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Long non-coding RNA SNHG15 interacts with and stabilizes transcription factor Slug and promotes colon cancer progression

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ABSTRACT

Slug is a fast-turnover transcription factor critical for controlling cell fate and cancer cell invasion and metastasis. The stability of Slug is important and maintained by diverse mechanisms. In this study, we presented a paradigm of this activity by identifying long noncoding RNA (lncRNA) small nucleolar RNA host gene 15 (SNHG15) that binds to and stabilizes Slug in colon cancer cells. LncRNA SNHG15 transcription is upregulated in a variety of human cancers according to The Cancer Genome Atlas. Here, ectopic expression of SNHG15 promoted colon cancer cell migration *in vitro*, accelerated xenografted tumor growth *in vivo*, and elevated levels of SNHG15 were associated with poor prognosis for colon cancer patients. Mechanistically, SNHG15 maintains Slug stability in living cells by impeding its ubiquitination and degradation through interaction with the zinc finger domain of Slug. These findings revealed a novel mechanism underlying the control of Slug stability by demonstrating that oncogenic lncRNA SNHG15 interacts with and blocks Slug degradation via the ubiquitin-proteasome system.