Correction

Correction: CD73 is associated with poor prognosis in HNSCC

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Published:

This article has been corrected: Due to an error during figure assembly, an incorrect immunohistochemical staining of CD73 was included in Figure 1A. In addition, due to errors during image selection, the CD 73 protein band in Figure 3B and the E-cadherin protein band in Figure 4C are accidental duplicates. The control group in Figure 2D and the control group in Figure 3C are also accidental duplicates. In Figure 5C, immunohistochemical staining images for "control" and "siRNACD73" are also accidentally duplicated. The corrected Figure 1A, Figure 3B and 3C, and Figure 5C, obtained from original data, are shown below. The authors declare that these corrections do not change the results or conclusions of this paper.

Original article: Oncotarget. 2016; 7:61690–61702. https://doi.org/10.18632/oncotarget.11435

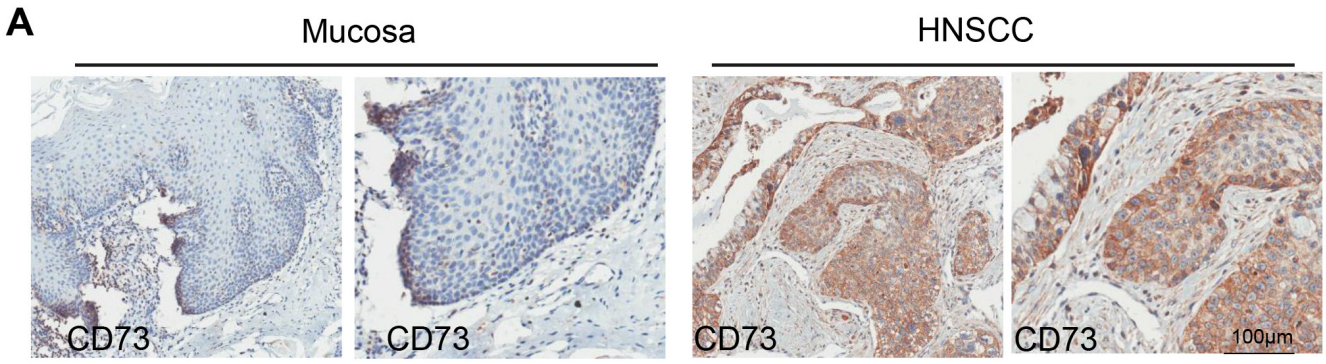

Figure 1: CD73 expression is upregulated and associated with poor prognosis in Head and Neck Squamous Cell Carcinoma (HNSC). (**A**) Representative immunohistochemical staining (IHC) of CD73 in head and neck squamous cell carcinoma tissue (left) as compared to normal oral mucosa (right) (*Scale bars* = 100 μm); (**B**) Quantification of histoscores of CD73 expression in normal oral mucosa ($n = 51$), epithelial dysplasia ($n = 11$) and HNSCC (n = 162), $^{*}P < 0.05$, $^{***}P < 0.001$; (**C**) Patients with high CD73 expression have a poor prognosis as compared to patients with low expression ($P = 0.002$, $n = 162$), Significance of differences in survival between patient groups was estimated by log-rank test; (**D**) Western blot analysis of the protein expression of CD73 in patients with/without Lymph node. GAPDH was used as loading control; (**E**) Relative CD73 mRNA expression was detected by RT-PCR in patients with versus without Lymph node metastasis. The data were presented as the means ± SEM. One-way ANOVA with post-Dunnett analysis was performed using GraphPad Prism 5. $^{*}P < 0.05$, $^{**}P < 0.01$ versus the control group. ($n = 3$).

