Targeting the Source of IgA Nephropathy

Saturday September 30 | 7:30–8:30 JST

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Agenda

The Potential Role of BAFF and APRIL in the Pathogenesis of IgA Nephropathy: Implications for Dual Inhibition

Chee Kay Cheung, MD, PhD, FRCP
Consultant Nephrologist and Honorary Associate Professor, The Mayer IgA Nephropathy Laboratories, University of Leicester, Leicester, UK

Emerging Therapies in IgA Nephropathy: Targeted Mechanisms and Clinical Implications

Richard Lafayette, MD, FACP
Professor, Medicine (Nephrology), Director, Glomerular Disease Center,
Stanford University Medical Center, San Francisco, USA

The Potential Role of BAFF and APRIL in the Pathogenesis of IgA Nephropathy: Implications for Dual Inhibition

Chee Kay Cheung, MD, PhD, FRCP

Consultant Nephrologist & Honorary Associate Professor,
The Mayer IgA Nephropathy Laboratories

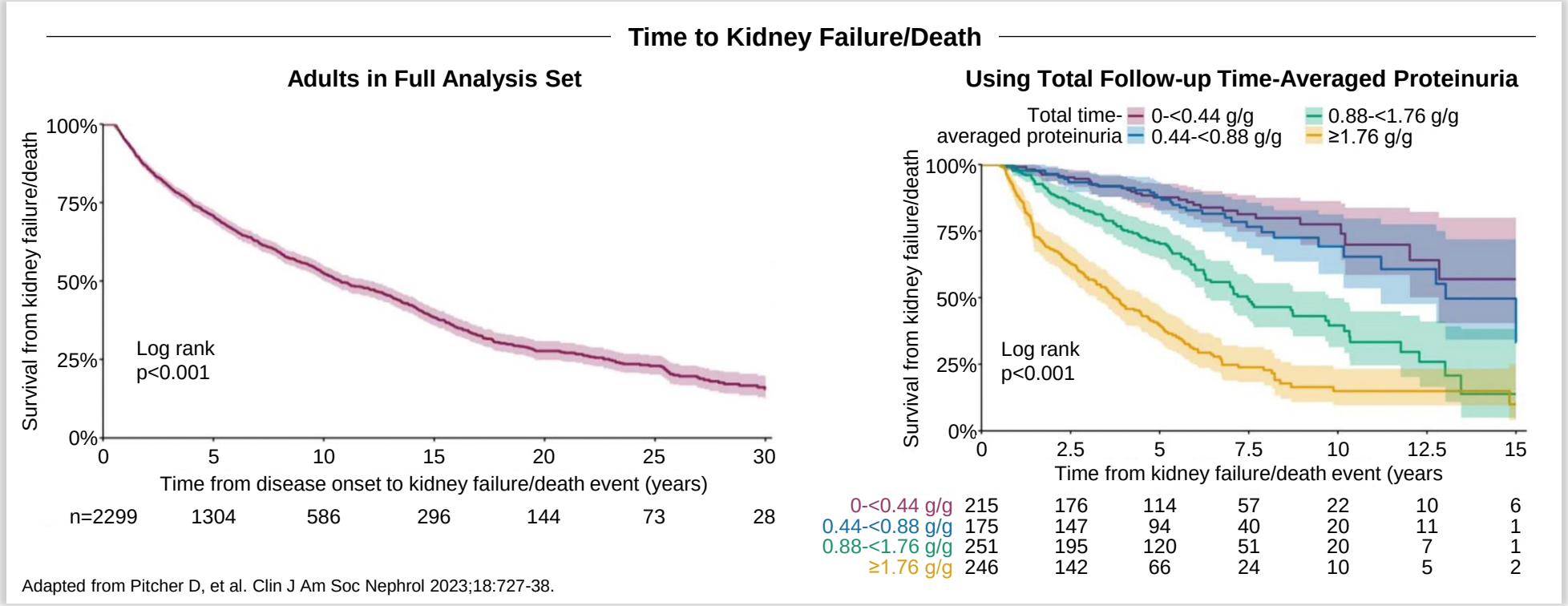
University of Leicester & John Walls Renal Unit, UK

COI Disclosure

- I have the following relationships to disclose any COI for this presentation within the period of 36 months.
- Consultancy agreements: Vera Therapeutics, George Clinical
- Advisory boards: Calliditas, CSL Vifor, Novartis
- Research funding: GlaxoSmithKline, Travere Therapeutics
- Steering Committees/DSMC: CSL Vifor, Alpine Immune Sciences, Roche
- Travel support: Otsuka, Chinook
- Speaker fees: Stada

