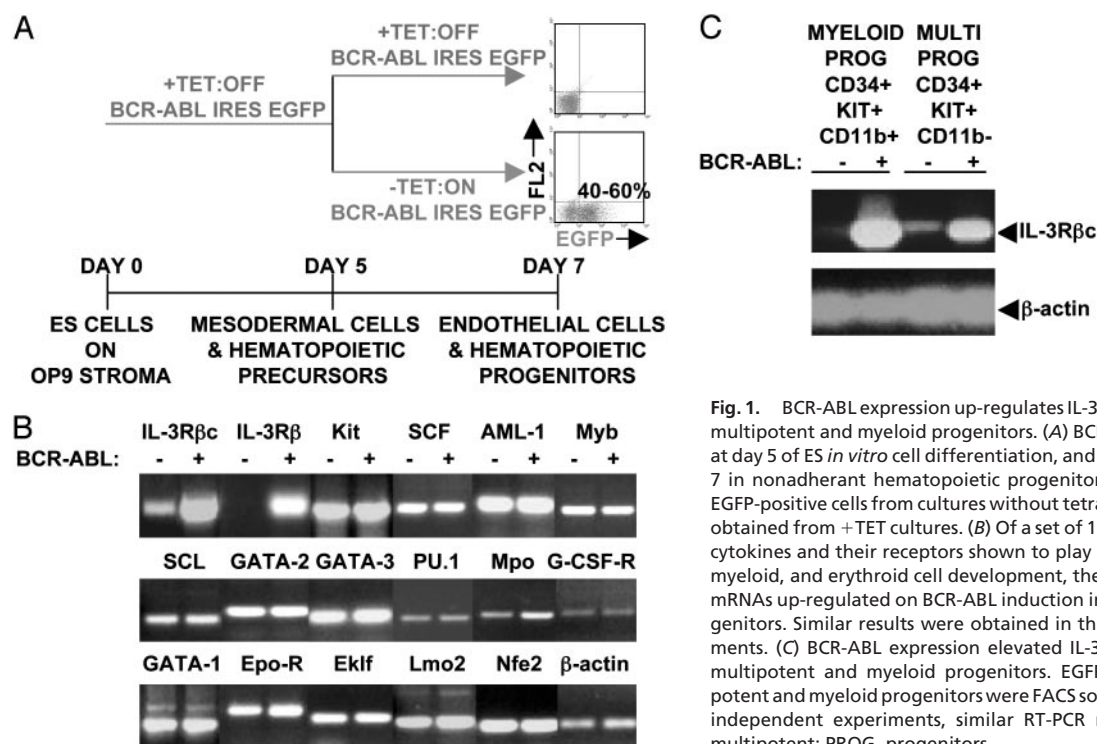


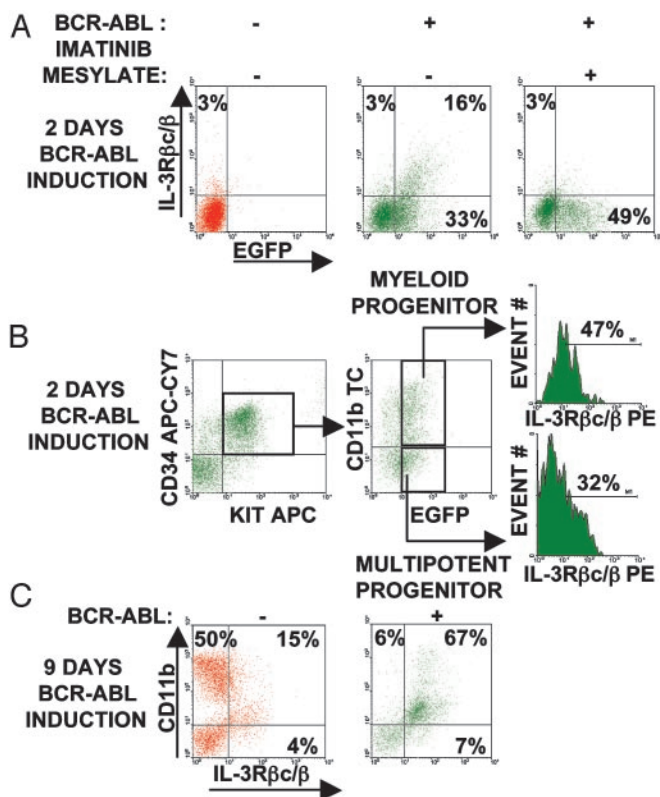
## Corrections

**MEDICAL SCIENCES.** For the article “IL-3 receptor signaling is dispensable for BCR-ABL-induced myeloproliferative disease,” by Stephane Wong, Jami McLaughlin, Donghui Cheng, Kevin Shannon, Lorraine Robb, and Owen N. Witte, which appeared in issue 20, September 30, 2003, of *Proc. Natl. Acad. Sci. USA* (100, 11630–11635; first published September 19,

2003; 10.1073/pnas.2035020100), Figs. 1*B* and 2*A* were printed incorrectly due to a printer’s error. Also, Fig. 2*B* middle plot, upper box, should have been labeled “myeloid progenitor,” and the middle plot, lower box, should have been labeled “multipotent progenitor.” The corrected figures and their legends appear below.



**Fig. 1.** BCR-ABL expression up-regulates IL-3R $\beta$ c/ $\beta$  mRNA levels in ES-derived multipotent and myeloid progenitors. (A) BCR-ABL expression was turned on at day 5 of ES *in vitro* cell differentiation, and its effects were analyzed on day 7 in nonadherent hematopoietic progenitors by gating and/or sorting for EGFP-positive cells from cultures without tetracycline (TET). Control cells were obtained from +TET cultures. (B) Of a set of 17 different transcription factors, cytokines and their receptors shown to play critical roles in HSC/progenitor, myeloid, and erythroid cell development, the IL-3R $\beta$ c/ $\beta$  chains were the only mRNAs up-regulated on BCR-ABL induction in ES-derived hematopoietic progenitors. Similar results were obtained in three sets of independent experiments. (C) BCR-ABL expression elevated IL-3R $\beta$ c mRNA levels in ES-derived multipotent and myeloid progenitors. EGFP-positive and -negative multipotent and myeloid progenitors were FACS sorted to >95% purity, and, in two independent experiments, similar RT-PCR results were obtained. MULTI, multipotent; PROG, progenitors.



