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Data in Brief





Data article

Data on cell cycle in breast cancer cell line, MDA-MB-231 with ferulic acid treatment



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ABSTRACT

Inhibition to repair DNA metabolism to respond to damaged DNA can lead to genetic instability, resulting in cancer cell death (Audeh et al., 2010; Bryant et al., 2005; Farmer et al., 2005; Lukas et al., 2003; Tutt et al., 2010) [1,2,6,8,11]. Despite of various studies demonstrating efficiency of combination therapy through down-regulation of DNA repair pathway, the suppression effects of DNA repair pathway by chemotherapeutic agents from natural bioactive compounds are less understood (Eitsuka et al., 2014; Kastan et al., 2004; Kawabata et al., 2000; Mancuso et al., 2014) [5,7,9].

Here, the data shows that ferulic acid reduced the S-phases post to UV treatment in breast cancer cells and was hypersensitive in breast cancer cells, MDA-MB-231.

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Specifications Table

Subject area

Biology

More specific sub-

Cancer biology

ject area

Type of data

Figure, graph

How data was

FACS analysis and colony assay

acquired

Data format Analyzed with FACS data, colony assay and statistical tests

Experimental factors Comparison of ferulic acid with UV-induced DNA damage in MDA-MB-231

cells

Experimental Cell cycle analysis and colony assay in breast cancer cell line, MDA-MB – 231

features with ferulic acid treatment post to UV.

Data source location Daejeon, Korea

Data accessibility Data is within this article

Value of the data

The data significantly extends ferulic acid treatment in breast cancer chemotherapy.

 The data provides the information of the effect of different UV irradiations in breast cancer cells with ferulic acid treatment.

1. Data

FACS profiles showed that ferulic acid in combination with UV irradiation reduced more the S-phase compared to the cells treated with UV irradiation, as well as in UV untreated cells [3] (Fig. 1). MDA-MB-231 cells with ferulic acid were more sensitive to UV treatment compared to the cells with DMSO by performing colony formation assays [4] (See Fig. 2).

2. Experimental design, materials and methods

2.1. Cell culture

MDA-MB-231 breast cancer cells were cultured in DMEM (Invitrogen) supplemented with 10% fetal bovine serum (Invitrogen) and 1% penicillin/streptomycin (Invitrogen) [3,4]. The cells were cultured with ferulic acid (Sigma) treatment for experiments. Ferulic acid was dissolved in DMSO was dissolved in PBS for the experiments. All of cell lines were incubated at 37 °C with 5% CO₂.

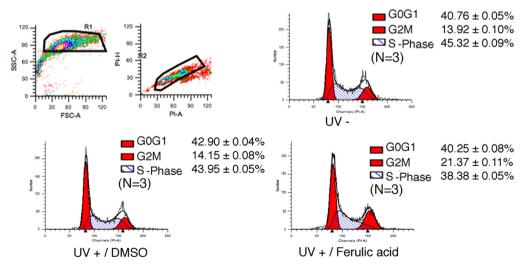


Fig. 1. Ferulic acid reduces S-phase cell cycle profiles post to UV treatment. MDA-MB-231 cells were cultured with $10 \,\mu$ M ferulic acid/or DMSO for 24 h. The cells were exposed to UV treatment and harvested. The cell pellets were fixed in 70% ethanol and stained with PI for FACS analysis. UV-; UV untreated, UV+; UV treated (20 mJ/s, harvest post to 3 h).

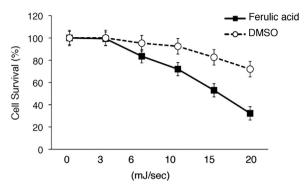


Fig. 2. Breast cancer cells with ferulic acid treatment are highly sensitive to UV treatment. MDA-MB-231 cells were pretreated with ferulic acid ($10~\mu M$) or DMSO for 24 h and re-plated in culture dishes. Then the cells were exposed with UV irradiation (0-20~mJ/s). Survival was determined using a colony assay from three independent experiments. The data are mean \pm -standard errors. *; p < 0.05.

