Corrections

COMMENTARY. For the article "Oxygen toxicity and the health and survival of eukaryote cells: A new piece is added to the puzzle," by F. Archibald, which appeared in issue 18, September 2, 2003, of *Proc. Natl. Acad. Sci. USA* (**100,** 10141–10143; first published August 25, 2003; 10.1073/pnas.1934513100), the author notes that Fig. 1 and its legend were incomplete and omitted critical components of the localization and activation of MnSOD. The correct figure and its legend appear below.

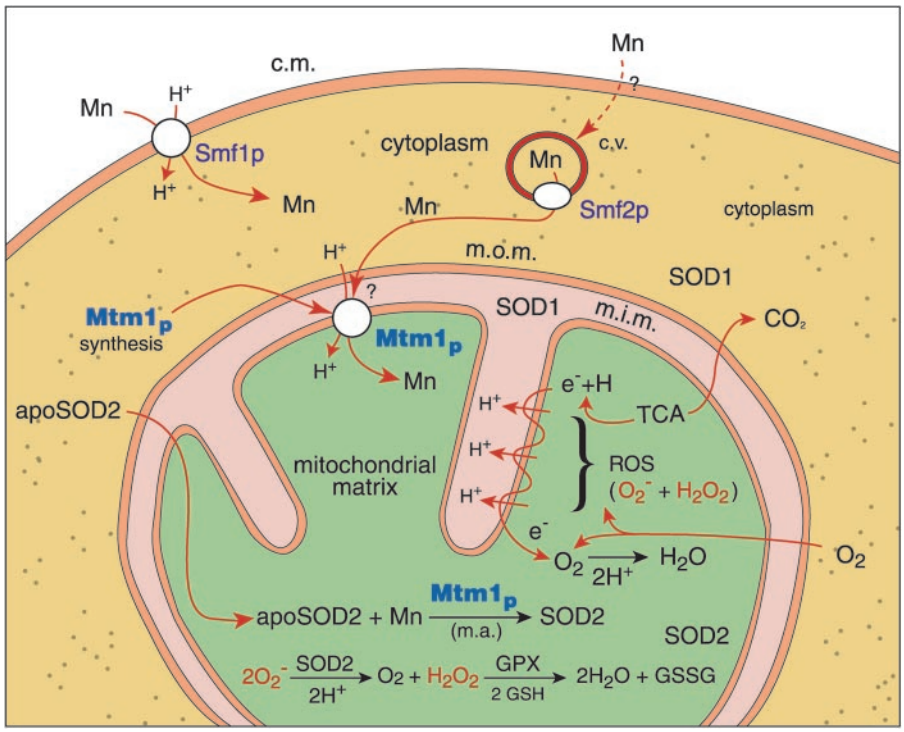

Fig. 1. Diagram of the postulated mechanisms of localization and activation of MnSOD (SOD2) in a cell of brewer's yeast (*S. cerevisiae*) based on the findings of Luk *et al*. (1). c.m., cell or plasma membrane; c.v., cytoplasmic vesicle (not Golgi); m.o.m., mitochondrial outer membrane; m.i.m., mitochondrial inner membrane; m.a., mitochondrial membrane-associated reaction; TCA, tricarboxylic acid cycle generating reducing equivalents for the electron transport (respiratory) chain (sinuous red line); ROS, reactive oxygen species, primarily O_2^- and H_2O_2; GPX, glutathione peroxidase; GSH, reduced glutathione monomer; GSSG, oxidized glutathione dimer; Smf1p, high-affinity Mn-uptake protein; Smf2p, hydrophobic cytoplasmic vesicle-associated protein providing Mn to mitochondrial SOD; Mtm1p, SOD-2 activating protein first reported by Luk *et al*. (1).

www.pnas.org/cgi/doi/10.1073/pnas.2536674100

MICROBIOLOGY. For the article "Divergent retroviral late-budding domains recruit vacuolar protein sorting factors by using alternative adaptor proteins," by Juan Martin-Serrano, Anton Yaravoy, David Perez-Caballero, and Paul D. Bieniasz, which appeared in issue 21, October 14, 2003, of *Proc. Natl. Acad. Sci. USA* (**100,** 12414–12419; first published September 30, 2003; 10.1073/pnas.2133846100), the author name Anton Yaravoy should have appeared as Anton Yarovoy. The online version has been corrected. The corrected author line appears below.

