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Corrigendum

In Yadav *et al.*, (2012), errors appeared in Figure 2E and Figure 5D. It was found that beta actin, the loading control in Figure 2E, was inadvertently duplicated from Figure 3A during figure assembly. An inadvertent error was also noted in Figure 5D with PARP bands. Thus, experiments representing these figures were repeated. The correct versions of these figures, are now reproduced below. The authors would like to apologize for these errors.

The editors of *BJP* believe that there should be a full explanation of any published corrigendum. After a full and thorough review of all the issues, the editors accept that errors in the figures were compiling errors by the authors. It is important to note that the correction of the errors does not change our judgement that this paper is suitable for publication nor does it alter the conclusions in the manuscript.

Reference

Yadav VR, Prasad S, Aggarwal BB (2012). Cardamonin sensitizes tumour cells to TRAIL through ROS- and CHOP-mediated up-regulation of death receptors and down-regulation of survival proteins. Br J Pharmacol 165: 741–753.

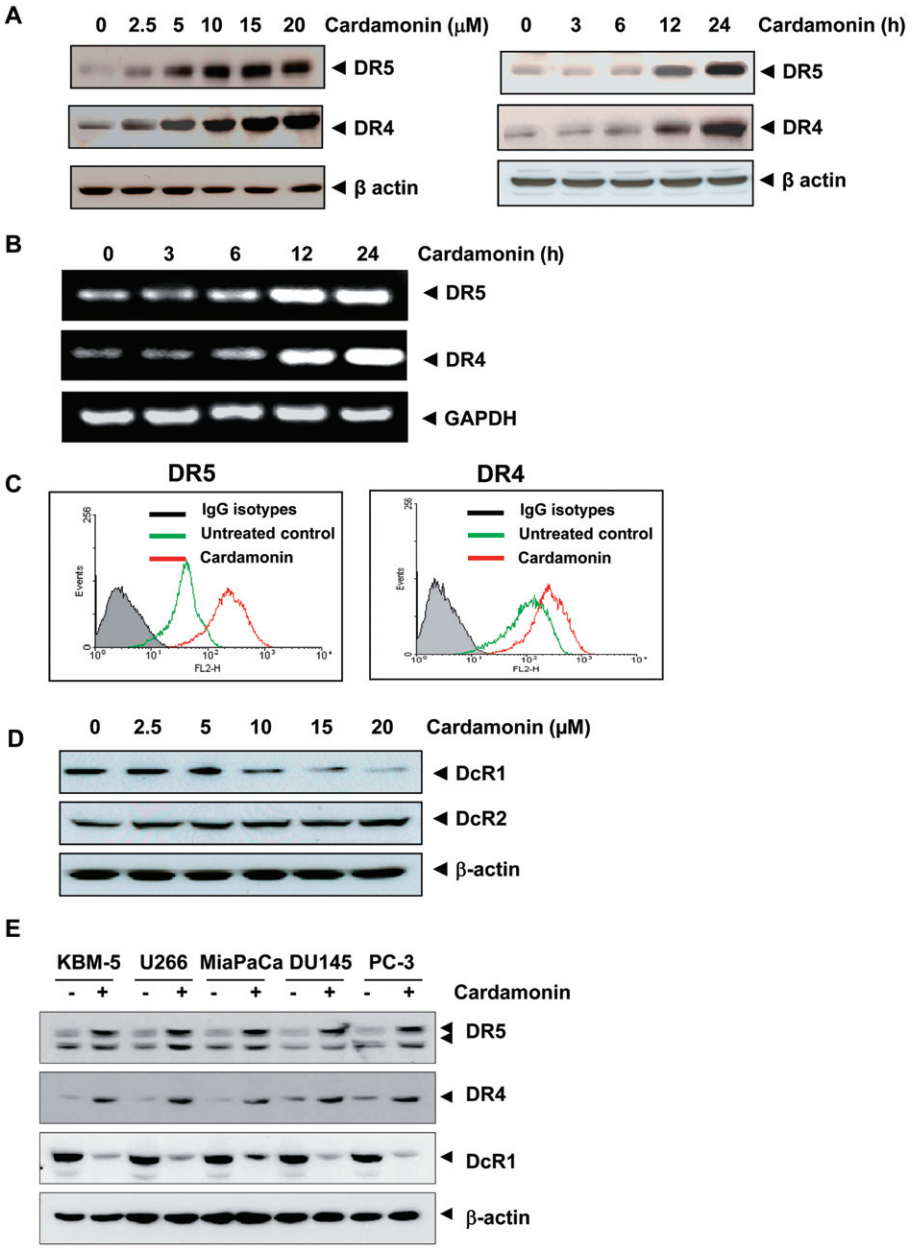

Figure 2

Cardamonin induces expression of death receptors and down-regulates decoy receptors. (A) Cardamonin induces expression of death receptor protein. HCT116 cells (1×10^6 cells per well) were treated with indicated dose of cardamonin for 24 h (left panel). HCT116 cells (1×10^6 cells per well) were treated with indicated time at the dose of 20 μM of cardamonin (right panel). Whole-cell extracts were then prepared and analysed for death receptor expression by Western blotting. (B) Cardamonin induces DR5 gene mRNA expression. Cells (1×10^6 cells mL^{-1}) were treated with 20 μM cardamonin for indicated times, and total RNA was extracted and examined for expression of DR4 and DR5 by RT-PCR. GAPDH was used as an internal control to equalize RNA loading. (C) Cardamonin induces cell surface expression of death receptors. HCT116 cells were treated with 20 μM cardamonin for 24 h and analysed for cell surface DR4 and DR5 by immunofluorescent staining and subsequent flow cytometry. Filled greyish peaks, cells stained with a matched control phycoerythrin-conjugated IgG isotype antibody. (D) Cardamonin down-regulates the expression of DcR1. HCT116 cells were pretreated with indicated dose of cardamonin for 24 h. Whole-cell extracts were prepared and subjected to Western blotting using antibodies specific to DcR1 and DcR2. (E) Cardamonin up-regulates DR5, DR4 and down-regulates DcR1 in various types of cancer cells. Cells (1×10^6 cells) were treated with 20 μM cardamonin for 24 h, after which whole-cell extracts were prepared and analysed by Western blotting. The same blots were stripped and reprobed with β-actin antibody to verify equal protein loading.

