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ERRATUM

Erratum to: Contribution of tumor endothelial cells to drug resistance: anti-angiogenic tyrosine kinase inhibitors act as p-glycoprotein antagonists

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**Erratum to: Angiogenesis
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In the original publication of the article, the panel B of Fig. 3 is incorrect. The correct version of the Fig. 3 is provided in this erratum.

The online version of the original article can be found under doi:10.1007/s10456-017-9549-6.

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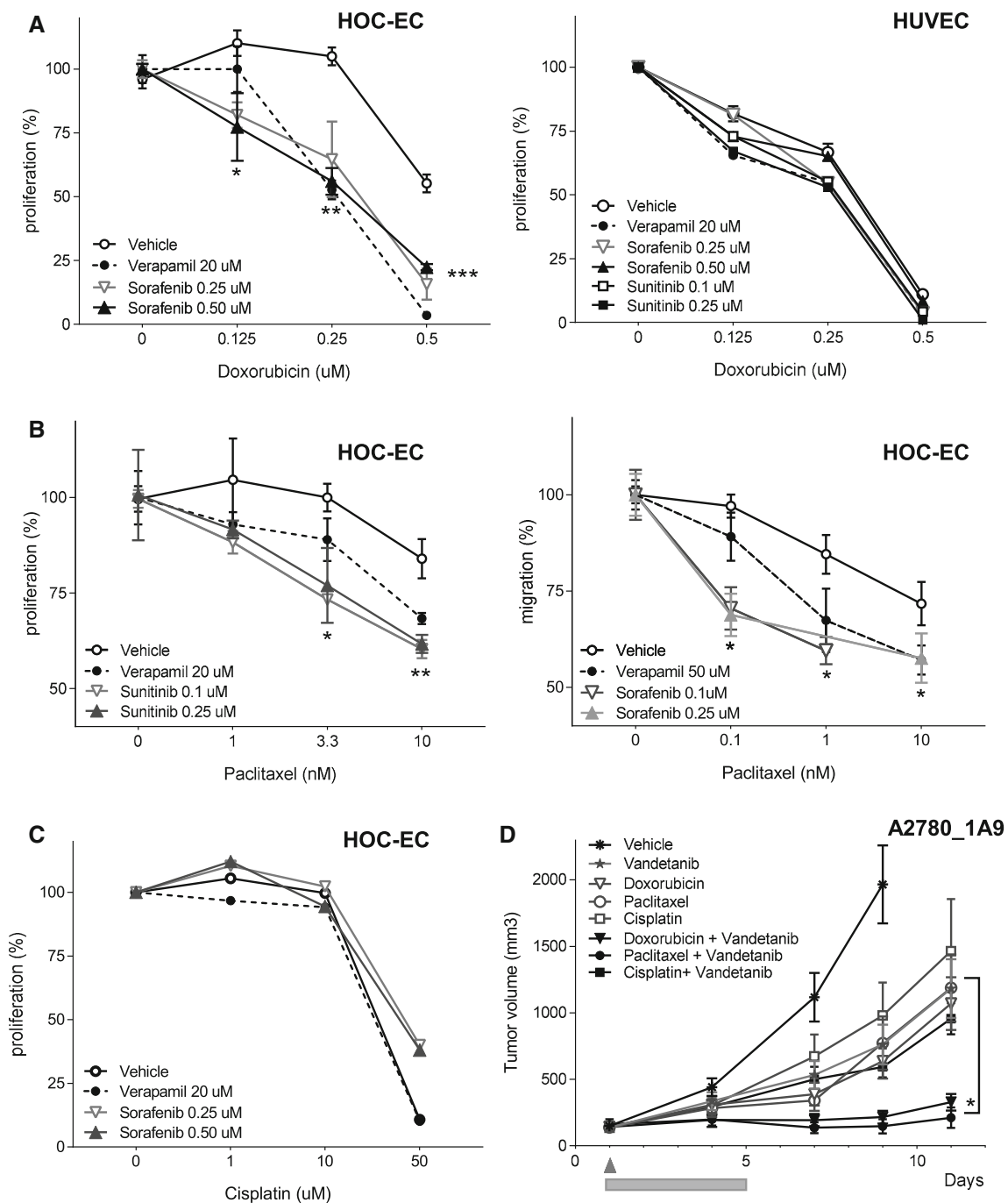
Fig. 3 TKI enhance HOC–EC responsiveness to doxorubicin, paclitaxel but not cisplatin. **a–c** In vitro responsiveness of TEC to chemotherapeutics. HOC–EC or HUVEC were exposed to increasing concentrations of doxorubicin (**a**), paclitaxel (**b**) or cisplatin (**c**) for the duration of the assays (72 h for proliferation and 4 h for migration). Verapamil, sorafenib or sunitinib at the indicated concentrations were added 2 h earlier. Proliferation and migration are expressed as percentage of control (in the absence of chemotherapeutic drugs). Data are representative of at least three independent experiments. **d** In vivo tumor growth inhibition. A2780-1A9 ovarian cancer cells (10×10^6) were transplanted subcutaneously in nude mice that were randomized to treatments at tumor volume of 150 mm^3 (eight mice per group). Vandetanib was administered p.o. at a dose of 50 mg/kg for 5 days. Doxorubicin (8 mg/kg), paclitaxel (20 mg/kg) or cisplatin (5 mg/kg) were administered i.v. as a single bolus (*arrowhead*). Response is shown as tumor volume over time. $P < 0.05$ (*), $P < 0.01$ (**), $P < 0.001$ (***)