Introduction

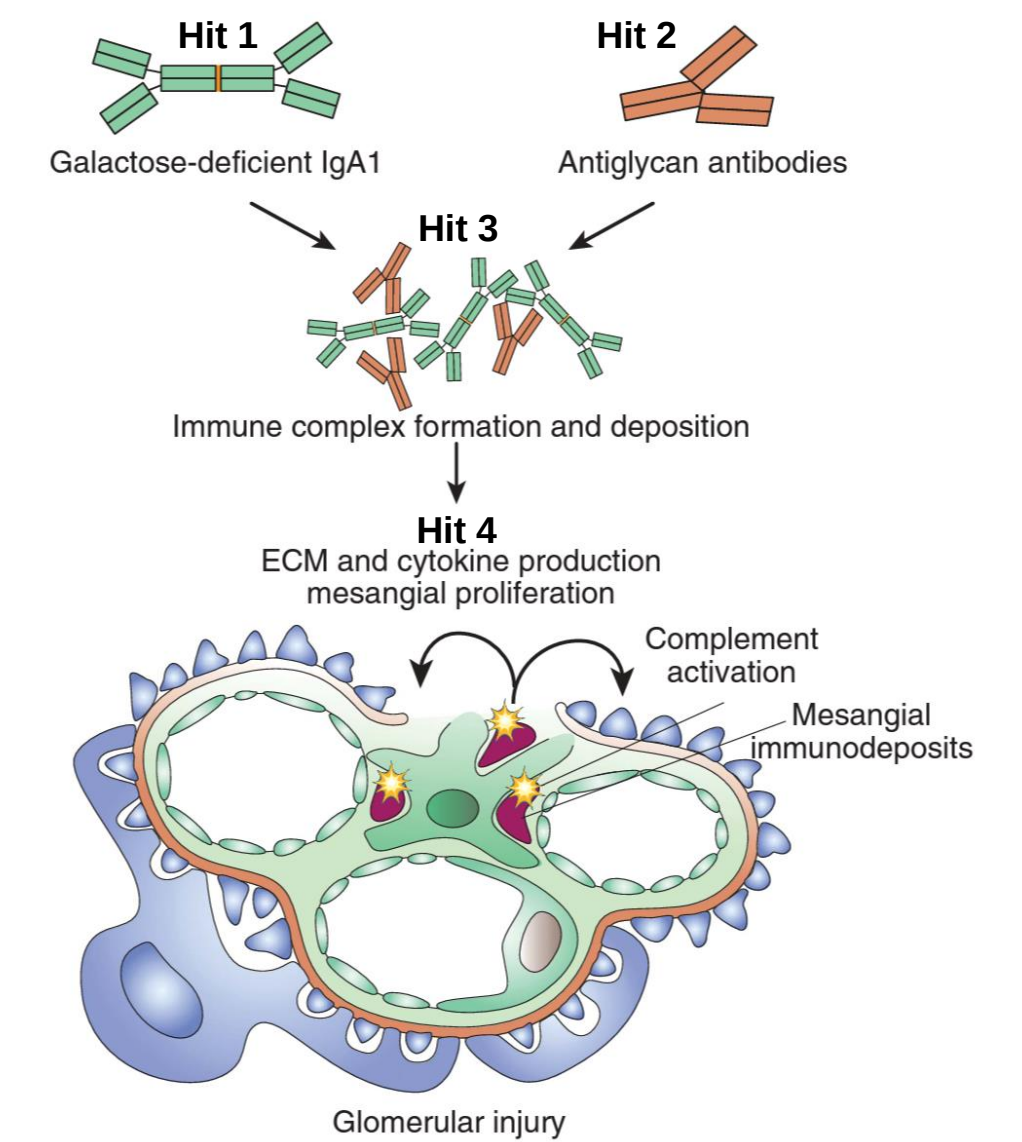
- IgAN is the most common form of primary glomerular disease worldwide
- Peak incidence is in 2nd to 3rd decade of life, but may occur at any age
- Marked variations in its clinical presentation and prognosis
- Overall, there is a substantial lifetime risk of developing kidney failure; up to 50% of patients with IgAN progress to ESKD, resulting in the need for dialysis or transplant



Adapted from Pitcher D, et al. Clin J Am Soc Nephrol 2023;18:727-38.

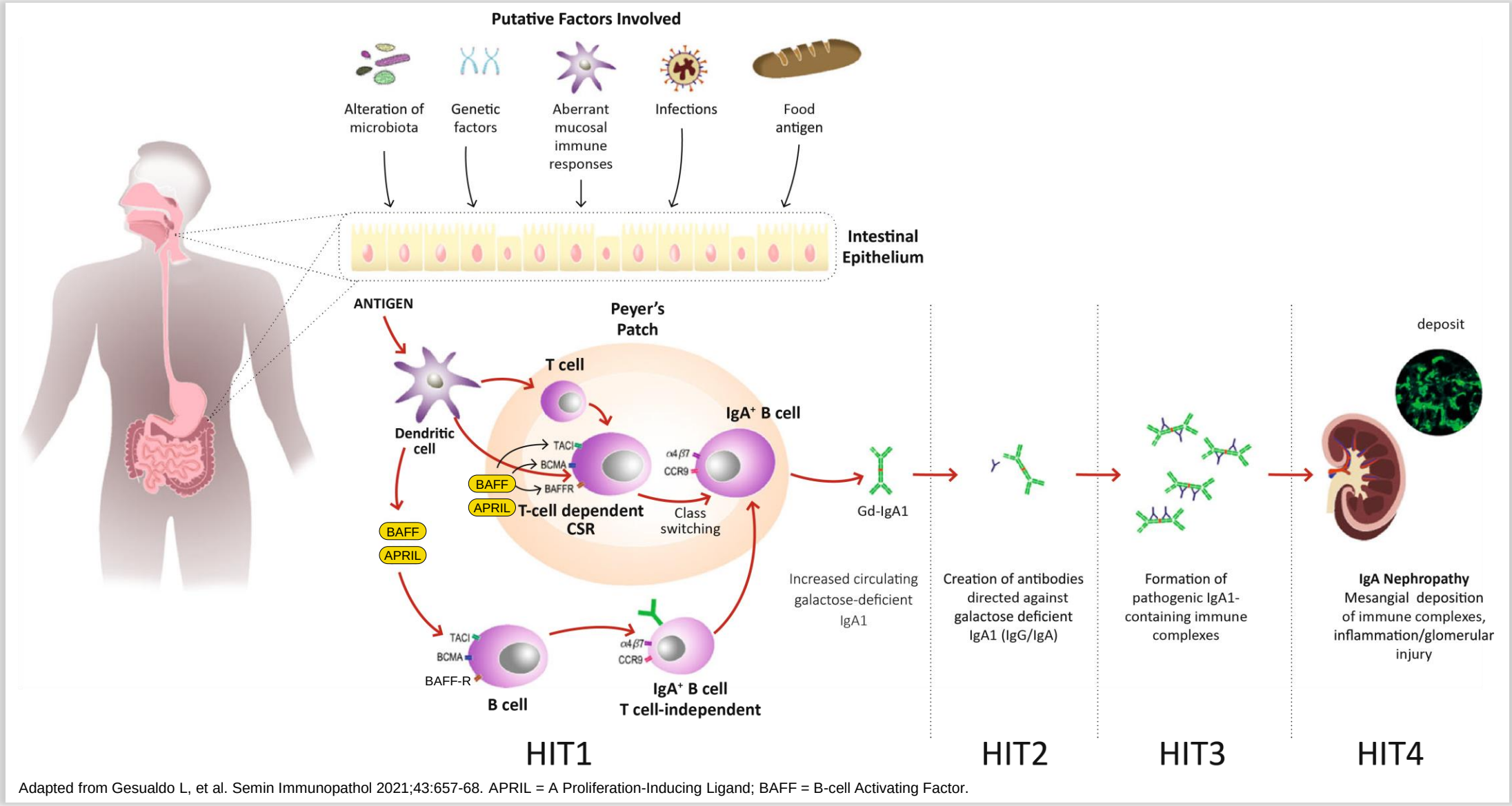
Pathogenesis of IgAN: Multi-Hit Hypothesis

