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Erratum
In vitro and *in vivo* antiangiogenic activity of desacetylvinblastine monohydrazide through inhibition of VEGFR2 and Axl pathways: Am J Cancer Res. 2016; 6(4): 843-858

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In this article, some mistakes have been found that the images in **Figures 3D** and **4C** were misassembled by the inadvertently forgotten of removing the overlay images during the preparation of figures with Photoshop software, due to the unfamiliarity of software operation in 2016.

The details of the mistakes during figure preparation are as follows:

1. The image of tube formation in Gas6+DAVLBH group in **Figure 3D** was wrongly duplicated with that in VEGF+DAVLBH group in **Figure 1D**.

2. The Western blot band of Active Cdc42 in **Figure 4C** was inadvertently duplicated with the band of Axl in **Figure 4B**.

So, we would like to publish this Erratum to reflect this change. The authors express regrets for this mistake. The correction does not affect our findings and conclusions.

The corrected **Figures 3** and **4** are shown below.

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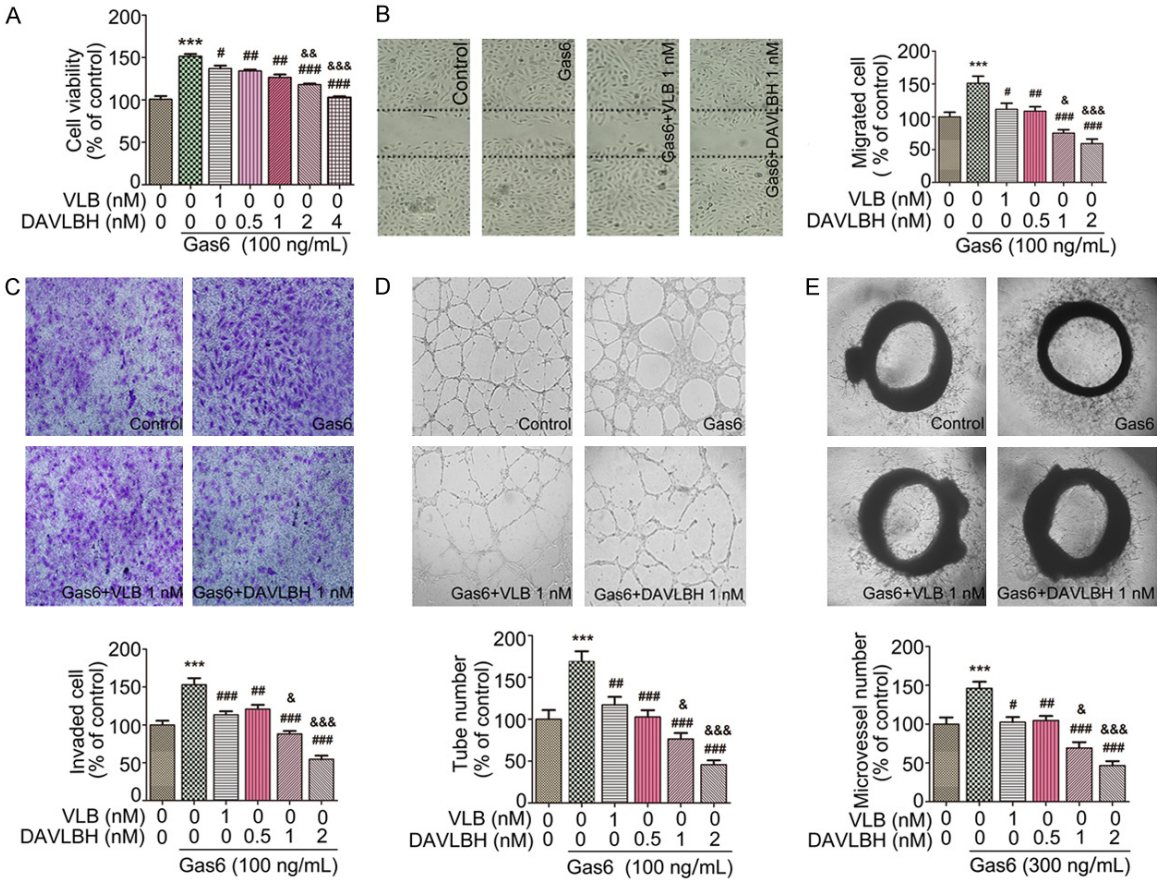

