Corrections

GENETICS. For the article "A common variant in combination with a nonsense mutation in a member of the thioredoxin family causes primary ciliary dyskinesia," by Bénédicte Duriez, Philippe Duquesnoy, Estelle Escudier, Anne-Marie Bridoux, Denise Escalier, Isabelle Rayet, Elisabeth Marcos, Anne-Marie Vojtek, Jean-François Bercher, and Serge Amselem, which appeared in issue 9, February 27, 2007, of *Proc Natl Acad Sci USA* (104:3336–3341; first published February 20, 2007; 10.1073/pnas.0611405104), Fig. 1 appeared incorrectly, due to a printer's error. The online version has been corrected. The corrected figure and its legend appear below.

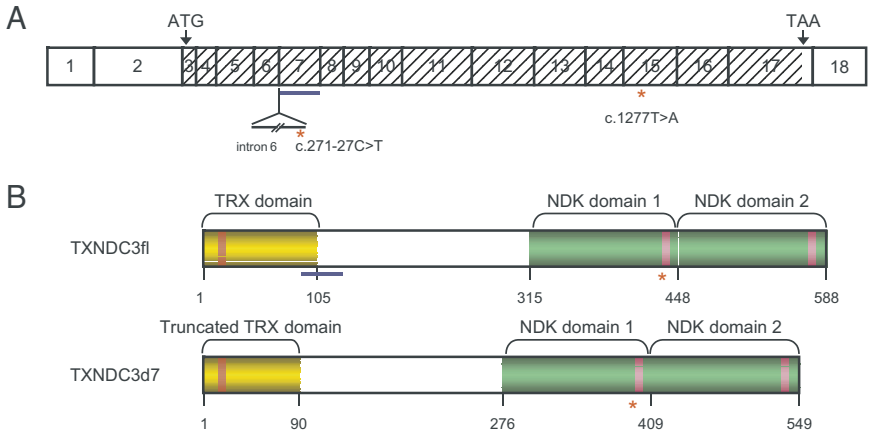

Fig. 1. The human *TXNDC3* gene and related products. (*A*) *TXNDC3* cDNA structure showing the location of the exons drawn to scale. The translation start and stop codons are labeled with ATG and TAA, respectively. The translated region is hashed. Exon 7 is underlined in blue, and intron 6 is shown as a thin line below exons 6 and 7. The red asterisks mark the locations of the c.271–27C>T and c.1277T>A nucleotide variations, located in intron 6 and exon 15, respectively. (*B*) Structure of the TXNDC3 isoforms: the TXNDC3fl isoform (*Upper*) and the TXNDC3d7 isoform (*Lower*). The thioredoxin (TRX) domain and the two NDK domains are shown in yellow and green, respectively. Within the TRX domain, the active site (GCPC) is shown by an orange box, and, within the NDK domains, the putative NDP kinase active sites are shown by pink boxes. The location of the region encoded by exon 7 is underlined in blue. The location of the p.Leu426X mutation is shown by a red asterisk.

www.pnas.org/cgi/doi/10.1073/pnas.0702345104

APPLIED BIOLOGICAL SCIENCES. For the article "An *in vitro* and *in vivo* disconnect uncovered through high-throughput identification of botulinum neurotoxin A antagonists," by Lisa M. Eubanks, Mark S. Hixon, Wei Jin, Sukwon Hong, Colin M. Clancy, William H. Tepp, Michael R. Baldwin, Carl J. Malizio, Michael C. Goodnough, Joseph T. Barbieri, Eric A. Johnson, Dale L. Boger, Tobin J. Dickerson, and Kim D. Janda, which appeared in issue 8, February 20, 2007, of *Proc Natl Acad Sci USA* (104:2602–2607; first published February 9, 2007; 10.1073/pnas.0611213104), the authors note that, due to a printer's error, the following statement was omitted from the Acknowledgments: "We also thank Laura A. McAllister, Jack P. Kennedy, and Grant E. Boldt (all of The Scripps Research Institute, La Jolla, CA) for providing the 2,4-dichlorocinnamic hydroxamic acid."