1. Increase in circulating galactose-deficient IgA1 (Gd-IgA1)
2. Formation of anti-Gd-IgA1 antibodies
3. Formation of Gd-IgA1-containing immune complexes
4. Mesangial deposition



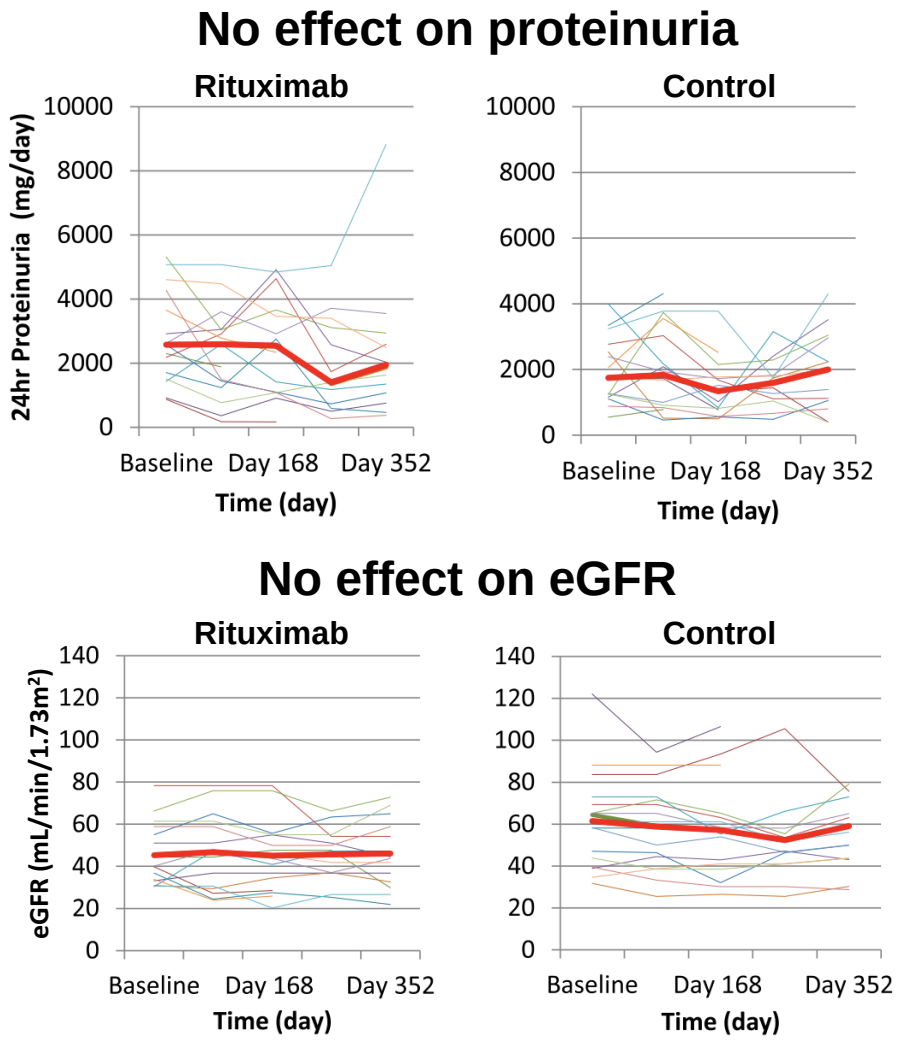
Adapted from Magistroni R, et al. Kidney Int 2015;88:974-89.

Pathogenesis of IgAN



Adapted from Gesualdo L, et al. Semin Immunopathol 2021;43:657-68. APRIL = A Proliferation-Inducing Ligand; BAFF = B-cell Activating Factor.

Rituximab Not Effective in IgAN

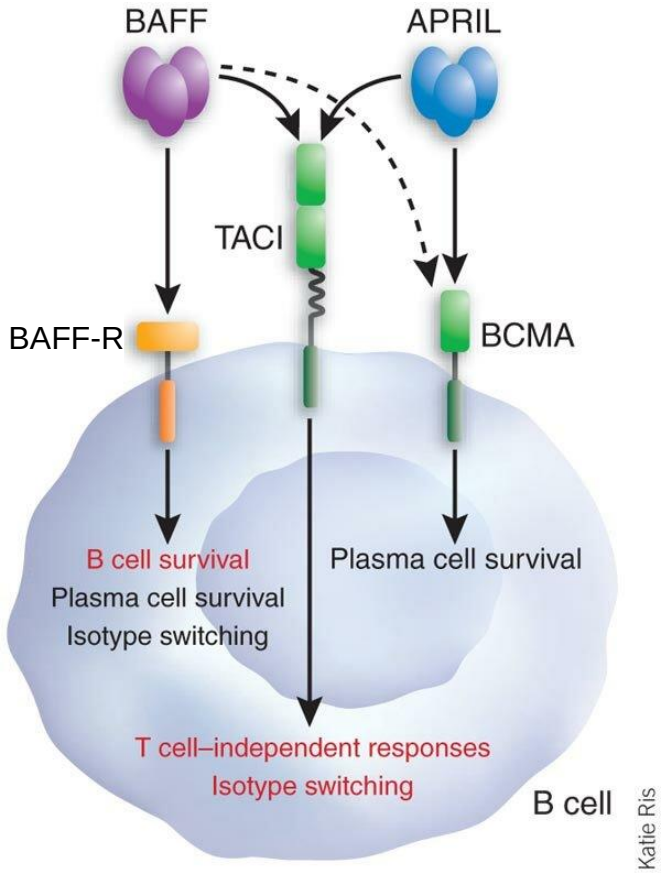


Red line represents average data

Adapted from Lafayette R, et al. J Am Soc Nephrol 2017;28:1306-13.

