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Data article

Data on the transcriptional regulation of DNA damage induced apoptosis suppressor (DDIAS) by ERK5/MEF2B pathway in lung cancer cells

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ABSTRACT

The data included in this article are associated with the article entitled "DNA-damage-induced apoptosis suppressor (DDIAS) is upregulated via ERK5/MEF2B signaling and promotes β-catenin-mediated invasion" (J.Y. Im, S.H. Yoon, B.K. Kim, H.S. Ban, K.J. Won, K. S. Chung, K.E. Jung, M. Won) [1]. Quantitative RT-PCR data revealed that genetic or pharmacological inhibition of extracellular signal-regulated kinase 5 (ERK5) suppresses DDIAS transcription in response to epidermal growth factor (EGF) in Hela cells. p300 did not interact with myocyte enhancer factor 2B (MEF2B), a downstream target of ERK5 and affect transcription of DDIAS. Moreover, DDIAS transcription is activated by ERK5/MEF2B signaling on EGF exposure in the non-small cell lung cancer cells (NSCLC) NCI-H1703 and NCI-H1299. DDIAS knockdown suppresses lung cancer cell invasion by decreasing β-catenin protein level on EGF exposure.

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Specifications Table

Subject area	Biology
More specific subject area	Cell biology, Molecular biology
Type of data	Image, graph, figure
How data was acquired	Quantitative PCR, Western blot, Transwell invasion assay
Data format	Raw
Experimental factors	Cells were overexpressed with ERK5, DDIAS, β-catenin or transfected with siRNA against ERK1, ERK2, ERK5, MEF2B, DDIAS
Experimental features	Samples were HeLa, non-small cell lung cancer cells, NCI-H1299, NCI-H1703 cells
Data source location	Daejeon, South Korea
Data accessibility	Data is available with the article

Value of the data

- Transcription of DDIAS is activated by ERK5/MEF2B pathway in lung cancer cells.
- Increase of DDIAS transcription activates β-catenin signaling to promote lung cancer cell invasion.
- The data provide evidence that DDIAS is a potential therapeutic target of lung cancer.

1. Data

DDIAS is highly expressed in lung cancers and is involved in cisplatin resistance [2,3]. In HeLa cells, genetic and pharmacological inhibition of MEK/ERK5 suppressed EGF-induced DDIAS transcription, whereas ERK5 overexpression increased DDIAS mRNA level (Fig. 1). DDIAS knockdown dramatically decreased β-catenin protein level in HeLa cells (Fig. 2). Consistent with data in HeLa cells, inhibition of ERK5 suppressed DDIAS transcription on EGF exposure in lung cancer cell lines (Fig. 3). In addition, MEF2B knockdown reduced EGF-induced DDIAS expression in lung cancer cells (Fig. 4). Furthermore, DDIAS knockdown inhibited β-catenin accumulation and lung cancer cell invasion (Fig. 5).

2. Experimental design, materials and methods

2.1. Cell culture and transfection

HeLa cells were cultured in Dulbecco's modified Eagle's medium and non-small cell lung cancer cell, NCI-H1703 and NCI-H1299 cells were cultured in RPMI-1640 containing 10% fetal bovine serum (FBS), 50 U/mL of penicillin, and 50 μg/mL of streptomycin (Invitrogen, Carlsbad, CA, USA) in an incubator at 37 °C and 5% CO_2. Knockdown and overexpression of target genes experiment were performed as described [1]. Cells were transiently transfected with HA-ERK5, HA-p300, Flag-DDIAS or HA-β-catenin using Turbofect (ThermoScientific, Rockford, IL) [4].

2.2. RT-PCR

Total RNA extraction and Real-time PCR were performed as described [1]. The cycling conditions were 95 °C for 15 min and 40 cycles of 95 °C for 15 s, 55 °C for 15 s and 72 °C for 15 s. All reactions were performed in triplicate and normalized to GAPDH as an internal control. The values are presented as the mean ± S.E.M.