Corrections

COLLOQUIUM PAPER. For the article "Synthetic zeolites and other microporous oxide molecular sieves" by John D. Sherman, which appeared in number 7, March 30, 1999, of *Proc. Natl. Acad. Sci. USA* (**96,** 3471–3478), the author notes the following corrections: (*i*) in *Linear Paraffins for Biodegradable Detergents*, OP ADS-34 should read UOP ADS-34; and (*ii*) in *Impacts of Molecular Sieves on Human Welfare*, the phrase, "From these numbers," should be deleted.

Correction published online before print: *Proc. Natl. Acad. Sci. USA*, 10.1073/pnas.110133597. Text and publication date are at www.pnas.org/cgi/doi/10.1073/pnas.110133597

CELL BIOLOGY. For the article "Tyrosine phosphorylation of $p62^{dok}$ by $p210^{bcr\text{-}abl}$ inhibits RasGAP activity" by Nobuhiro Kashige, Nick Carpino, and Ryuji Kobayashi, which appeared in number 5, February 29, 2000, of *Proc. Natl. Acad. Sci. USA* (**97**, 2093–2098), the authors note that the image in lane 12 of Fig. 6*a* was mistakenly deleted in the printing process. The complete figure and its legend are shown below.

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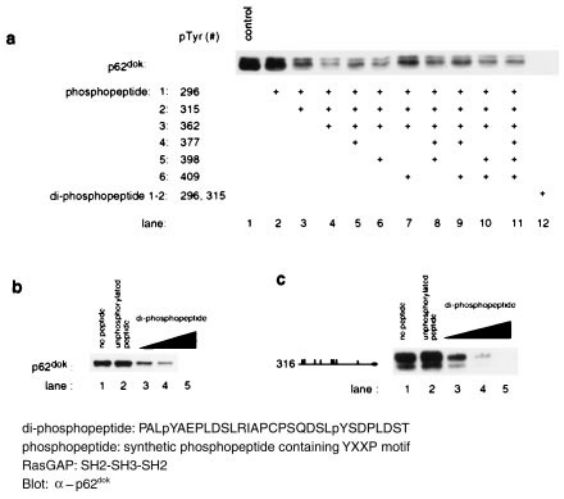

Fig. 6. Peptide competition analysis indicates that Tyr-296 and Tyr-315 play a critical role in the binding of $p62^{dok}$ to RasGAP. The ability of the diphosphopeptide to inhibit binding of Dok-1 to GAP suggests that the proper positioning of pTyr-296 and pTyr-315 in tandem is critical for the interaction of the two molecules. (*a*) Combinations of phosphopeptides corresponding to the regions surrounding individual tyrosines in $p62^{dok}$ fail to inhibit the binding of $p62^{dok}$ to RasGAP. However, a diphosphopeptide corresponding to residues 293–322 is able to inhibit binding of Dok-1 to the GAP SH2-SH3-SH2 region. Binding analysis was conducted as described. Synthetic phosphopeptides used for this experiment were: 1, SPPALpYAEPLDS (pTyr-296); 2, SQDSLpYSDPLDS (pTyr-315); 3, PKEDPIpYDEPEGL (pTyr-362); 4, VPPQG LpYDLPREPK (pTyr-377); 5, RVKEEGpYELPYNPATDD (pTyr-398); 6, NPATDD pYAVPPPR (pTyr-409); and diphosphopeptide, PALpYAEPLDSLRIAPCPSQDS LpYSDPLDST (pTyr-296 and pTyr-315). For control, unphosphorylated peptides were used. Each phosphopeptide was added in concentration of 50 μM. (*b*) Dose-dependent inhibition of $p62^{dok}$ binding to RasGAP by diphosphopeptide (Dok-1 aa 293–322). The diphosphopeptide was added in concentration of 0.5, 5, or 50 μM. The unphosphorylated peptide was added in concentration of 50 μM. (*c*) Dose-dependent inhibition of a truncated Dok-1 (the truncation construct 316: residues 316–481) binding to RasGAP by diphosphopeptide. The diphosphopeptide was added in concentration of 0.5, 5, or 50 μM. The unphosphorylated peptide was added in concentration of 50 μM.

