

Correction

Correction: Singlár et al. Revealing the Specific Contributions of Mitochondrial CB₁ Receptors to the Overall Function of Skeletal Muscle in Mice. *Cells* 2025, 14, 1517

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Missing Citation

In the original publication [1], Ref. [2] was not cited. The citation has now been inserted in the Discussion, 2nd paragraph, and should read:

We replicated the genetic engineering strategy described by Hebert-Chatelain in 2016 [39], which is aimed at interfering with mitochondrial targeting by specifically disrupting only the mitochondrial pool of CB₁Rs while leaving plasma membrane CB₁Rs intact (Figure 1A). The strategy involves removing 63 nucleotides (21 amino acids) that have been identified as being responsible for the mitochondrial targeting of the receptor. The exact mutation was published in an elegant study by Soria-Gomez et al. in 2021 [58].

Text Correction

There was an error in the original publication. We incorrectly claimed that we investigated the first-ever global mitochondrial CB₁ knockout mouse model.

A correction has been made to the last paragraph of the Introduction:

In the current study we created a global mitochondrial CB₁ receptor mouse model. By using a combination of genetic and pharmacological approaches, we report a key role of mtCB₁Rs in skeletal muscle physiology. Our findings offer novel insights into the complex involvement of mtCB₁Rs in ECS-mediated regulation of skeletal muscle contraction, force generation, mitochondrial morphology, and energetics.

A correction has been made to the first paragraph of the Discussion:

In this work, employing a systemic mitochondrial CB₁ deletion murine model, we explored the multifaceted consequences of the suppression of mtCB₁ expression, primarily impacting muscle force, muscle mitochondrial morphology, respiration, membrane potential, as well as calcium levels.



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A correction has been made to the first paragraph of the Conclusion:

Our findings obtained on the newly generated systemic mtCB₁-KO mouse model align with the hypothesis that mitochondrial CB₁ receptors play a critical role in maintaining mitochondrial health and function, which in turn impacts overall muscle performance. Suppression of mtCB₁ expression leads to mitochondrial dysfunction at a very fundamental level, directly impacting energy production and, consequently, skeletal muscle performance.

References

Added Reference 58: Soria-Gomez, E.; Pagano Zottola, A.C.; Mariani, Y.; Desprez, T.; Barresi, M.; Río, I.B.-D.; Muguruza, C.; Bon-Jego, M.L.; Julio-Kalajzić, F.; Flynn, R. Subcellular specificity of cannabinoid effects in striatonigral circuits. *Neuron* **2021**, *9*, 1513–1526.e11. <https://doi.org/10.1016/j.neuron.2021.03.007>. With this correction, the order of some references has been adjusted accordingly.

The authors state that the scientific conclusions are unaffected. This correction was approved by the Academic Editor. The original publication has also been updated.

References

1. Singlár, Z.; Szentesi, P.; Ganbat, N.; Horváth, B.; Juhász, L.; Gönczi, M.; Keller-Pintér, A.; Oláh, A.; Máté, Z.; Erdélyi, F.; et al. Revealing the Specific Contributions of Mitochondrial CB₁ Receptors to the Overall Function of Skeletal Muscle in Mice. *Cells* **2025**, *14*, 1517. [[CrossRef](#)] [[PubMed](#)]
2. Soria-Gomez, E.; Pagano Zottola, A.C.; Mariani, Y.; Desprez, T.; Barresi, M.; Río, I.B.-D.; Muguruza, C.; Bon-Jego, M.L.; Julio-Kalajzić, F.; Flynn, R. Subcellular specificity of cannabinoid effects in striatonigral circuits. *Neuron* **2021**, *9*, 1513–1526.e11. [[CrossRef](#)] [[PubMed](#)]

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