Corrections

CELL BIOLOGY. For the article "Retinoblastoma protein functions as a molecular switch determining white versus brown adipocyte differentiation," by Jacob B. Hansen, Claus Jørgensen, Rasmus K. Petersen, Philip Hallenborg, Rita De Matteis, Hans A. Bøye, Natasa Petrovic, Sven Enerbäck, Jan Nedergaard, Saverio Cinti, Hein te Riele, and Karsten Kristiansen, which appeared in issue 12, March 23, 2004, of *Proc. Natl. Acad. Sci. USA* (**101,** 4112–4117; first published March 15, 2004; 10.1073/pnas.0301964101), the authors note that the units in the last sentence of the Fig. 5 legend should be "μm" instead of "mM." The figure and its corrected legend appear below.

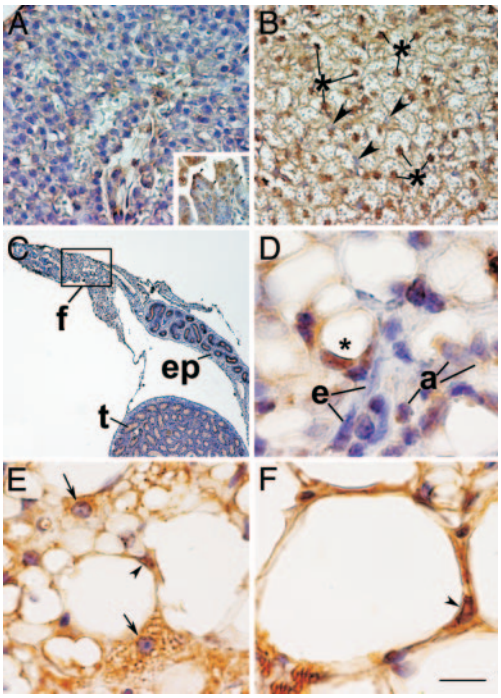

Fig. 5. Differential expression of pRB during development of WAT and BAT and during transdifferentiation of white into brown adipocytes. (*A*) BAT anlage of an embryonic day 19 mouse fetus. No nuclear pRB immunoreactivity is observed. (*Inset*) Internal positive control of the same fetus showing pRB-positive nuclei of apical cells of intestinal villi (arrowheads). (*B*) BAT of a 10-day-old mouse. Nuclei of well differentiated brown adipocytes are pRB-positive (*), and nuclei of endothelial cells are negative (arrowheads). (*C*) Epididymal fat pad of a 9-day-old mouse. f, fat pad; ep, epididymus; t, testis. (*D*) Epididymal WAT of a 9-day-old mouse (enlargement of the squared area in *C*). Adipocyte precursors with lipids droplets (*) show nuclear staining. Endothelial cells (e) and adipoblasts (a) are pRB-negative. (*E*) Retroperitoneal WAT of a 20-week-old rat treated with the β3-adrenergic agonist CL-316243 for 7 days. Most of the transdifferentiating multilocular adipocytes exhibit pRB negative nuclei (arrows). A unilocular adipocyte with positive nucleus is visible (arrowhead). (*F*) Same tissue as in *E*. A unilocular adipocyte with positive nucleus is visible (arrowhead). (Bars: *A* and *B* = 30 μm; *A Inset* = 60 μm; *D* and *F* = 10 μm; *C* = 200 μm; *E* = 15 μm.)

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CELL BIOLOGY. For the article "Cholesterol depletion induces PKA-mediated basolateral-to-apical transcytosis of the scavenger receptor class B type I in MDCK cells," by Patricia V. Burgos, Carla Klattenhoff, Erwin de la Fuente, Attilio Rigotti, and Alfonso González , which appeared in issue 11, March 16, 2004, of *Proc. Natl. Acad. Sci. USA* (**101,** 3845–3850; first published March 8, 2004; 10.1073/pnas.0400295101), the authors note that the ordinate for the graphs in Fig. 5 *B* and *C* should read "cAMP levels (pmol/μg)" instead of "PKA activity (pmol/min/μg)." The corrected figure and its legend appear below.

