

Errata

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High levels of gut-homing immunoglobulin A⁺ B lymphocytes support the pathogenic role of intestinal mucosal hyperresponsiveness in immunoglobulin A nephropathy patients, *Nephrol Dial Transplant* 2020; gfaa264. doi: 10.1093/ndt/gfaa264

In the above article, the graphical abstract has been updated as follows online:

High levels of gut-homing IgA⁺ B lymphocytes support the pathogenic role of intestinal mucosal hyperresponsiveness in IgA nephropathy patients

Blood samples collected from:

- 44 IgAN patients
- 23 healthy subjects
- 8 non-IgA glomerulonephritis controls

Increased level of circulating gut-homing Bregs, memory B cells and IgA⁺ memory B cells in IgAN patients

Increased serum levels of BAFF and APRIL cytokines in IgAN patients

Correlation with higher amounts of 5 specific microbiota metabolites

Intestinal mucosal hyperresponsiveness

The diagram illustrates the interaction between the gut microbiome and the immune system. It shows the intestinal epithelium, Peyer's patches, and the lamina propria. Key components include:

- Microbes and dietary antigens entering the gut.
- Antigen presentation to T cells (Treg, Th17, Th1, Th22, Th17/22, Th17/17, Th17/17/22, Th17/17/22/22).
- Secretory IgA production.
- Interaction with B cells (naïve B cell, T cell-dependent CSR, T cell-independent CSR, IgA⁺ plasma cell).
- Production of IgA⁺ memory B cells.
- Release of cytokines like APRIL and BAFF.

Conclusions: The results of this study show for the first time a significant difference in the amount of intestinal-activated B lymphocytes in IgAN patients, confirming the hypothesis of the pathogenic role of intestinal mucosal hyperresponsiveness in the IgAN

Sallustio, F., et al. *NDT* (2020)
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