Shaping Tomorrow's Nephrology: Insight-Driven Kidney Care
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Misplaced Immunity: the Role of Defective B-Cell Homing in IgAN Pathogenesis

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Title: Misplaced Immunity: The Role of Defective B-Cell Homing in IgAN Pathogenesis Abstract: IgA nephropathy (IgAN) is a complex immune-mediated glomerular disease and the most common form of glomerulonephritis that leads to end stage kidney disease in about 20 to 40% of patients. IgA1 molecules play a key role in the pathogenesis of IgAN, and serum IgA1 and galactose deficient IgA1 (Gd-IgA1) levels are elevated in IgAN patients. IgAN patients have a hyper-responsiveness limited to the IgA1 subclass to viral and bacterial mucosal challenges that are absent in non-mucosal infections. This has been attributed to genetic predisposition, coupled with environmental factors upon an immunological challenge. However, the mechanisms for the elevated IgA1 and Gd-IgA1 in the circulation of IgAN patients are poorly understood. The immune system is complex, composed of many cells including B-cells, that play a critical role in tissue homeostasis including mucosal sites such as the gastrointestinal tract and lungs. Genome wide association studies have identified common genetic variants that have connected both the innate and adaptive immune response and mucosal immunity, for example human genetic studies have identified variants, including GALNT14, involved in O-glycosylation. We have demonstrated dysregulated B-cell homing in mouse models null for Galnt14. Other studies have demonstrated mislocalized mucosal immune cells in the circulation of IgAN patients. Together, these studies implicate abnormal B-cell homing as one of the pathogenic mechanisms in IgAN. To better understand the mechanisms of defective B-cell homing in IgAN pathogenesis, better insights into the biology of the IgA B-cells is required, and this can be achieved by combining genetics, genomic and immunological approaches.

Keywords: IgA Nephropathy, B-cells, homing