

Corrections

GENETICS. For the article “*miR-15* and *miR-16* induce apoptosis by targeting BCL2,” by Amelia Cimmino, George Adrian Calin, Muller Fabbri, Marilena V. Iorio, Manuela Ferracin, Masayoshi Shimizu, Sylwia E. Wojcik, Rami I. Aqeilan, Simona Zupo, Mariella Dono, Laura Rassenti, Hansjuerg Alder, Stefano Volinia, Chang-gong Liu, Thomas J. Kipps, Massimo Negrini, and Carlo M. Croce, which appeared in issue 39, September 27, 2005,

of *Proc. Natl. Acad. Sci. USA* (**102**, 13944–13949; first published September 15, 2005; 10.1073/pnas.0506654102), the authors note that Fig. 1C incorrectly shows the direct correlation between the Bcl2 levels and levels of *miR-15a* and *miR-16-1* instead of the indirect correlation, as presented in the article. The corrected figure and legend appear below. This error does not affect the conclusions of the article.

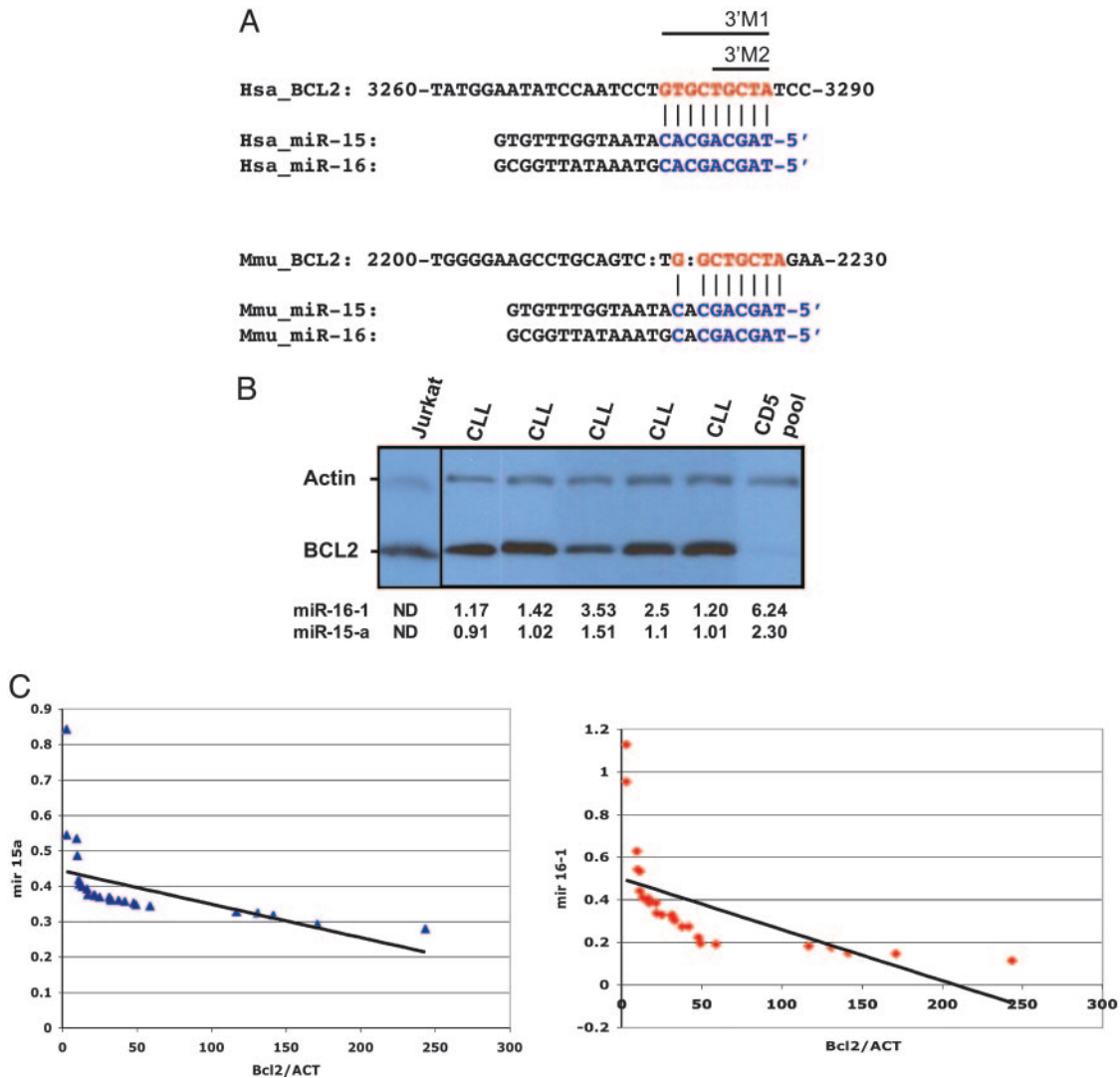


Fig. 1. Bcl2 protein expression is inversely correlated with *miR-15a* and *miR-16-1* miRNAs expression in CLL patients. (A) The unique site of complementarity miR::mRNA is conserved in human and mouse and is the same for all four human members of the family. The sites of target mutagenesis are indicated (3'M1 and 3'M2). (B) In CLL patients the levels of Bcl2 protein are inversely correlated with *miR-15a* and *miR-16-1* expression. Five different CLL cases are presented, and the normal cells were pools of CD5⁺ B lymphocytes. The T cell leukemia Jurkat was used as control for Bcl2 protein expression. For normalization we used β -actin. The numbers represent normalized expression on miRNACHIP. ND, not determined. (C) The inverse correlation in the full set of 26 samples of CLL between *miR-15a*/*miR-16-1* and Bcl2 protein expressions. The normalized Bcl2 expression is on abscissa vs. *miR-15a* (Left) and *miR-16-1* (Right) levels by miRNA chip on ordinates. ACT, β -actin.

