

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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Following the publication of erratum in 2023 (Am J Cancer Res 2023; 13(1): 352-354), two additional mistakes were identified. The Western blot bands for β -actin in **Figure 2B** and Total Rac1 in the Gas6+DAVLBH group in **Figure 4C**, were inadvertently misused due to an error during figure preparation in 2016. We are issuing this erratum to correct the figures. The authors sincerely apologize for this oversight and any confusion that may have caused. This correction does not affect the findings or conclusions of this study. The corrected **Figures 2, 4** are shown below.

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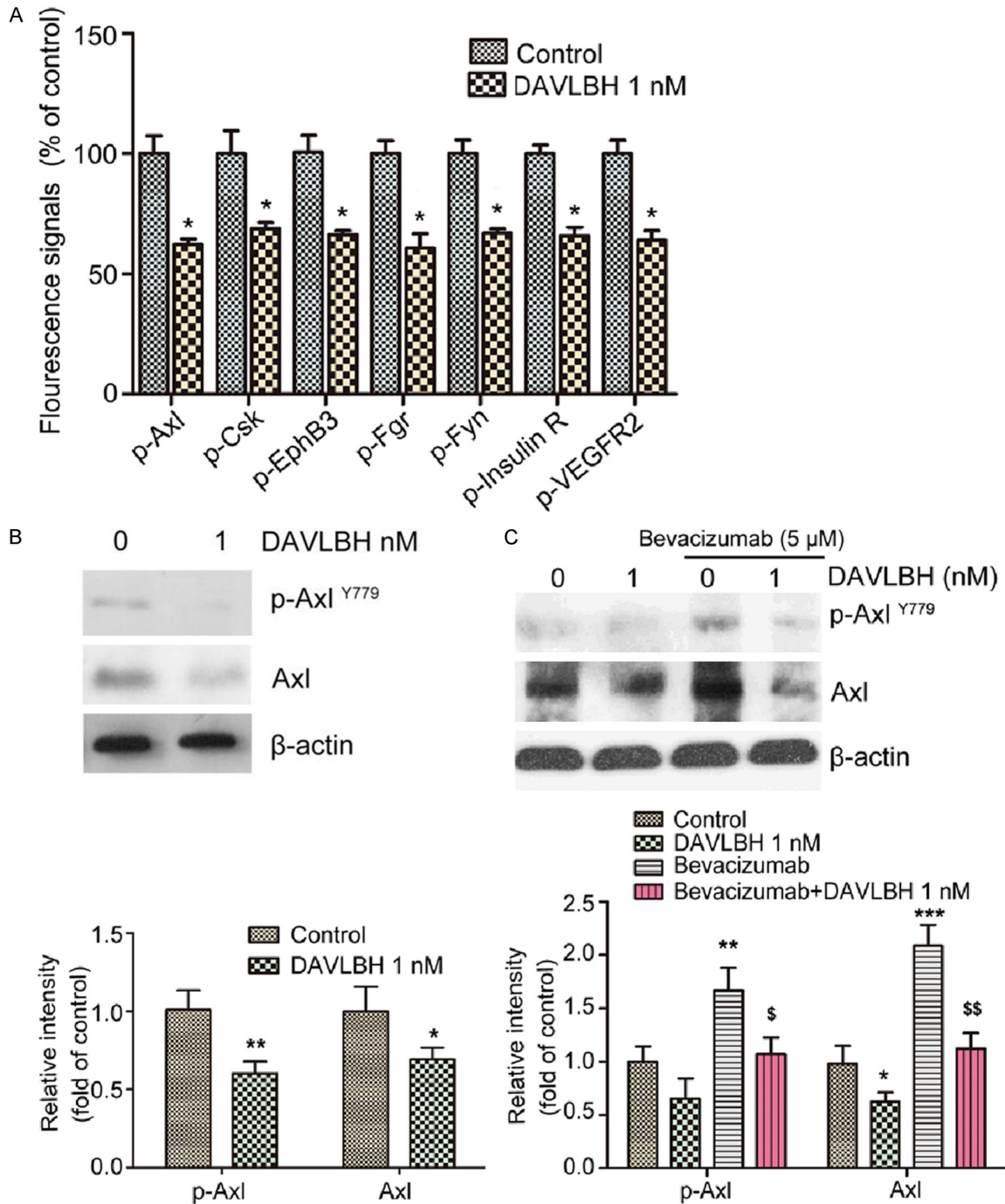


