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Treatment of immunoglobulin A nephropathy with targeted-release budesonide (Kinpeygo®▼)

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Treatment of immunoglobulin A nephropathy with targeted-release budesonide (Kinpeygo®▼)

This article explores the evolving treatment of IgA nephropathy, highlighting targeted-release budesonide as the first licensed therapy to reduce proteinuria and slow disease progression when combined with optimised supportive care.

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■ glomerular disease ■ IgA nephropathy ■ proteinuria ■ target-release budesonide

The treatment landscape for the world's most common primary glomerular disease, immunoglobulin A nephropathy (IgAN), is rapidly evolving. The clinical presentation of IgAN can be highly variable, ranging from patients with minor urinary abnormalities to those with advanced chronic kidney disease requiring renal replacement therapy shortly after diagnosis. Patients are typically diagnosed at the age of 30–50 years and their significant lifetime risk of kidney failure alongside its socioeconomic impact has driven a search for ways to alter disease outcomes (Rodrigues et al, 2017).

Previous large trials such as TESTING (therapeutic evaluation of steroids in IgAN global; Wong et al, 2021) and STOP-IgAN (supportive versus immunosuppressive therapy of progressive IgAN; Rauen et al, 2020) demonstrated that systemic corticosteroids can reduce proteinuria in IgAN, but these benefits were offset by a substantial increase in serious adverse events, including infections and metabolic derangements. This risk–benefit profile has driven the search for safer, targeted therapies. The development of targeted-release budesonide (Kinpeygo®), the first disease-modifying therapy for IgAN, has led to a new treatment paradigm, changing the approach to the care of this patient population.

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Immunoglobulin A nephropathy (IgAN)

Normal function of kidneys and IgA

The nephrology community is familiar with the wide spectrum of IgAN presentations, which range from isolated microscopic haematuria noted incidentally to rapidly progressive glomerulonephritis. Subclinical disease may persist for many years, underscoring the importance of vigilant monitoring, allowing early intervention should deterioration occur.

Pathophysiology

IgA plays a key role in defending mucosal surfaces against bacteria (De Sousa-Pereira and Woof, 2019), and originates from antibody-secreting cells located either within the mucosal-associated lymphoid tissue, or from bone marrow resident cells that are of mucosal origin (Reich and Makita, 2024). In

addition to being the most prevalent antibody class at mucosal sites it is present at significant serum concentrations. The histopathological hallmark of IgAN is of IgA dominant or co-dominant mesangial immune-complex deposition within the glomerulus (Suzuki et al, 2011).

The 'multi-hit' hypothesis of IgAN pathophysiology first proposed by Suzuki et al (2011) forms the basis of the current understanding of the disease's development and informs therapeutic targets (Rodrigues et al, 2017; Gleeson et al, 2023). The first hit is the excess production of galactose deficient (Gd) IgA1. The second hit is the production of anti-Gd-IgA1 autoantibodies. IgA1 complexes are formed in hit three, followed by the deposition of these complexes in the mesangium in hit four. This triggers an inflammatory cascade which leads to progressive kidney damage.

Prevalence and risk factors

Although recognised as the most common primary glomerular disease, there are challenges in estimating the true prevalence of IgAN given the varying thresholds for kidney biopsy. The prevalence of IgAN is highest in East and South-East Asia, intermediate in Europe and lowest in individuals of Black African descent (Willey et al, 2023). Across Europe, annual incidence is estimated to be 0.76 per 100 000 with a point prevalence of 2.53 per 10 000 (Willey et al, 2023), although some authors suggest that the incidence is as high as 2.5 per 100 000 (Rodrigues et al, 2017). IgAN prevalence is highest in north-eastern Europe and lowest in southern Europe (Willey et al, 2023).

Susceptibility to IgAN and the risk of disease progression is thought to be influenced by a combination of genetic and environmental factors (Rodrigues et al, 2017). Environmental factors such as recurrent mucosal infections, dietary antigens and possibly alterations in the gut microbiome have been implicated in the pathogenesis and progression of IgAN (Rodrigues et al, 2017).

