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Erratum
AGR2 promotes the proliferation, migration and regulates epithelial-mesenchymal transition in salivary adenoid cystic carcinoma: Am J Transl Res. 2017; 9(2): 507-519

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Following a thorough review of the published article, we have identified that there were several inadvertent misuses of images in our publication. Specifically, in **Figure 2A**, the immunofluorescence image of the TGF-β1 in the AdCC tissue was mistakenly used. In **Figure 3B**, the colony formation image in the shAGR2 group of the SACC-LM cell line was also mistakenly used. In **Figures 4C** and **5D**, the transwell images of the SACC-LM and SACC-38 cell lines were mistakenly used. These errors occurred during the preparation of the manuscript and unfortunately went unnoticed prior to publication. The corrected Figures are displayed below. We sincerely apologize for any confusion the errors may have caused. All authors have confirmed the mistakes did not affect the conclusions of this article.

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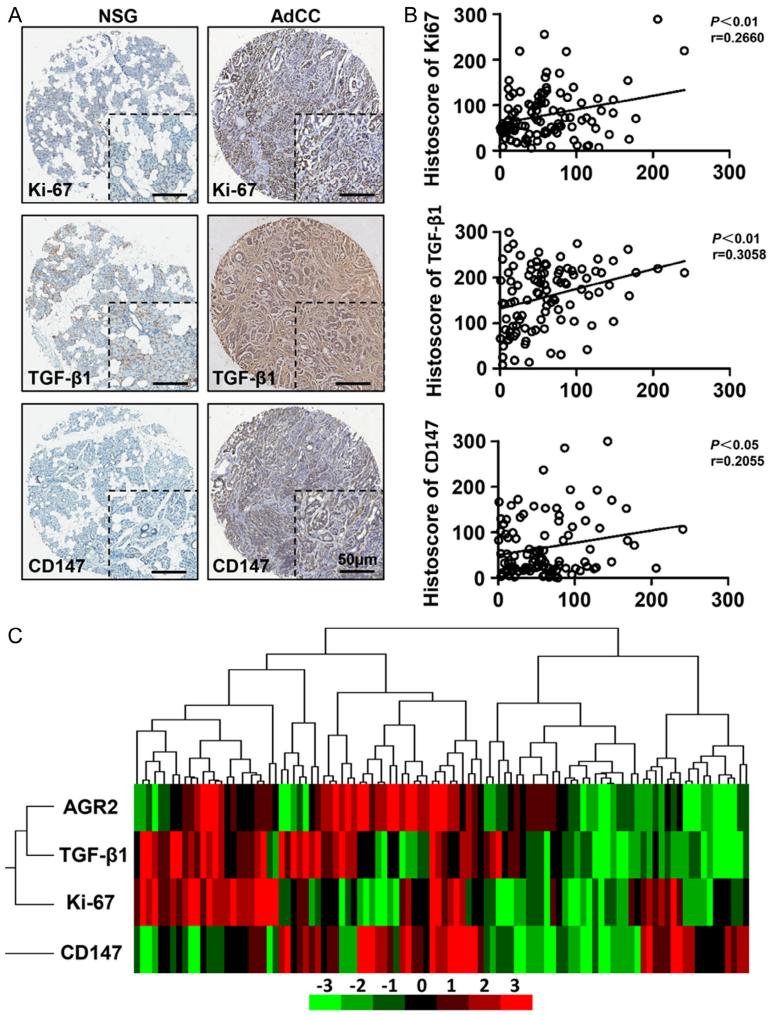

Figure 2. Overexpression of AGR2 is correlated with Ki-67, TGF-β1, and CD147 in human salivary AdCC tissue microarray. A. Representative photos of Ki-67, TGF-β1, and CD147 of normal salivary gland (NSG) or adenoid cystic carcinoma (AdCC) tissues (scale bar = 50 μm). B. Two-tailed Pearson's statistics showed a significant correlation of AGR2 with Ki-67, TGF-β1, and CD147. C. Hierarchical clustering of AGR2, Ki-67, TGF-β1, and CD147 histoscore results in human AdCC tissue microarrays.

