

CORRECTION

Open Access



Correction to: Pathophysiology of and therapeutic options for a *GABRA1* variant linked to epileptic encephalopathy

Yun-Fei Bai^{1,2†}, Michelle Chiu^{3†}, Elizabeth S. Chan^{1†}, Peter Axerio-Cilies¹, Jie Lu¹, Linda Huh³, Mary B. Connolly³, Ilaria Guella⁴, Matthew J. Farrer^{4,5}, Zhi-Qing David Xu², Lidong Liu^{1*}, Michelle Demos^{3*} and Yu Tian Wang^{1*}

Correction to: Mol Brain (2019) 12:92
<https://doi.org/10.1186/s13041-019-0513-9>

Following publication of the original article [1], the authors reported errors in Fig. 4. Specifically, a wrong actin blot is presented in Fig. 4a. In this Correction, the corrected version of Fig. 4 is shown.

Author details

¹Djavad Mowafaghian Centre for Brain Health and Department of Medicine, University of British Columbia, Vancouver, Canada. ²Department of Neurobiology, Beijing Key Laboratory of Neural Regeneration and Repair, Beijing Laboratory of Brain Disorders (Ministry of Science and Technology), Beijing Institute for Brain Disorders, Capital Medical University, Beijing, China. ³Division of Neurology, Department of Paediatrics, BC Children's Hospital, University of British Columbia, Vancouver, Canada. ⁴Centre for Applied Neurogenetics, University of British Columbia, Vancouver, Canada. ⁵McKnight Brain Institute, University of Florida, Gainesville, USA.

Published online: 27 March 2020

Reference

1. Bai YF, et al. Pathophysiology of and therapeutic options for a *GABRA1* variant linked to epileptic encephalopathy. *Mol Brain*. 2019;12:92. <https://doi.org/10.1186/s13041-019-0513-9>.

The original article can be found online at <https://doi.org/10.1186/s13041-019-0513-9>

* Correspondence: lidong@mail.ubc.ca; mdemos@cw.bc.ca; ytwang@brain.ubc.ca

[†]Yun-Fei Bai, Michelle Chiu and Elizabeth S. Chan contributed equally to this work.

¹Djavad Mowafaghian Centre for Brain Health and Department of Medicine, University of British Columbia, Vancouver, Canada

³Division of Neurology, Department of Paediatrics, BC Children's Hospital, University of British Columbia, Vancouver, Canada

Full list of author information is available at the end of the article



© The Author(s). 2020 **Open Access** This article is licensed under a Creative Commons Attribution 4.0 International License, which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if changes were made. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit <http://creativecommons.org/licenses/by/4.0/>. The Creative Commons Public Domain Dedication waiver (<http://creativecommons.org/publicdomain/zero/1.0/>) applies to the data made available in this article, unless otherwise stated in a credit line to the data.