Disease prognosis

IgAN may progress slowly, and the lifetime risk of renal replacement therapy for those with relatively low-grade proteinuria has only recently been fully appreciated. A large population-based cohort study (Pitcher et al, 2023) has shown that the median time from diagnosis to kidney failure was 11.4 years for those with an eGFR <60 ml/min and proteinuria >0.5 g/day. In this analysis, the mean age of patients reaching the primary endpoint of kidney failure or death was 48 years, and most patients progressed to kidney failure within 10–15 years (Pitcher et al, 2023). The study found that 30% of patients with time-averaged proteinuria of only 0.44–0.88 g/g (approximately 50–100 mg/mmol) and around 20% of patients with time-averaged proteinuria of <0.44 g/g (<50 mg/mmol) developed kidney failure within 10 years (Pitcher et al, 2023). These findings challenged the earlier view that prognosis was benign if proteinuria level of <1 g/day occurred.

Diagnosis

Signs and symptoms

Patients may present with hypertension associated with an abnormal urinalysis, sympharyngitic haematuria or nephrotic syndrome, but similarly to many forms of kidney disease, patients may not present until they develop symptoms associated with advanced kidney disease. A reduction in kidney function, non-visible haematuria or proteinuria may be found incidentally but should prompt further evaluation.

Diagnosis via biopsy

There are no validated diagnostic serum or urine biomarkers for IgAN; a kidney biopsy is required for diagnosis (Devuyst et al, 2025; Floege et al, 2025). The KDIGO guidelines encourage a lower proteinuria threshold for biopsy if IgAN is suspected (proteinuria 0.5 g/day or equivalent) (Devuyst et al, 2025). The aim of this recommendation is to ensure early diagnosis and prompt treatment of IgAN. Reporting should include mesangial (M) and endocapillary (E) hypercellularity, segmental sclerosis (S), interstitial fibrosis/tubular atrophy (T) and crescents (C) (MEST-C) scores (Trimarchi et al, 2017), and that secondary causes of IgAN should be excluded.

Assessing prognosis

As with diagnosis, there are no novel validated biomarkers for IgAN prognosis; this is predicted from eGFR, proteinuria and histological findings. MEST-C scores can be used to predict outcomes regarding kidney damage (Barbour et al, 2016). Clinicians can use the IgAN prediction tool to aid with prognosis: https://qxmd.com/calculate/calculator_499/international-igan-prediction-tool-at-biopsy-adults

However, the KDIGO guideline highlights that the MEST-C score has not been evaluated as a means of determining the likely impact of any particular treatment regimen and, at present, should not be used to decide on a specific therapy (Devuyst et al, 2025).

Treatment paradigm

The treatment goal in patients with IgAN at risk of progressive loss of kidney function is to reduce the rate of loss of function to normal age-related change (i.e. <1 ml/min/year). The KDIGO guideline emphasises the importance of proteinuria in

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guiding clinical decision making. This should be maintained at a minimum of <0.5 g/day (or equivalent) and ideally at <0.3 g/day (Devuyst et al, 2025).

Managing IgAN-induced nephron loss

A supportive management approach has historically been the mainstay of IgAN treatment. The supportive management approach focuses on the reduction of glomerular hyperfiltration and the impact it has on proteinuria reduction through the use of renin-angiotensin system (RAS) blockade or the use of a novel dual endothelin angiotensin receptor antagonism (DEARA) sparsentan (Rovin et al, 2023), which was approved for use in the UK in June 2025 (NICE, 2025). The KDIGO guideline (Devuyst et al, 2025) also suggests that patients who are at risk of progressive loss of kidney function with IgAN should be treated with a sodium–glucose co-transporter 2 inhibitor (SGLT2i) based on subgroup analysis of the DAPA-CKD trial (dapagliflozin and prevention of adverse outcomes in chronic kidney disease; Heerspink et al, 2020, Wheeler et al, 2021). Controlling blood pressure is also key, with a target proposed by KDIGO of ≤120/70 mmHg (Devuyst et al, 2025). The supportive approach also includes lifestyle advice such as dietary sodium restriction (<2 g/day), smoking and vaping cessation, weight control and endurance exercise as appropriate.

