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## Author Correction: Ionizing radiation modulates human macrophages towards a pro-inflammatory phenotype preserving their pro-invasive and pro-angiogenic capacities

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This Article contains an error in the description of the data presented in Figure 2.

Each blot demonstrating a protein of interest, or of its phosphorylated form, is matched with the expression of  $\beta$ -actin, used as loading control. The majority of the proteins were separated in different gels, apart from proteins p105, p50 and Bcl-xL which were run in the same gel and have the same loading control.

As a result, the Figure 2 legend,

“Ionizing radiation induces macrophage NF- $\kappa$ B activation and increases Bcl-xL expression. (A) Evaluation of RelA phosphorylation (Ser536) and RelB, cRel, p100/p52 and p105/p50 subunit expression, 1 and 6 h after irradiation (2, 6 and 10 Gy). (B) RelB nuclear translocation 6 h after macrophage irradiation (10 Gy). Histone deacetylase 1 (HDAC1) and  $\beta$ -actin were used as loading controls for nuclear and cytoplasmic fractions, respectively. (C) Evaluation of Bcl2 and Bcl-xL expression after macrophage irradiation. Western blot images are representative of protein expression/phosphorylation status in distinct donors (at least  $n = 4$ ), evaluated in two independent experiments.”

should read:

“Ionizing radiation induces macrophage NF- $\kappa$ B activation and increases Bcl-xL expression. (A) Evaluation of RelA phosphorylation (Ser536) and RelB, cRel, p100/p52 and p105/p50 subunit expression, 1 and 6 h after irradiation (2, 6 and 10 Gy). (B) RelB nuclear translocation 6 h after macrophage irradiation (10 Gy). Histone deacetylase 1 (HDAC1) and  $\beta$ -actin were used as loading controls for nuclear and cytoplasmic fractions, respectively. (C) Evaluation of Bcl2 and Bcl-xL expression after macrophage irradiation. Western blot images are representative of protein expression/phosphorylation status in distinct donors (at least  $n = 4$ ), evaluated in two independent experiments. The  $\beta$ -actin loading control of the panels comprised by p105, p50 (2A) and Bcl-xL (2C) is the same, since proteins were separated in the same gel electrophoresis.”

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