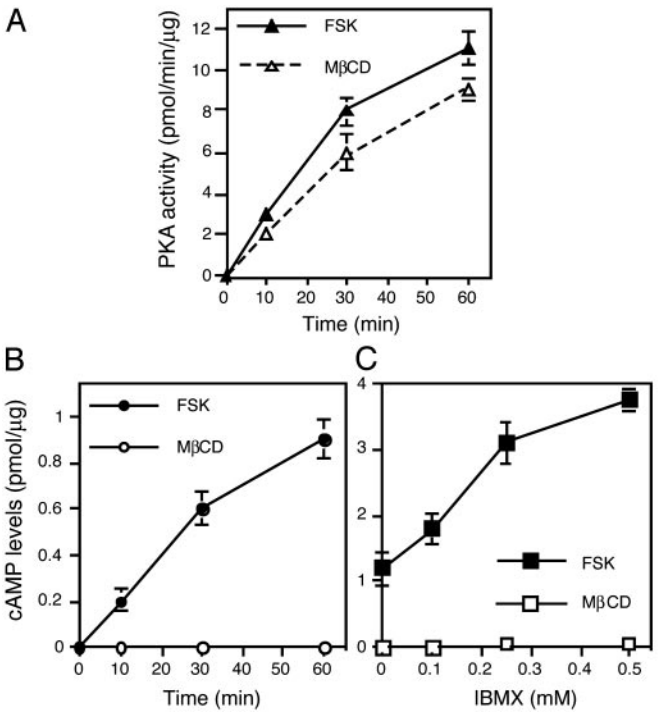

Fig. 5. Acute cholesterol depletion by MβCD activates PKA without increasing intracellular cAMP levels. MDCK cells were incubated at 37°C with 10 mM MβCD or 50 μM FSK. (*A*) Both treatments increased PKA activity. (*B*) In contrast with FSK, cells treated with MβCD showed undetectable levels of cAMP levels. (*C*) In the presence of increasing concentrations of phosphodiesterase inhibitor IBMX, cAMP levels elicited by 1 h of FSK treatment increased progressively whereas, during treatment with MβCD, they remained almost undetectable. Each point represents average and SE.

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CORRECTIONS

CELL BIOLOGY. For the article "The BRCA1-associated protein BACH1 is a DNA helicase targeted by clinically relevant inactivating mutations," by Sharon Cantor, Ronny Drapkin, Fan Zhang, Yafang Lin, Juliana Han, Sushmita Pamidi, and David M. Livingston, which appeared in issue 8, February 24, 2004, of *Proc. Natl. Acad. Sci. USA* (**101,** 2357–2362; first published February 17, 2004; 10.1073/pnas.0308717101), the authors note that the *x* axis of the left graph in Fig. 2*B* is numbered incorrectly. The corrected figure and its legend appear below.

