Corrigendum

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

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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

**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 (death 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 Th1, Th17, and cytotoxic T cells. Th17 cells have Tbet transcription factor; Th1 cells have RORc transcription factor. Cytotoxic T cells secrete IFN, TNF, granzyme, and perforins. Regulatory T cells (Treg = CD4CD25highCD127low) 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.