- No effect on proteinuria or eGFR
- No reductions in Gd-IgA1 or IgG autoantibodies despite adequate depletion of peripheral B cells

BAFF and APRIL

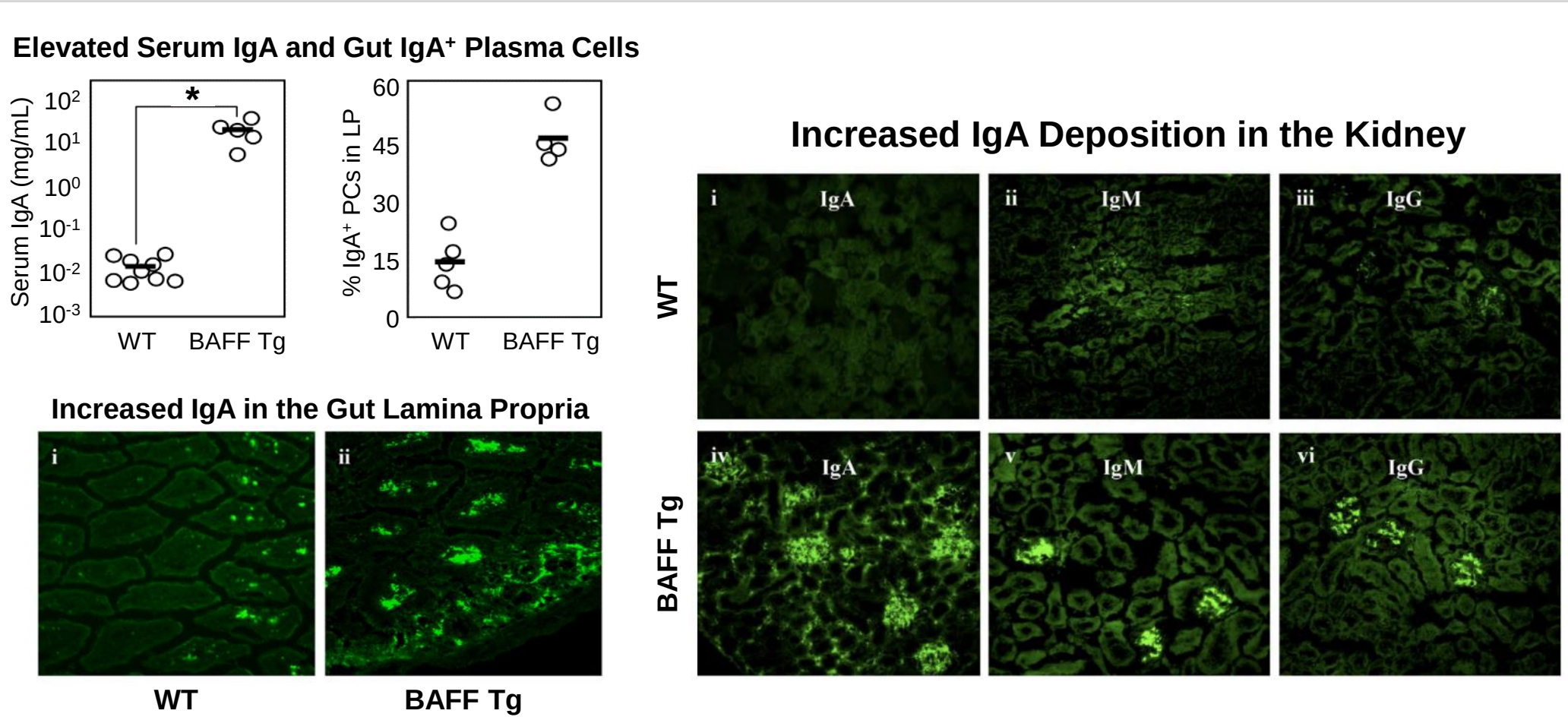


Adapted from Martin F, Dixit VM. Nat Genet 2005;37:793-4.

- B-cell Activating Factor (BAFF) and A PRoliferation-Inducing Ligand (APRIL) are members of the TNF family of signaling proteins
- Act via shared B cell receptors
- Important roles in
  - B cell proliferation
  - B cell survival and maturation
  - Class switching
- Inhibitors of APRIL and/or BAFF studied in other autoimmune diseases (eg, SLE)

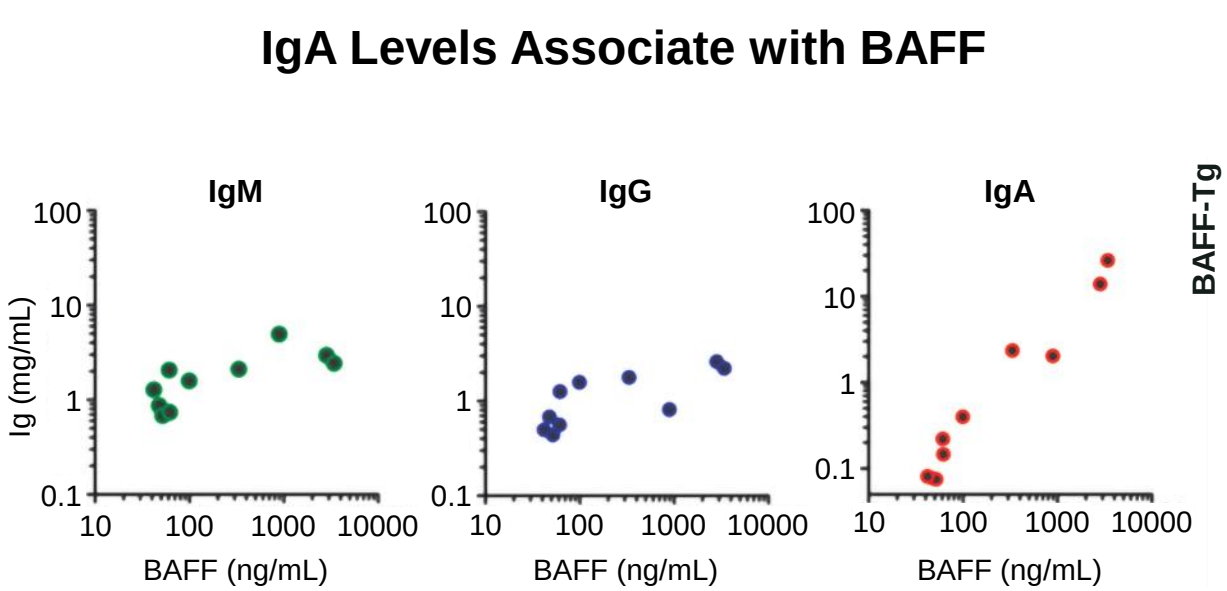
SLE = systemic lupus erythematosus; TNF = tumor necrosis factor.