GENETICS. In the article "Toward *Anopheles* transformation: *Minos* element activity in anopheline cells and embryos" by Flaminia Catteruccia, Tony Nolan, Claudia Blass, Hans-Michael Müller, Andrea Crisanti, Fotis C. Kafatos, and Thanasis G. Loukeris, which appeared in number 5, February 29, 2000, of *Proc. Natl. Acad. Sci. USA* (**97,** 2157–2162), the authors note that three mistakes were introduced inadvertently in assembling Fig. 4. The revised Fig. 4 printed below includes the correct photograph of E7 insertion at 21E, as well as the correct chromosomal locations of E4 at 25D (2L) and E5 at 36B (3R).

A

Sua 5.1*	*flanking sequence*	location
E1	ggttggggctcg**TA***ACCACGGAACAG*	43D (3L)
E2	ggttggggctcg**TA***AAGCACCACGCT*	13E (2R)
E3	ggttggggctcg**TA***GACCCAGACCAC*	35B (3R)
E4	ggttggggctcg**TA***GATAAACCTTTA*	25D (2L)
E5	ggttggggctcg**TA***CAGTACACATCG*	36B (3R)
E6	ggttggggctcg**TA***ATGTGCTGCATC*	34A (3R)
E7	ggttggggctcg**TA***TGTAGATCGGTT*	21E (2L)
E8	ggttggggctcg**TA***ATTAAACTTCCG*	36C (3R)
E9	ggttggggctcg**TA***TGTAGGTCAGTG*	28B (2L)
E10	ggttggggctcg**TA***AACACGCTCGAA*	41D (3L)

Sua 4.0	*flanking sequence*	location
S1	ggttggggctcg**TA***GATGGATCACGC*	44B (3L)
S2	ggttggggctcg**TA***CTAACCTAACAG*	25C (2L)
S3	ggttggggctcg**TA***ATGCAATTAATG*	31C (3R)
S4	*GAGATGTTGTAA***TA**cgagccccaacc	32D (3R)

B

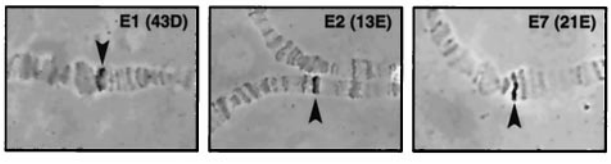

Fig. 4. (*A*) Sequences of the *Minos* insertion sites in the genome of Sua 5.1* and Sua 4.0 cells. Chromosomal flanking sequences are represented with capital letters in italics. Small lettering represents the sequences of the *Minos* end. The expected TA dinucleotide of the insertion site is shown in bold. The chromosomal divisions and subdivisions from which the flanking sequences were derived are indicated with the chromosomal arm listed in parenthesis. (*B*) Typical results of determining the location of origin of the rescued genomic fragments by *in situ* localization to polytene chromosomes of the Suakoko mosquito strain.

PLANT BIOLOGY. For the article "*Oryza sativa PSK* gene encodes a precursor of phytosulfokine-α, a sulfated peptide growth factor found in plants" by Heping Yang, Yoshikatsu Matsubayashi, Kenzo Nakamura, and Youji Sakagami, which appeared in number 23, November 9, 1999, of *Proc. Natl. Acad. Sci. USA* (**96,** 13560–13565), the authors note the following correction. In line 18 of the first column on page 13565, "+5 positions" should read "+3 positions."

Correction published online before print: *Proc. Natl. Acad. Sci. USA*, 10.1073/pnas.120161097. Text and publication date are at www.pnas.org/cgi/doi/10.1073/pnas.120161097