UAB IgAN Research Team

UAB’s inter-disciplinary and collaborative approach has paved the way for improving outcomes for IgAN patients. UAB is the recognized leader in the field of IgAN research.

Bruce A. Julian, MD. Clinical Nephrologist who has been treating patients with IgAN and investigating the cause of their kidney disease for more than 30 years.

Dana Rizk, MD. Clinical Nephrologist who focuses on monitoring outcomes of patients with IgAN.

Jan Novak, PhD. Molecular Immunologist who clarified the autoimmune nature of IgAN and developed a variety of disease-specific assays.

Matthew B. Renfrow, PhD. Analytical Biochemist who specializes in the analysis of IgA sugars and other components of the disease.

You Can Make a Difference

Philanthropy is key to changing the outcomes for patients with IgAN. UAB is currently working with private foundations and other universities to find ways to treat this disease.

“We are confident that further research will lead us to potentially life-saving treatments,” reports Dr. Bruce A. Julian. “We have already developed a blueprint for this disease that is widely accepted by the scientific community, and we have a clear path in front of us. We hope that we can find partners to join us and accelerate our research.”

To discuss the many options for supporting the IgA Nephropathy Research Program at UAB, please contact:

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With your help, we can make a difference.
What Is IgA Nephropathy?

IgA nephropathy (IgAN) is a renal disease that affects how blood is filtered in the small blood vessels of the kidneys. IgAN occurs when an abnormal protein damages the filtering unit (glomerulus) inside the kidneys. It is one of the most common kidney diseases in the world.

It is estimated that 20-40% of the people who have IgAN will develop end-stage kidney disease, which means they will need dialysis or kidney transplantation to survive.

Detection & Treatment

Although IgAN is a serious health threat, the symptoms of this disease often go unrecognized. Blood and protein in the urine are common, and many patients have high blood pressure.

Currently, the only way to confirm the diagnosis of IgAN is to perform an invasive procedure—a biopsy of a kidney. It is also very difficult to monitor the progression of the disease, because there is no easy method of assessing the ongoing damage. Treatment normally focuses on controlling blood pressure and reducing the amount of protein in the urine.

A closer look at kidney structure

A simplified illustration of the glomerulus, the filtering unit inside a kidney. The mesangium is the structure (shown in yellow) inside the glomerulus between the small blood vessels (shown in pink) where the IgA deposits (red stars) accumulate.

Next Steps

These seminal discoveries have led UAB’s researchers to develop a minimally invasive means (blood test) to diagnose and possibly monitor the progression of IgAN. Standardization of these tests will have a great impact on the disease. These tests will help define targets for treatment.

UAB Seminal Discoveries

As IgAN is often a precursor to life-threatening kidney failure, UAB researchers have paved the way in understanding the mechanisms of the disease. They discovered that IgAN is actually an autoimmune disease that damages the kidneys as innocent bystanders.

The body’s immune system normally produces an antibody known as immunoglobulin A1 (IgA1). This protein has a special chemical makeup that includes attached sugar molecules. When the sugar content of IgA1 is altered, other antibodies bind to it to form immune complexes that deposit in the mesangium of the glomerular units of the kidneys. Over time, these deposits cause kidney damage.