



# *Ligusticum wallichii* inhibits renal carcinoma progression by downregulating UBE3A and through suppression of NF- $\kappa$ B signaling

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**ABSTRACT.** Renal carcinoma accounts for a fifth of the morbidity among malignant tumors in China. Ubiquitin-protein ligase E3A (UBE3A) plays an important role in the occurrence and development of gene mutation-induced diseases. This study was designed to investigate the mechanism of *Ligusticum wallichii* in treating renal carcinoma. Hematoxylin and eosin staining was applied to detect the pathological changes in a rat renal carcinoma model. The experimental group received *L. wallichii* treatment at 100 mg/kg every 48 h for 4 weeks, while the control group only received normal saline. The proliferation index Ki67 was measured by immunohistochemistry. Primary renal carcinoma cells were isolated and UBE3A expression was measured by quantitative polymerase chain reaction. The related signaling pathway was screened by the Pathway Finder Array. pP65 nuclear import was

detected by immunofluorescence. A total of 60 rats were used for the renal carcinoma model, of which 58 rats were successfully established and equally divided into two groups: *L. wallichii* and normal saline. Ki67 expression decreased in the *L. wallichii* group and was upregulated in the normal saline group. Histological analysis showed significant renal cell nucleus division in the normal saline group. The UBE3A level decreased after *L. wallichii* treatment compared to the level in the normal saline group. The Pathway Finder Array revealed that the NF- $\kappa$ B signaling pathway was activated, and pP65 presented obvious nuclear import in the normal saline group. In conclusion, *L. wallichii* inhibits renal carcinoma progression by downregulating UBE3A and suppressing the NF- $\kappa$ B signaling pathway.

**Key words:** *Ligustium wallichii*; Renal carcinoma; UBE3A; NF- $\kappa$ B