



OPEN ACCESS

EDITED AND REVIEWED BY
Veronica De Rosa,
National Research Council (CNR), Italy

*CORRESPONDENCE

Motoko Unoki
✉ unokim@m.u-tokyo.ac.jp

RECEIVED 08 June 2024

ACCEPTED 17 June 2024

PUBLISHED 28 June 2024

CITATION

Unoki M (2024) Corrigendum: Exploring the intersection of epigenetics, DNA repair, and immunology from studies of ICF syndrome, an inborn error of immunity. *Front. Immunol.* 15:1445756. doi: 10.3389/fimmu.2024.1445756

COPYRIGHT

© 2024 Unoki. 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.

Corrigendum: Exploring the intersection of epigenetics, DNA repair, and immunology from studies of ICF syndrome, an inborn error of immunity

Motoko Unoki*

Department of Human Genetics, School of International Health, Graduate School of Medicine, The University of Tokyo, Tokyo, Japan

KEYWORDS

ICF syndrome, epigenetics, DNA methylation, DNA repair, NHEJ, class-switch recombination, hypoglobulinemia, immunodeficiency

A Corrigendum on

Exploring the intersection of epigenetics, DNA repair, and immunology from studies of ICF syndrome, an inborn error of immunity

By Unoki M (2024). *Front. Immunol.* 15:1405022. doi: 10.3389/fimmu.2024.1405022

In the published article, there was an error. The author accidentally overlooked an important recent work by Prof. Sébastien Storck's group (Université Paris Cité).

A correction has been made to the newly generated section, "Postscript", which has been inserted after "6 Conclusions" and before "Author Contributions".

The corrected sentence appears below:

"One recent excellent work by Cousu et al. (31) was accidentally overlooked in the main text. Using B-cell-specific *Hells* cKO mice, they found that HELLS plays a pivotal role in T-dependent B-cell responses; HELLS deficiency induces the accelerated decay of germinal center (GC) B cells and impairs the generation of high-affinity memory B cells and circulating IgGs. In addition, mutant GC B cells undergo dramatic DNA hypomethylation, leading to the premature upregulation of either memory B cell markers or the transcription factor ATF4, which drives an mTORC1-dependent metabolic program typical of plasma cells. Although CSR is unlikely affected by the absence of HELLS in the mice, as IgM and IgA levels are unchanged, their findings shed light on why IgG levels are frequently decreased in all types of ICF patients. I appreciate Prof. Sébastien Storck (Université Paris Cité) for notifying me of this oversight."

Missing Citation

Citation (31) has been added according to the addition of the "Postscript".

31. Cousu C, Mulot E, Smet AD, Formichetti S, Lecoecueche D, Ren J, et al. Germinal center output is sustained by HELLS-dependent DNA-methylation-maintenance in B cells. *Nat. Commun* (2023) 14:5695. doi: 10.1038/s41467-023-41317-3

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.

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.