CORRIGENDUM

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Dexamethasone-induced inhibition of miR-132 via methylation promotes TGF-β-driven progression of pancreatic cancer

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Subsequently to the publication of the above article, an interested reader drew to the authors' attention that two pairs of the culture plate images in Fig. 4A-C on p. 60 appeared to be the same, although the images were shown in different orientations; moreover, the 'NC/0 and DEX+miR132' and 'DEX and miR132' pairings of images in the scratch-wound assay experiments shown in Fig. 4B also appeared to be overlapping, such that these were apparently derived from the same original source where the results of differently performed experiments were intended to have been portrayed.

After re-examining their original data, the authors have realized that some of the data in Fig. 4A and B were inadvertently assembled incorrectly. The revised version of Fig. 4, showing all the correct data for the culture plate images in Fig. 4A-C (specifically, the images fifth along on the right for Fig. 4B and C have been revised) and the correct images for 'NC/0' and 'DEX/0' in Fig. 4D is shown on on the next page. The authors are grateful to the Editor of *International Journal of Oncology* for allowing them this opportunity to publish a Corrigendum, and all the authors agree with its publication. Furthermore, the authors apologize to the readership for any inconvenience caused.



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Figure 4. Cancer progression features are inhibited by miR-132. (A) AsPC-1, PANC-1 and ASAN-PaCa cells were transfected with 50 nM miR-132 mimics or a negative miR control (NC). At 8 h later, the cells were treated with 1 μ M DEX in the presence or absence of miR-132. After 48 h, the cells were resuspended in complete medium and plated at a low clonogenic density in 6-well tissue culture plates. After 14 days, colony-forming assays were performed and evaluated as described in the materials and methods. (B) PANC-1 cells were transfected as aforementioned. At 48 h after transfection, the cells were seeded at a high density in ibidi culture insert 24-well plates. After 24 h, once the cells had attached and reached ~90% confluency, the inserts were removed to leave a defined 500- μ m-thick scratch. Images of the cell-free gap were obtained immediately (0 h), and at 24 and 48 h after removal of the inserts. (C) PANC-1 cells were treated as afore-described, and cell viability was measured using a RealTime-GloTM MT Cell Viability Assay. (D) The number of PANC-1 cells was determined by the use of a Coulter counter after 24, 48 and 72 h of treatment. Data are presented as the means \pm SD. *P<0.05. DEX, dexamethasone; NC, negative miR control.