Data in Brief 6 (2016) 710–714

Contents lists available at ScienceDirect

Data in Brief

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

Data article

Data on the DNA damaging and mutagenic potential of the BH3-mimetics ABT-263/Navitoclax and TW-37

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ARTICLE INFO

Article history:
Received 22 September 2015
Received in revised form
5 January 2016
Accepted 8 January 2016
Available online 16 January 2016

ABSTRACT

Unfortunately, the mutagenic activities of chemotherapy and radiotherapy can provoke development of therapy-induced malignancies in cancer survivors. Non-mutagenic anti-cancer therapies may be less likely to trigger subsequent malignant neoplasms. Here we present data regarding the DNA damaging and mutagenic potential of two drugs that antagonize proteins within the Bcl-2 family: ABT-263/Navitoclax and TW-37. Our data reveal that concentrations of these agents that stimulated Bax/Bak-dependent signaling provoked little DNA damage and failed to trigger mutations in surviving cells. The data supplied in this article is related to the research work entitled "Inhibition of Bcl-2 or IAP proteins does not provoke mutations in surviving cells" [1].

Specifications table

Subject area	*Biology*
More specific subject area	*Mutagenesis, cancer biology, apoptosis research*
Type of data	*Graphs*

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http://dx.doi.org/10.1016/j.dib.2016.01.013

How data was acquired	*Clonogenicity assays, HPRT mutagenesis assays, γH2AX flow cytometry quantitation*
Data format	*Normalized data*
Experimental factors	*Murine embryonic fibroblasts from wild type or $Bax^{-/-}$, $Bak^{-/-}$ mice were treated with various concentrations of ABT-263 or TW-37, prior to the assays listed above.*
Experimental features	*Cells were exposed to drugs (or not) for various periods of time, then washed and either stained with anti-γH2AX for flow cytometry, seeded into normal media to quantitate clonogenicity, or seeded into media containing 6-thioguanine to count 6-thioguanine-resistant clones (presumably reflecting mutagenesis at the HPRT locus).*
Data source location	*La Trobe University, Bundoora, Australia*
Data accessibility	*Data is included within this article*

Value of the data

- These data can be used to compare the mutagenic potentials of anti-cancer drugs that employ different mechanisms of action
- Future research could define the pathways through which high concentrations of some BH3-mimetics kill cells and damage DNA
- Researchers could use this data to design animal-based experiments to evaluate the mutagenic and oncogenic activity of Bcl-2 antagonists in vivo

1. Data

Embryonic fibroblasts derived from wildtype or Bax/Bak-deficient mice were treated with ABT-263 (Fig. 1) or TW-37 (Fig. 2), or incubated in normal medium. We measured the impact of these treatments on survival, DNA damage and mutagenicity at the HPRT locus.

2. Experimental design, materials and methods

2.1. Cell lines and materials

SV-40 transformed Mouse Embryonic Fibroblasts (MEF) were kindly provided by Anissa Jabbour and Paul Ekert [2] and were cultured in DMEM high glucose (Invitrogen; Carlsbad, California, USA) containing 10% fetal calf serum (Invitrogen). ABT-263 and TW-37 were purchased from Selleck Chemicals (Houston, Texas, USA). The following antibodies were used: anti-H2AX (Ser 139) clone 20E3 (Cell Signaling Technology) and goat anti-rabbit FITC (Chemicon).

2.2. Cell survival assays

The toxicity of the drugs was assessed by comparing the clonogenic survival of treated and untreated cells. Cells were incubated with drugs, then washed and seeded at various densities in 6-well plates. After seven days, cells were stained with methylene blue (Sigma Aldrich) 1.25 g/l in 50% methanol, incubated for 5 min and washed twice with water, then the numbers of colonies were counted.