CORRECTION



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Correction: Reduced expression of AMPK-ß1 during tumor progression enhances the oncogenic capacity of advanced ovarian cancer

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Correction

After publication of this article [1] the authors noticed an error in Figure 5A, B and D (Figure 1 here). In Figure 5A (Figure 1 here), the same image from AMPK- β 1 was erroneously used for pAMPK α of A2780cp- β 1 panel. The correct image for pAMPK α is now provided. In Figure 5B and D (Figure 1 here), the panel of OV2008-sh β 1 used two different sets of b-actin. The b-actin in Figure 5B (Figure 1 here) is now used for the whole panel of OV2008-sh β 1 in Figure 5B and D (Figure 1 here). These errors were unintentionally made during figure preparation and do not in any way alter the results or conclusions of this study. The authors apologize that these errors were not detected earlier.

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C25) cells by shrink suppressed AMPK activity (decrease of pAMPKd and pACC (left panel)) but elevated AKT (pAKT) and miOK (pmTOK and pP70S6K) activities (right panel). (C) AMPK- β 1 overexpression sensitizes ovarian cancer cells to an AMPK activator, metformin, during AMPK activation. SKOV3 cells were treated with the AMPK activator, metformin, at 0-, 2-, and 10-mM concentrations. Stable clones overexpressing AMPK- β 1 (C1, C2, C4, and C5) were more sensitive to metformin (2 mM) in the presence of elevated pAMPKa compared with the two empty vector controls (V2 and V3). (D) Depletion of AMPK- β 1 activates the ERK and JNK pathways, and knockdown of AMPK- β 1 in OV2008 (C2, C5 and C32) and OVCA433 (C1, C12 and C23) cells led to an increase in JNK (pJNK) and ERK (pERK) signaling activities.

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