Corrections and Retraction

CORRECTIONS

BIOPHYSICS. For the article "Microviscometry reveals reduced blood viscosity and altered shear rate and shear stress profiles in microvessels after hemodilution," by David S. Long, Michael L. Smith, Axel R. Pries, Klaus Ley, and Edward R. Damiano, which appeared in issue 27, July 6, 2004, of *Proc. Natl. Acad. Sci. USA* (**101,** 10060–10065; first published June 25, 2004; 10.1073/pnas.0402937101), the first two authors, David S. Long and Michael L. Smith, contributed equally to this work.

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INAUGURAL ARTICLE, DEVELOPMENTAL BIOLOGY. For the article "Nuclear cloning of embryonal carcinoma cells," by Robert H. Blelloch, Konrad Hochedlinger, Yasuhiro Yamada, Cameron Brennan, Minjung Kim, Beatrice Mintz, Lynda Chin, and Rudolf Jaenisch, which appeared in issue 39, September 28, 2004, of *Proc. Natl. Acad. Sci. USA* (**101,** 13985–13990; first published August 11, 2004; 10.1073/pnas.0405015101), all authors agree to this correction. The mouse embryonal carcinoma cell line referred to in the publication as METT-1 was in fact a derivative designated METT-1a, and it should have been referred to as such throughout the paper. The METT-1 cell line was isolated from an induced teratocarcinoma and found to be karyotypically normal (29). When injected into blastocysts, the cells contributed to all somatic tissues and to the germ line (25). The cells in the present study were grown from a thawed aliquot of the METT-1 line, previously tested *in vivo* and frozen at culture passage 12 (25). In the experiments we performed, the cells were grown in a medium different from the one to which they had been adapted, and they were passaged repeatedly to obtain the data reported in the present study. Genetic and developmental differences, relative to METT-1, may have arisen during these passages. This correction does not change the conclusions in the present study.

25. Stewart, T. A. & Mintz, B. (1981) *Proc. Natl. Acad. Sci. USA* **78,** 6314–6318.
29. Mintz, B. & Cronmiller, C. (1981) *Somatic Cell Genet.* **7,** 489–505.

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RETRACTION

BIOCHEMISTRY. For the article "Molecular dissection of the roles of nucleotide binding and hydrolysis in dynein's AAA domains in *Saccharomyces cerevisiae*," by Samara L. Reck-Peterson and Ronald D. Vale, which appeared in issue 6, February 10, 2004, of *Proc. Natl. Acad. Sci. USA* (**101,** 1491–1495; first published January 30, 2004; 10.1073/pnas.2637011100), the undersigned authors wish to note the following: "In conjunction with a follow-up project regarding these mutations, we resequenced several of the mutant yeast strains and found that the genes in two of the eight analyzed mutant strains (the E2488Q mutation in the AAA3 domain and the K2766A mutation in the AAA4 domain) no longer contained the mutation and instead were wild type. The correct K2766A mutation does not have a defect in either nuclear segregation or microtubule dissociation as originally reported; however, there is a modest defect (≈50% decrease) in microtubule binding. More significantly, the true E2488Q mutation demonstrated a severe nuclear segregation phenotype and a defect in the release of dynein from microtubules with ATP, comparable to that reported for the nuclear hydrolysis mutation in AAA1. Thus, our initial conclusion that the AAA site 3 ATP hydrolysis mutation has no phenotype is incorrect. We now know that ATP hydrolysis in both AAA1 and AAA3 is essential for dynein function and that nucleotide binding at AAA2 and AAA4 is necessary for maximal levels of microtubule binding *in vitro*."

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