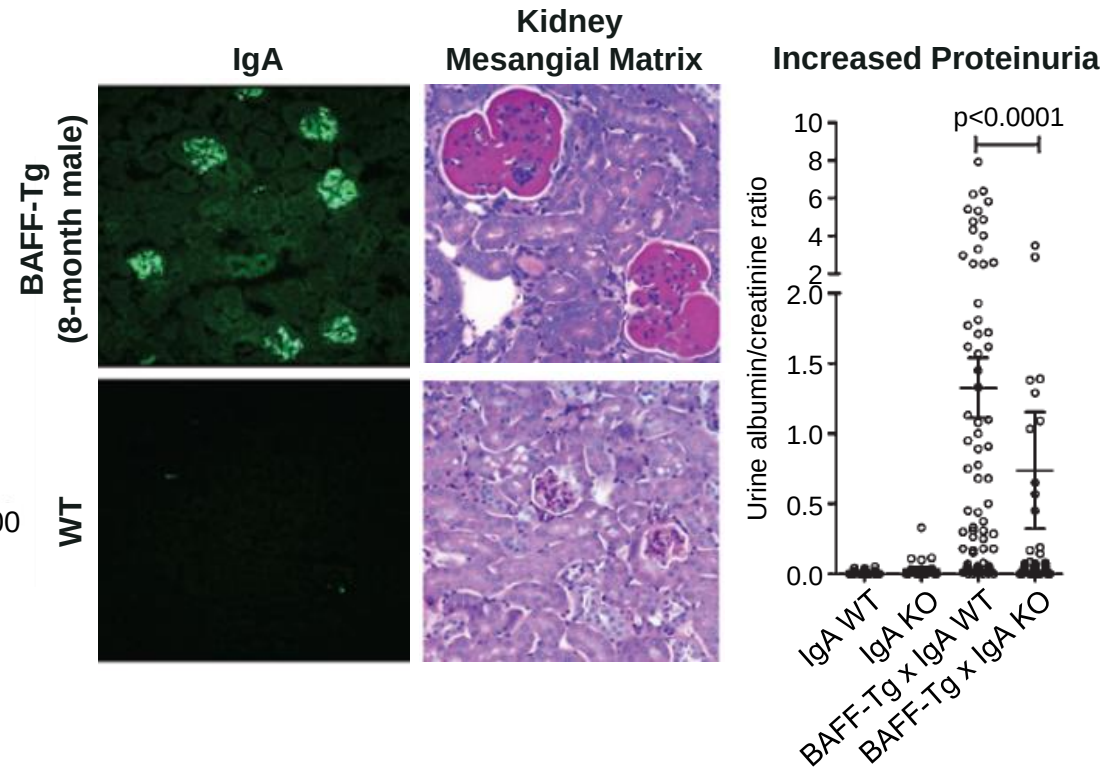
BAFF Overexpression Leads to Increased IgA From the Intestinal Lamina Propria that Deposits in the Kidney



Adapted from McCarthy D, et al. Cell Immunol 2006;241:85-94.

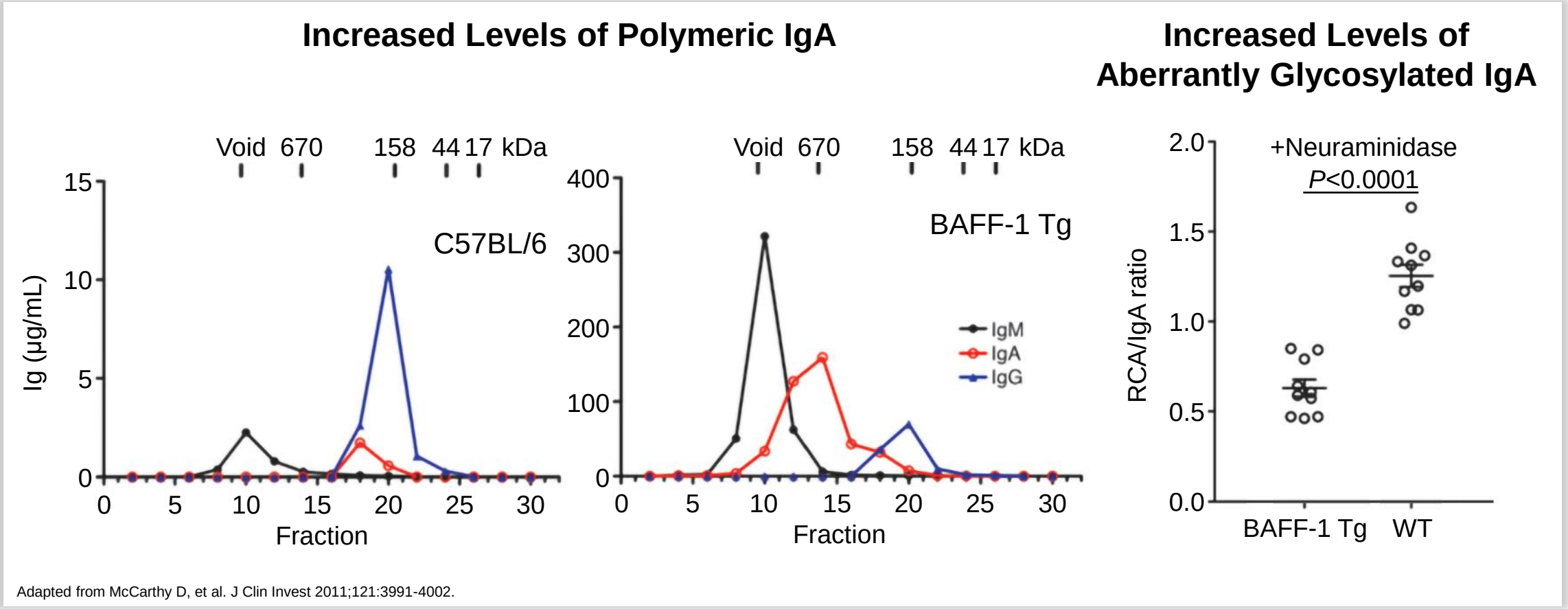
Serum BAFF Overexpression Correlates with Serum IgA, and May Lead to IgA-associated Nephropathy





