Errata

Cappellini MD, Bejaoui M, Agaoglu L, et al. Iron chelation with deferasirox in adult and pediatric patients with thalassemia major: efficacy and safety during 5 years' follow-up. *Blood*. 2011;118(4):884-893.

On page 884 in the 28 July 2011 issue, there is an error in the affiliation of the first author (Cappellini). The word "Scientifico" is misspelled. The affiliation should have read: "[1]Universitá di Milano, Ca Granda Foundation Istituto di Ricovero e Cura a Carattere Scientifico (IRCCS), Milan, Italy." On pages 885, 887, 889, 891, and 893, there is an error in the running title of the article; the word "EFFICIENCY" should be "EFFICACY." The running title should have read: "DEFERASIROX 5-YEAR EFFICACY AND SAFETY." The errors were corrected in the online version, which now differs from the print version.

Booth C, Gilmour KC, Veys P, et al. X-linked lymphoproliferative disease due to SAP/SH2D1A deficiency: a multicenter study on the manifestations, management and outcome of the disease. *Blood*. 2011;117(1):53-62.

On page 53 of the 6 January 2011 issue, the 17th author's last name was misspelled Pachlopnick-Schmid. The correct name is Pachlopnik Schmid. The error has been corrected in the online version, which now differs from the print version.

Gregori S, Tomasoni D, Pacciani V, et al. Differentiation of type 1 T regulatory cells (Tr1) by tolerogenic DC-10 requires the IL-10–dependent ILT4/HLA-G pathway. *Blood*. 2010;116(6):935-944.

On page 935 in the 12 August 2010 issue, one of the affiliations of the third author (Valentina Pacciani) is incorrect. The second affiliation listed as "Department of Pediatrics, Università di Tor Vergata, Rome" should have been: "University Department of Pediatrics (DPUO), Bambino Gesù Children's Hospital, Rome, Italy." The correct byline and affiliations are shown. The error has been corrected in the online version, which now differs from the print version.

Silvia Gregori,[1] Daniela Tomasoni,[1] Valentina Pacciani,[1,2] Miriam Scirpoli,[3] Manuela Battaglia,[1,3] Chiara Francesca Magnani,[1] Ehud Hauben,[1] and Maria-Grazia Roncarolo[1,4]

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Gomes AL, Carvalho T, Serpa J, Torre C, Dias S. Hypercholesterolemia promotes bone marrow cell mobilization by perturbing the SDF-1:CXCR4 axis. *Blood*. 2010;115(19):3886-3894.

On pages 3888, 3889, and 3892 in the 13 May 2010 issue, there are errors in the color of the bars in the plots of several figure panels. In Figures 1C, 1D, 2B, 6B, and 6C, when parameters are compared between the 2 conditions, "Normal diet" on the left should be gray, and "High-cholesterol diet" on the right should be black. The corrected Figures 1, 2, and 6 are shown.

