Data in Brief 7 (2016) 1073–1077

Contents lists available at ScienceDirect

Data in Brief

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

Data Article

DNA damage response and repair data with pharmacological modulators of Tousled

Prakash Srinivasan Timiri Shanmugam [a], Renjith P. Nair [a], Arrigo DeBenedetti [a], Gloria Caldito [b], Fleurette Abreo [c], Gulshan Sunavala-Dossabhoy [a,*]

[a] Department of Biochemistry and Molecular Biology, USA
[b] Department of Computational Biology and Bioinformatics, USA
[c] Department of Pathology, LSU Health Sciences Center, Shreveport, LA, USA

ARTICLE INFO

Article history:
Received 5 January 2016
Received in revised form
23 March 2016
Accepted 24 March 2016
Available online 30 March 2016

Keywords:
Tousled
Ionizing radiation
DNA repair
Gallic acid
Thioridazine

ABSTRACT

Human Tousled kinase 1 (TLK1) plays an important role in chromatin remodeling, replication, and DNA damage response and repair. TLK1 activity is immediately, but transiently, down-regulated after genotoxic insult, and its recovery is important for exit from checkpoint arrest and cell survival after radiation. The data in this article compliments research presented in the paper titled, "Tousled kinase activator, gallic acid, promotes DNA repair and suppresses radiation cytotoxicity in salivary gland cells" [1]. The identification of small molecule activators and inhibitors of TLK1 provided an opportunity to pharmacologically alter the protein's activity to elucidate its role in DNA damage response pathways. TLK1 effectors, gallic acid (GA) and thioridazine (THD) activate and inhibit the kinase, respectively, and the data report on the impact of these compounds and the significance of TLK1 to DNA break repair and the survival of human salivary acinar cells.

DOI of original article: http://dx.doi.org/10.1016/j.freeradbiomed.2015.12.029
* Corresponding author. Tel.: 318 675 8216; fax 318 675 5180.
E-mail address: gsunav@lsuhsc.edu (G. Sunavala-Dossabhoy).

http://dx.doi.org/10.1016/j.dib.2016.03.075

Specifications Table

Subject area	*Molecular biology*
More specific subject area	*DNA break repair*
Type of data	*Image, graph, figure*
How data was acquired	*Olympus Provis AX70 microscope, BD LSR II flow cytometer, BioTek Synergy 4 hybrid microplate reader.*
Data format	*Raw*
Experimental factors	*Cells were treated with 10 μM THD 1 h before drug washout and irradiation, whereas cell treatment with 50 μM GA followed 30 minutes after radiation and extended up to indicated experimental time points or 16 h before placement in drug-free medium.*
Experimental features	*NS-SV-AC and stably transfected shRNA TLK1 cells were grown in complete KGM-2 medium. Cells were treated with drug before or after radiation, and cells were analyzed by colony formation assays, single cell alkaline gel electrophoreses, or Annexin V staining at indicated times. Radiation-generated reactive oxygen species was analyzed using CM-H2DCFDA. Repair of restriction enzyme (I SceI)- induced double strand break was studied in HEK293-PC222 cells. Cell population that became RFP positive was quantified by FACS analyses.*
Data source location	*Shreveport, LA*
Data accessibility	*Data accessible in the article*

Value of the data

- Influence of small molecule TLK1 effectors on cell survival highlight their clinical utility in altering tissue response to radiation.
- Studies that assess synthetic lethality of TLK1 inhibitors and genotoxic therapeutics can initiate the development of more effective cancer treatment regimens.
- Enhanced TLK1 activity leads to improved DNA break repair kinetics, and an examination of TLK1-dependent dynamics at DNA breaks may lead to the identification of its yet unknown protein targets.

1. Data

The data in the article demonstrate the effects of small molecule modulators of TLK1 activity in DNA damage response to and repair of radiation-induced DNA breaks. Unrepaired DNA breaks activate apoptosis, and the consequential effect of TLK1 function on cell survival and programmed cell death was demonstrated. The repair of DNA double-strand breaks predominantly occurs by non-homologous end joining (NHEJ), and using a cell-based reporter assay we reported the effect of TLK1 function on NHEJ repair of I-SceI-induced chromosomal breaks.

2. Experimental design, materials and methods

2.1. Cell culture

The acinar cells of the salivary glands are fluid-producing cells that are highly sensitive to radiation. Their inadvertent damage and resultant death during head and neck radiotherapy results in an