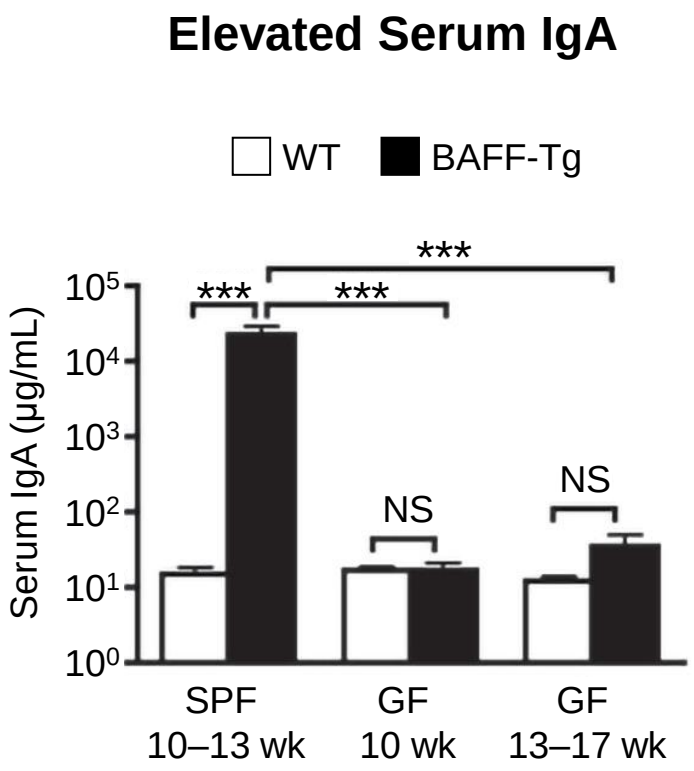
Adapted from McCarthy D, et al. J Clin Invest 2011;121:3991-4002.

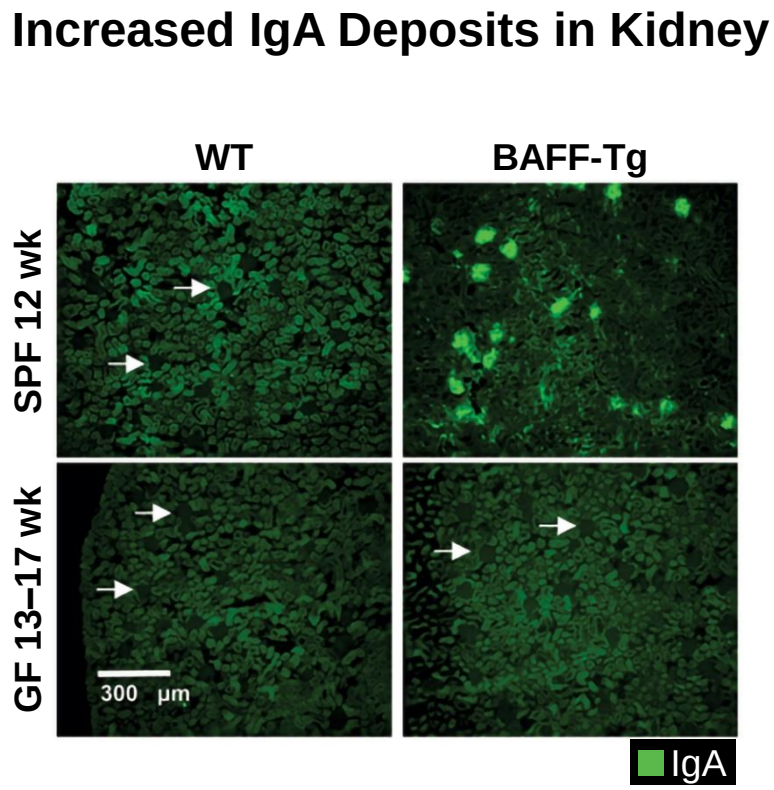
BAFF Overexpression Leads to Increased Levels of Polymeric Aberrantly Glycosylated IgA

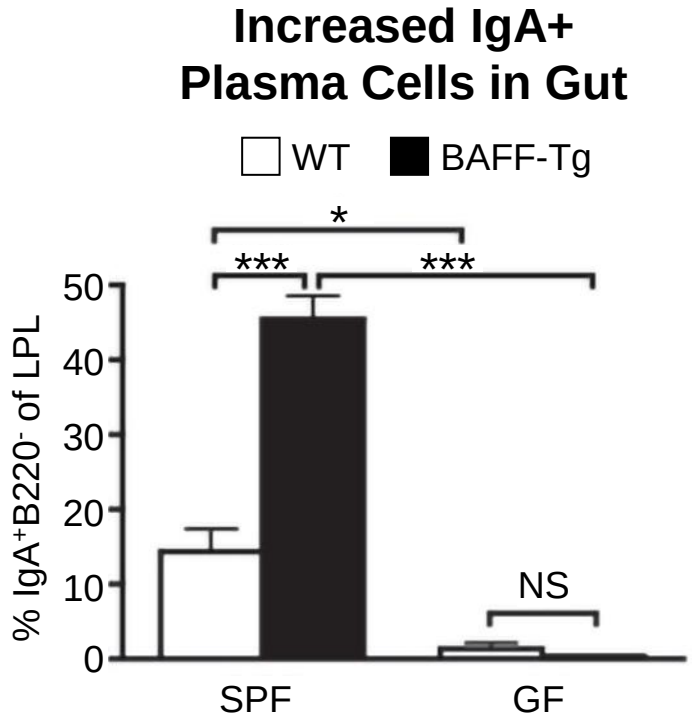


Adapted from McCarthy D, et al. J Clin Invest 2011;121:3991-4002.

