



ELSEVIER

Contents lists available at ScienceDirect

## Data in Brief

journal homepage: [www.elsevier.com/locate/dib](http://www.elsevier.com/locate/dib)

## Data Article

# Data on the effects of anti-cancer drug of resveratrol in breast cancer cells, MDA-MB-231 cells



Eunmi Park

Department of Food and Nutrition, School of Life Science and Nano-Technology, Hannam University, Daejeon, South Korea

## ARTICLE INFO

## Article history:

Received 3 February 2016

Received in revised form

14 March 2017

Accepted 15 March 2017

Available online 21 March 2017

## Keywords:

Resveratrol

Chemotherapy

Natural bioactive compounds

## ABSTRACT

The data here is related to the article, “Curcumin enhances poly (ADP-ribose) polymerase inhibitor sensitivity to chemotherapy in breast cancer cells” (Y.E Choi, and E. Park, 2015) [1]. The article shows that curcumin, as a natural bioactive compound, enhanced DNA damage response and induced cell death in MDA-MB-231 cells [1]. This data includes that breast cancer cells, MDA-MB-231 respond to DNA damage after UV irradiation, post to resveratrol treatment. The data shows that resveratrol treatment results in reduction of S-phase cell cycle and induction of  $\gamma$ -H2AX, which is a hallmark of DNA damage after UV irradiation in breast cancer cells, MDA-MB-231. Moreover, resveratrol sensitizes breast cancer cells to respond to UV treatment as a natural bioactive compound.

© 2017 Published by Elsevier Inc. This is an open access article under the CC BY license

(<http://creativecommons.org/licenses/by/4.0/>).

## Specifications Table

Subject area	Biology
More specific subject area	Cancer biology
Type of data	Figure, graph

DOI of original article: <http://dx.doi.org/10.1016/j.jnutbio.2015.07.015>

E-mail address: [eunmi\\_park@hnu.kr](mailto:eunmi_park@hnu.kr)

<http://dx.doi.org/10.1016/j.dib.2017.03.029>

2352-3409/© 2017 Published by Elsevier Inc. This is an open access article under the CC BY license (<http://creativecommons.org/licenses/by/4.0/>).

How data was acquired	FACS analysis, colony assay and western blot
Data format	Analyzed
Experimental factors	Comparison of resveratrol with UVC irradiation in MDA-MB-231 cells
Experimental features	Cell cycle analysis, survival data and western blotting in breast cancer cell line, MDA-MB-231 with resveratrol treatment post to UVC irradiation
Data source location	Daejeon, Korea
Data accessibility	Data is within this article

### Value of the data

- Dana can be used for investigate the cell cycle effects of resveratrol on UVC irradiation.
- The data provides the information of the synergistic effect of resveratrol in combination of the UVC irradiation in breast cancer cell culture system.
- Data will be useful for investigating the effect of resveratrol on DNA damage response in breast cancer cells.
- This data significantly extends the effects of resveratrol in breast chemotherapy.

## 1. Data

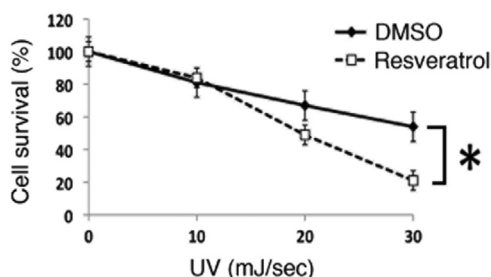
Resveratrol hyposensitized breast cancer cells to various UVC irradiations (0–30 mJ/sec) compared to cells treated with DMSO (Fig. 1). Next, resveratrol reduced more the S-phase cell cycle profiles post to 30 mJ/sec of UVC-induced DNA damage, compared to the cells treated with UVC alone (See Fig. 2A and B).

In addition, the effect of resveratrol on response of  $\gamma$ -H2AX induced by UVC treatment was provided in the western blotting (Fig. 2C).

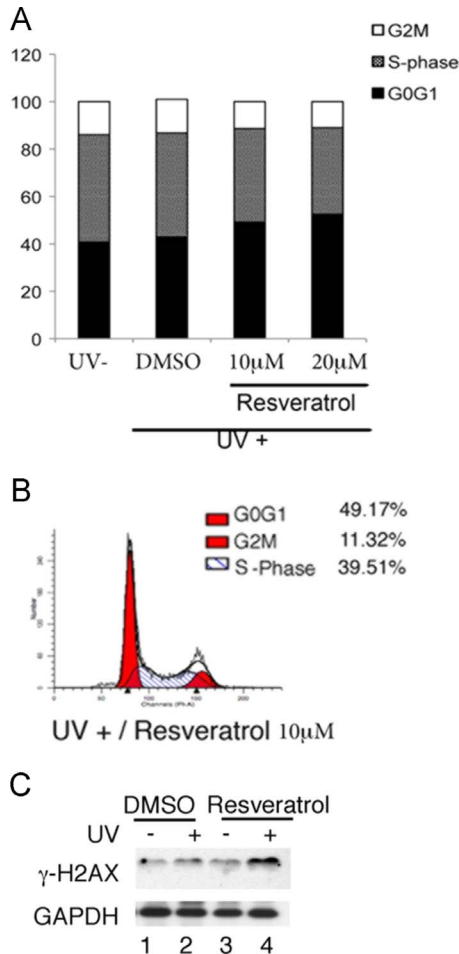
## 2. Experimental design, materials and methods

### 2.1. Cell culture

Breast cancer cell line, MDA-MB-231 cells were cultured in Dulbecco's modified Eagle's medium (DMEM) (Invitrogen) containing 1% penicillin/streptomycin and 10% fetal bovine serum (Invitrogen) [1–3]. Resveratrol (Sigma) was dissolved in DMSO. The MDA-MB-231 cells were treated with resveratrol for all of experiments. The cells were incubated at 37 °C with 5% CO<sub>2</sub>.