The KDIGO guideline has proposed a radical change to the current treatment paradigm (Devuyst et al, 2025). The major new concept is a dual and simultaneous focus to IgAN treatment that aims to firstly prevent or reduce IgA-containing immune complex formation and associated glomerular injury while also managing the consequences of existing IgAN-induced nephron loss.

Targeted-release budesonide

Disease-modifying mode of action

A targeted-release formulation of budesonide is designed to release the active compound in the distal part of the ileum and proximal part of the colon, where Peyer's patches are located (Smerud et al, 2011). It was hypothesised that the targeted release would exert its effects by local immunosuppression and reducing the production of Gd-IgA1, hence targeting the initiating event that leads to the development of IgAN (Smerud et al, 2011). A randomised clinical trial (Wimbury et al, 2024) showed that the drug significantly reduced levels of both serum Gd-IgA1 and IgA-/IgG-immune complexes, supporting the hypothesis of the compound may lead to a disease-modifying action in IgAN.

Evidence base

The recommended dose for targeted-release budesonide is 16 mg once daily, for an initial duration of 9 months (European Medicines Agency, 2024). When discontinuing treatment, the dose should be reduced to 8 mg once daily for 2 weeks and may be reduced to 4 mg once daily for an additional 2 weeks, at the discretion of the treating physician (European Medicines Agency, 2024). The pivotal phase three NefIgArd study (targeted-release formulation of budesonide) in patients with primary IgA nephropathy) showed that a 9-month treatment period provided a clinically-relevant reduction in eGFR decline and a durable reduction in proteinuria compared with placebo (Lafayette et al, 2023). Patients receiving renin-angiotensin system (RAS) blockade with proteinuria greater than 1 g/day and an eGFR of 35–90 mL/min/1.73 m^2 were randomised to 16 mg/day targeted-release budesonide or placebo for 9 months. Following 9 months of treatment, targeted-release budesonide treatment resulted in a 31% reduction in proteinuria compared with 5% in the placebo group. The longer-term follow up data (Lafayette et al, 2023) showed that the time-weighted average of eGFR over 2 years demonstrated a statistically significant treatment benefit with budesonide versus placebo, with a difference of around 5 mL/min. However, there was still a decline in GFR in the budesonide-treated group that paralleled the placebo group, with a rise in proteinuria after the 9-month treatment course finished.

Adverse events were consistent with those expected for exposure to a low glucocorticoid dose (approximately 8 mg equivalent of prednisolone), including peripheral oedema, hypertension, acne and headache (each occurring in 10–17% of participants). There was no increase in mortality or serious infectious events, potentially showing an important safety signal compared to systemic corticosteroids. Of note, only 5% of patients in the budesonide arm were receiving an SGLT2i, which is now considered part of standard nephro-protection.

Clinical and cost efficacy

The National Institute for Health and Care Excellence (NICE, 2026) has recommended targeted-release budesonide as an option for treating primary IgAN when there is a risk of rapid disease progression in adults with a urine protein-to-creatinine ratio of 90 mg/mmol or more. Targeted-release budesonide was recommended as an add-on to optimised standard care, including the highest tolerated licensed dose of angiotensin-converting enzyme (ACE) inhibitors or angiotensin-receptor blockers (ARBs), unless these are contraindicated.

This recommendation was based on the clinical trial evidence that targeted-release budesonide plus standard care is more effective than standard care alone (Lafayette et al, 2023). Targeted-release budesonide is the first licensed treatment that specifically treats IgAN, increasing the likelihood that people may avoid or delay the need for dialysis or a kidney transplant. NICE (2026) considered the cost-effectiveness estimate for targeted-release budesonide to be within an acceptable range for the NHS, resulting in its recommendation for use.

Patient selection

Indications and eligibility

Patients with a urine protein-to-creatinine ratio of 90 mg/mmol or more are eligible to receive targeted-release budesonide, as this is the group at the highest risk of rapid disease progression (NICE, 2026). Alongside the proteinuria threshold for treatment, patients should receive optimised standard care, including the highest tolerated licensed dose of ACE inhibitors or ARBs and SGLT2i, unless these are contraindicated (NICE, 2026). This recommendation was based on the clinical trial evidence that targeted-release budesonide plus standard care is more effective than standard care alone (Lafayette et al, 2023).

