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Erratum: Mouse and human strategies identify *PTPN14* as a modifier of angiogenesis and hereditary haemorrhagic telangiectasia

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This article contains four typographical errors in the abstract. Hereditary haemorrhagic telangiectasia is incorrectly abbreviated as HTT instead of HHT. The correct version of the abstract follows.

Hereditary haemorrhagic telangiectasia (HHT) is a vascular dysplasia syndrome caused by mutations in transforming growth factor-β/bone morphogenetic protein pathway genes, *ENG* and *ACVRL1*. HHT shows considerable variation in clinical manifestations, suggesting environmental and/or genetic modifier effects. Strain-specific penetrance of the vascular phenotypes of $Eng^{+/-}$ and $Tgfb1^{-/-}$ mice provides further support for genetic modification of transforming growth factor-β pathway deficits. We previously identified variant genomic loci, including *Tgfbm2*, which suppress prenatal vascular lethality of $Tgfb1^{-/-}$ mice. Here we show that human polymorphic variants of *PTPN14* within the orthologous *TGFBM2* locus influence clinical severity of HHT, as assessed by development of pulmonary arteriovenous malformation. We also show that *PTPN14*, *ACVRL1* and *EFNB2*, encoding EphrinB2, show interdependent expression in primary arterial endothelial cells *in vitro*. This suggests an involvement of PTPN14 in angiogenesis and/or arteriovenous fate, acting *via* EphrinB2 and ACVRL1/activin receptor-like kinase 1. These findings contribute to a deeper understanding of the molecular pathology of HHT in particular and to angiogenesis in general.