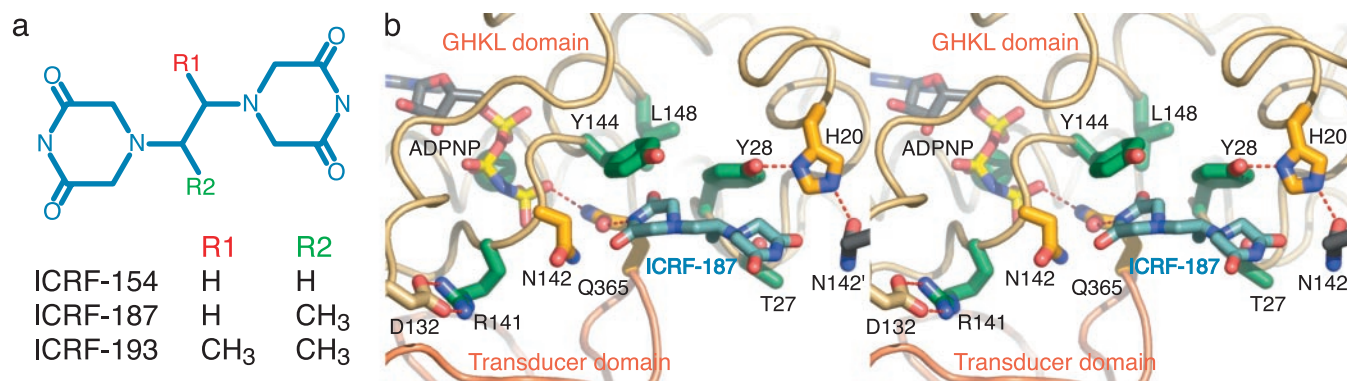


## Corrections

**BIOCHEMISTRY, MEDICAL SCIENCES.** For the article “Structure of the topoisomerase II ATPase region and its mechanism of inhibition by the chemotherapeutic agent ICRF-187,” by Scott Classen, Stephane Olland, and James M. Berger, which appeared in issue 19, September 16, 2003, of *Proc. Natl. Acad. Sci. USA* (**100**, 10629–10634; first published September 8, 2003; 10.1073/pnas.1832879100), the authors note that the recently published structure of the *Saccharomyces cerevisiae* topoisomerase II ATPase region bound to adenosine 5'-[ $\beta,\gamma$ -imino]triphosphate (ADPNP) and ICRF-187 contains an error. The drugs ICRF-187 and ICRF-186 are the respective (*S*)- and (*R*)-specific enantiomers of the racemic compound ICRF-159 (1, 2). During model building of our drug-inhibited complex, we relied on coordinates downloaded from the National Cancer Institute database for ICRF-187. We now have discovered that this file actually contains the ICRF-186 stereoisomer, and that the structure of the drug bound to topoisomerase II was correspondingly modeled incorrectly. Because the electron density for the ethanediyl linker region of the drug that contains the chiral center is poorly defined, the mistake was not immediately evident.

We have now rebuilt and refined the correct (*S*)-enantiomeric ICRF-187 compound into the ATPase region (see corrected Fig. 5 and legend below). Compared with the original model containing (*R*)-ICRF-186, the new model with (*S*)-ICRF-187 alters the angle at which the single methyl group extends from the chiral center of the linker. Despite this change, the well defined electron density of the two dioxopiperazine rings constrains the ethanediyl linker of ICRF-187 to adopt a slightly different twist than observed previously, shifting the coordinate position of the methyl substituent by only  $\approx 0.5$  Å. As a consequence, the methyl- $\pi$  interaction postulated to exist between the ICRF-187 ethanediyl linker and Tyr28 is maintained. Other interactions observed between the drug and topoisomerase II are similarly unaffected, and all discussions and conclusions of the paper still stand. Indeed, both ICRF-186 and ICRF-187 inhibit topoisomerase II with virtually the same  $K_i$  values (2–4), an observation that can be explained by the absence of stereospecific interactions between these drugs and the enzyme. The new coordinates have been deposited in the Protein Data Bank, www.rcsb.org (PDB ID code 1QZR).



**Fig. 5.** Details of bisdioxopiperazine compounds and the ICRF-187-binding site. (a) Schematic of different bisdioxopiperazine compounds. (b) Stereo figure of drug-binding site (compare with Fig. 5 of the original paper). One protomer has been removed for clarity. ADPNP, ICRF-187, and residues within 5 Å of the drug are shown in stick representation and colored as in the original paper.

1. Hempel, A., Camerman, N. & Camerman, A. (1982) *J. Am. Chem. Soc.* **104**, 3453–3456.
2. Hasinoff, B. B., Kuschak, T. I., Yalowich, J. C. & Creighton, A. M. (1995) *Biochem. Pharmacol.* **50**, 953–958.
3. Hasinoff, B. B., Kuschak, T. I., Creighton, A. M., Fattman, C. L., Allan, W. P., Thampatty, P. & Yalowich, J. C. (1997) *Biochem. Pharmacol.* **53**, 1843–1853.
4. Snapka, R. M., Woo, S. H., Blokhin, A. V. & Witak, D. T. (1996) *Biochem. Pharmacol.* **52**, 543–549.

www.pnas.org/cgi/doi/10.1073/pnas.2436153100

**EVOLUTION.** For the article “Active self-splicing group I introns in 23S rRNA genes of hyperthermophilic bacteria, derived from introns in eukaryotic organelles,” by Camilla L. Nesbø and W. Ford Doolittle, which appeared in issue 19, September 16, 2003, of *Proc. Natl. Acad. Sci. USA* (**100**, 10806–10811; first published August 28, 2003; 10.1073/pnas.1434268100), the authors note that, due to an earlier misalignment, the position of the intron should be L1917, not L1926, and the intron should be annotated Tsu.bL1917 in Fig. 1*b*. The annotation of the sequence has been corrected in the GenBank database (accession no. AJ556793). Tsu.bL1917 is very similar in structure to two other introns in position L1917 (*Chlorosarcina brevispinosa*, GenBank accession no. L49150; and *Chaetosphaeridium globosum*, GenBank accession no. AF494279). The ORF in Tsu.bL1917 (i-Tsu1917*b*) clusters with the ORFs in these introns, as shown in Fig. 5, further supporting a recent transfer from a eukaryote.

