CORRIGENDUM

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Adenosine induces intrinsic apoptosis via the PI3K/Akt/mTOR signaling pathway in human pharyngeal squamous carcinoma FaDu cells

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An interested reader drew to the authors' attention that, in the published version of the above article, the data shown in Fig. 4A for p-Akt and total phosphoinositide 3-kinase (PI3K) were strikingly similar. After having re-examined their source data, the authors were able to confirm that the data correctly shown for total PI3K had also inadvertently been included in the Figure as the data for p-Akt.

A corrected version of Fig. 4, including the correct data for p-Akt for Fig. 4A, is shown opposite. Note that this change does not affect the results or the conclusions reported in this paper, and all the authors agree to this correction. The authors thank the reader for drawing this error to their attention, and apologize to the Editor and to the readership of the Journal for any inconvenience caused.

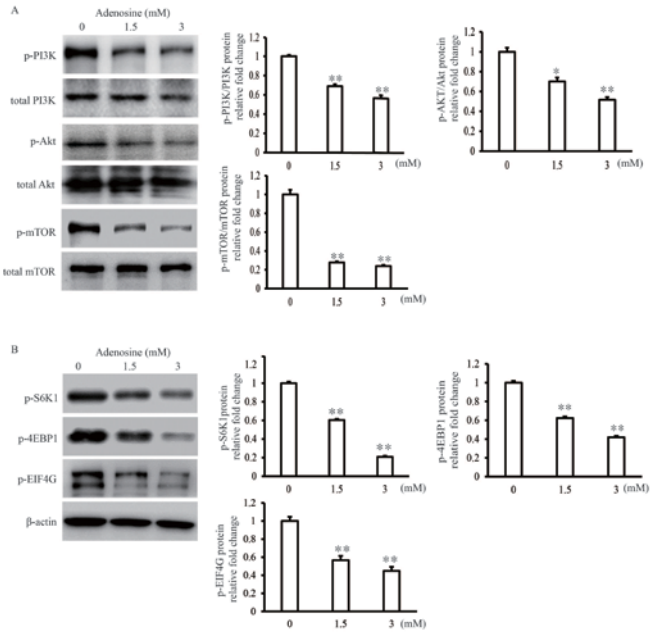

Figure 4. Adenosine treatment of FaDu cells suppresses the PI3K/Akt/mTOR signaling pathway. FaDu cells were treated with 3 mM adenosine for 24 h prior to collection of whole-cell lysates. Samples were separated using 8-15% SDS-PAGE, and resolved by incubation with primary antibodies against (A) phospho-PI3K, total PI3K, phospho-Akt, total Akt, phospho-mTOR, total mTOR, and (B) phospho-S6K1, phospho-4EBP1, and phospho-EIF4 G. β-actin was used as an internal control for the western blot analysis. Data are representative of three experiments that produced similar results. Sample bands (n=3) were densitometrically evaluated. *P<0.05 and **P<0.01. PI3K, phosphoinositide 3-kinase; Akt, RAC serine/threonine-protein kinase; mTOR, mechanistic target of rapamycin; 4EBP1, eukaryotic translation initiation factor 4E-binding protein 1; EIF4 G, eukaryotic translation initiation factor 4 γ1.