Corrections

BIOCHEMISTRY. For the article "Interaction of RNA polymerase with forked DNA: Evidence for two kinetically significant intermediates on the pathway to the final complex," by Laura Tsujikawa, Oleg V. Tsodikov, and Pieter L. deHaseth, which appeared in number 6, March 19, 2002, of Proc. Natl. Acad. Sci. USA (99, 3493-3498; First Published March 12, 2002; 10.1073/ pnas.062487299), the authors note the following concerning RNA polymerase (RNAP) concentrations. No correction was made for the fraction of RNAP (0.5) that is active in promoter binding. With this correction, the values of K_1 and K_{app} (but not $K_{\rm f}$) would increase by about a factor of 2. The relative values would remain essentially unchanged. Also, the legends to Figs. 2, 3, and 5 contain errors pertaining to the symbols used for data obtained with and without heparin challenge, the duration of the challenge, and the concentration of added heparin. The figures and the corrected legends appear below.



Fig. 2. Determination of equilibrium affinities by titration of wt Fork with RNAP. The reactions contained 1 nM wt Fork and variable amounts of RNAP as shown and were analyzed by electrophoretic mobility shift immediately (Δ ; data shown are averages of three independent experiments) or after a challenge with 100 μ g/ml heparin for 10 min (\bullet ; data shown are averages of four independent experiments). The curves shown reflect the simultaneous errorweighted fits of the data to Eqs. **3** and **4–7**. The parameters are shown in Table 1 (line 1).



Fig. 3. Kinetics of complex formation. RNAP (65 nM) and wt forked DNA (1 nM) were incubated for various time intervals and then complex formation was determined immediately (-heparin) or after a 2-min challenge with 100 μ g/ml heparin (+heparin). The -heparin data (**■**) were fit (error-weighted) with Eq. **8** with $a_2 = 0$ ($k_{a-} = 0.10 \pm 0.01 \text{ s}^{-1}$) and the +heparin data (**▲**) with both single ($k_{a+} = 0.036 \pm 0.004 \text{ s}^{-1}$; thin line) and double-exponential ($k_{a_1} = 0.044 \pm 0.002 \text{ s}^{-1}$; $k_{a_2} = (5 \pm 3) \times 10^{-4} \text{ s}^{-1}$; thick line) equations.



Fig. 5. Comparison of the kinetics for formation and dissociation of competitor-resistant complexes between RNAP and wt Fork. Association data were obtained as described in the text and the legend for Fig. 3 except the concentration of forked DNA was 10 nM. Dissociation kinetics were obtained by challenging with 100 μ g/ml heparin a mixture of RNAP and forked DNA that had been incubated for 30 min. The curves represent double-exponential fits of the data to Eq. **10**. (A) wt RNAP. The observed association rate constants (**II**) are shown in the legend for Fig. 3; for the slow phase of the dissociation of the wt Fork–wt RNAP complex (**O**), $k_{d_2} = (1.3 \pm 0.2) \times 10^{-4} s^{-1}$. (B) YYW RNAP. The slow phase of the dissociation reaction (**O**) has a $k_{a_2} = (1.1 \pm 0.3) \times 10^{-3} s^{-1}$; the slow phase of the dissociation reaction (**II**), a $k_{d_2} = (6 \pm 1) \times 10^{-4} s^{-1}$.

CELL BIOLOGY. For the article "Nrdp1/FLRF is a ubiquitin ligase promoting ubiquitination and degradation of the epidermal growth factor receptor family member, ErbB3," by Xiao-Bo Qiu and Alfred L. Goldberg, which appeared in number 23, November 12, 2002, of *Proc. Natl. Acad. Sci. USA* (**99**, 14843–14848; First Published October 31, 2002; 10.1073/pnas.232580999), Fig. 4 should have appeared in color. In addition, the marker for the Ub band of Fig. 6*A* should be 14. The corrected figures and their legends appear below.



