THE JOURNAL OF BIOLOGICAL CHEMISTRY VOL. 284, NO. 31, p. 21100, July 31, 2009 © 2009 by The American Society for Biochemistry and Molecular Biology, Inc. Printed in the U.S.A.

### VOLUME 278 (2003) PAGES 51722–51734 DOI 10.1074/jbc.A308740200

## Dsl1p, an essential component of the Golgi-endoplasmic reticulum retrieval system in yeast, uses the same sequence motif to interact with different subunits of the COPI vesicle coat.

#### Uwe Andag and Hans Dieter Schmitt

On page 51726, we misleadingly stated that the central acidic domain in Dsl1p is essential for viability. Evidence for this was presented in Fig. 3*C*. This figure shows that a mutant carrying two Trp-to-Ala replacements in this region did not support growth of a *dsl1* deletion mutant. However, we recently created a GAL-regulated TAP-tagged version of *DSL1* carrying five Trp-to-Ala substitutions in this region to use as a negative control in pulldown experiments. Surprisingly, this mutant complemented the *dsl1* knock-out. Even a single-copy untagged version of this allele could replace the wild-type gene. However, these cells grow poorly at all temperatures tested and show phenotypes similar to those of Dsl1p-depleted cells.

Sequencing showed that the plasmid used for the growth assay in our previous work contained a C-terminal truncation in addition to the Trp-to-Ala substitutions at positions 413 and 455. A mutant clone with an intact C terminus supported growth of a *dsl1* deletion mutant.

Our recent data are still consistent with the notion that the outer tryptophan motifs in the acidic domain of Dsl1p (Trp<sup>413</sup>/Trp<sup>415</sup> and Trp<sup>455</sup>/Trp<sup>459</sup>) mediate binding to  $\delta$ -COP, whereas the central tryptophan residue at position 425 is involved in the interaction with  $\alpha$ -COP. Obviously, all motifs must be destroyed to affect the growth of cells.

We sincerely apologize for any confusion that this may have caused.

VOLUME 284 (2009) PAGES 4857-4864 DOI 10.1074/jbc.A807610200

## Angiotensin II type 2 receptor blockade increases bone mass.

#### Yayoi Izu, Fumitaka Mizoguchi, Aya Kawamata, Tadayoshi Hayata, Testuya Nakamoto, Kazuhisa Nakashima, Tadashi Inagami, Yoichi Ezura, and Masaki Noda

This work was supported by Grants-in-aid 17012008, 18109011, 18659438, and 18123456 from the Japanese Ministry of Education (21st Century Center of Excellence Program, Frontier Research for Molecular Destruction and Reconstitution of Tooth and Bone); by grants from the Japan Space Forum, NASDA, and the Advanced Bone and Joint Science Strategic Research Networks Projects (Japan Society for Promotion of Science Core to Core Program, Research for the Future Program, Genome Science); and by National Institutes of Health Grant HL58205.

## VOLUME 280 (2005) PAGES 24839-24848 DOI 10.1074/jbc.A500253200

# The ATPase activity of BfpD is greatly enhanced by zinc and allosteric interactions with other Bfp proteins.

Lynette J. Crowther, Atsushi Yamagata, Lisa Craig, John A. Tainer, and Michael S. Donnenberg

This article has been withdrawn at the request of the authors.

VOLUME 284 (2009) PAGES 13602–13609 DOI 10.1074/jbc.A900894200

# Determination of *in vivo* dissociation constant, $K_D$ , of Cdc42-effector complexes in live mammalian cells using single wavelength fluorescence cross-correlation spectroscopy.

#### Thankiah Sudhaharan, Ping Liu, Yong Hwee Foo, Wenyu Bu, Kim Buay Lim, Thorsten Wohland, and Sohail Ahmed

On page 13603, the following sentence should be added to the legend of Fig. 1: The SH3 in the N-WASP schematic indicates a polyproline sequence that binds SH3 domains.

VOLUME 284 (2009) PAGES 7385-7394 DOI 10.1074/jbc.A807820200

# Autotaxin/lysopholipase D and lysophosphatidic acid regulate murine hemostasis and thrombosis.

Zehra Pamuklar, Lorenzo Federico, Shuying Liu, Makiko Umezu-Goto, Anping Dong, Manikandan Panchatcharam, Zachary Fulkerson, Evgeny Berdyshev, Viswanathan Natarajan, Xianjun Fang, Laurens A. van Meeteren, Wouter H. Moolenaar, Gordon B. Mills, Andrew J. Morris, and Susan S. Smyth

Dr. Fulkerson's name was misspelled. The correct spelling is shown above.

We suggest that subscribers photocopy these corrections and insert the photocopies in the original publication at the location of the original article. Authors are urged to introduce these corrections into any reprints they distribute. Secondary (abstract) services are urged to carry notice of these corrections as prominently as they carried the original abstracts.