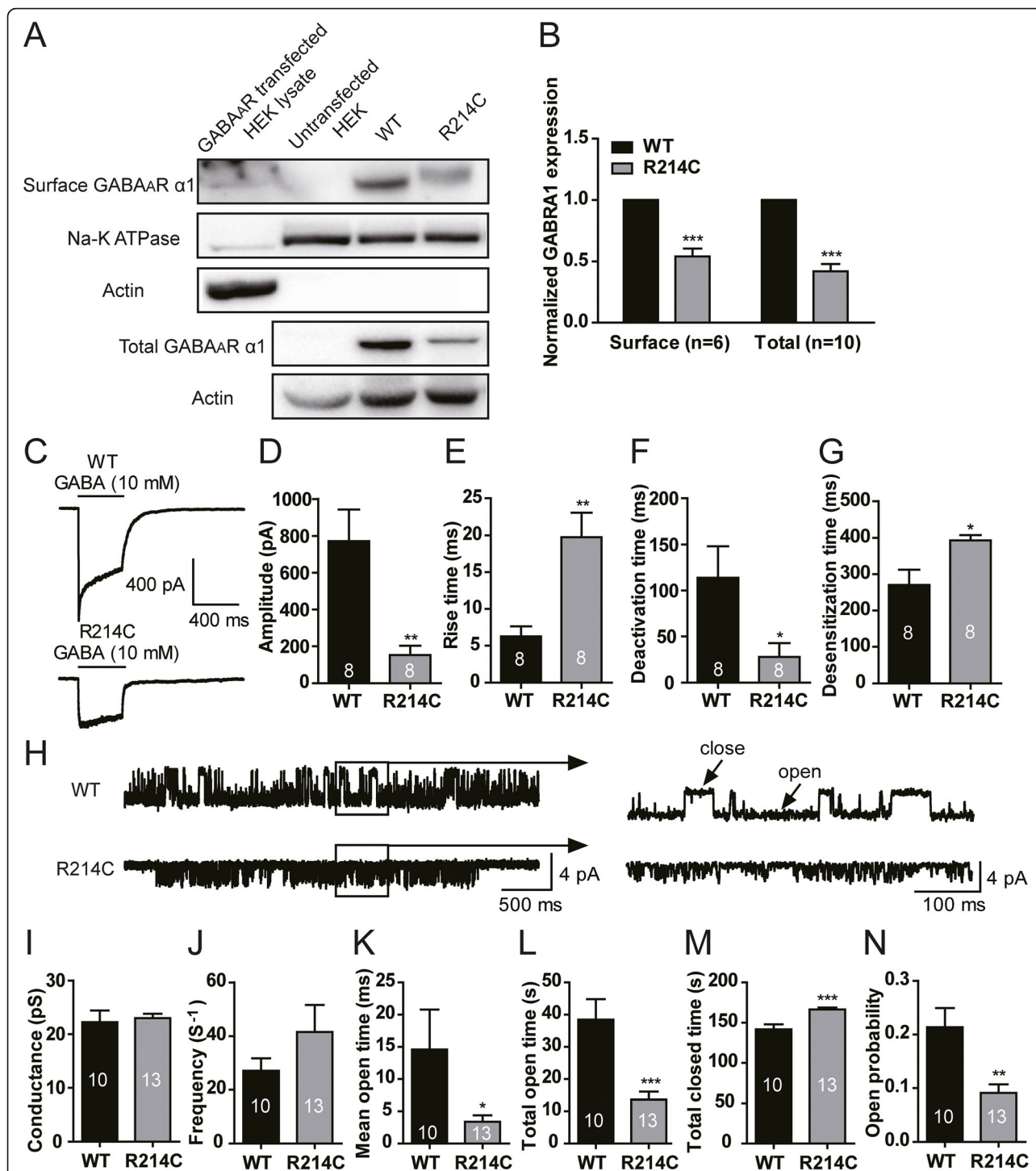


Fig. 4 The R214C mutation resulted in reduced surface and total expression levels of the α1 subunit, and altered the kinetic and single channel properties of GABA_ARs. **a** Representative blots of biotinylation samples for surface receptor expression and cell lysates for total receptor expression from HEK293 cells expressing either WT or R214C GABA_ARs. **b** Quantification of surface α1 subunits normalized to Na⁺/K⁺ ATPase (n = 6), and total α1 subunits normalized to β-actin (n = 10). Statistical differences were determined using student's t-test by comparing to expression levels of WT GABA_AR expressing cells (***p < 0.001). **c** Representative traces of GABA currents recorded in excised macro-patch membrane under outside-out configuration from WT or R214C GABA_AR expressing cells. Currents were evoked by rapidly perfusion of 10 mM GABA to the membrane patch for 400 ms. Quantification of averaged peak current amplitudes (**d**), 10–90% rise time (**e**), deactivation rate (**f**) and desensitization (**g**) in WT (n = 8) or R214C (n = 8) GABA_AR expressing cells. **h** Representative single channel current traces recorded under cell-attached configuration with a pipette containing GABA (1 mM) at a holding potential of +100 mV from cells expressing WT or R214C GABA_ARs. Quantified average of conductance (**i**), opening frequency (**j**), mean open time (**k**), total open time (**l**), total closed time (**m**), and open channel probability (**n**) of WT (n = 10) or R214C (n = 13) GABA_ARs. Statistical differences were determined using student's t-test by comparing to WT GABA_AR cells (*p < 0.05, **p < 0.01, ***p < 0.001)