

Fraser SR, Booth NA, Mutch NJ. The antifibrinolytic function of factor XIII is exclusively expressed through α_2 -antiplasmin cross-linking. *Blood*. 2011;117(23):6371-6374.

On pages 6372 and 6373 in the 9 June 2011 issue, there are errors in the third and fourth sentences in the third paragraph of “Results and discussion” and in the Figure 2 legend. The incorrect sentences read, “In contrast, when a neutralizing antibody to α_2 AP was also added, no change in lysis was observed (Figure 2A open circles). We verified that the addition of FXIII had no effect on lysis of thrombi formed from α_2 AP depleted plasma (Figure 2A closed triangles).” The sentences should have read, “In contrast, when a neutralizing antibody to α_2 AP was also added, no change in lysis was observed (Figure 2A closed triangles). We verified that the addition of FXIII had no effect on lysis of thrombi formed from α_2 AP depleted plasma (Figure 2A open circles).” In the Figure 2 legend, the symbol for α_2 AP depleted plasma should be an open circle and the symbol for FXIII depleted plasma with neutralizing antibody to α_2 AP should be a closed triangle. The first sentence of the Figure 2 legend should have read, “(A) Plasma thrombi were prepared from FXIII depleted plasma in the absence (●) and presence (▲) of a neutralizing antibody to α_2 AP or α_2 AP depleted plasma (○).”

Montserrat E, Gribben JG. Autografting CLL: the game is over! *Blood*. 2011;117(23):6057-6058.

On page 6057 of the 9 June 2011 Inside *Blood*, the table legend that read “FCR versus ASCT for CML” should have been titled “FCR versus ASCT for CLL.”

Merryweather-Clarke AT, Atzberger A, Soneji S, et al. Global gene expression analysis of human erythroid progenitors. *Blood*. 2011;117(13):e96-e108.

On pages e96, e98, and e107 in the 31 March 2011 issue, there is an error in the Web link www.cellline.molbiol.ox.ac.uk/eryth/index.html; the “www.” should have been “https://.” The URL should have been “<https://cellline.molbiol.ox.ac.uk/eryth/index.html>.”

The link appears 3 times in the article. On page e96 in the last sentence of the “Abstract,” the sentence should have read: “Our Human Erythroid Maturation database is available at <https://cellline.molbiol.ox.ac.uk/eryth/index.html>.”

In “Methods” on page e98 in the second sentence of the second paragraph under the heading “Web-accessible database,” the sentence should have read: “The Human Erythroblast Maturation database is available at <https://cellline.molbiol.ox.ac.uk/eryth/index.html>.”

In the third paragraph of “Acknowledgments” on page e107, the sentence should have read: “The Human Erythroblast Maturation (HEM) Database is available at: <https://cellline.molbiol.ox.ac.uk/eryth/index.html>.”

Sehrawat S, Hernandez T, Cullere X, et al. AKAP9 regulation of microtubule dynamics promotes Epac1-induced endothelial barrier properties. *Blood*. 2011;117(2):708-718.

On page 712 of the 13 January 2011 issue, the data from independent experiments used to plot histograms in Figure 3A and B were incorrectly compiled and therefore contained errors. The original data have been re-plotted and, although the correction results in a change in individual data points plotted, the profile of the histograms remains the same as those in Figure 3 of the parent article. Thus, the results of Figure 3 and conclusions stated in the original article remain unchanged. The legend for the corrected figure is as published in the parent article. The corrected figure is shown.