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**EVOLUTION.** For the article “Synteny perturbations between wheat homoeologous chromosomes caused by locus duplications and deletions correlate with recombination rates,” by Eduard D. Akhunov, Alina R. Akhunova, Anna M. Linkiewicz, Jorge Dubcovsky, David Hummel, Gerry Lazo, Shiaoman Chao, Olin D. Anderson, Jacques David, Lili Qi, Benjamin Echaliier, Bikram S. Gill, Miftahudin, J. Perry Gustafson, Mauricio La Rota, Mark E. Sorrells, Deshui Zhang, Henry T. Nguyen, Venugopal Kalavacharla, Khwaja Hossain, Shahryar F. Kianian, Junhua Peng, Nora L. V. Lapitan, Emily J. Wennerlind, Vivienne Nduati, James A. Anderson, Deepak Sidhu, Kulvinder S. Gill, Patrick E. McGuire, Calvin O. Qualset, and Jan Dvorak, which appeared in issue 19, September 16, 2003, of *Proc. Natl. Acad. Sci. USA* (**100**, 10836–10841; first published September 5, 2003; 10.1073/pnas.1934431100), the author name Gerry Lazo should have appeared as Gerard R. Lazo. The online version has been corrected. The corrected author line appears below.

**Eduard D. Akhunov, Alina R. Akhunova, Anna M. Linkiewicz, Jorge Dubcovsky, David Hummel, Gerard R. Lazo, Shiaoman Chao, Olin D. Anderson, Jacques David, Lili Qi, Benjamin Echaliier, Bikram S. Gill, Miftahudin, J. Perry Gustafson, Mauricio La Rota, Mark E. Sorrells, Deshui Zhang, Henry T. Nguyen, Venugopal Kalavacharla, Khwaja Hossain, Shahryar F. Kianian, Junhua Peng, Nora L. V. Lapitan, Emily J. Wennerlind, Vivienne Nduati, James A. Anderson, Deepak Sidhu, Kulvinder S. Gill, Patrick E. McGuire, Calvin O. Qualset, and Jan Dvorak**

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**GENETICS.** For the article “The presence of p53 mutations in human osteosarcomas correlates with high levels of genomic instability,” by Michael Overholtzer, Pulivarthi H. Rao, Reyna Favis, Xin-Yan Lu, Michael B. Elowitz, Francis Barany, Marc Ladanyi, Richard Gorlick, and Arnold J. Levine, which appeared in issue 20, September 30, 2003, of *Proc. Natl. Acad. Sci. USA* (**100**, 11547–11552; first published September 12, 2003; 10.1073/pnas.1934852100), the authors note that several tumor samples in Table 1 were numbered incorrectly. OS17 should have read OS18, OS22 should have read OS24, OS27 should have read OS29, OS28 should have read OS30, and OS31 should have read OS33. This correction does not affect the conclusion that no tumors contain both a mutation of p53 and an amplification of *HDM2*. The corrected table and its legend appear below.

**Table 1. p53 mutation screening**

	Tumor	p53 mutation	SSCP	PCR/LDR
1	OS2	Exon 8, D281H G → C	–	+
2	OS3	Exon 8, V272M G → A	–	+
3	OS6	Exon 5, R175H G → A	+	+
4	OS7	Exon 6, frameshift	+	NT
5	OS8	Exon 8, R273H G → A	–	+
6	OS11	Exon 5, frameshift, del 17nt	+	NT
7	OS18	Exon 6, E224D G → C	+	NT
8	OS19	Exon 6, Y220C A → G	+	NT
9	OS24	Exon 5, V173M G → A	+	NT
10	OS29	Exon 5, deletion codon 190	+	NT
11	OS30	Exon 5, V173G T → G	+	NT
12	OS33	Exon 8, R273C C → T	–	+

SSCP, single-strand conformation polymorphism; LDR, ligase-detection reaction; +, found by technique; –, not found; NT, not tested; del 17nt, deletion of 17 nucleotides.

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**SPECIAL FEATURE, CHEMISTRY.** For the article “Nutritional supplement chromium picolinate causes sterility and lethal mutations in *Drosophila melanogaster*,” by Dion D. D. Hepburn, Jiarong Xiao, Sharell Bindom, John B. Vincent, and Janis O’Donnell, which appeared in issue 7, April 1, 2003, of *Proc. Natl. Acad. Sci. USA* (**100**, 3766–3771; first published March 20, 2003; 10.1073/pnas.0636646100), it should have been noted that the corresponding author, John B. Vincent, holds four patents dealing with isolated oligopeptides or synthetic chromium (III) complexes and their potential use as nutritional supplements or drugs. However, to date neither Dr. Vincent nor the University of Alabama has marketed or formed a company to market the chromium-containing species.

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