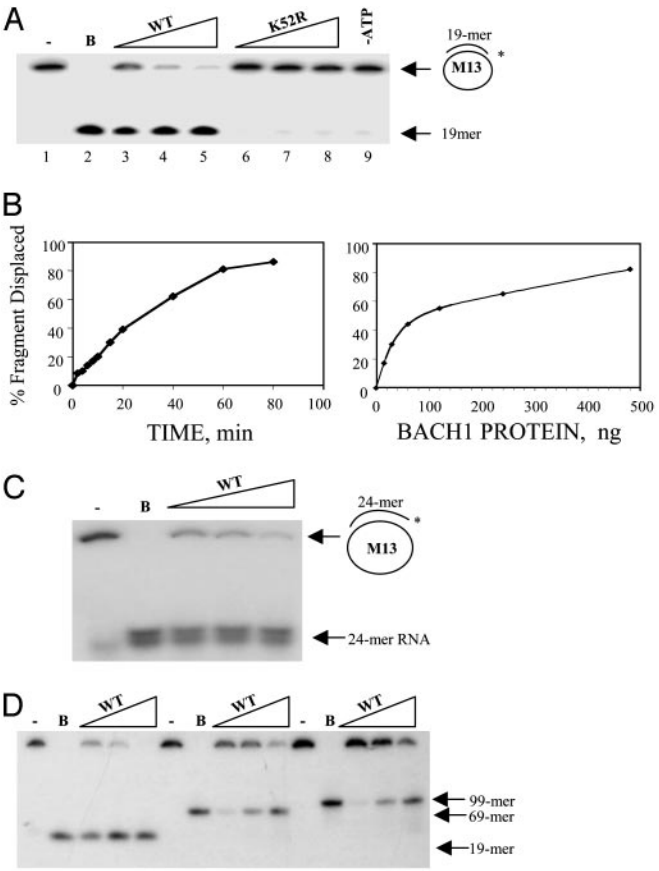

Fig. 2. BACH1 is an ATP-dependent helicase. (*A*) Increasing amounts of WT and K52R mutant BACH1 were incubated with a DNA helicase substrate containing an annealed radiolabeled 19-nt oligomer (see *Materials and Methods*). Lane 1, annealed substrate (−); lane 2, heat-denatured substrate (B, for boiled); lanes 3–5, BACH1 (60, 180, and 450 ng, respectively); lanes 6–8, K52R BACH1 (200, 400, and 600 ng, respectively); lane 9, WT with no ATP. (*B*) BACH1 unwinds DNA in a time- and dose-dependent manner. BACH1 protein (150 ng) was incubated with the 19-mer helicase substrate for the indicated times. Independently, increasing amounts of BACH1 (15, 30, 60, 120, 240, and 480 ng) were incubated with substrate for 30 min. (*C*) Increasing amounts of BACH1 (60, 180, and 450 ng) were incubated with a RNA:DNA helicase substrate and helicase activity was measured. (*D*) Increasing quantities of BACH1 (60, 180, and 450 ng) were incubated with DNA helicase substrates of increasing partial duplex length, as indicated. In all cases, reaction products were resolved in an 8% native polyacrylamide gel containing 15% glycerol. Results were quantitated by using a Molecular Dynamics STORM PhosphorImager.

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MICROBIOLOGY. For the article "Pneumococcal carriage results in ganglioside-mediated olfactory tissue infection," by Frederik W. van Ginkel, Jerry R. McGhee, James M. Watt, Antonio Campos-Torres, Lindsay A. Parish, and David E. Briles, which appeared in issue 24, November 25, 2003, of *Proc. Natl. Acad. Sci. USA* (**100,** 14363–14367; first published November 10, 2003; 10.1073/pnas.2235844100), the authors note that the day 4 colonization data of strain EF3030 of the nasal washes have been omitted from Fig. 1*B*. The corrected figure and its legend appear below.

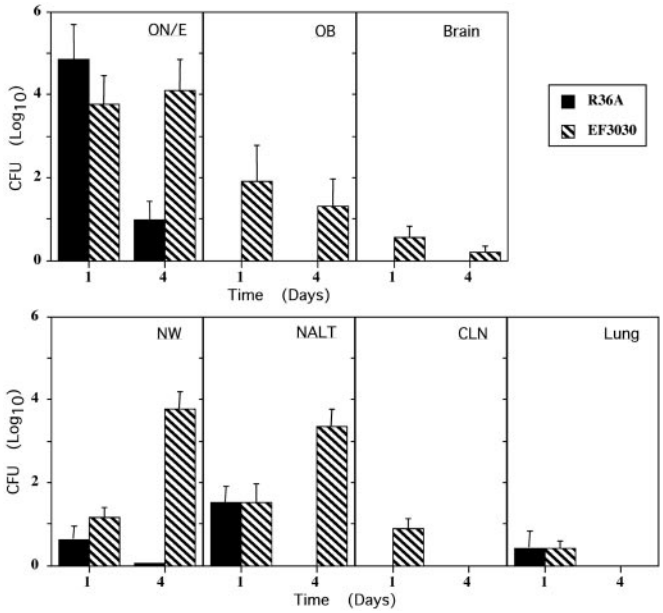

Fig. 1. Nasal delivery of 3×10^6 cfu of either the nonencapsulated R36A strain or the virulent EF3030 strain of *S. pneumoniae* to *xid* mice. The neuronal tissues (ON/E, OB, and brain) and the lymphoid tissues (NALT, CLN, and lungs) were collected, minced, and analyzed for the presence of live pneumococci at 1 and 4 days after nasal challenge. Indicated is the mean of $\log_{10}$ cfu ± 1 SE. The 0 value on the *y*-axis represents the absence of detectable cfu. Indicated are the mean cfu ± SE of five mice per group; data are representative of three different experiments.

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MICROBIOLOGY. For the article "Mycobacterial polyketide-associated proteins are acyltransferases: Proof of principle with *Mycobacterium tuberculosis* PapA5," by Kenolisa C. Onwueme, Julian A. Ferreras, John Buglino, Christopher D. Lima, and Luis E. N. Quadri, which appeared in issue 13, March 30, 2004, of *Proc. Natl. Acad. Sci. USA* (**101,** 4608–4613; first published March 18, 2004; 10.1073/pnas.0306928101), due to a printer's error, the word "acyltransferase" incorrectly appeared as "acetyltransferase" in the eighth line of the abstract.

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