

CORRECTION

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Correction to: Loss of homeostatic microglial phenotype in CSF1R-related Leukoencephalopathy

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Correction to: *Acta Neuropathol Commun* (2020) 8:72
<https://doi.org/10.1186/s40478-020-00947-0>

In the original publication of this article [1] the reference & citations of Chitu et al. 2020 [2] was unintentionally omitted. The original publication [1] has used data from the same tissue samples.

In this correction article the reference & citations have been published to rectify this omission.

Abstract

1. **Incorrect:** “We observed increased expression of CSF-2 in gray matter compared to affected white matter, which may contribute to selective vulnerability of white matter in HDLS”
2. **Correct:** “Previously we observed increased expression of CSF-2 in the gray matter but not the white matter of HDLS patients (Chitu et al. 2020) which may explain the selective vulnerability of white matter microglia in HDLS”.

Discussion

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The original article can be found online at <https://doi.org/10.1186/s40478-020-00947-0>.

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1. **Incorrect:** “The unique vulnerability of frontal white matter in HDLS may also be accounted for by differences in abundance of the two main microglial trophic factors between brain regions, with CSF-2 being significantly up-regulated in gray matter compared to white matter and vice versa for CSF-1 [26].”
2. **Correct:** “The unique vulnerability of frontal white matter in HDLS may also be accounted for by differences in abundance of microglial trophic factors between brain regions. Compared to healthy individuals, ALSP patients significantly up-regulate the expression of with CSF-2 in gray matter but not in the white matter (Chitu et al. 2020) and this may compensate for reduced signaling via the CSF-1R in microglia.”

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1. **Incorrect:** “Combined with developmental issues highlighted in *Csf1r*^{+/-} mice, where it has been suggested that aberrant energy metabolism may play a pathogenetic role, additional studies are warranted on these factors in HDLS [9].”
2. **Correct:** “Combined with developmental issues highlighted in *Csf1r*^{+/-} mice, where it has been suggested that aberrant energy metabolism may play a pathogenetic role, additional studies are warranted on these factors in HDLS (Chitu et al. 2020).”

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