Figure 2. DAVLBH suppressed the expression of Axl in HUVECs. **A.** Quantitative analysis of the changes in human phosphorylated RTKs associated with angiogenesis in DAVLBH-treated HUVECs. **B.** DAVLBH inhibited the expression of Axl in normal HUVECs. HUVECs were treated with or without DAVLBH (1 nM) for 4 h. Cells were collected, lysed and subjected to western blot analysis. **C.** DAVLBH antagonized the increased expression of Axl in bevacizumab-treated HUVECs. Cells were continuously stimulated with bevacizumab for 48 h and then treated with or without DAVLBH for 4 h. The levels of Axl and p-Axl in the collected cell supernatants were analyzed by western blotting. The quantitative analysis of Western blot was shown. Data are ratios of p-Axl/ β -actin and Axl/ β -actin, and are presented as mean $\pm$ SEM, $n = 5$. * $P < 0.05$, ** $P < 0.01$ and *** $P < 0.001$ compared with the control group; \$ $P < 0.05$ and \$\$ $P < 0.01$, compared with the bevacizumab-treated group.

DAVLBH inhibits angiogenesis

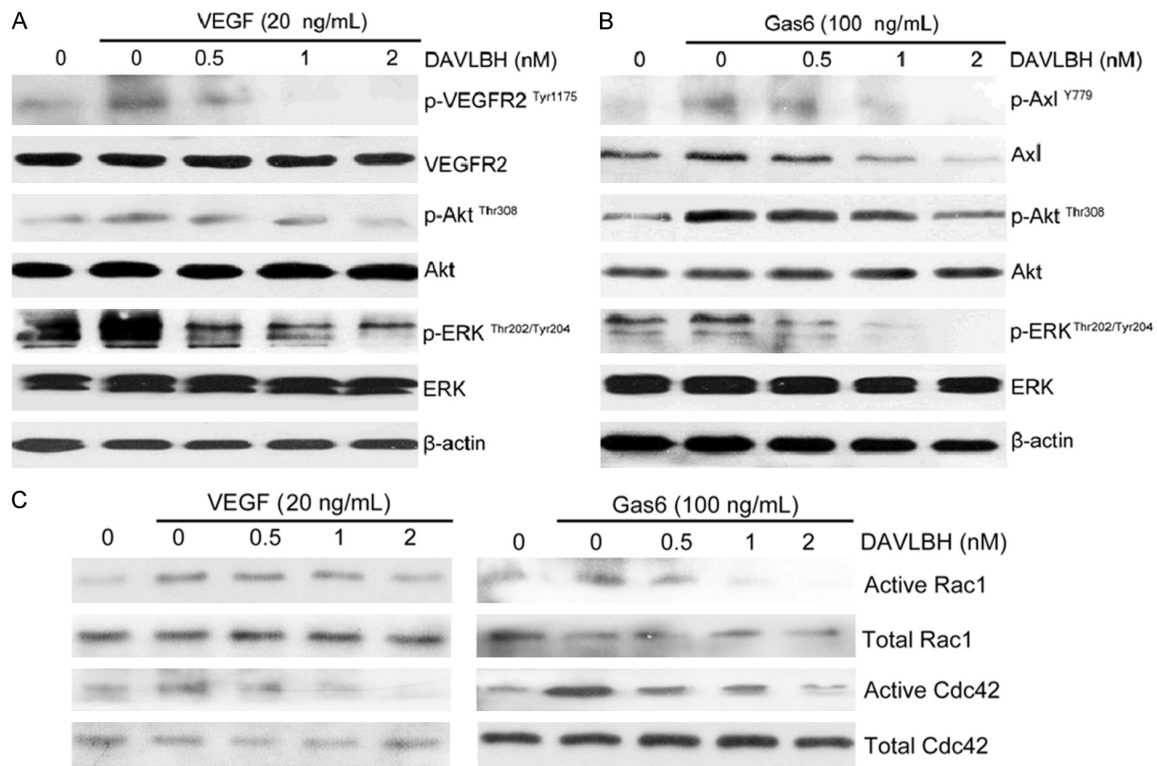


Figure 4. DAVLBH suppressed VEGF/VEGFR2 and Gas6/Axl signaling pathways. DAVLBH inhibited VEGFR2, Axl, Akt and ERK in (A) VEGF-treated HUVECs and (B) Gas6-treated HUVECs. HUVECs were pre-treated with various concentrations of DAVLBH for 4 h and then stimulated with VEGF or Gas6 for 1 h. Protein was collected 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.