



Corrigendum: Lymphocyte Autophagy in Homeostasis, Activation, and Inflammatory Diseases

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Keywords: autophagy, mitophagy, metabolism, unfolded protein response, autoimmunity, lymphocytes

A Corrigendum on

Lymphocyte Autophagy in Homeostasis, Activation, and Inflammatory Diseases

by Arbogast, F., and Gros, F. (2018). *Front. Immunol.* 9:1801. doi: 10.3389/fimmu.2018.01801

In the original article, two clarifications about cited references are necessary.

First, the sentence “As a consequence, autophagy-deficient T cells show impaired TH9 differentiation and antitumor responses (63)” should be “As a consequence, autophagy-deficient T cells show enhanced TH9-dependent anti-tumor responses (63)”. A correction has been made to the section *Autophagy in Peripheral T Cells, Macroautophagy in T Cell Activation*, paragraph 2.

Moreover, even if mechanisms are not totally understood, Chen et al. indeed found experimental evidence in (42), for a role played by autophagy in limiting lipid peroxidation toxicity induced by reactive oxygen species. The sentence “To date, no mechanism linking autophagy and memory B cell survival has been proposed. It is possible that mitophagy and mobilization of lipids through lipophagy might be important, as for T cells” has been corrected to “Chen et al. (42) showed that autophagy in memory B cells limits mitochondrial ROS production and toxicity of peroxidized lipids. It is also possible that mobilization of lipids through lipophagy might be required for the survival of both memory B and T cells”. A correction has been made to the section *Autophagy in peripheral B Cells, Macroautophagy in Memory B Cell and Plasma Cell Survival*, paragraph 2.

The authors apologize for these errors and state that they do not change the scientific conclusions of the article in any way. The original article has been updated.

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Edited and reviewed by:

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Specialty section:

This article was submitted to
Immunological Tolerance and
Regulation,
a section of the journal
Frontiers in Immunology

Received: 24 August 2018

Accepted: 25 October 2018

Published: 16 November 2018

Citation:

Arbogast F and Gros F (2018)
Corrigendum: Lymphocyte Autophagy
in Homeostasis, Activation, and
Inflammatory Diseases.
Front. Immunol. 9:2627.
doi: 10.3389/fimmu.2018.02627

REFERENCES

42. Chen M, Hong MJ, Sun H, Wang L, Shi X, Gilbert BE, et al. Essential role for autophagy in the maintenance of immunological memory against influenza infection. *Nat Med.* (2014) 20:503–10. doi: 10.1038/nm.3521
63. Rivera Vargas T, Cai Z, Shen Y, Dosset M, Benoit-Lizon I, Martin T, et al. Selective degradation of PU.1 during autophagy represses the differentiation and antitumor activity of TH9 cells. *Nat Commun.* (2017) 8:559. doi: 10.1038/s41467-017-00468-w

Conflict of Interest Statement: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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