IgA-associated Nephropathy in a BAFF Overexpressing Mouse Model is Dependent on Commensal Flora

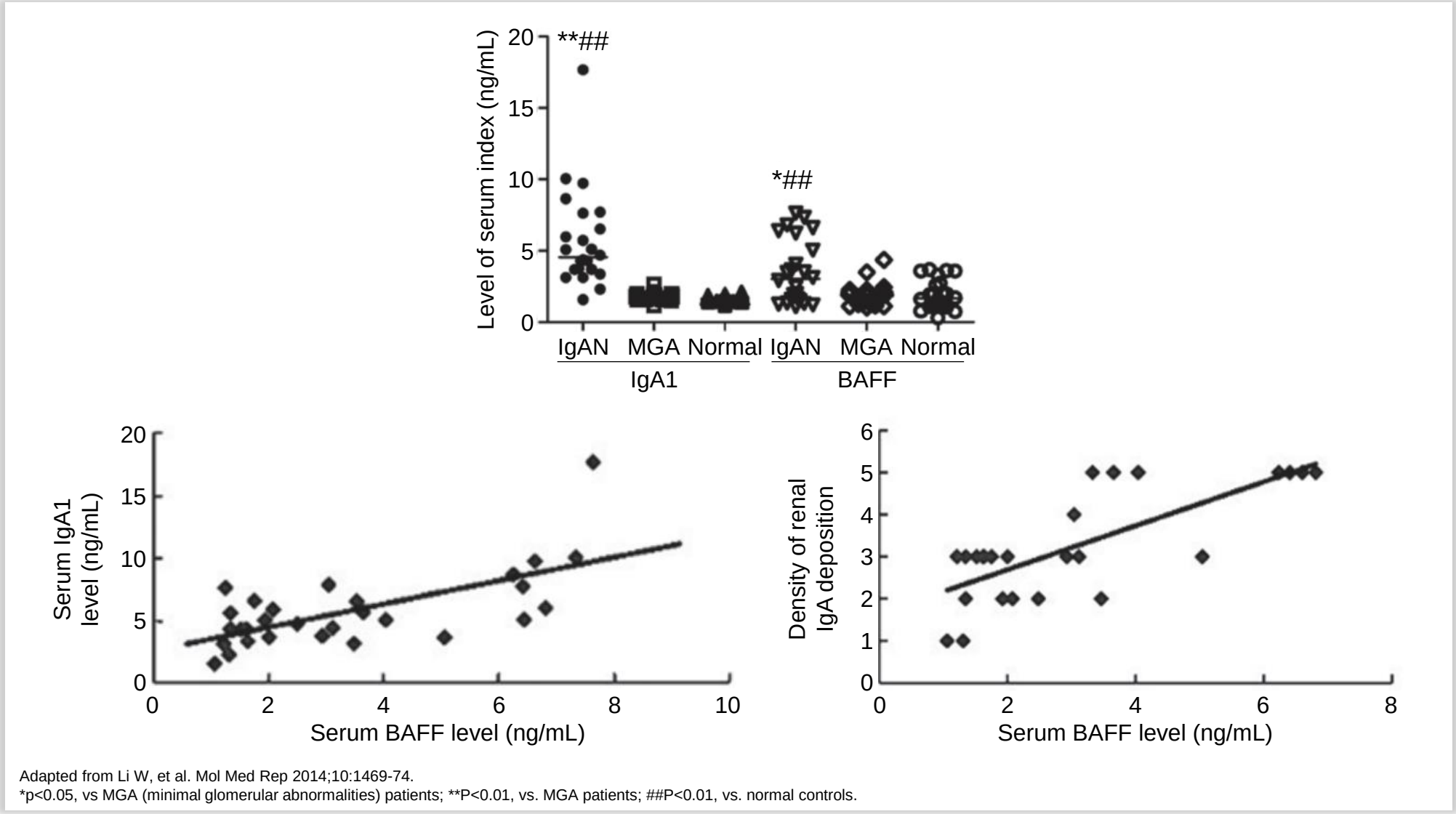






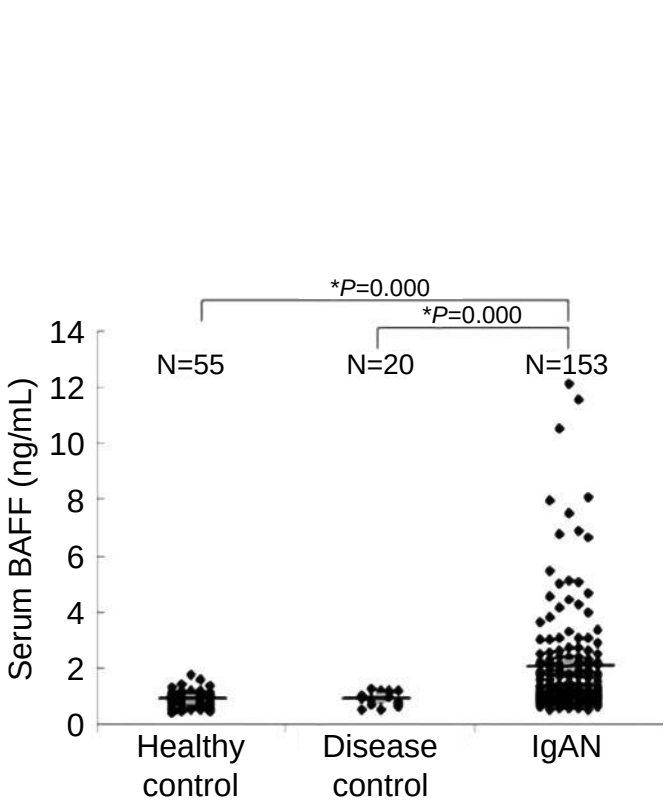
Adapted from McCarthy D, et al. J Clin Invest 2011;121:3991-4002. GF = germ-free; LPL = lamina propria lymphoyte; SPF = specific pathogen free.

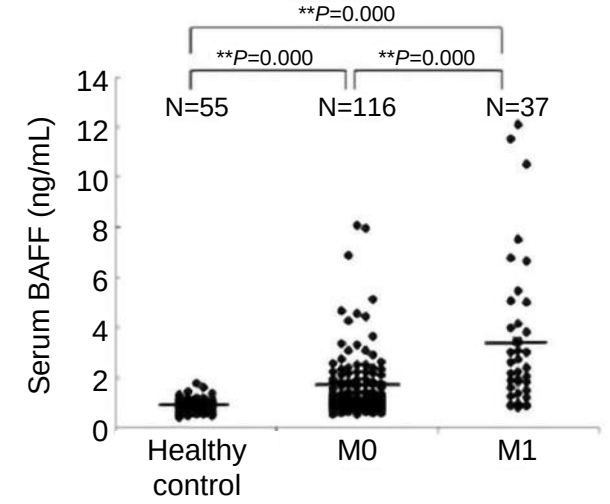
BAFF Associates with Circulating IgA1 and Kidney IgA Deposits in Patients with IgAN

