

Corrigendum

Corrigendum to “Autoimmune Hepatitis: Progress from Global Immunosuppression to Personalised Regulatory T Cell Therapy”

Nwe Ni Than,^{1,2} Hannah C. Jeffery,¹ and Ye H. Oo^{1,2}

¹Centre for Liver Research and NIHR Liver BRU, University of Birmingham, Edgbaston, Birmingham B15 2TT, UK

²Liver and Hepatobiliary Unit, University Hospital Birmingham National Health Service Foundation Trust, Birmingham B15 2WB, UK

Correspondence should be addressed to Ye H. Oo; y.h.oo@bham.ac.uk

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In the article titled “Autoimmune Hepatitis: Progress from Global Immunosuppression to Personalised Regulatory T Cell Therapy” [1], the word “adenosis” is misspelled in the legend of Figure 1 and should be corrected as “adenosine.” The figure’s legend is corrected as follows.

References

- [1] N. N. Than, H. C. Jeffery, and Y. H. Oo, “Autoimmune hepatitis: progress from global immunosuppression to personalised regulatory T cell therapy,” *Canadian Journal of Gastroenterology and Hepatology*, vol. 2016, Article ID 7181685, 12 pages, 2016.

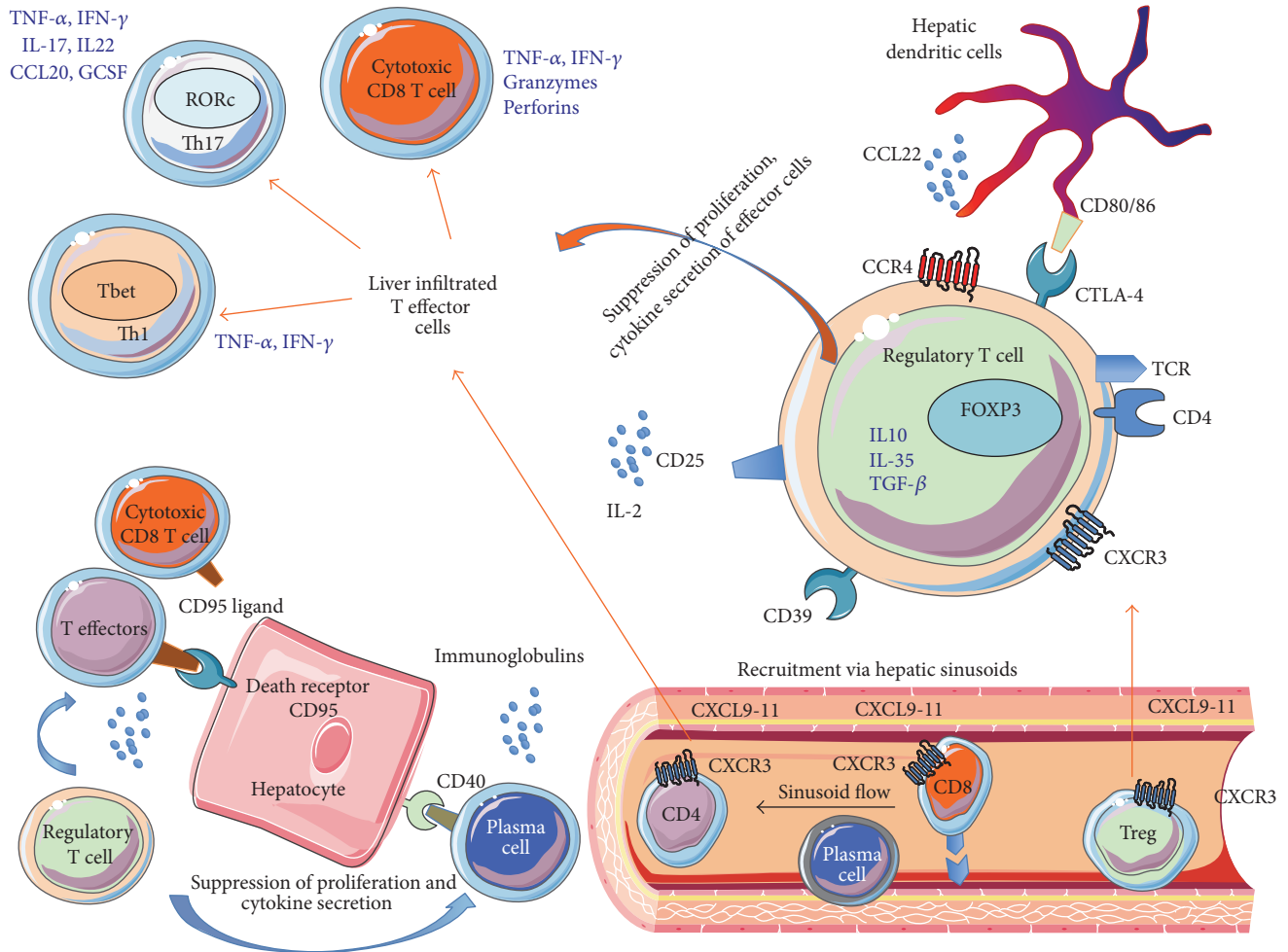


FIGURE 1: Pathogenesis of autoimmune hepatitis. Both effector T cells (CD4, CD8) and regulatory T cells (Treg) are recruited to inflamed autoimmune hepatitis liver via hepatic sinusoids. T effector cells lead to apoptosis of hepatocytes via CD95 ligand (dead ligand) expressed on them, which binds to CD95 on the hepatocytes. This killing action of T effector cells is regulated by regulatory T cells, which suppress proliferation and cytokine secretion of effector T cells. Plasma cells are also involved in immune-pathogenesis and they secrete immunoglobulin. Liver infiltrated T effector cells consist of Th17, Th1, and cytotoxic T cells. Th1 cells have T bet transcription factor; Th17 cells have transcription factor RORc. Cytotoxic T cells secrete IFN, TNF, granzyme, and perforins. Regulatory T cells (Treg = CD4CD25^{high}CD127^{low}) express liver tissue homing chemokine receptor CXCR3, which binds to its ligands CXCL9-11 expressed on inflamed hepatic sinusoid, hepatocytes, and bile ducts. Treg also expresses its functional markers CTLA4 (interacting with CD80/CD86 on dendritic cells). Dendritic cells secrete chemokine CCL22, which binds to chemokine receptor CCR4 on the regulatory T cells. CD39 on the Treg can generate immunosuppressive adenosine from ATP in the hepatic microenvironment. IL-2, which acts on its receptor CD25, is crucial for intrahepatic Treg survival and function.



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