Contraindications and cautions

As per the summary of product characteristics (Electronic Medicines Compendium, 2025), the safety and efficacy of targeted-release budesonide has not been studied in patients with hepatic impairment; it is therefore contraindicated in patients with severe hepatic impairment (Child-Pugh class C). It is also contraindicated in patients who have hypersensitivity to the active substance or to any of the excipients (Electronic Medicines Compendium, 2025).

General advice concerning long-term steroid treatment should be provided, including the risk of adrenal suppression and need for additional steroid at times of stress. Patients should also be advised of their increased susceptibility to infections. Concomitant treatment with potent CYP3A4 inhibitors, including ketoconazole and cobicistat containing products, is expected to increase the risk of systemic side effects attributable to targeted-release budesonide (Electronic Medicines Compendium, 2025).

Case studies

This case series presents two adults with immunoglobulin A nephropathy (IgAN) treated with targeted-release budesonide at Cambridge University Hospitals between 2024 and 2025. The cases illustrate the variability of clinical response, particularly in relation to proteinuria reduction and estimated glomerular filtration rate (eGFR) trajectory. Each case highlights the importance of optimising supportive care, addressing modifiable risk factors and considering targeted-release budesonide as part of a multimodal strategy to slow disease progression.

The two cases were selected as illustrative examples to highlight the range of clinical presentations and outcomes observed in the cohort. They were chosen to demonstrate outcome variability. We acknowledge that this method of case selection introduces potential selection bias.

Case study 1

Presentation

A 77-year-old man with longstanding hypertension, ischaemic heart disease, benign prostate hyperplasia and lichen simplex chronicus was noted to have renal impairment during an admission for myocardial infarction in 2013. He underwent percutaneous coronary intervention to the right coronary artery and permanent pacemaker implantation.

Subsequent nephrology assessment identified chronic kidney disease (CKD) stage 3b, microscopic haematuria and significant proteinuria, with a urine protein:creatinine ratio (UPCR) of 237 mg/mmol. Vasculitis and immunological screening were negative, and renal imaging showed normal kidney size and corticomedullary differentiation. A native kidney biopsy in June 2018 demonstrated IgAN with mild parenchymal scarring (Oxford classification M1 E1 S1 T0). There was no significant chronic damage, as the T score was 0.

Intervention

Proteinuria gradually increased following biopsy, with UPCR rising to 278 mg/mmol. Ramipril was titrated to the maximum tolerated dose, but blood pressure remained above recommended targets. Amlodipine was added in May 2021, and dapagliflozin was introduced in May 2024 to reduce glomerular hyperfiltration and proteinuria.

He was enrolled in the open-label extension of a Phase 2 trial of iptacopan; however, treatment was discontinued following pneumococcal pneumonia in 2023. Despite maximal supportive therapy, proteinuria persisted (UPCR 213 mg/mmol). Following discussion at the regional multidisciplinary team (MDT) meeting, the patient commenced targeted-release budesonide in February 2025.

Outcomes

After 9 months of treatment with targeted-release budesonide, the patient's UPCR improved from 213 mg/mmol to 114 mg/mmol, representing a 46.5% reduction (**Figure 1**); eGFR levels remained

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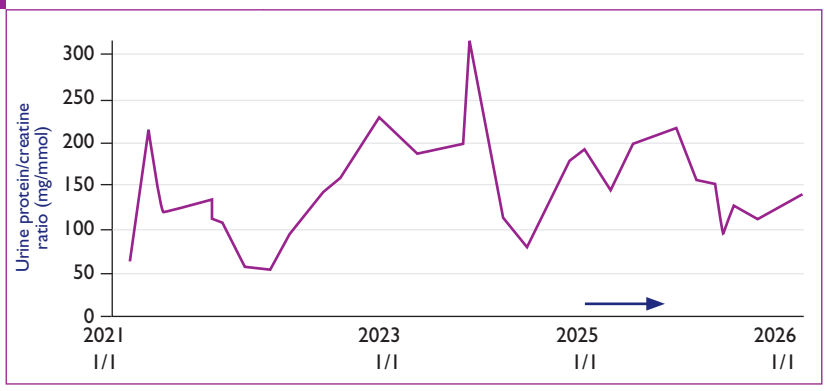