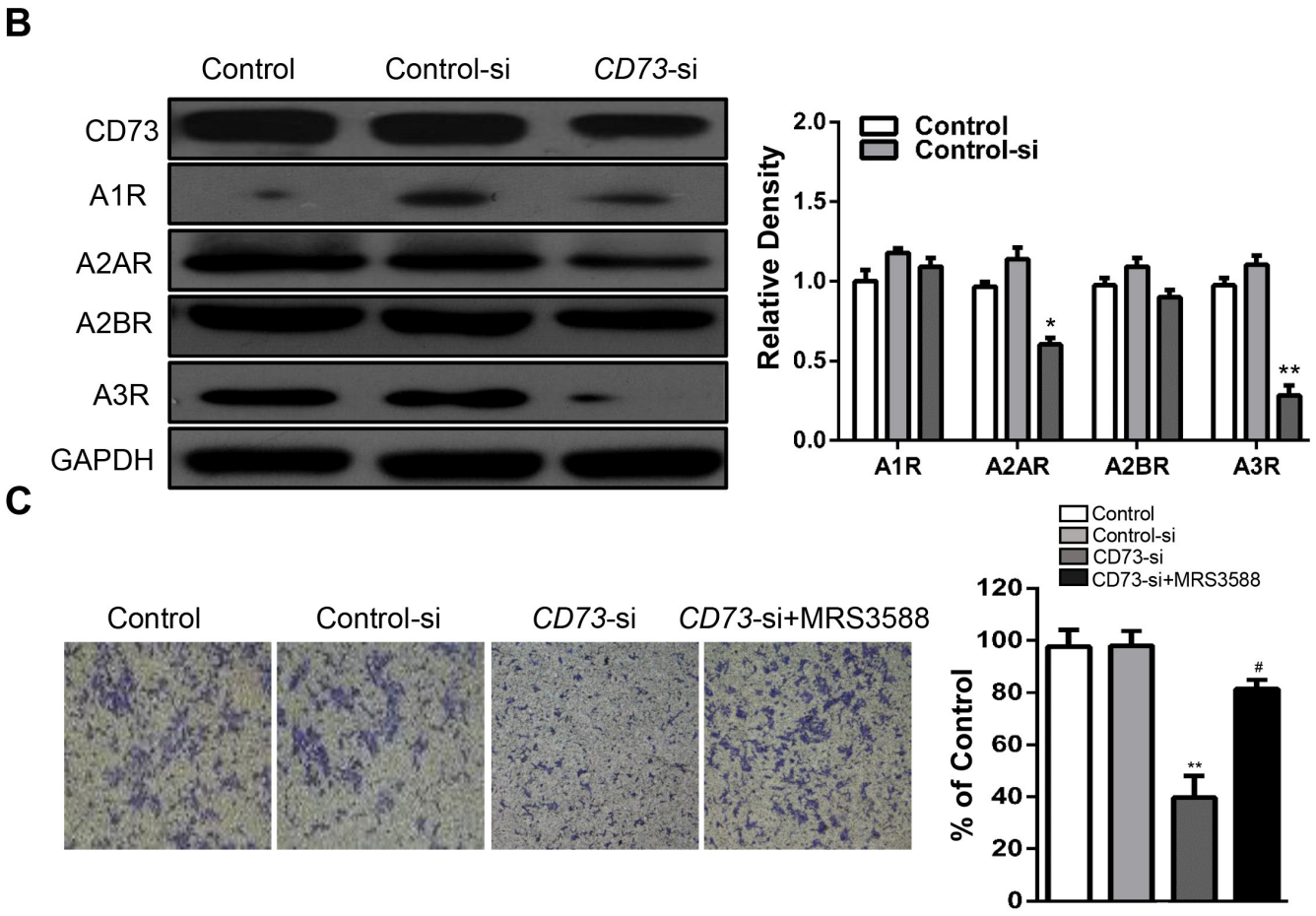

Figure 3: CD73 promotes invasion and metastasis of HNSC through adenosine A3 receptor stimulation. (**A**) Confocal immunofluorescence microscopy of CD73 and A1R, A2AR, A2BR, A3R co-expression in CAL27 cells; (**B**) CAL27 cells were treated with siRNA for CD73, then the A1R, A2AR, A2BR and A3R levels were determined. GAPDH was the internal standard for protein loading. The values are presented as the means ± SEM. One-way ANOVA with post-Dunnett analysis was performed using GraphPad Prism5. $^{*}P < 0.05$, $^{**}P < 0.01$ versus the control group. ($n = 3$); (**C**) Transwell assay demonstrated that CAL27 cell invasion was impaired following knocking down of CD73 compared with those of control group. These findings could be reversed using MRS3588, an agonist of A3R, quantification of cell numbers with Image J "cell counter" module (Mean ± SEM; $^{**}P < 0.01$, versus control-si group, $^{\#\#}P < 0.01$, versus the CD73-si group, student *t*-test with GraphPad Prism5.0), (*Scale bars* = 200 μm).