**Fig. 2.** BCR-ABL induces cell surface expression of IL-3Rβc/β in ES-derived hematopoietic cells. (A) BCR-ABL tyrosine kinase activity is required to up-regulate IL-3Rβc/β chain expression in ES-derived hematopoietic progenitors. Imatinib mesylate was added to BCR-ABL cultures at final concentrations of 1 and 10 μM with similar results in triplicate wells. (B) BCR-ABL up-regulates IL-3Rβc/β chain expression in both multipotent and myeloid ES-derived progenitors as defined by cell surface marker analysis. (C) Nine-day induction of BCR-ABL leads to 60–70% of ES-derived hematopoietic cells expressing IL-3Rβc/β chains coincident with low levels of CD11b expression. PE, phycoerythrin.

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**NEUROSCIENCE.** For the article “Conformation-dependent hydrophobic photolabeling of the nicotinic receptor: Electrophysiology-coordinated photochemistry and mass spectrometry,” by John F. Leite, Michael P. Blanton, Mona Shahgholi, Dennis A. Dougherty, and Henry A. Lester, which appeared in issue 22, October 28, 2003, of *Proc. Natl. Acad. Sci. USA* (**100**, 13054–13059; first published October 20, 2003; 10.1073/pnas.2133028100), the authors note that the following funding acknowledgement was omitted from the article: “This work was supported by grants from the National Institutes of Health (NS11756, NS34407, NS35786, and NRSA) to J.F.L.”

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