Figure 1. Case study 1 urine protein/creatine ratio

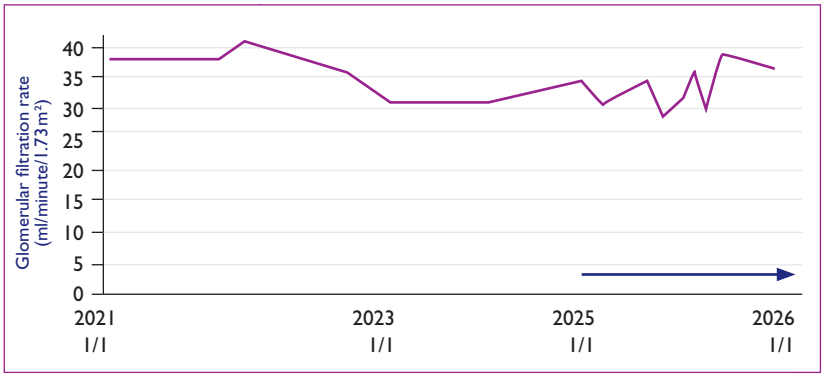

Figure 2. Case study 1 estimated glomerular filtration rate

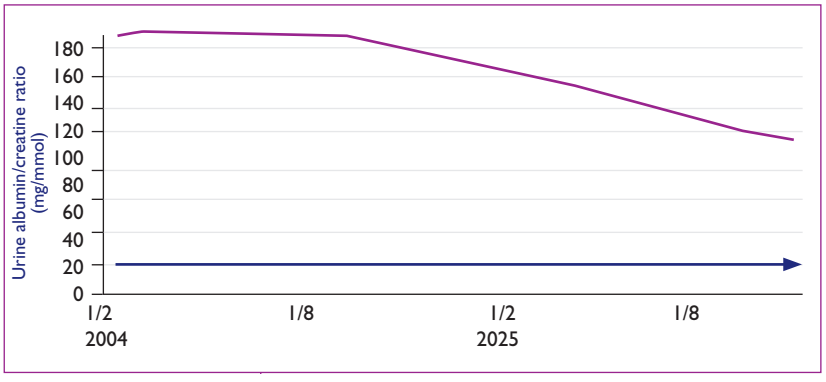

Figure 3. Case study 2 urine albumin/creatinine ratio

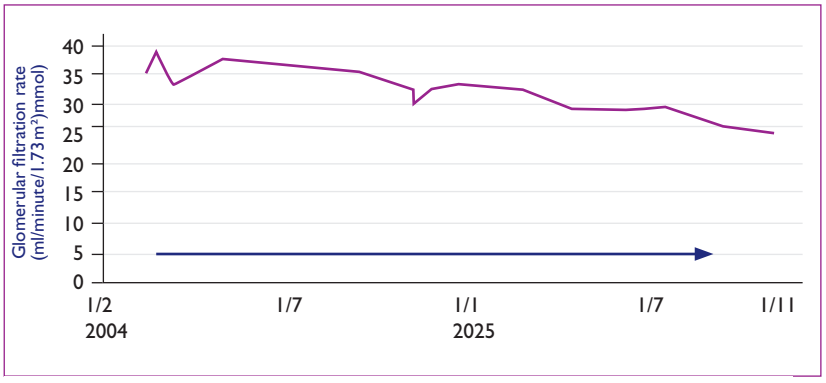

Figure 4. Case study 2 estimated glomerular filtration rate

stable at 34–36 ml/minute/1.73 m² (**Figure 2**), and serum creatinine decreased from 194 µmol/l to 152 µmol/l. Given the limited treatment and follow-up time frame, formal calculation of an eGFR slope was not possible and potential confounding factors, such as periods of uncontrolled blood pressure and chronicity in pre-treatment kidney biopsy, may have influenced interim eGFR changes. Treatment was well tolerated, with no infective, metabolic or cardiovascular adverse events reported.

Discussion

The patient demonstrated a favourable response to targeted-release budesonide, with substantial proteinuria reduction and stable kidney function. Early identification of persistent proteinuria despite optimal supportive care, combined with MDT review, facilitated timely initiation of targeted therapy. The patient's stable eGFR suggests that targeted-release budesonide may enable further decline in individuals with moderate CKD.

