



Corrigendum: IRSp53 Deletion in Glutamatergic and GABAergic Neurons and in Male and Female Mice Leads to Distinct Electrophysiological and Behavioral Phenotypes

Yangsik Kim^{1†}, Young Woo Noh^{2†}, Kyungdeok Kim², Esther Yang³, Hyun Kim³ and Eunjoon Kim^{2,4*}

¹ Graduate School of Medical Science and Engineering, Korea Advanced Institute of Science and Technology (KAIST), Daejeon, South Korea, ² Department of Biological Sciences, Korea Advanced Institute of Science and Technology (KAIST), Daejeon, South Korea, ³ Department of Anatomy, College of Medicine, Korea University, Seoul, South Korea, ⁴ Center for Synaptic Brain Dysfunctions, Institute for Basic Science (IBS), Daejeon, South Korea

OPEN ACCESS

Edited and reviewed by: Lei Shi, Jinan University, China

> ***Correspondence:** Eunjoon Kim kime@kaist.ac.kr

[†]These authors have contributed equally to this work

Specialty section:

This article was submitted to Cellular Neuropathology, a section of the journal Frontiers in Cellular Neuroscience

Received: 24 September 2021 Accepted: 15 October 2021 Published: 22 November 2021

Citation:

Kim Y, Noh YW, Kim K, Yang E, Kim H and Kim E (2021) Corrigendum: IRSp53 Deletion in Glutamatergic and GABAergic Neurons and in Male and Female Mice Leads to Distinct Electrophysiological and Behavioral Phenotypes. Front. Cell. Neurosci. 15:782716. doi: 10.3389/fncel.2021.782716 Keywords: autism, synapse, IRSp53, mPFC, social interaction, hyperactivity

A Corrigendum on

IRSp53 Deletion in Glutamatergic and GABAergic Neurons and in Male and Female Mice Leads to Distinct Electrophysiological and Behavioral Phenotypes

by Kim, Y., Noh, Y. W., Kim, K., Yang, E., Kim, H., and Kim, E. (2020). Front. Cell Neurosci. 14:23. doi: 10.3389/fncel.2020.00023

In the original article, there was a mistake in **Figure 3** as published. It was due to an inadvertent mistake in the quantification process. The new quantification indicates that there is no statistical difference in the NMDA/AMPA ratio between WT and IRSp53-KO mice; previous **Figure 3**C indicated a decrease in the mutant mice. The correct **Figure 3** and legend appears below.

To reflect this change a correction has also been made to the Results, *Emx1-Cre; Irsp53*^{fl/fl} and *Viaat-Cre; Irsp53*^{fl/fl} Mice Show Distinct Changes in Synaptic Transmission and Intrinsic Excitability in mPFC Pyramidal Neurons, Second paragraph:

"When evoked synaptic transmission was measured, the ratio of NMDAR-mediated EPSCs and AMPA receptor (AMPAR)-mediated EPSCs was not altered in *Emx1-Cre; Irsp53*^{fl/fl} layer V pyramidal neurons (**Figure 3C**). These results collectively suggest that Irsp53 deletion in glutamatergic neurons leads to reduced spontaneous excitatory but not inhibitory synaptic transmission, increased ratio of evoked EPSCs/IPSCs, and increased neuronal excitability without affecting evoked NMDAR-EPSC/AMPAR-EPSC ratio in layer V mPFC neurons."

The authors apologize for this error and state that this does not change the scientific conclusions of the article in any way. The original article has been updated.

