

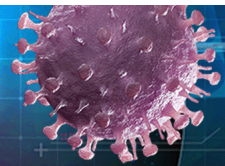
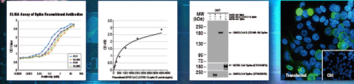


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## Correction: Transcription Factors Directing Th2 Differentiation: Gata-3 Plays a Dominant Role

This information is current as  
of October 30, 2020.

Anne O'Garra and Leona Gabrysová

*J Immunol* 2016; 197:4504; ;

doi: 10.4049/jimmunol.1601671

<http://www.jimmunol.org/content/197/11/4504>

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## Corrections

O'Garra, A., and L. Gabryšová. 2016. Transcription factors directing Th2 differentiation: Gata-3 plays a dominant role. *J. Immunol.* 196: 4423–4425.

It has been brought to our attention by individuals involved in these studies that the timing of the work was not entirely accurate as described. To address this issue, the authors would like to update the first two sentences of the second paragraph on page 4424 as follows:

Concurrent and subsequent studies using alternate technologies to those of the *Pillars of Immunology* article by Zheng and Flavell (15) clearly support the premise of this paper, confirming that GATA-3 is the key transcription factor regulating the differentiation and maintenance of Th2 cytokine production. A separate study by Ray et al. was also published in 1997, and showed that the transcription factor GATA-3 was upregulated in murine Th2 cells (while repressed in Th1 cells) and controlled Th2-specific expression of the *IL5* gene, independently and concurrently demonstrating the importance of this transcription factor in Th2 differentiation (21).

For clarity, Refs. 15 and 21 from the original article are included below.

This text has been corrected in the online version of the article, which now differs from the print version as originally published.

## References

15. Zheng, W., and R. A. Flavell. 1997. The transcription factor GATA-3 is necessary and sufficient for Th2 cytokine gene expression in CD4 T cells. *Cell* 89: 587–596.
21. Zhang, D. H., L. Cohn, P. Ray, K. Bottomly, and A. Ray. 1997. Transcription factor GATA-3 is differentially expressed in murine Th1 and Th2 cells and controls Th2-specific expression of the interleukin-5 gene. *J. Biol. Chem.* 272: 21597–21603.

[www.jimmunol.org/cgi/doi/10.4049/jimmunol.1601671](http://www.jimmunol.org/cgi/doi/10.4049/jimmunol.1601671)