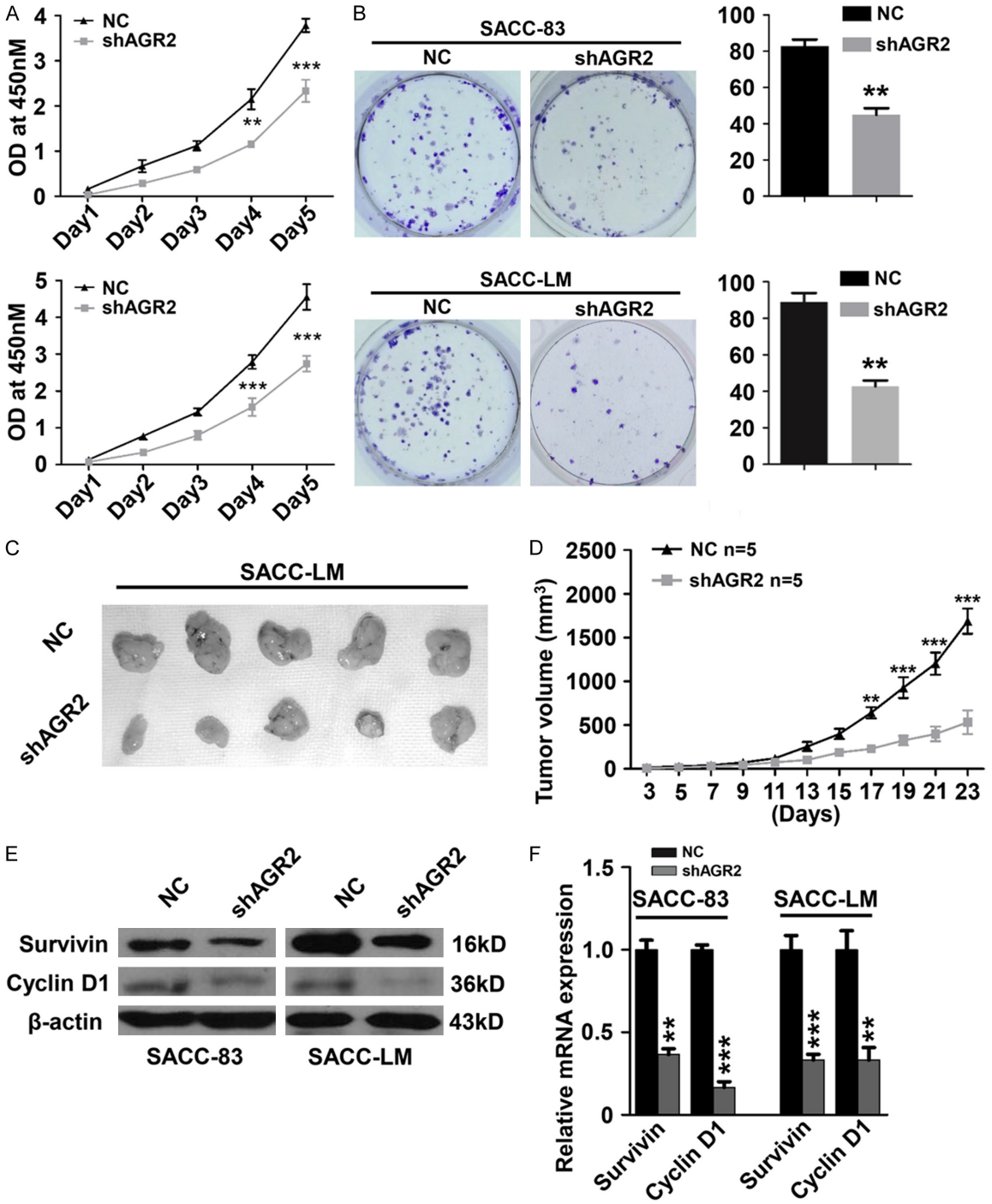

Figure 3. Knockdown of AGR2 represses cell proliferation of AdCC cell lines *in vitro* and *in vivo*. A. Cell viability assays on SACC-83 and SACC-LM cells. Knockdown of AGR2 significantly repressed the proliferation of SACC-83 and SACC-LM cells on Days 4 and 5 (Mean ± SEM, **P<0.01, ***P<0.001). B. Anchorage-dependent colony formation assay of SACC-83 and SACC-LM cells. Knockdown of AGR2 significantly attenuated the colony formation ability of both SACC-83 and SACC-LM cells (Mean ± SEM, **P<0.01, unpaired t test). C. Representative images of the tumors of negative control group (NC, n = 5) and AGR2 shRNA group (shAGR2, n = 5). D. Tumor size of NC group and shAGR2 group was assessed (Mean ± SEM, **P<0.01, ***P<0.001 unpaired t test). E. Western blotting indicated that knockdown of AGR2 reduced the protein level of Survivin and Cyclin D1 in SACC-83 cells and SACC-LM cells. β-actin was used as loading control. F. Knockdown of AGR2 significantly reduced the mRNA levels of Survivin and Cyclin D1 in SACC-83 cells and SACC-LM cells (Mean ± SEM, **P<0.01, ***P<0.001, unpaired t test).