Frontiers in Cellular Neuroscience | www.frontiersin.org

1

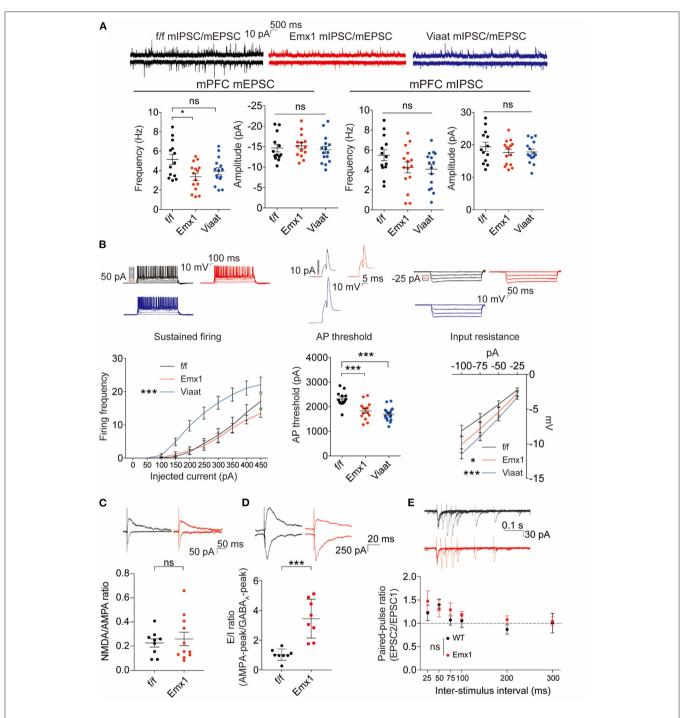


FIGURE 3 [*Emx1-Cre;Irsp53^{#/#}* and *Viaat-Cre;Irsp53^{#/#}* mice show distinct changes in synaptic transmission and intrinsic excitability in medial prefrontal cortex (mPFC) pyramidal neurons. **(A)** Miniature excitatory postsynaptic currents (mEPSCs) and miniature inhibitory postsynaptic currents (mIPSCs) in layer V pyramidal neurons in the prelimbic region of the mPFC in *Emx1-Cre;Irsp53^{#/#}* and *Viaat-Cre;Irsp53^{#/#}* mice (3 months; male). Note that the frequency of mEPSCs is significantly decreased in *Emx1-Cre;Irsp53^{#/#}* mice. *n* = 13 neurons from three mice for *t/f*-mEPSC, 14, 3 for Emx1-mEPSC, 15, 3 for Viaat-mEPSC, 13, 3 for *t/f*-mIPSC, 15, 3 for Emx1-mIPSC, and 15, 3 for Viaat-mIPSC, '*P* < 0.05, ns, not significant, one-way ANOVA with Bonferroni's test. mEPSC frequency, *F_(2, 39)* = 4.119; mEPSC amplitude, *F_(2, 39)* = 0.342; mIPSC frequency, *F_(2, 40)* = 2.012; mIPSC amplitude, *F_(2, 40)* = 0.7806. **(B)** Intrinsic excitability in layer V pyramidal neurons in the prelimbic region of the mPFC in *Emx1-Cre;Irsp53^{#/#}* and *Viaat-Cre;Irsp53^{#/#}* mice (3 weeks; male). Note that intrinsic excitability is increased both in *Emx1-Cre;Irsp53^{#/#}* and *Viaat-Cre;Irsp53^{#/#}* mice (3 weeks; male). Note that intrinsic excitability is for *Cre;Irsp53^{#/#}* and *Viaat-Cre;Irsp53^{#/#}* mice (3 weeks; male). Note that intrinsic excitability is increased both in *Emx1-Cre;Irsp53^{#/#}* and *Viaat-Cre;Irsp53^{#/#}* mice (3 weeks; male). Note that intrinsic excitability is increased both in *Emx1-Cre;Irsp53^{#/#}* and *Viaat-Cre;Irsp53^{#/#}* mice (3 weeks; male). Note that intrinsic excitability is noreased both in *Emx1-Cre;Irsp53^{#/#}* and *Viaat-Cre;Irsp53^{#/#}* mice (3 weeks; male). Note that intrinsic excitability is noreased both in *Emx1-Cre;Irsp53^{#/#}* and *Viaat-Cre;Irsp53^{#/#}* mice (3 weeks; male). Note that intrinsic excitability is noreased both in *Emx1-Cre;Irsp53^{#/#}* and *Viaat-Cre;Irsp53^{#/#}* mice (3 metra-1-firing frequency, 18, 3 for Viaat-input resistance, *i* = 13, 3 for *f/f*

FIGURE 3 (AMPAR)-EPSCs in Emx1- $Cre;Irsp53^{d/fl}$ layer V pyramidal neurons in the prelimbic region of the mPFC (2 months; male). n = 9 neurons for three mice for f/f, 11, 3 for Emx1, ns, not significant, Student's t-test, t = 0.2447, df = 18. (D) Increased ratio of evoked EPSCs and IPSCs in Emx1- $Cre;Irsp53^{d/fl}$ layer V pyramidal neurons in the prelimbic region of the mPFC (2 months; male). n = 8 neurons for three mice for f/f, 8, 3 for Emx1, ""P < 0.001, Student's t-test, t = 5.019, df = 14. (E) Normal paired-pulse ratio in Emx1- $Cre;Irsp53^{d/fl}$ layer V pyramidal neurons in the prelimbic region of the mPFC (2 months; male). n = 10 neurons for three mice for f/f, 9, 3 for Emx1, ns, not significant, two-way ANOVA with Bonferroni's test, interaction $F_{(5,85)} = 0.6379$, time $F_{(5,85)} = 4.100$, genotype $F_{(1,17)} = 0.7348$.

Publisher's Note: All claims expressed in this article are solely those of the authors and do not necessarily represent those of their affiliated organizations, or those of the publisher, the editors and the reviewers. Any product that may be evaluated in this article, or claim that may be made by its manufacturer, is not guaranteed or endorsed by the publisher.

Copyright © 2021 Kim, Noh, Kim, Yang, Kim and Kim. This is an open-access article distributed under the terms of the Creative Commons Attribution License (CC BY). The use, distribution or reproduction in other forums is permitted, provided the original author(s) and the copyright owner(s) are credited and that the original publication in this journal is cited, in accordance with accepted academic practice. No use, distribution or reproduction is permitted which does not comply with these terms.