

## Corrections

### BIOCHEMISTRY

Correction for “Mechanism of ligand-gated potassium efflux in bacterial pathogens,” by Tarmo P. Roosild, Samantha Castronovo, Jess Healy, Samantha Miller, Christos Pliotas, Tim Rasmussen, Wendy Bartlett, Stuart J. Conway, and Ian R. Booth, which appeared in issue 46, November 16, 2010, of *Proc Natl Acad Sci USA* (107:19784–19789; first published November 1, 2010; 10.1073/pnas.1012716107).

The undersigned authors wish to note, “The KeffC system of *E. coli* is maintained in an inactive state by the binding of glutathione (GSH) and is activated by the formation of GSH adducts (GSX), particularly those with bulky substituents. We described two crystal structures with density present in the ligand-binding domain that we interpreted as GSH and GSX. Recently, an independent, experienced crystallographer, who had viewed the structures from our study in a different context, made representations to us that cast doubt on position of the succinimido ring of GSX. We have further reviewed the density maps with the aid of an experienced crystallographer. As a consequence, we believe it is important to draw this altered interpretation of the crystal structures to the attention of readers. In both structures, the density for the backbone of GSH is clear and allows unequivocal assignment of the position of the tripeptide. In PDB coordinate set 3L9X, the density for the succinimido ring is very weak, making interpretation very speculative and the assignment rests on the identity of the ligand added to the crystallization mixture, for which there are two diastereomers in the solution—a possibility that provides some basis for weakening the density. However, in 3L9W there are two anomalies that affect the interpretation of the bound ligand. First, there is no density for the carbon atom attached to the sulfur of GSH and second, there is extra density adjacent to the position of sulfur that could be modelled as a constrained succinimido ring. However, this density could also be water or any other molecule that is trapped in the structure. Thus, while there is good evidence for the peptide, the evidence that it is in the GSH form is uncertain.

“There are no new data on either the structures or on the gating mechanism. However, we believe that we should be cautious in interpreting the structural data and that the field in general should be made aware of the alternative views of the electron density data. Note that the mutagenesis and spectroscopic data that were presented in the original manuscript are not affected by this alternative interpretation.”

Tarmo P. Roosild and Samantha Castronovo have decided not to sign this statement.

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Jess Healy  
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Christos Pliotas  
Tim Rasmussen  
Wendy Bartlett  
Stuart J. Conway

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### NEUROSCIENCE

Correction for “Bioluminescence imaging of A $\beta$  deposition in bi-genic mouse models of Alzheimer’s disease,” by Joel C. Watts, Kurt Giles, Sunny K. Grillo, Azucena Lemus, Stephen J. DeArmond, and Stanley B. Prusiner, which appeared in issue 6, February 8, 2011, of *Proc Natl Acad Sci USA* (108:2528–2533; first published January 24, 2011; 10.1073/pnas.1019034108).

The authors note that the following grant should be added to the Acknowledgments: “NIH Grant AG002132.”

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### PHARMACOLOGY

Correction for “Structural insights into gene repression by the orphan nuclear receptor SHP,” by Xiaoyong Zhi, X. Edward Zhou, Yuanzheng He, Christoph Zechner, Kelly M. Suino-Powell, Steven A. Kliewer, Karsten Melcher, David J. Mangelsdorf, and H. Eric Xu, which appeared in issue 2, January 14, 2014, of *Proc Natl Acad Sci USA* (111:839–844; first published December 30, 2013; 10.1073/pnas.1322827111).

The authors note that the following statement should be added to the Acknowledgments: “We want to thank the generosity of Drs. Marcia I. Dawson and Zebin Xia (Sanford-Burnham Medical Research Institute) for providing us with 3-Cl-AHPC and for their discussion on its use.”

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### PHYSIOLOGY

Correction for “Phosphorylation sites required for regulation of cardiac calcium channels in the fight-or-flight response,” by Ying Fu, Ruth E. Westenbroek, Todd Scheuer, and William A. Catterall, which appeared in issue 48, November 26, 2013, of *Proc Natl Acad Sci USA* (110:19621–19626; first published November 11, 2013; 10.1073/pnas.1319421110).

The authors note “The method used for exogenous expression of Ca<sub>v</sub>1.2 channels in ref. 32 was incorrectly described as ‘viral transduction’ in the text. In fact, Yang et al. created transgenic mice with inducible, cardiomyocyte-specific expression of exogenous Ca<sub>v</sub>1.2 channels regulated by a tetracycline-inducible promoter. When crossed with a transgenic mouse line expressing doxycycline-regulated reverse transcriptional activator under control of the  $\alpha$ -myosin heavy chain promoter, the resulting double transgenic offspring expressed exogenous Ca<sub>v</sub>1.2 channels in their cardiac myocytes after treatment with doxycycline. The authors regret the error in describing these methods.”

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