLC proliferation induced by oxidative stress-mediated upregulation of the Wnt/ β -catenin pathway. Previous studies have reported that the Wnt/ β -catenin signaling pathway is abnormally active in hepatic carcinoma¹⁶. In recent decades, lung cancer death rates have increased yearly in an apparent trend, particularly nonsmall-cell lung cancer (NSCLC)³. β -catenin, which is deregulated in many cancers, is a multifunctional adaptor protein/transcription factor. Apoptosis is a programmed cell death process related to tumor malignancy, and is very important in embryonic development and tissue homeostasis.