Errata

Duh EJ. Sema 3A resists retinal revascularization. *Blood*. 2011;117(22):5785-5786.

On page 5785 in the 2 June 2011 issue, there is an error in the Inside *Blood* title. There should be no spaces in the abbreviation for Semaphorin 3A (Sema3A). The title reads, "Sema 3A resists retinal revascularization." The title should have read, "Sema3A resists retinal revascularization."

Hole PS, Darley RL, Tonks A. Do reactive oxygen species play a role in myeloid leukemias? *Blood*. 2011;117(22):5816-5826.

On page 5817 in the 2 June 2011 issue, there is an error in Figure 1: the Fenton chemistry cycle is drawn with the arrows in the opposing direction to the true nature of the cycle. The corrected Figure 1 is shown.

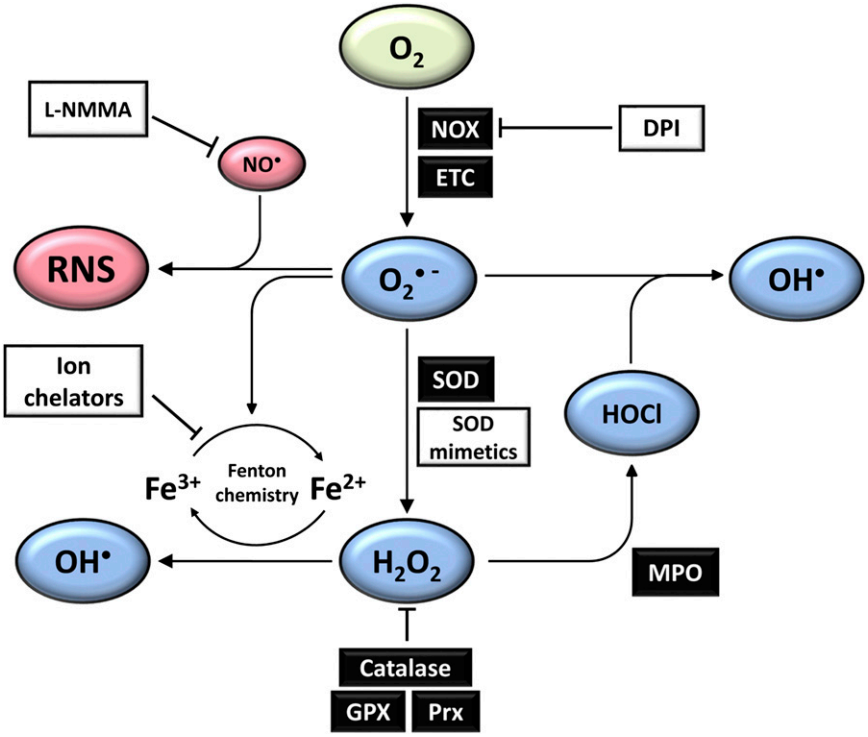

Figure 1. Physiologic ROS homeostasis networks. Univalent reduction of oxygen results in the formation of superoxide ($O_2^{\bullet -}$), which can occur as a result of NADPH oxidase (NOX) activity, and also as a by-product of oxidative phosphorylation, primarily at complex I in the mitochondrial electron transport chain (ETC). Superoxide may act as a reductant or an oxidant and is a key molecule in several subsequent physiologic reactions. Most of the superoxide generated in vivo is converted into hydrogen peroxide (H_2O_2) primarily by the actions of superoxide dismutases, which exist in cytosolic (SOD1), mitochondrial (SOD2), and extracellular (SOD3) isoforms. H_2O_2 levels are tightly regulated by several mechanisms, including the actions of catalase, the glutathione peroxidase (GPX) system, and peroxiredoxins (Prx). H_2O_2 may be further processed by the actions of myeloperoxidase (MPO) during an immune response to form hypochlorous acid (HOCl), which may in turn react with superoxide to form hydroxyl radicals. Hydroxyl radicals may also be formed from H_2O_2 by Fenton chemistry, which may occur in the presence of free metal cations such as Fe^{2+} or Cu^{+}. Where superoxide production and production of nitric oxide ($NO^{\bullet}$) are colocalized, reactive nitrogen species (RNS) may be formed, with the proximal species being peroxynitrite. Various RNS may then form via further chemical reactions with other ROS or RNS. This network of ROS and RNS production can be disrupted or biased in the presence of various compounds such as diphenyleneiodonium (DPI), which inhibits flavoproteins including the NOX oxidase family, ion chelators that can terminate Fenton chemistry cycles, and L-arginine analogs such as L-monomethyl arginine (L-NMMA), which inhibits nitric oxide synthase. Green represents molecular oxygen, blue are ROS derived from O_2, and red represents nitric oxide and other RNS.

Hsu LL. Hydroxyurea makes inflammation "just right"? *Blood*. 2012;119(8):1796-1798.

On page 1797 in the 23 February 2012 issue, there is an error in the text. The first sentence of the seventh paragraph of the article reads, "Their final experiment used clinical SCD blood samples obtained during the Pediatric Hydroxyurea Phase III Clinical (BABY HUG) randomized trial.[7]" The sentence should have read, "Their final experiment used clinical SCD blood samples obtained during the pediatric Hydroxyurea Study of Long-Term Effects (HUSTLE, NCT00305175).[7]"

Furthermore, on page 1798, the citation at the end of the corrected sentence is incorrect in "References." Reference 7 reads, "Wang WC, Ware RE, Miller ST, et al. Hydroxycarbamide in very young children with sickle-cell anaemia: a multicentre, randomised, controlled trial (BABY HUG). *Lancet.* 2011;377(9778):1663-1672." Reference 7 should have read, "Flanagan JM, Steward S, Howard TA, et al. Hydroxycarbamide alters erythroid gene expression in children with sickle cell anaemia. *Br J Haematol*. 2012;157(2):240-248."