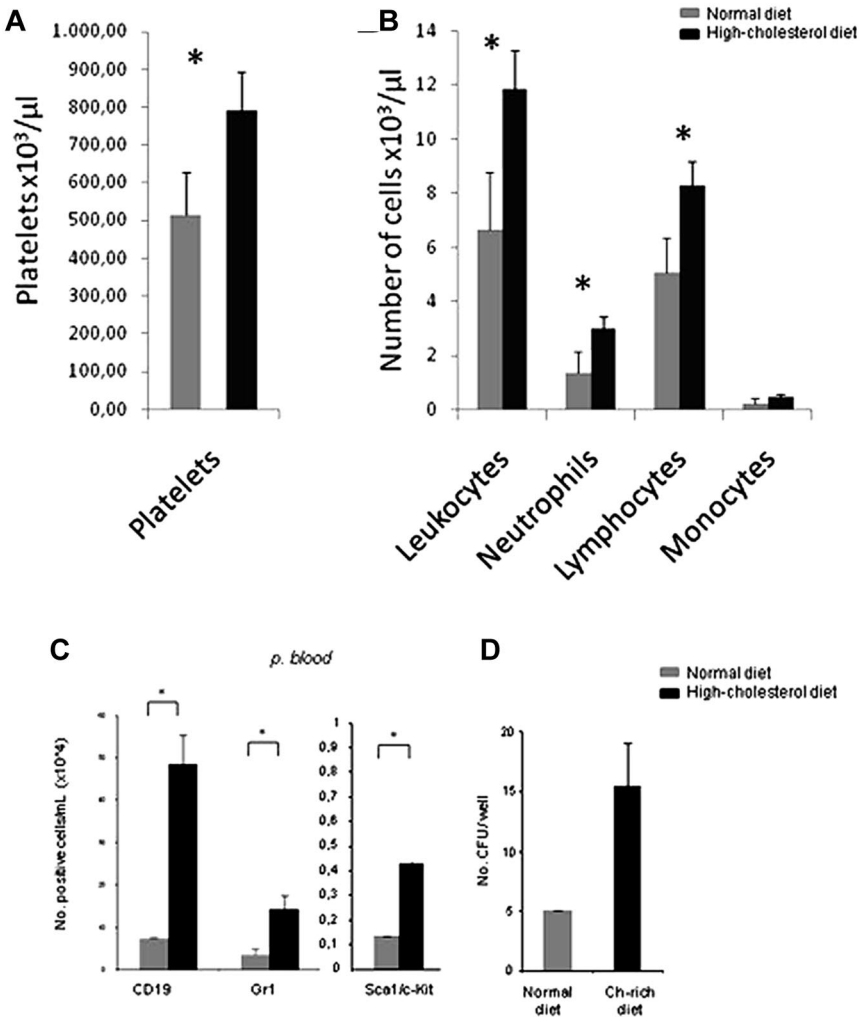

Figure 1. A high-cholesterol diet is associated with thrombocytosis, lymphocytosis, and increased circulating progenitor cells. (A) Hypercholesterolemia was accompanied by increased platelet counts (thrombocytosis; $\times 10^3/\mu L$). (B) Besides thrombocytosis, leukocytosis also was observed in mice fed a high-cholesterol diet. The leukocytosis in mice fed a high-cholesterol diet was mainly caused by the significant increase in circulating lymphocytes and neutrophils ($\times 10^3/\mu L$). (C) Flow cytometry analysis with Gr-1 (neutrophils), Sca1/c-Kit (progenitors), and CD19 (B lymphocytes) cell-surface markers confirms that the leukocytosis is mainly attributable to a massive increase in circulating lymphocytes (lymphocytosis) and neutrophils (neutrophilia; $\times 10^3/\mu L$). In addition, hypercholesterolemia was also accompanied by an increase in the number of circulating progenitor cells ($\times 10^4$/mL;$^*P < .05$). (D) Isolated Lin$^-$Sca1$^+$c-Kit$^+$ cells from the PB of mice fed a normal diet and a high-cholesterol diet form CFUs in methylcellulose cultures, demonstrating their progenitor potential ($^*P < .05$). These experiments were performed 3 times with groups of 6 mice/experimental condition with consistent results.

