Data in Brief 13 (2017) 77–84

Contents lists available at ScienceDirect

Data in Brief

journal homepage: www.elsevier.com/locate/dib

Data Article

Data on the association of CMPK1 with clinicopathological features and biological effect in human epithelial ovarian cancer

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ARTICLE INFO

Article history:
Received 12 April 2017
Received in revised form
3 May 2017
Accepted 9 May 2017
Available online 12 May 2017

Keywords:
TGF-β signaling
UMP/CMP kinase
Tissue microarray
Tumorigenesis
miRNA
Therapeutic target

ABSTRACT

Human epithelial ovarian cancer (EOC) is the most lethal gynecological disease. However, the molecular mechanisms by which transforming growth factor-β (TGF-β) regulates ovarian tumor progression markers remain unclear. The present data show cytidine monophosphate kinase (CMPK) as an EOC biomarker and are related to the article entitled "Cytidine monophosphate kinase is inhibited by the TGF-β signalling pathway through the upregulation of miR-130b-3p in human epithelial ovarian cancer" [1]. CMPK, as well as cystatin B [2] and β-2-microglobulin [3], is overexpressed in human epithelial-type ovarian tumors. CMPK is an enzyme required for nucleic acid biosynthesis [4] and is regulated by the TGF-β signaling pathway in EOC cells [1]. Furthermore, the data show the effect of CMPK-shRNA on EOC cell apoptosis and TGF-β-induced Smad2 phosphorylation. CMPK expression in two EOC cell lines OVCAR-3 and SK-OV-3 is regulated by multiple miRNAs and some of these miRNAs may affect EOC chemoresistance [5].

DOI of original article: http://dx.doi.org/10.1016/j.cellsig.2017.04.009
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http://dx.doi.org/10.1016/j.dib.2017.05.022

Specifications Table

Subject area	*Cell biology; Health science*
More specific subject area	*Apoptosis; Ovarian cancer*
Type of data	*Table and figure*
How data was acquired	*Tissue microarray, human EOC cell lines OVCAR-3 and SK-OV-3 (ATCC, Manassas, VA, USA), Transfection, Western blot, Flow cytometry (Becton Dickinson Beckman Coulter, Inc., Brea, CA, USA)*
Data format	*Analyzed*
Experimental factors	*Cells were transfected with siRNA or shRNA; Cells were treated with 10 ng/ml TGF-β1 for 24 h*
Experimental features	*The tissue microarray included 100 paraffin-embedded ovarian tissues; Screen 9 miRNAs that potentially target CMPK1*
Data source location	*Shanghai, China*
Data accessibility	*The data are with this article*
Related research article	*Zhou et al. [1] "Cytidine monophosphate kinase is inhibited by the TGF-β signalling pathway through the upregulation of miR-130b-3p in human epithelial ovarian cancer" j.cellsig 35:197–207.*

Value of the data

- Data present CMPK as an ovarian serous tumor progression marker.
- The location of CMPK protein expression in the cytoplasm and nucleus of epithelial-type ovarian tumor cells is shown.
- Suppression of CMPK affects the doubling time of EOC cells.
- Data describe for the first time that knockdown of CMPK influences EOC cell apoptosis.
- Data show the effect of CMPK-shRNA on TGF-β-induced Smad2 phosphorylation.

1. Data

The data represent the observation from experiments of tissue microarray, Western blot and flow cytometry. Data in Table 1 are the list of sequences of siRNA, shRNA, miRNA and PCR primer used in a related research article [1]. The data of the association of CMPK protein expression with clinicopathological features of patients with epithelial ovarian tumours are shown in Table 2. Data in Fig. 1 show the positive rate for CMPK staining in the cytoplasm and nucleus. Fig. 2 confirms the knockdown of CMPK protein by Western blot after CMPK-siRNA transfection in OVCAR-3 and SK-OV-3 cells. Doubling times (DT) based on the optical density (OD) values at the time of measurement is shown in Fig. 3. Data in Fig. 4 represent the proportion of early apoptotic cells detected by flow cytometry and the expression of cleaved caspase-3 protein, an active form of apoptotic protein, detected by Western blot after CMPK-shRNA infection. Data of phospho-Smad2 detection by Western blot are shown in Fig. 5. Screening data of the effect of miRNAs on CMPK expression are shown in Fig. 6.