Fig. 4. Nrdp1 reduces levels of endogenous ErbB3. MDA-MB-468 cells were transfected with FLAG-tagged Nrdp1 or Nrdp1C. Localization of Nrdp1, Nrdp1C, ErbB3, or EGFR was visualized by confocal fluorescence microscopy after incubation with appropriate primary antibodies and FITC or Cy3 conjugates of secondary antibodies. Different images on each row, which are representative of many (>30) microscopic fields, are the same cells with different kinds of staining. The cells transfected with Nrdp1 or Nrdp1C are indicated by arrowheads. The nuclei of cells were visualized under UV light after staining with 4',6-diamidino-2-phenylindole.



Fig. 6. Nrdp1 is a ubiquitin ligase promoting ubiquitination of itself and ErbB3. (A) Nrdp1-catalyzed formation of [125] ubiquitin conjugates was assayed in vitro. All of the components were expressed and purified from bacteria. (B) Autoubiquitination of Nrdp1 requires its RING finger domain. Wild-type, mutant (C34S/H36Q), and truncated Nrdp1 were immunopurified from the 293T cells transfected with their FLAG-tagged form and were used for in vitro ubiquitination assay. (Left) Nrdp1 was labeled in vivo with ³⁵S. (Right) Ubiquitin was labeled with ¹²⁵I. (C) Nrdp1 stimulates ErbB3 ubiquitination in vivo. The 293T cells were transfected with ErbB3, or cotransfected with ErbB3 and the wild-type or mutant (C34S/H36Q) Nrdp1-FLAG in the presence (Left) or the absence (Right) of pCMV-myc-Ub (kindly provided by Ron Kopito, Stanford University, Stanford, CA). ErbB3 ubiquitination was detected by Western blotting by using either anti-myc antibodies (Oncogene Research Products) or anti-Ub antibodies (Zymed) after immunoprecipitation by anti-ErbB3 antibodies. Arrows indicate 191-kDa markers. (D) Nrdp1 ubiquitinates endogenous ErbB3 in vitro. ErbB3 immunoprecipitated from the MDA-MB-453 cells was incubated with the lysates of 293T cells untransfected or transfected with wild-type, mutant (C34S/H36Q), or truncated Nrdp1-FLAG in the presence of [125] ubiquitin (K48R mutant). The 125I-labeled ErbB3ubiquitin conjugates on beads were separated on SDS/PAGE and analyzed by PhosphorImager after ubiquitination assays and precipitation from the reaction mix. Arrow indicates the 191-kDa marker.

GENETICS. For the article "Genetic and physiological data implicating the new human gene G72 and the gene for D-amino acid oxidase in schizophrenia," by Ilya Chumakov, Marta Blumenfeld, Oxana Guerassimenko, Laurent Cavarec, Marta Palicio, Hadi Abderrahim, Lydie Bougueleret, Caroline Barry, Hiroaki Tanaka, Philippe La Rosa, Anne Puech, Nadia Tahri, Annick Cohen-Akenine, Sylvain Delabrosse, Sébastien Lissarrague, Françoise-Pascaline Picard, Karelle Maurice, Laurent Essioux, Philippe Millasseau, Pascale Grel, Virginie Debailleul, Anne-Marie Simon, Dominique Caterina, Isabelle Dufaure, Kattayoun Malekzadeh, Maria Belova, Jian-Jian Luan, Michel Bouillot, Jean-Luc Sambucy, Gwenael Primas, Martial Saumier, Nadia Boubkiri, Sandrine Martin-Saumier, Myriam Nasroune, Hélène Peixoto, Arnaud Delaye, Virginie Pinchot, Mariam Bastucci,