Figure 3. DAVLBH inhibited Gas6-induced angiogenesis in vitro and ex vivo. DAVLBH inhibited Gas6-stimulated HUVEC (A) proliferation, (B) migration, (C) invasion, (D) capillary-structure formation and (E) aortic ring microvessel sprouting. HUVECs or rat aortic rings were treated with VLB or various concentrations of DAVLBH in the presence or absence of Gas6. Representative figures (100 × magnification in B-D and 40 × magnification in E) are shown, and the quantitative data were analyzed with GraphPad Prism 5.0. The data are presented as mean ± SEM, n = 5. $^{***}P < 0.001$ versus the control group; $^{\#}P < 0.05$, $^{\#\#}P < 0.01$, and $^{\#\#\#}P < 0.001$ versus the Gas6-treated group; and $^{\&}P < 0.05$, $^{\&\&}P < 0.01$, and $^{\&\&\&}P < 0.001$ compared with the VLB-treated group.

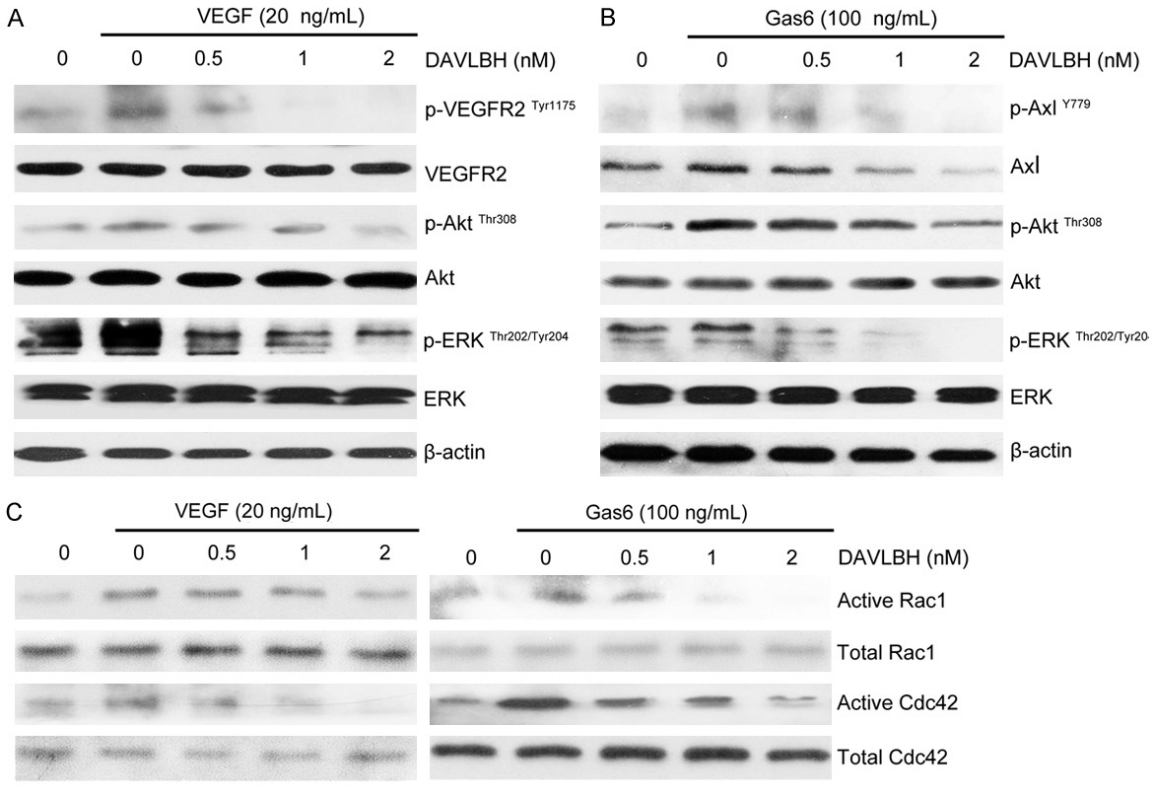

Figure 4. DAVLBH suppressed VEGF/VEGFR2 and Gas6/Axl signaling pathways. DAVLBH inhibited VEGFR2, Axl, Akt and ERK in (A) VEGF-treated HUVECs and (B) Gas6-treated HUVECs. HUVECs were pre-treated with various concentrations of DAVLBH for 4 h and then stimulated with VEGF or Gas6 for 1 h. Protein was collected and subjected to Western blot analysis. (C) DAVLBH inhibited the VEGF- and Gas6-induced activation of Rac1 and Cdc42. HUVECs were starved with serum-free ECM and treated with different concentrations of DAVLBH for 4 h and then stimulated with VEGF or Gas6 for 1 h. The cells were then lysed, and active Rac1 and Cdc42 were pulled down and applied to Western blotting.