2.2. Cell cycle analysis

MDA-MB-231 breast cancer cells were pretreated with ferulic acid or DMSO for 24 h. The cells were exposed to 20 mJ/s UV treatment and harvested post to 3 h. For fluorescence-activated cell sorting (FACS) analysis, MDA-MB-231 cells were fixed overnight at 4C in 70% ethanol, stained with propidium iodine (PI) for 1 h. The cells analyzed for DNA content using a FACS Calibur machine (BD Biosciences).

2.3. Colony assay (Cell survival analysis)

MDA-MB-231 breast cancer cells were prepared for colony assay [10]. After the pre-treatments with ferulic acid or DMSO, the cells were plated in plates with UV irradiation (0-20 mJ/s). The cells were cultured for clonogenic assay in triplicates. After 2 weeks in culture, colonies were fixed with methanol and stained with crystal violet.

2.4. Statistical analysis

All data are representative of at least three independent experiments. Data are mean \pm SEM unless otherwise indicated. Statistical significance of comparison between two groups was determined by two-tailed Student's t-test where indicated. For comparing more than one group, one-way ANOVA was used. Significant differences were considered at p-values of less than 0.05.

Acknowledgments

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Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at http://dx.doi. org/10.1016/j.dib.2016.02.001.

References

- [1] M.W. Audeh, J. Carmichael, R.T. Penson, M. Friedlander, B. Powell, K.M. Bell-McGuinn, C. Scott, J.N. Weitzel, A. Oaknin, N. Loman, et al., Oral poly(ADP-ribose) polymerase inhibitor olaparib in patients with BRCA1 or BRCA2 mutations and recurrent ovarian cancer: a proof-of-concept trial, Lancet 376 (2010) 245–251.
- [2] H.E. Bryant, N. Schultz, H.D. Thomas, K.M. Parker, D. Flower, E. Lopez, S. Kyle, M. Meuth, N.J. Curtin, T. Helleday, Specific killing of BRCA2-deficient tumours with inhibitors of poly(ADP-ribose) polymerase, Nature 434 (2005) 913–917.
- [3] 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.
- [4] Y.E. Choi, E. Park, Curcumin enhances poly (ADP-ribose) polymerase inhibitor sensitivity to chemotherapy in breast cancer cells, J. Nutr. Biochem. (2015), http://dx.doi.org/10.1016/j.jnutbio.2015.07.015.
- [5] T. Eitsuka, N. Tatewaki, H. Nishida, T. Kurata, K. Nakagawa, T. Miyazawa, Synergistic inhibition of cancer cell proliferation with a combination of delta-tocotrienol and ferulic acid, Biochem. Biophys. Res. Commun. 453 (2014) 606–611.
- [6] 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.
- [7] (a) M.B. Kastan, J. Bartek, Cell-cycle checkpoints and cancer, Nature 432 (2004) 316–323;
 - (b) K. Kawabata, T. Yamamoto, A. Hara, M. Shimizu, Y. Yamada, K. Matsunaga, T. Tanaka, H. Mori, Modifying effects of ferulic acid on azoxymethane-induced colon carcinogenesis in F344 rats, Cancer Lett. 157 (2000) 15–21.
- [8] C. Lukas, J. Falck, J. Bartkova, J. Bartek, J. Lukas, Distinct spatiotemporal dynamics of mammalian checkpoint regulators induced by DNA damage, Nat. Cell Biol. 5 (2003) 255–260.
- [9] C. Mancuso, R. Santangelo, Ferulic acid: pharmacological and toxicological aspects, Food Chem. Toxicol. 65 (2014) 185–195.
- [10] 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.
- [11] A. Tutt, M. Robson, J.E. Garber, S.M. Domchek, M.W. Audeh, J.N. Weitzel, M. Friedlander, B. Arun, N. Loman, R.K. Schmutzler, et al., Oral poly(ADP-ribose) polymerase inhibitor olaparib in patients with BRCA1 or BRCA2 mutations and advanced breast cancer: a proof-of-concept trial, Lancet 376 (2010) 235–244.