Sophie Guillou, Magali Chevillon, Ricardo Sainz-Fuertes, Said Meguenni, Joan Aurich-Costa, Dorra Cherif, Anne Gimalac, Cornelia Van Duijn, Denis Gauvreau, Gail Ouelette, Isabel Fortier, John Realson, Tatiana Sherbatich, Nadejda Riazanskaia, Evgeny Rogaev, Peter Raeymaekers, Jeroen Aerssens, Frank Konings, Walter Luyten, Fabio Macciardi, Pak C. Sham, Richard E. Straub, Daniel R. Weinberger, Nadine Cohen, and Daniel Cohen, which appeared in number 21, October 15, 2002, of *Proc. Natl. Acad. Sci. USA* (**99**, 13675–13680; First Published October 3, 2002; 10.1073/pnas.182412499), the author name Gail Ouelette should have appeared as Gail Ouellette and the author name John Realson should have appeared as John Raelson. The corrected author line appears below. The online version has been corrected.

Ilya Chumakov, Marta Blumenfeld, Oxana Guerassimenko, Laurent Cavarec, Marta Palicio, Hadi Abderrahim, Lydie Bougueleret, Caroline Barry, Hiroaki Tanaka, Philippe La Rosa, Anne Puech, Nadia Tahri, Annick Cohen-Akenine, Sylvain Delabrosse, Sébastien Lissarrague, Françoise-Pascaline Picard, Karelle Maurice, Laurent Essioux, Philippe Millasseau, Pascale Grel, Virginie Debailleul, Anne-Marie Simon, Dominique Caterina, Isabelle Dufaure, Kattayoun Malekzadeh, Maria Belova, Jian-Jian Luan, Michel Bouillot, Jean-Luc Sambucy, Gwenael Primas, Martial Saumier, Nadia Boubkiri, Sandrine Martin-Saumier, Myriam Nasroune, Hélène Peixoto, Arnaud Delaye, Virginie Pinchot, Mariam Bastucci, Sophie Guillou, Magali Chevillon, Ricardo Sainz-Fuertes, Said Meguenni, Joan Aurich-Costa, Dorra Cherif, Anne Gimalac, Cornelia Van Duijn, Denis Gauvreau, Gail Ouellette, Isabel Fortier, John Raelson, Tatiana Sherbatich, Nadejda Riazanskaia, Evgeny Rogaev, Peter Raeymaekers, Jeroen Aerssens, Frank Konings, Walter Luyten, Fabio Macciardi, Pak C. Sham, Richard E. Straub, Daniel R. Weinberger, Nadine Cohen, and Daniel Cohen

IMMUNOLOGY. For the article "Negative autoregulation of BCL-6 is bypassed by genetic alterations in diffuse large B cell lymphomas," by Xing Wang, Zhiping Li, Akira Naganuma, and B. Hilda Ye, which appeared in number 23, November 12, 2002, of *Proc. Natl. Acad. Sci. USA* (**99**, 15018–15023; First Published October 29, 2002; 10.1073/pnas.232581199), the left axis label of Fig. 6 appeared incorrectly due to a printer's error. The corrected figure and its legend appear below.

COMMENTARY. For the article "The inner inner core of Earth," by Don L. Anderson, which appeared in number 22, October 29, 2002, of *Proc. Natl. Acad. Sci. USA* (**99**, 13966–13968; First Published October 21, 2002; 10.1073/pnas.232565899), the legend to Fig. 1 should have included the following statement. "Figure courtesy of M. Ishii."

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Fig. 6. BCL-6 preferentially binds to the wild-type exon 1 in Ly1 cells. Both Ly1 and the control Ly7 cells were analyzed for association of BCL-6 and acetylated H3 with the exon 1 sequence. Location of the PCR primers is given with respect to exon 1 and the surrounding mutations. Various amounts of recovered genomic DNA as well as a fraction of the total chromatin input were used in PCR reactions with the BCL-6-1 and BCL-6 i2 primer pairs. IgG, normal rabbit IgG used as control antibody; N3, polyclonal anti-BCL-6 antibody; N.C., negative control PCR reaction with no template.