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BIOCHEMISTRY. For the article “The crystal structure of CREG, a secreted glycoprotein involved in cellular growth and differentiation,” by Michael Sacher, Alessandra Di Bacco, Vladimir V. Lunin, Zheng Ye, John Wagner, Grace Gill, and Miroslaw Cygler, which appeared in issue 51, December 20, 2005, of *Proc. Natl. Acad. Sci. USA* (**102**, 18326–18331; first published December 12, 2005; 10.1073/pnas.0505071102), the last sentence of the Abstract was inadvertently truncated, due to a printer’s error. “These findings indicate that CREG utilizes a known fold” should have read: “These findings indicate that CREG utilizes a known fold for a previously undescribed function.”

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MICROBIOLOGY. For the article “EST-based genome-wide gene inactivation identifies ARAP3 as a host protein affecting cellular susceptibility to anthrax toxin,” by Quan Lu, Wensheng Wei, Paul E. Kowalski, Annie C. Y. Chang, and Stanley N. Cohen, which appeared in issue 49, December 7, 2004, of *Proc. Natl. Acad. Sci. USA* (**101**, 17246–17251; first published November 29, 2004; 10.1073/pnas.0407794101), the authors note that on page 17247, the last sentence of the second paragraph, left column, the sequence of the Lenti3 primer was incorrectly written as the complement of the primer that actually was used in the study. The correct sequence for the Lenti3 primer is 5'-CATAGCG-TAAAAGGAGCAACA. This error does not affect the conclusions of the article.

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NEUROSCIENCE. For the article “Antidepressant-like activity and modulation of brain monoaminergic transmission by blockade of anandamide hydrolysis,” by G. Gobbi, F. R. Bambico, R. Mangieri, M. Bortolato, P. Campolongo, M. Solinas, T. Cassano, M. G. Morgese, G. Debonnel, A. Duranti, A. Tontini, G. Tarzia, M. Mor, V. Trezza, S. R. Goldberg, V. Cuomo, and D. Piomelli, which appeared in issue 51, December 20, 2005, of *Proc. Natl. Acad. Sci. USA* (**102**, 18620–18625; first published December 13, 2005; 10.1073/pnas.0509591102), the authors note that a patent on the subject of this publication has been filed by the University of California, Irvine (inventors: D.P., A.D., A.T., G.T., and M.M.). D.P. is a cofounder of and consultant for Kadmus Pharmaceuticals, Inc.

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