Juan Martin-Serrano, Anton Yarovoy, David Perez-Caballero, and Paul D. Bieniasz

www.pnas.org/cgi/doi/10.1073/pnas.2637115100

NEUROBIOLOGY. For the article "Inhibition of calcium/calmodulin kinase II alpha subunit expression results in epileptiform activity in cultured hippocampal neurons," by Severn B. Churn, Sompong Sombati, Emma R. Jakoi, Lawrence Sievert, and Robert J. DeLorenzo, which appeared in issue 10, May 9, 2000, of *Proc. Natl. Acad. Sci. USA* (**97,** 5604–5609; first published April 25, 2000; 10.1073/pnas.080071697), the author name Lawrence Sievert should have appeared as Lawrence Severt. The online version has been corrected. The corrected author line appears below.

Severn B. Churn, Sompong Sombati, Emma R. Jakoi, Lawrence Severt, and Robert J. DeLorenzo

www.pnas.org/cgi/doi/10.1073/pnas.2335809100

NEUROSCIENCE. For the article "Activation of ATP-sensitive K^+ (K_{ATP}) channels by H_2O_2 underlies glutamate-dependent inhibition of striatal dopamine release," by Marat V. Avshalumov and Margaret E. Rice, which appeared in issue 20, September 30, 2003, of *Proc. Natl. Acad. Sci. USA* (**100,** 11729–11734; first published September 17, 2003; 10.1073/pnas.1834314100), the scale bar on the voltammograms in Fig. 1*B* on page 11730 was incorrectly labeled as mM instead of μM due to a printer's error. The corrected figure and its legend appear below.

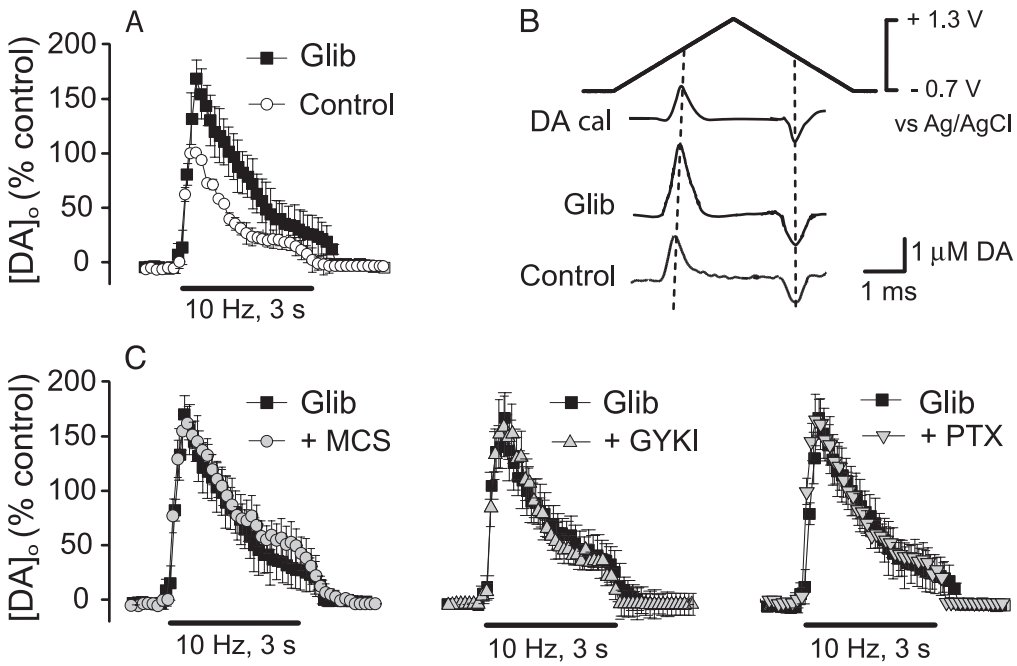

Fig. 1. Glutamate–H_2O_2-dependent modulation of striatal DA release is blocked by glibenclamide. (*A*) Glibenclamide (Glib; 3 μM) caused a significant increase in evoked DA release ($P < 0.01$, glibenclamide vs. control; $n = 5$). (*B*) Applied voltage waveform and representative voltammograms of DA obtained during DA calibration (DA cal; 1 μM) and at maximum evoked $[DA]_o$ during stimulation (10 Hz, 30 pulses) in normal aCSF (control) and in the presence of glibenclamide in the same striatal slice. Sampling interval was 100 ms; voltage scan rate was 800 V/s. (*C*) In the presence of glibenclamide, the usual effects of MCS (1 mM), GYKI-52466 (GYKI; 50 μM), and picrotoxin (100 μM) on DA release were prevented (P 0.05, each agent vs. glibenclamide alone; $n = 5$). Data are given as mean ± SEM, illustrated as percentage of same-site control. Solid bars indicate the stimulation period.

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