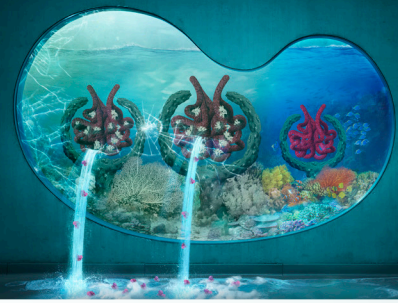


IgA NEPHROPATHY (IgAN) EXPLAINED



EPIDEMIOLOGY



IgAN is the most prevalent primary glomerulonephritis worldwide¹

- Mean age at **diagnosis is 41**²
- 2.5/100,000** people affected globally per year³
- Detected in **19–51%** of kidney biopsies in Europe⁴



A kidney biopsy is required for confirmation of IgAN diagnosis¹

Risk factors include:⁵⁻⁸



Genetic



Environmental



Lifestyle factors

DISEASE PROGRESSION

IgAN is a long-term, progressive disease with a relentless clinical course^{2,9}



The median age of kidney failure or death is 48²



Over a median follow-up of 9.5 years, 51% of adult patients (N=2168) progressed to ESKD^{10*}



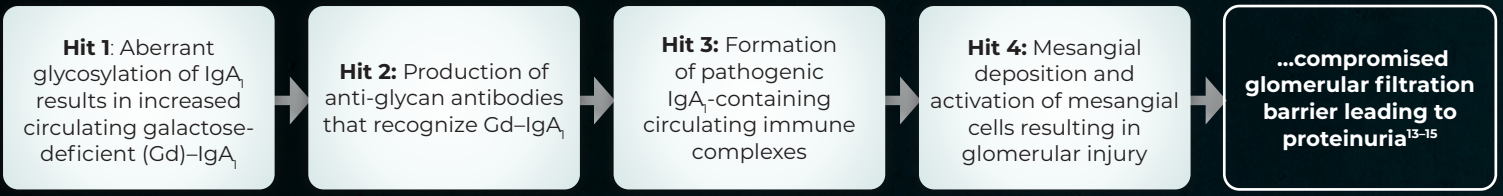
At diagnosis, 2/3 of patients are already at CKD stage ≥ 3 ^{2,11}



Sustained proteinuria of >1g/day is the strongest modifiable predictor of the rate of progression in IgAN¹²

PATHOPHYSIOLOGY

There are 4 Hits involved in the pathogenesis of IgAN:⁵



This deposition increases the production of **endothelin-1 (ET-1)** and **angiotensin II (ANG II)**, which act in tandem to amplify inflammation and damage to the glomerular filtration barrier and tubulointerstitial compartment, leading to increased proteinuria and ultimately kidney failure¹⁶⁻¹⁸

TREATMENT TARGETS

According to the KDIGO guidelines, the reduction of proteinuria is a key treatment target in IgAN:¹



However, with current supportive care **63%** of patients[†] (N=96) **DO NOT reach the KDIGO recommended target** with treatment¹⁹

There are limited treatment options indicated for IgAN, such as generically used renin-angiotensin-aldosterone system (RAAS) inhibition and glucocorticoids, which leaves patients at high risk of progression^{19†}

THERE IS A HIGH CLINICAL UNMET NEED FOR DISEASE COURSE MODIFYING TREATMENTS THAT PRESERVE KIDNEY FUNCTION FOR PATIENTS WITH IgAN

Visit targetproteinuria.com for more information

*UK-based study of biopsy-verified patients with IgAN (N=2,168).¹⁰ †Prospective study of patients with biopsy-proven IgAN (urinary protein ≥ 1 g/day). 63.5% of patients treated with an ACEi or ARB did not achieve remission after 3 months of treatment. Partial remission (defined as <1g/day and $\geq 50\%$ decrease from baseline with stable kidney function [$\leq 25\%$ reduction in eGFR]) of proteinuria was seen in 29 patients (30.2%) after 3 months of treatment.¹⁹ ‡The KDIGO Guidelines define high risk of progression in IgAN as proteinuria >0.75–1g/day, despite at least 90 days of optimized supportive care¹

ACEi, angiotensin-converting-enzyme inhibitor; ANG II, angiotensin II; ARB, angiotensin receptor blocker; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; ESKD, end-stage kidney disease; ET-1, endothelin-1; Gd, galactose-deficient; IgA, immunoglobulin A; IgA₁, immunoglobulin A subclass 1; IgAN, IgA nephropathy; KDIGO, Kidney Disease: Improving Global Outcomes; RAAS, renin-angiotensin-aldosterone system

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