醫學檢驗生物技術學系碩士班
研究生參加研討會資料表

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論文摘要：

Backgrounds: Heat shock factor-1 (HSF1) coordinates a conserved, genome wide-transcription program known as the heat shock response that not only restores the normal protein folding environment but modulates cellular signaling pathways and metabolism to maintain cell survival under stresses. HSF1 is also activated by cancer-associated stresses and supports cellular transformation and cancer progression. In the present study, whether and how HSF1 may modulate oncogenic Ras^{G12V}-induced cell transformation was investigated. **Materials & Methods:** An H-Ras^{G12V}-induced transformed/tumorigenic cell line (Ras-AIT2) was established from a human bronchial epithelial cell line (BEAS2B). The transcription activity of HSF1 before and after Ras transformation was measured using a HSPA1A promoter-luciferase reporter. Cellular activity of HSF1 was modulated by a specific shRNA or chemical inhibitor. The effects of HSF1 on Ras-AIT2 cell proliferation and Ras-related signaling pathway were assessed by cell viability assay, immunoblotting, and immunofluorescence staining. **Results:** Ectopic overexpression of the oncogenic Ras^{G12V} in BEAS2B cells significantly increased the transcription activity of HSF1. The enhanced activity of HSF1 in Ras-AIT2 cells was not due to the activation of PI3K/AKT or MAPK pathways. In addition, depletion of HSF1 by shRNA or inhibition of HSF1 by a chemical inhibitor in Ras-AIT2 cells not only reduced the cell proliferation rate and induced apoptosis but decreased the stability of H-Ras^{G12V} and impaired autophagy. **Conclusion:** HSF1 was activated by oncogenic Ras and required for maintaining the stability of oncogenic Ras and the survival of transformed cells.

指導教授簽名：