

<https://doi.org/10.1038/s41467-020-15097-z>

OPEN

Author Correction: A phenotypic and genomics approach in a multi-ethnic cohort to subtype systemic lupus erythematosus

Cristina M. Lanata , Ishan Paranjpe, Joanne Nititham, Kimberly E. Taylor, Milena Gianfrancesco, Manish Paranjpe, Shan Andrews, Sharon A. Chung, Brooke Rhead, Lisa F. Barcellos, Laura Trupin, Patricia Katz, Maria Dall'Era, Jinoos Yazdany, Marina Sirota & Lindsey A. Criswell

Correction to: *Nature Communications* <https://doi.org/10.1038/s41467-019-11845-y>, published online 29 August 2019.

In the original version of this manuscript, in the discussion section in the seventh paragraph, the gene symbol for *PARP14* was incorrectly given as *PAR14* and incorrect citations of the literature were given. The incorrect version read ‘We would like to highlight variants in *HLA-F*, *PAR14* and *GAB2* controlled methylation sites in *USP35*. *HLA-F* is part of the nonclassical *HLA-Ib* genes, which are mono- or oligomorphic⁴⁶. Surface expression of *HLA-F* has been demonstrated on activated T, B and NK cells, and serum IgG autoantibodies against *HLA-F* have been detected in SLE patients and correlated with disease activity^{63–65}. *PARP14* encodes for poly (ADP-ribose) polymerase (PARP) protein family 14 and is involved in cellular maintenance and cell fate decisions, such as cell-cycle progression, metabolic pathways and ribosome biogenesis⁶⁶. Its role in SLE and autoimmune disease has not been defined but it has been shown to regulate glycolysis via IL-4 in B lymphocytes⁶⁷ and to promote survival of cancer cells^{67–69}.’

The correct version replaces these sentences with ‘We would like to highlight variants in *HLA-F*, *PARP14* and *GAB2* controlled methylation sites in *USP35*. *HLA-F* is part of the nonclassical *HLA-Ib* genes, which are mono- or oligomorphic⁴⁶. Surface expression of *HLA-F* has been demonstrated on activated T, B and NK cells, and serum IgG autoantibodies against *HLA-F* have been detected in SLE patients and correlated with disease activity^{63–65}. *PARP14* encodes for poly(ADP-ribose) polymerase (PARP) protein family 14 and assists in post-translational ribosylation modification of target proteins. Its role in SLE and autoimmune disease has not been defined but it has been shown to regulate glycolysis via IL-4 in B lymphocytes⁶⁶, promote survival of cancer cells⁶⁷, and regulate macrophage activation⁶⁸.’

Further, the original refs. ^{66–69} were replaced with the following corrected refs. ^{66–68} and all following references were renumbered.

All of these errors have now been corrected in the HTML and PDF versions of the article.

References

66. Cho, S. H. et al. Glycolytic rate and lymphomagenesis depend on PARP14, an ADP ribosyltransferase of the B aggressive lymphoma (BAL) family. *Proc. Natl. Acad. Sci. USA* **108**, 15972–15977 (2011).
67. Iansante, V. et al. PARP14 promotes the Warburg effect in hepatocellular carcinoma by inhibiting JNK1-dependent PKM2 phosphorylation and activation. *Nat. Commun.* **6**, 7882 (2015).
68. Iwata, H. et al. PARP9 and PARP14 cross-regulate macrophage activation via STAT1 ADP-ribosylation. *Nat. Commun.* **7**, 12849 (2016).

Published online: 27 February 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 license, and indicate if changes were made. The images or other third party material in this article are included in the article's Creative Commons license, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons license 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 license, visit <http://creativecommons.org/licenses/by/4.0/>.

© The Author(s) 2020