www.pnas.org/cgi/doi/10.1073/pnas.0702406104

MEDICAL SCIENCES. For the article "c-Myc-mediated genomic instability proceeds via a megakaryocytic endomitosis pathway involving Gp1bα," by Youjun Li, Jie Lu, and Edward V. Prochownik, which appeared in issue 9, February 27, 2007, of *Proc Natl Acad Sci USA* (104:3490–3495; first published February 20, 2007; 10.1073/pnas.0610163104), the authors note that, due to a printer's error, the curves in several panels of Fig. 4 were shifted to the left. The corrected figure and its legend appear below. This error does not affect the conclusions of the article.

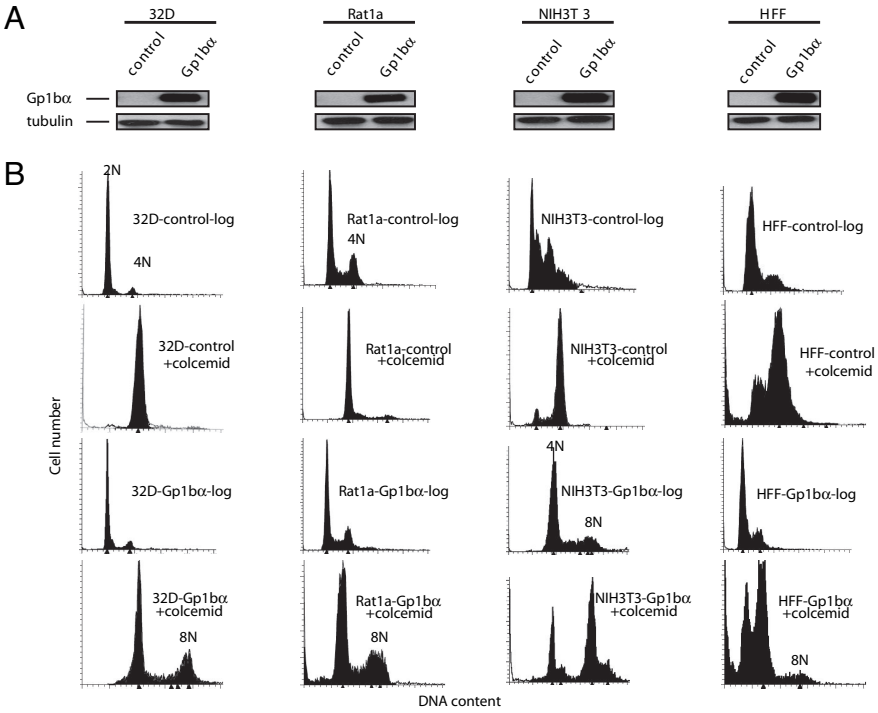

Fig. 4. Enforced expression of Gp1bα is sufficient for the induction of tetraploidy in various cell types. Stable expression of Gp1bα in the indicated immortalized or primary cells was accomplished through transduction with a bicistronic LXSN retroviral vector expressing myc-epitope-tagged Gp1bα and enhanced yellow fluorescent protein (EYFP, ref. 23). Control cell lines were derived after transduction with the empty parental vector. In both cases, pure populations of EYFP-positive cells were obtained by fluorescence-activated cell sorting (23). (*A*) Immunoblotting for Gp1bα and tubulin expression after expansion of EYFP-positive cells. (*B*) Cell cycle analyses. The indicated cells were examined during log-phase growth or after exposure to colcemid. Similar results were seen after stable transfection of several cell lines with a pcDNA-based, nonepitope-tagged Gp1bα expression vector, thus indicating that the method of delivery, the nature of the vector, and the presence of an epitope tag were not important for conferring the tetraploid genotype (data not shown and Fig. 3 *B–D*).

www.pnas.org/cgi/doi/10.1073/pnas.0702407104