

Correction to “Mending Leaky Blood Vessels: The Angiopoietin-Tie2 Pathway in Sepsis”

In the above article [David S, Kumpers P, van Slyke P, and Parikh SM (2013) *J Pharmacol Exp Ther* 345:2–6], the abstract was omitted from the XML and PDF versions of the article published on March 19, 2013. The abstract was included in the Fast Forward version of the article published on February 1, 2013. The abstract is as follows:

Sepsis is a systemic inflammatory response to infection. A common end-feature, these patients regularly suffer from is the so-called multiple organ dysfunction syndrome, an often fatal consequence of organ hypoperfusion, coagulopathy, immune dysregulation, and mitochondrial dysfunction. Microvascular dysfunction critically contributes to the morbidity and mortality of this disease. The angiopoietin (Angpt)/Tie2 system consists of the transmembrane endothelial tyrosine kinase Tie2 and its circulating ligands (Angpt-1, -2, and -3/4). The balance between the canonical agonist Angpt-1 and its competitive inhibitor, Angpt-2, regulates basal endothelial barrier function and the leakage and vascular inflammation that develop in response to pathogens and cytokines. Here we summarize recent work in mice and men to highlight the therapeutic potential in this pathway to prevent or even reverse microvascular dysfunction in this deadly disease.

Revised XML and PDF versions of the article that include the abstract have replaced those published on March 19.

We regret this error and any inconvenience it may have caused.