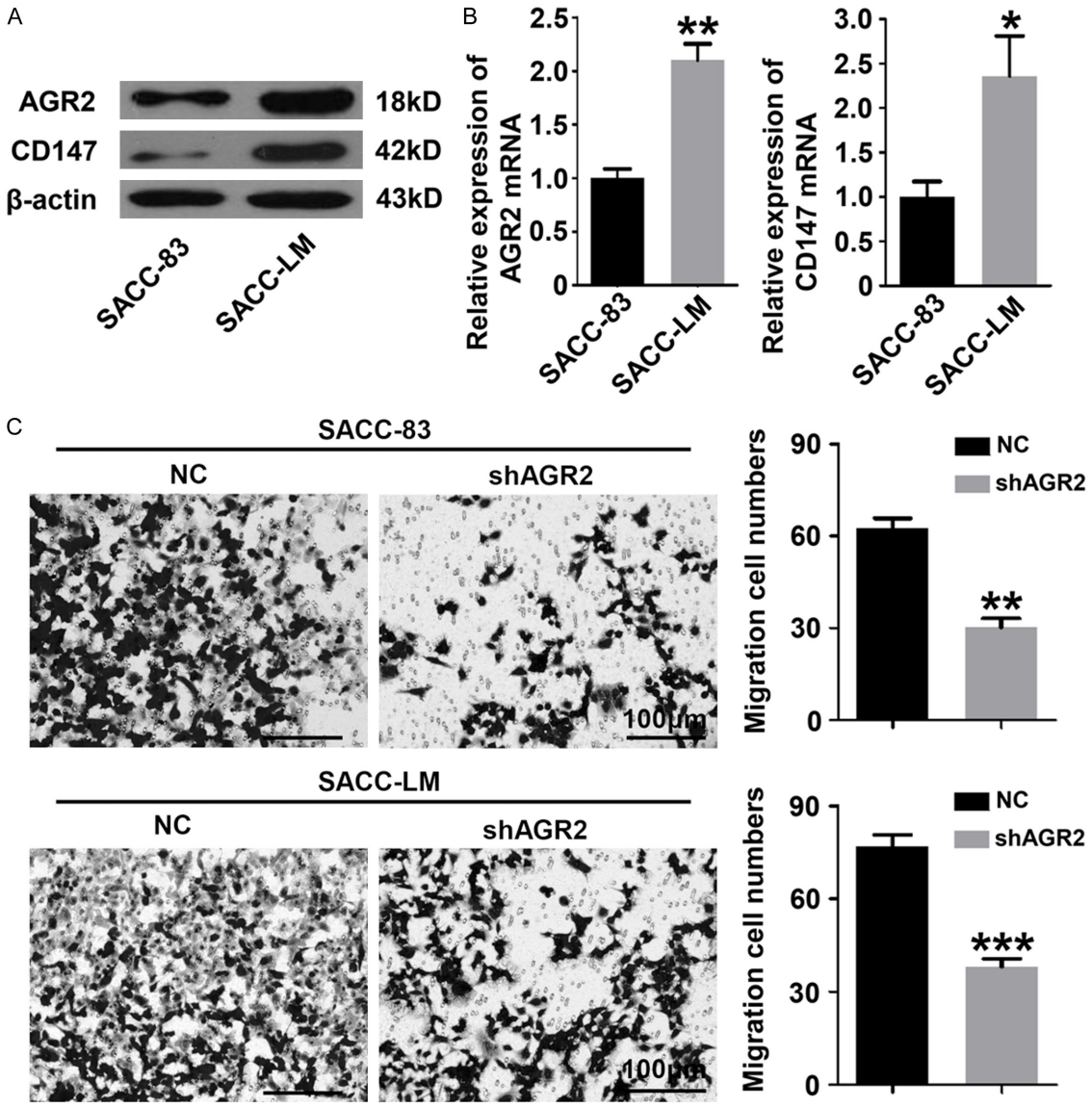

Figure 4. Knockdown of AGR2 inhibits cell migration of AdCC cell lines. A. Western blotting showed that the protein levels of AGR2 and CD147 in SACC-LM cells were higher. β-actin was used as loading control. B. The mRNA levels of AGR2 and CD147 were significantly increased in SACC-LM cells (Mean ± SEM, *P<0.05, **P<0.01, unpaired t test). C. Transwell migration assays revealed that knockdown of AGR2 significantly inhibited the migration ability of SACC-83 and SACC-LM cells (scale bar = 100 μm).

