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Analysis of IgA+ B-cells, IgA, and Gd-IgA1 in Monozygotic Twins Discordant for IgA Nephropathy

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Objectives : IgA nephropathy (IgAN), a common form of glomerulonephritis, is caused by deposition of IgA1 immune complexes in the glomerulus. Many IgAN patients have elevated IgA1 and galactose deficient IgA1 (Gd-IgA1) in the serum, and blood relatives have elevated Gd-IgA1 in the serum, compared to healthy individuals.
Methods : We studied five sets of discordant monozygotic twins for IgA nephropathy. IgA+ B-cells were analyzed by flow cytometry, IgA and Gd-IgA1 were analyzed by ELISA, and cell co-culture experiments were performed.
Results : Using flow cytometry, the number and the percentage of IgA antibody secreting cells were evaluated in monozygotic twins discordant for IgA nephropathy. No significant differences were observed between the IgAN-twins and the healthy-twins. Analysis of IgA and Gd-IgA1 demonstrated no differences in the serum concentrations of IgA and Gd-IgA1, in concordance with the published literature reporting elevated Gd-IgA1 in the serum of blood relatives. As expected, the Gd-IgA1 serum concentrations from the IgAN-twins were significantly elevated compared to healthy individuals, however no significant differences were observed between the healthy twins and healthy individuals. To determine whether there is preferential IgA production in IgAN, autologous circulating T-follicular like helper cell – naïve B-cell co-cultures were performed on cells derived from the discordant monozygotic twins. We detected significantly more IgA production in the autologous co-cultures of cells from the IgAN-twins compared to co-cultures of the healthy-twins.
Conclusions : Studying monozygotic twins discordant for IgAN presents a unique opportunity to dissect the B and T cells, and IgA and Gd-IgA1 while controlling for genetic background. Our data demonstrates an essential interaction of naïve B-cells and T-follicular like helper cells in the increased generation of IgA in IgAN, and the need for additional molecular studies to understand the IgA1 and Gd-IgA1 hyperresponsiveness in IgAN.