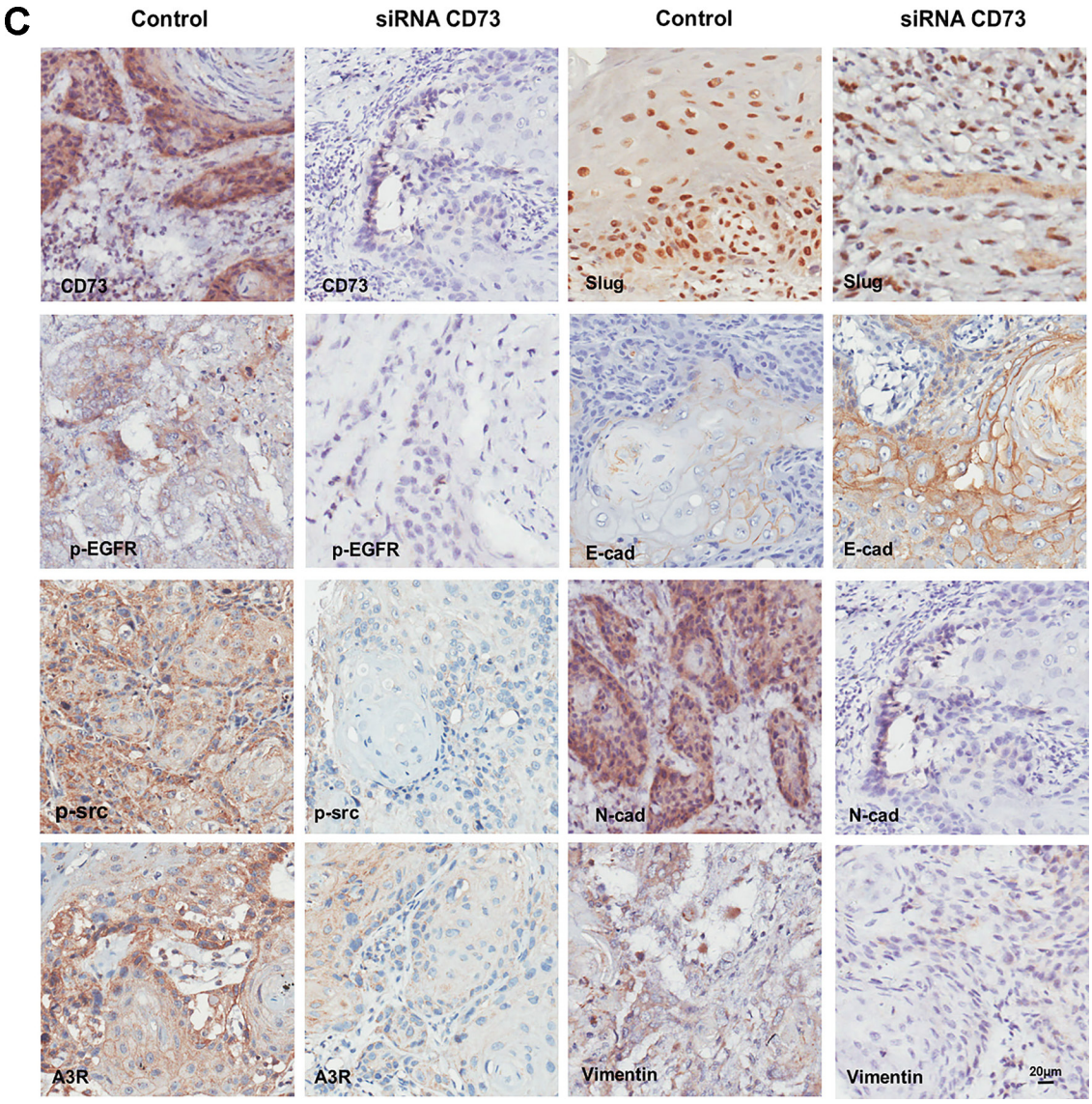

Figure 5: si-CD73 inhibits HNSC tumor growth and suppress EMT *in vivo*. (**A**) Tumor growth curve of siRNA-CD73 mice and control mice. Data represent the mean ± SEM. of eight mice in each group. $^{*}P < 0.05$, $^{**}P < 0.01$, $^{***}P < 0.001$ by the Student's *t*-test; (**B**) Dissected tumors were photographed. The tumor volume and weight were measured. $^{***}P < 0.001$ by the Student's *t*-test; (**C**) Representative images of immunohistochemical analysis of CD73, p-EGFR, p-Src, A3R, Slug, E-cad, N-cad and Vimentin in tumors, (*Scale bars* = 100 μm).