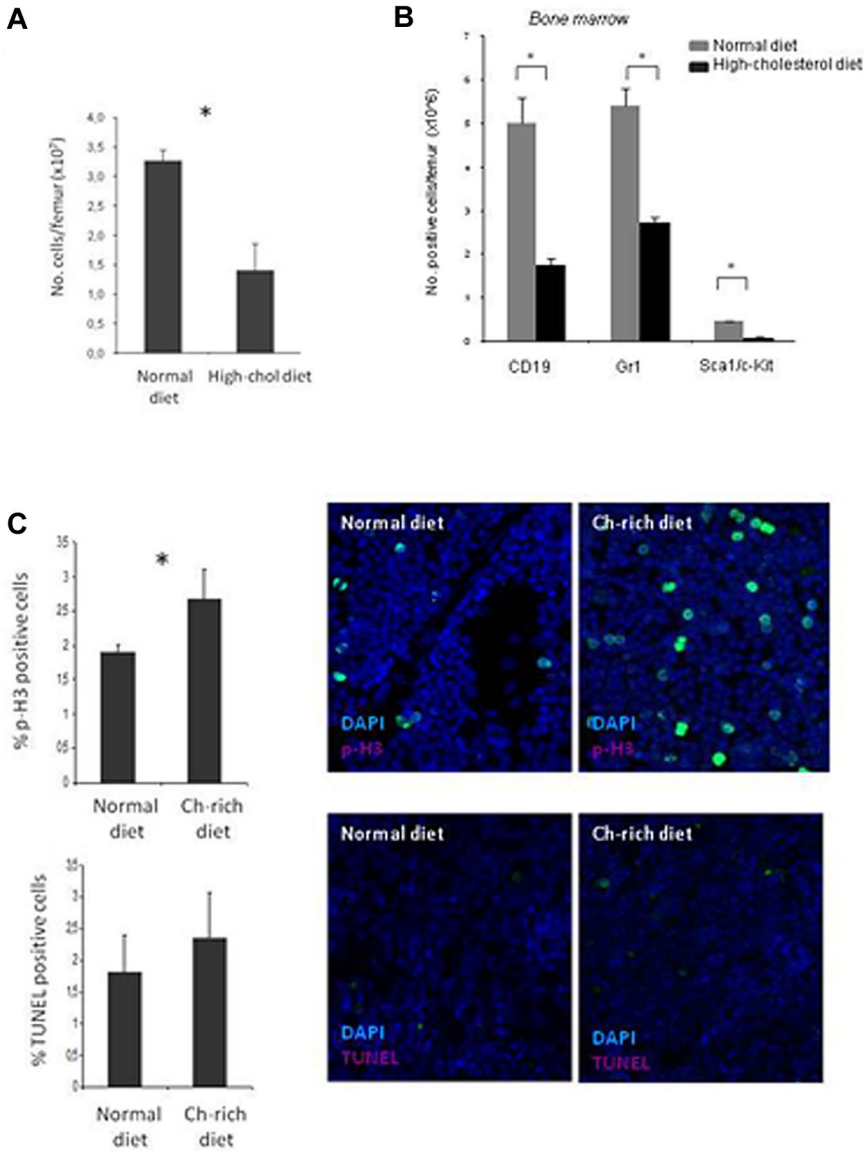

Figure 2. A high-cholesterol diet is associated with decreased total BM-cell counts. (A) Hypercholesterolemic mice present reduced cell numbers per femur ($\times 10^7$). (B) Flow cytometric analysis with Gr-1 (neutrophils), Sca1/c-Kit (progenitors), and CD19 (B lymphocytes) cell-surface markers shows reduced numbers per femur ($\times 10^6$) of all cell lineages tested. (C) Hypercholesterolemia induces cell proliferation (p-H3 immunostaining, top) without altering cell apoptosis (TUNEL assay, bottom; $^*P < .05$). These experiments were performed 3 times with groups of 6 mice/experimental condition with consistent results. Ch indicates cholesterol.