**Fig. 1.** Resveratrol sensitizes MDA-MB-231 breast cancer cells to UVC treatment. MDA-MB-231 cells were pretreated with resveratrol (10  $\mu$ M) or DMSO for 24 h and re-plated in culture dishes. Then the cells were exposed with various UVC irradiations (0–30 mJ/sec). Survival was determined using a colony assay from three independent experiments. The data are mean  $\pm$  standard errors. \*,  $p < 0.05$ .



**Fig. 2.** Resveratrol shows the reduction of S-phase cell cycle profiles post to UVC treatment. (A) MDA-MB-231 cells were cultured with 10 µM, 20 µM resveratrol/or DMSO for 24 h. The cells were exposed to UVC treatment and harvested. The cell pellets were fixed in 70% ethanol and stained with PI for FACS analysis. (B) Representative data of FACS analysis data in MDA-MB-231 cells with 10 µM resveratrol treatment post to UVC treatment. (C) Western blotting in MDA-MB-231 cells with 10 µM resveratrol treatment post to UVC treatment. UV-; UV untreated, UV+; UV treated (30 mJ/sec, harvest post to 3 h).

## 2.2. Cell cycle analysis

For fluorescence-activated cell sorting (FACS) analysis, MDA-MB-231 cells were fixed overnight at 4 °C in 70% ethanol, stained with propidium iodide (PI) for 1 h. The cells analyzed for DNA content using a FACS Calibur machine (BD Biosciences) [4].

## 2.3. Colony assay (Cell survival analysis)

Cell survival analysis was performed as described previously [3,5,6]. MDA-MB-231 breast cancer cells were prepared and exposed to resveratrol or DMSO for 12 h. After the treatment with UV irradiation (0–30 mJ/sec), the cells were re-plated in 6-well plates for clonogenic assays in triplicate. After 2 weeks later, cell culture were stopped and fixed with methanol. The colonies were stained with crystal violet. Data are mean ± SEM as indicated. Statistical significance of comparison between

two groups was determined by two-tailed Student's *T*-test. The *p*-values of less than 0.05 considered significant differences of statistical analysis.

#### 2.4. Western blot

Cell lysates (30 µg) was applied to 10% SDS-PAGE gel, and then transferred to nitrocellulose membranes. Specific protein levels were measured by Western blotting as described previously [1–3] with the antibodies against  $\gamma$ H2AX (Cell signaling) and GAPDH (Bethyl laboratory).

### Acknowledgments

This study was supported by Basic Science Research Program through the National Research of Korea (NRF) funded by the Ministry of Science, ICT & Future Planning (Grant no. 2015R1C1A1A02037579) and Hannam University Research Fund (No. 2016).

### Transparency document. Supporting information

Transparency data associated with this article can be found in the online version at <http://dx.doi.org/10.1016/j.dib.2017.03.029>.

### References

- [1] Y.E. Choi, E. Park, Curcumin enhances poly (ADP-ribose) polymerase inhibitor sensitivity to chemotherapy in breast cancer cells, *J. Nutr. Biochem.* 26 (2015) 1442–1447.
- [2] E. Park, J.M. Kim, B. Primack, D.M. Weinstock, L.A. Moreau, K. Parmar, A.D. D'Andrea, Inactivation of Uaf1 causes defective homologous recombination and early embryonic lethality in mice, *Mol. Cell. Biol.* 33 (2013) 4360–4370.
- [3] E. Park, H. Kim, J.M. Kim, B. Primack, S. Vidal-Cardenas, Y. Xu, B.D. Price, A.A. Mills, A.D. D'Andrea, FANCD2 activates transcription of TAp63 and suppresses tumorigenesis, *Mol. Cell* 50 (6) (2013) 908–918.
- [4] Y.E. Choi, E. Park, Ferulic acid in combination with PARP inhibitor sensitizes breast cancer cells as chemotherapeutic strategy, *Biochem. Biophys. Res. Commun.* 458 (2015) 520–524.
- [5] H. Farmer, N. McCabe, C.J. Lord, A.N. Tutt, D.A. Johnson, T.B. Richardson, M. Santarosa, K.J. Dillon, I. Hickson, C. Knights, et al., Targeting the DNA repair defect in BRCA mutant cells as a therapeutic strategy, *Nature* 434 (2005) 917–921.
- [6] J. Cho, E. Park, Curcumin utilizes the anti-inflammatory response pathway to protect the intestine against bacterial invasion, *Nutr. Res. Pract.* 9 (2) (2015) 117–122.