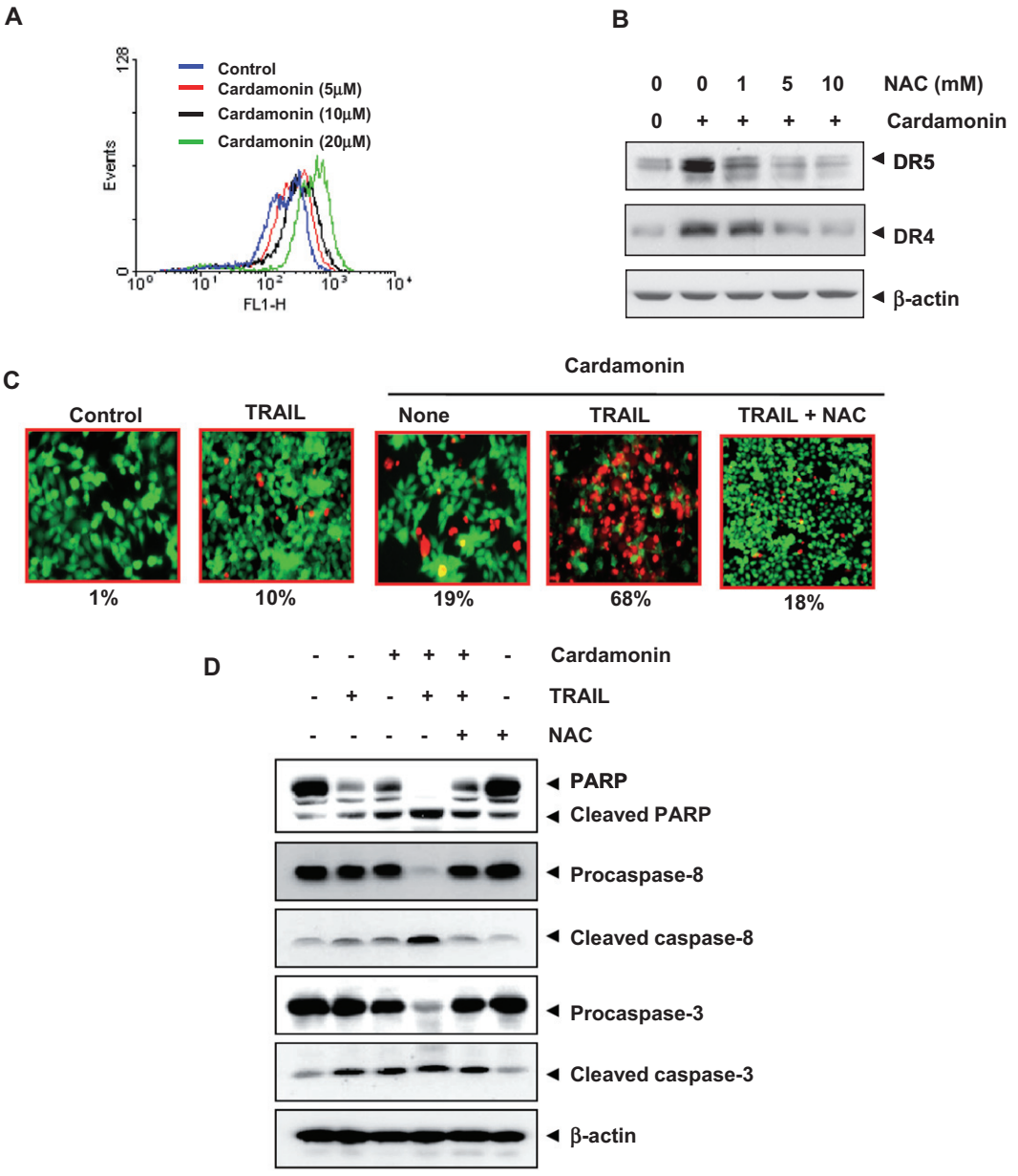

Figure 5

The effect of cardamonin on TRAIL-induced apoptosis is dependent on ROS. (A) Cardamonin induces ROS production. HCT116 (1×10^6 cells) cells were labelled with DCF-DA, treated with indicated concentration of cardamonin for 1 h, and examined for ROS production by flow cytometer. (B) Cardamonin-induced up-regulation of DR5 and DR4 is mediated by ROS. HCT116 cells (1×10^6 cells) were pretreated with various concentrations of NAC for 1 h and then the cells were treated with 20 μM cardamonin for 24 h. Whole-cell extracts were prepared and analysed by Western blotting for DR5 and DR4. (C) NAC reverses cell death induced by combination of cardamonin and TRAIL. HCT116 cells were pretreated with NAC (10 mM) for 1 h and then treated with 20 μM cardamonin for 12 h. After being washed with PBS, cells were treated with TRAIL (25 ng·mL^{-1}) for 24 h. Cell death was determined by the live/dead assay. (D) NAC inhibits caspase activation and PARP cleavage induced by combination of TRAIL and cardamonin. HCT116 cells were treated with NAC, 20 μM cardamonin and TRAIL as indicated above. Whole-cell extracts were prepared and analysed by Western blotting using the relevant antibodies. β-actin was used as a loading control.