Case study 2

Presentation

A 38-year-old man with no significant medical history apart from a high body mass index was found to have elevated blood pressure during an occupational health assessment. His general practitioner diagnosed hypertension and prescribed amlodipine 5 mg once daily. Routine investigations to exclude secondary causes revealed impaired renal function, with a serum creatinine of 180 µmol/l and an eGFR of 39 ml/minute/1.73 m² in February 2024. No previous creatinine results were available.

Over the following month, kidney function deteriorated, with serum creatinine increasing to 205 µmol/l. Microscopic haematuria and sub-nephrotic proteinuria were present, with a urinary albumin:creatinine ratio (UACR) of 178.7 mg/mmol. At this stage, eGFR was 34 mL/min/1.73 m2.

A native kidney biopsy in March 2024 showed acute-on-chronic mesangial proliferative glomerulonephritis with IgA and C3 deposition, consistent with IgAN (Oxford classification M0 E1 S1 T0 C0).

Intervention

The patient was started on ramipril and dapagliflozin, and ramipril was titrated to the maximum tolerated dose. Amlodipine was increased to 10 mg daily to optimise blood pressure control.

Despite 6 months of maximal supportive therapy, proteinuria persisted (UACR 180 mg/mmol in September 2024), and eGFR declined to 33 ml/minute/1.73 m². Following MDT review, the patient was approved for a 9-month course of targeted-release

budesonide and commenced treatment in November 2024. Blood pressure control remained challenging, and doxazosin was added to the treatment regimen in October 2025.

Outcome

At completion of the 9-month course in September 2025, the patient's UACR had decreased from 180 mg/mmol to 107 mg/mmol, representing a 40.6% reduction (**Figure 3**). Serum creatinine increased from 214 µmol/l to 255 µmol/l, with a corresponding decline in eGFR from 33 to 26 ml/minute/1.73 m² (**Figure 4**). This deterioration likely reflected ongoing suboptimal blood pressure control rather than a treatment-related effect.

Discussion

The patient experienced a reduction in proteinuria following targeted-release budesonide, consistent with trial data. However, the patient's eGFR continued to decline, highlighting the importance of optimal blood pressure management alongside disease-modifying therapy. Persistent hypertension is a recognised driver of CKD progression, and achieving target blood pressure remains essential to maximising the benefits of targeted-release budesonide.

Conclusion

Across the case studies, targeted-release budesonide was associated with meaningful reductions in proteinuria. In the first case, eGFR remained stable, whereas in the second case, eGFR decline slowed but did not meet the target of <1 ml/minute/year. Factors contributing to ongoing decline included suboptimal blood pressure control and advanced CKD.

These cases reinforce that targeted-release budesonide is most effective when initiated early, before irreversible structural damage occurs. For patients with more advanced CKD, comprehensive optimisation of supportive care—including strict blood pressure control and consideration of endothelin receptor antagonists—remains essential. Early identification, timely MDT review and individualised treatment planning are key to improving long-term outcomes in IgAN.

Conflicts of interest

This article was sponsored by Stada UK. Stada UK had no involvement in the creation of the content, which was independently developed by the authors Richard Hull, Ayat Faggad and Lisa Willcocks in partnership with MA Healthcare. Stada UK reviewed the article for technical accuracy and compliance only. Prescribing information and adverse event reporting can be found at the end of this article.

Key points

- IgAN is the most common primary glomerular disease with high risk of kidney failure.
- Targeted-release budesonide reduces galactose-deficient IgA1 and proteinuria, slowing eGFR decline with fewer adverse effects than systemic corticosteroids.
- Targeted-release budesonide is most effective when started early alongside optimised supportive care.

CPD reflective questions

- How might earlier initiation of targeted-release budesonide change long-term outcomes for patients with IgAN?
- What role does strict optimisation of supportive care (RAS blockade, SGLT2i, blood pressure control) play in maximising the benefits of targeted-release budesonide?
- How should clinicians balance proteinuria reduction with ongoing eGFR decline when evaluating the effectiveness of targeted-release budesonide in different patients?

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AF has no conflicts of interest to declare.

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