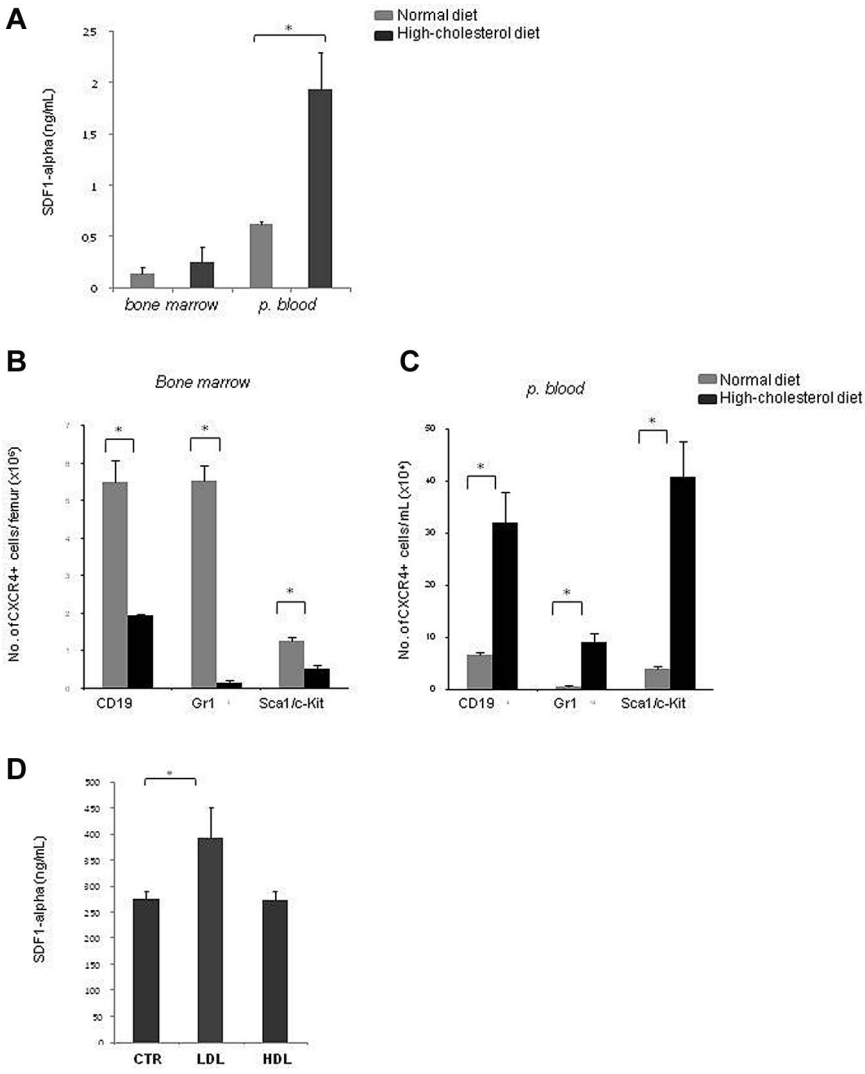

Figure 6. A high-cholesterol diet induces increased SDF-1 plasma levels, favors CXCR4$^+$ cell mobilization to PB, and favors HC migration toward SDF1. (A) Hypercholesterolemia is accompanied by an increase in PB plasma SDF-1 levels, as determined by ELISA quantification. (B) Flow cytometry analysis using Sca1/c-Kit (progenitor), CD19 (lymphocyte), and Gr-1 (neutrophils) cell-surface markers together with CXCR4 shows reduced numbers of double-positive cells per femur ($\times 10^6$) for all cell lineages tested. (C) Flow cytometry analysis with Lin$^-$Sca1$^+$c-Kit$^+$ (progenitor), CD19$^+$ (lymphocyte), and Gr-1$^+$ (neutrophil) cell-surface markers together with CXCR4 shows increased numbers of double-positive B lymphocytes, neutrophils, and progenitor cells ($\times$ 10^4) in the PB of high-cholesterol mice. (D) LDL exposure (100 μg/mL) increased SDF-1 production by HUVEC in vitro, as determined by ELISA (*$P < .05$). These data were obtained from 3 separate experiments in which we used 6 mice per experimental condition with consistent results. (E) LDL (100 μg/mL) induces and HDL (100 μg/mL) reduces progenitor cells (Lin$^-$Sca1$^+$ c-Kit$^+$) migration toward SDF-1. (F) LDL (100 μg/mL) induced B-lymphocyte (CD19$^+$) migration toward SDF-1 is reversed when SR-BI is inhibited. LDL effect is reverted when SR-BI is inhibited (*$P < .05$). The data are shown as the number of migrating cells in relation to the control condition (SDF-1 alone). These data were obtained from 2 separate experiments with consistent results. CTR, control.