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Corrigendum

Corrigendum to "Adaphostin and bortezomib induce oxidative injury and apoptosis in imatinib mesylate-resistant hematopoietic cells expressing mutant forms of Bcr/Abl" [Leuk. Res. 30 (2006) 1263–1272]

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A research misconduct finding against the first author affected one of the figures in the above article, as detailed in the Office of Research Integrity: Findings of Research Misconduct, NIH Guide Grants Contracts. 2015 Dec 18: NOT-OD-16-040:

"Based on the report of an inquiry conducted by Virginia Commonwealth University (VCU), the willingness of the Respondent to settle this matter, and analysis conducted by ORI in its oversight review, ORI found that Dr. Girija Dasmahapatra, former Instructor, Department of Internal Medicine, VCU, engaged in research misconduct in research supported by National Cancer Institute (NCI), National Institutes of Health (NIH), grants R01 CA063753, R01 CA093738, and R01 CA100866."

ORI found that false data were included in 11 publications, including the above paper. As they stated:

"ORI found that Respondent falsified and/or fabricated data by reporting the results of Western blot experiments and mouse imaging experiments that examined interactions between multiple histone deacetylase and/or proteasome inhibitors in several cancer models. Specifically, Respondent duplicated, reused, and/or relabeled Western blot panels and mouse images and claimed they represented different controls and/or experimental result in" [those 11 papers].

Specifically, a blot labeled actin (Fig 3D) in the above paper was identical to other loading controls (e.g., tubulin) or in one case, c-Jun, which appeared elsewhere.

In the interim, the remaining authors have repeated this experiment and offer a corrected version (Fig. 3A–D). In brief, the effects of the indicated concentrations of adaphostin (10 h) on multiple signaling and apoptotic pathways were compared in wild-type versus T315I mutant BCR/ABL BaF/3 cells. In summary, the effects of adaphostin on apoptotic (e.g., cytochrome c, SMAC, PARP, caspase3/8, MCL-1, and BCL-xL) and signaling (e.g., pStat3, p-c-Jun, and p-Lyn) pathways were essentially equivalent in the two lines. These results were also very similar to those reported in the original manuscript figure. Based on these findings, the authors believe that the original results reported in this article are valid, and apologize for any inconvenience caused.

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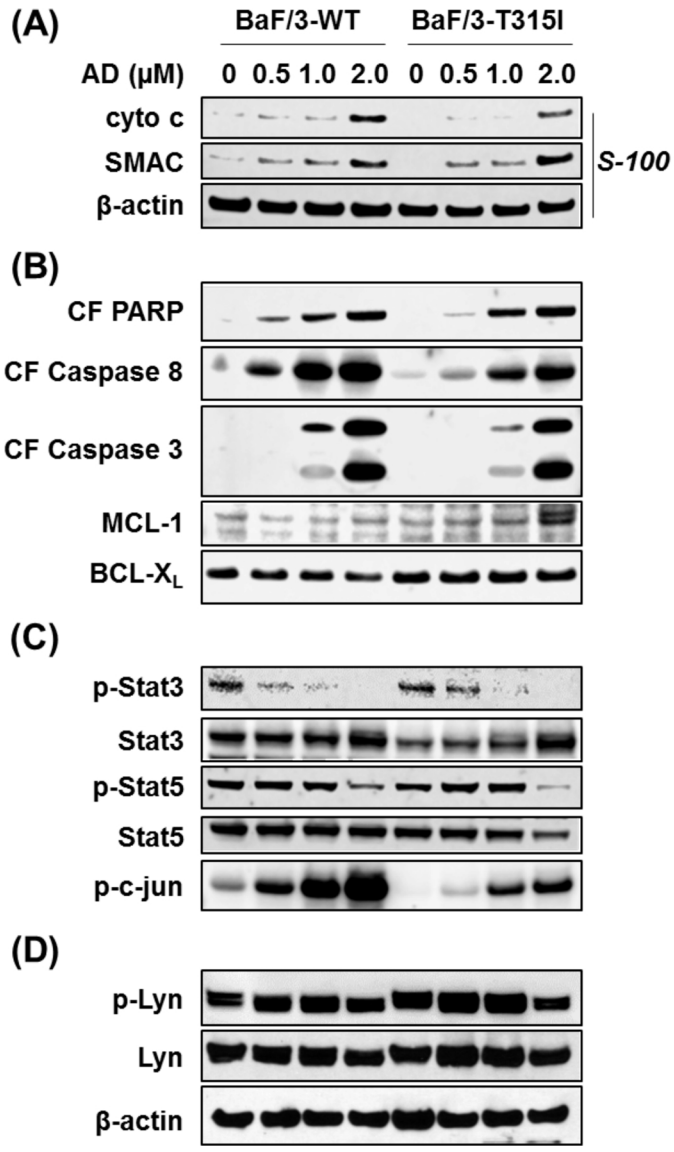

Fig. 3. Treatment with adaphostin induces equivalent effects on mitochondrial damage, caspase activation, and perturbations in various signaling cascades in wild-type and T315I BaF/3 cells. BaF/3 cells expressing wild-type or a mutant form of Bcr/Abl (T315I) were exposed to various concentrations of adaphostin as indicated for 10 h. (A) Cytosolic (S-100) fractions were obtained as described in Section 2. (B–D) At the end of drug exposure, cells were lysed and total protein contents were subjected to Western blot analysis using the indicated primary antibodies. Each lane was loaded with 20μg of protein. Blots were stripped and re-probed with anti-actin antibodies to ensure equal loading and transfer of protein. Results are representative of three separate experiments.