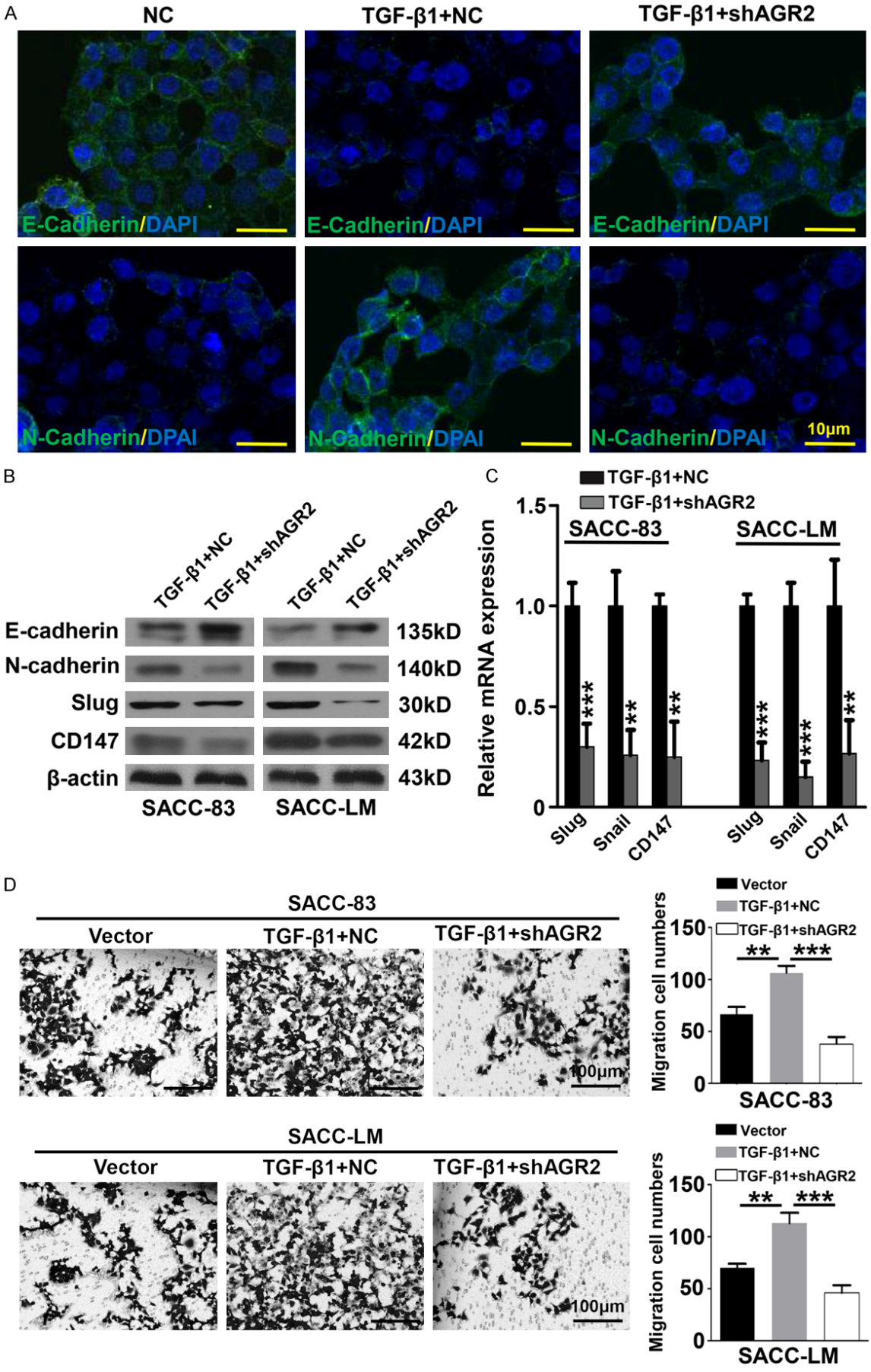
A
NC
TGF-β1+NC
TGF-β1+shAGR2
E-Cadherin/DAPI
N-Cadherin/DPAI
10μm
B
TGF-β1+NC
TGF-β1+shAGR2
E-cadherin
N-cadherin
Slug
CD147
β-actin
135kD
140kD
30kD
42kD
43kD
SACC-83
SACC-LM
C
TGF-β1+NC
TGF-β1+shAGR2
SACC-83
SACC-LM
Relative mRNA expression
1.5
1.0
0.5
0
Slug
Snail
CD147
D
SACC-83
Vector
TGF-β1+NC
TGF-β1+shAGR2
100μm
Migration cell numbers
150
100
50
0
SACC-LM

Figure 5. Knockdown of AGR2 reverses the EMT induced by TGF-β1. A. Representative Immunofluorescence photos of SACC-LM treated with negative control shRNA (NC), TGF-β1 with or without AGR2 shRNA (shAGR2) are displayed. The expression of E-cadherin was reduced by TGF-β1 but reversed by AGR2 knockdown. The expression of N-cadherin was up-regulated by TGF-β1 but recovered by AGR2 knockdown (scale bar = 10 μm). B. Western blotting showed the expression of E-cadherin, N-cadherin, Slug, and CD147 in SACC-83 and SACC-LM cells tranfected with negative control shRNA (NC) or with AGR2 shRNA (shAGR2) in the presence of TGF-β1. β-actin was used as loading control. C. The mRNA levels of Slug, Snail, and CD147 in SACC-83 and SACC-LM cells were significantly reduced by AGR2 silencing in the presence of TGF-β1 (Mean ± SEM, **P<0.01, ***P<0.001, unpaired t test). D. Knockdown of AGR2 reversed the enhanced ability of migration which was induced by TGF-β1 in SACC-83 cells and SACC-LM cells in the indicated treatment group (NC, Negative control; shAGR2, AGR2 short haripin RNA, scale bar = 100 μm).