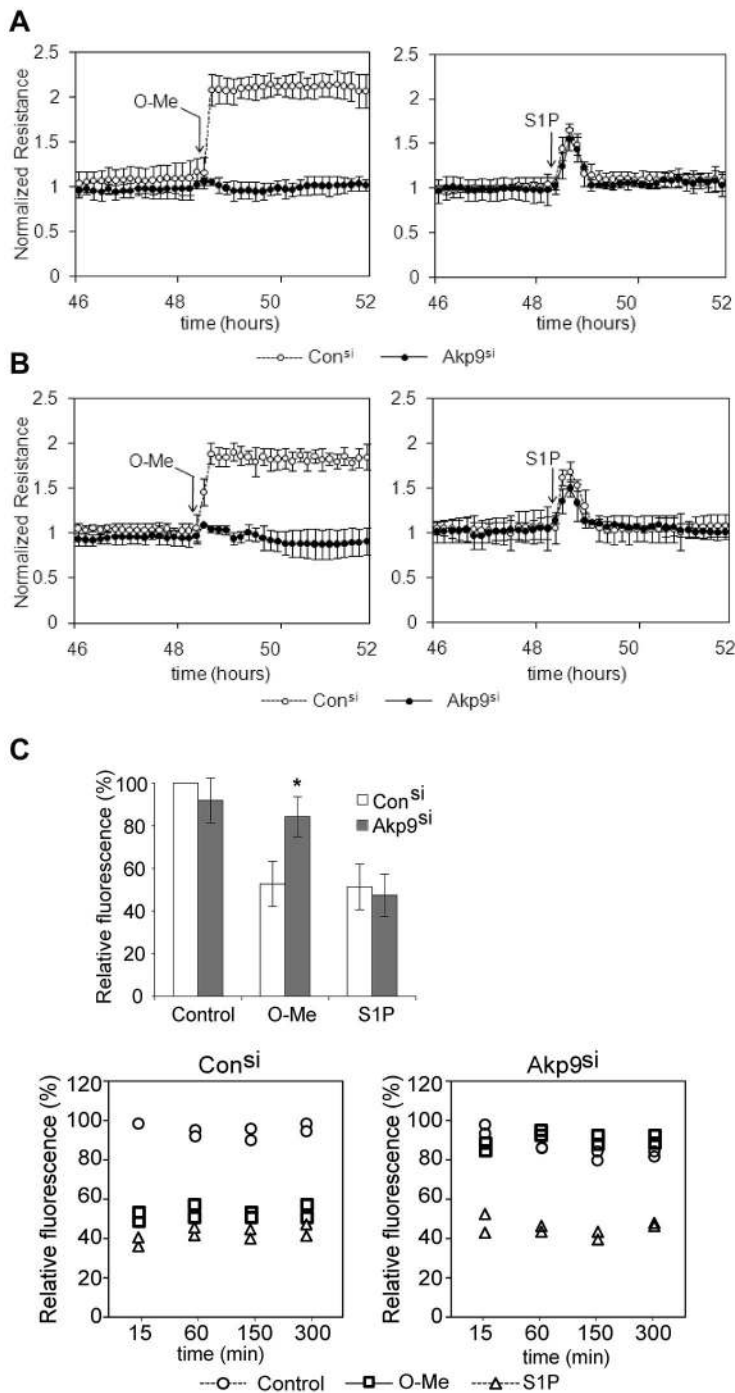


Figure 3. AKAP9-depleted cells display defects in Epac-induced enhancement of barrier properties. (A) TER of AKAP9 (Akp9^{si}) and control (Con^{si}) siRNA-treated HUVEC monolayers at baseline and following the addition of O-Me-cAMP (left panel) or S1P (right panel) at the times indicated by arrows. N = 5 experiments. (B) TER in HDMECs in response to indicated agonists. N = 3 experiments. In panels A and B, TER measurement was normalized to the resistance of the sample at time 0, which was taken 10 minutes following the addition of siRNA duplexes. Data points are average \pm SD. TER data points at 45 hours and greater reflect the effects of AKAP9 silencing on resistance. AKAP9 silencing did not significantly affect basal permeability in HUVECs (A) or HDMECs (B). However, AKAP9-silenced cells fail to respond to O-Me-cAMP. (C) Top panel, dextran leakage across control siRNA and AKAP9 siRNA cell monolayers after 15 minutes of treatment with vehicle control, O-Me, or S1P (n = 3). Bottom panels, time course of dextran leakage in control (left) and AKAP9-silenced (right) cells following agonist treatment for the indicated times in minutes. One of 2 representative experiments is shown; the data represent 2 determinations (in duplicate wells) at each time point. Results are reported as a percentage of the value obtained with control siRNA at the 15-minute time point, which was set at 100%. *P < .05.

Allen A, Fisher C, Premawardhena A, et al. Adaptation to anemia in hemoglobin E- β thalassemia. *Blood*. 2010;116(24):5368-5370.

On page 5368 of the 9 December 2010 issue, the affiliation of the second author (Fisher) is incomplete. He is correctly assigned to the WIMM in Oxford but not to the particular unit in which he worked within the Institute, which is the MRC Molecular Haematology Unit. The correct affiliation for Dr Fisher should have been: MRC Molecular Haematology Unit, Weatherall Institute of Molecular Medicine, John Radcliffe Hospital, Headington, Oxford, United Kingdom, and the corrected affiliation list is shown. The error has been corrected in the online version, which now differs from the print version.

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