OFFICIAL ABSTRACT and CERTIFICATION

Identifying Functional Disease Drivers in Lupus Nephritis Associated with Glomerular Remodeling

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Lupus Nephritis (LN) is an autoimmune disease that affects the healthy tissue of the kidney, leading to chronic inflammation and excessive leakage of protein in the urine (proteinuria). There is no cure; instead, current methods of treatment involve management of symptoms and slowing the progression toward renal failure. Although the genes linked to LN have long been been identified, limited research has highlighted which specific genes may be driving the physical changes caused by this disease.

This research used provided tissue samples from NZB/W female mice, a confirmed LN model, which had been previously sacrificed at 36 weeks at the first indication of excess proteinuria. The purpose of this study was to identify the specific morphological changes within the glomerulus that led to protein leakage, and then to use qPCR to identify the specific genes that are driving this conformational change.

Histochemistry with Trichrome staining revealed, for the first time, a significant expansion of the Bowman's capsule within the LN group which may correlate with excessive protein leakage. In addition, by correlating qPCR expression results with the increase in Bowman's space, this novel research revealed that ROCK2, ICAM, cMET, TGFβ, acox, and Col1 may be disease drivers for LN.

Recent research has focused on understanding how disease progression differs from person to person. By having a better understanding of the underlying mechanisms of LN, we can create precision medication tailored to each patient's genotype, thereby increasing the effectiveness of treatment.

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