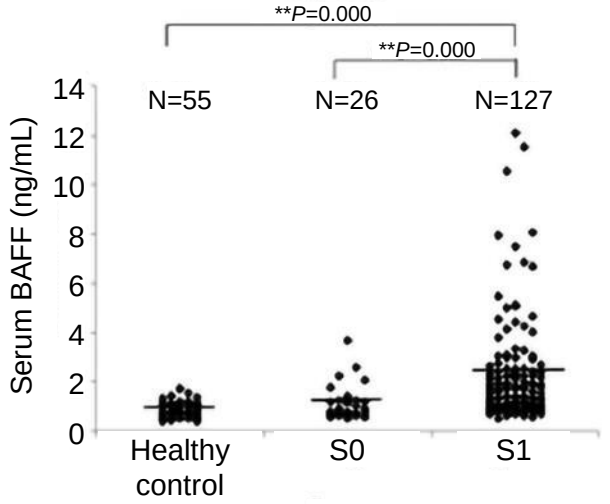


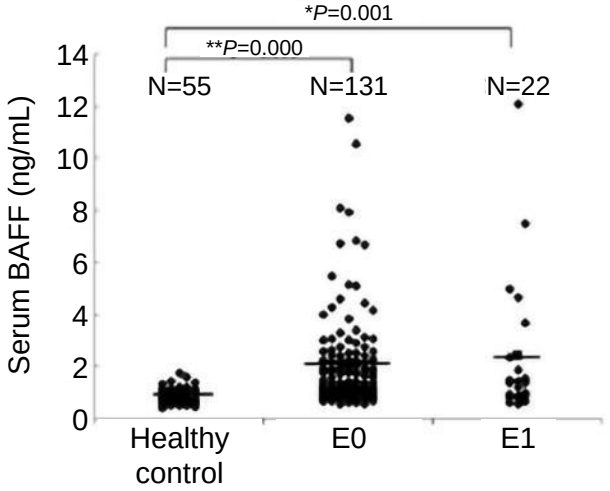
Adapted from Li W, et al. Mol Med Rep 2014;10:1469-74.
*p<0.05, vs MGA (minimal glomerular abnormalities) patients; **P<0.01, vs. MGA patients; ##P<0.01, vs. normal controls.

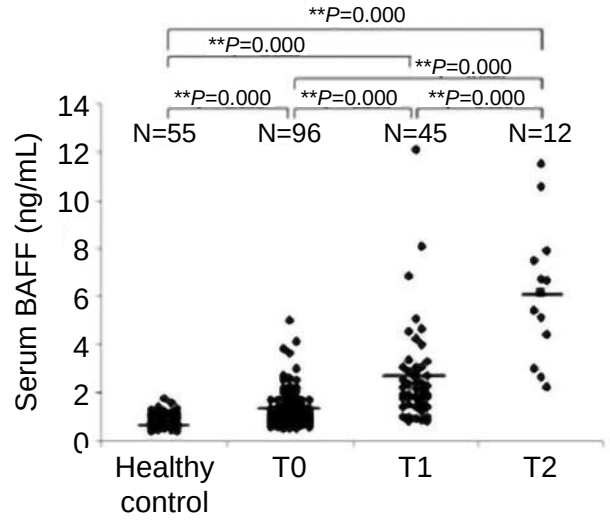
Serum BAFF is Elevated in Patients with IgAN and is Associated with Adverse Pathological Features











Adapted from Xin G, et al. J Nephrol 2013;26:683-90.
M=mesangial hypercellularity grade; E=endocapillary hypercellularity score grade; S=segmental glomerulosclerosis grade; T=tubular atrophy/interstitial fibrosis grade.

Serum BAFF is Increased in IgAN and is Associated with Reduced Kidney Function

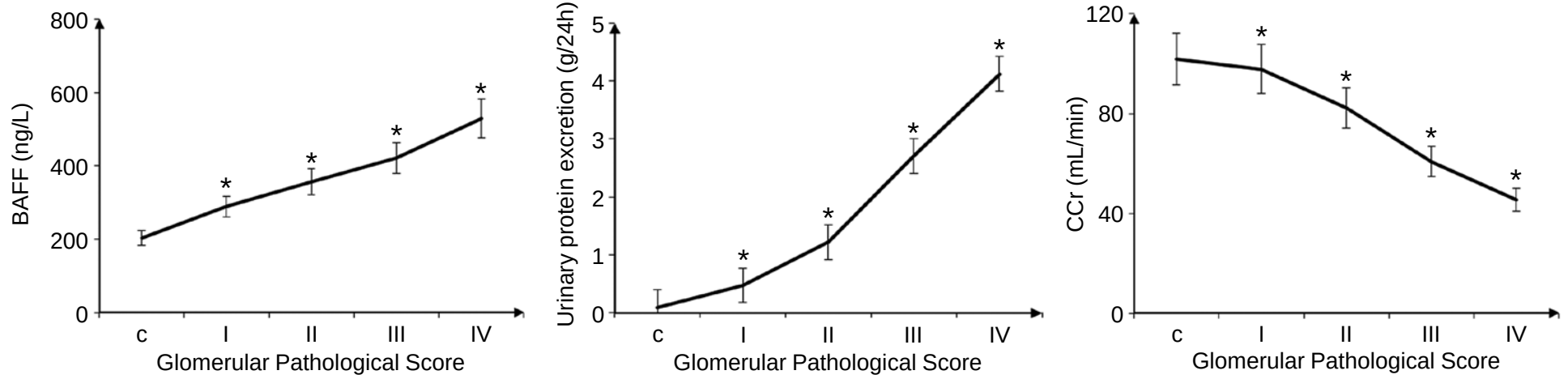
TABLE II
CORRELATIONS BETWEEN SERUM BAFF LEVELS AND CLINICAL PARAMETERS

Patients	Univariant analysis		Multiple stepwise regression analysis	
	r	p Value	r	p Value
Age, years	0.177	0.016*	0.020	0.840
Systolic BP, mm Hg	0.364	0.000**	-0.135	0.242
Diastolic BP, mm Hg	0.280	0.002*	-0.093	0.421
eGFR	-0.776	0.000**	-0.338	0.001*
Proteinuria, g/24 hours	0.454	0.000**	0.099	0.232
Serum creatinine, μmol/L	0.771	0.000**	0.775	0.000**
Serum BUN, mmol/L	0.650	0.000**	0.186	0.052
Serum uric acid, mmol/L	0.484	0.000**	0.084	0.377

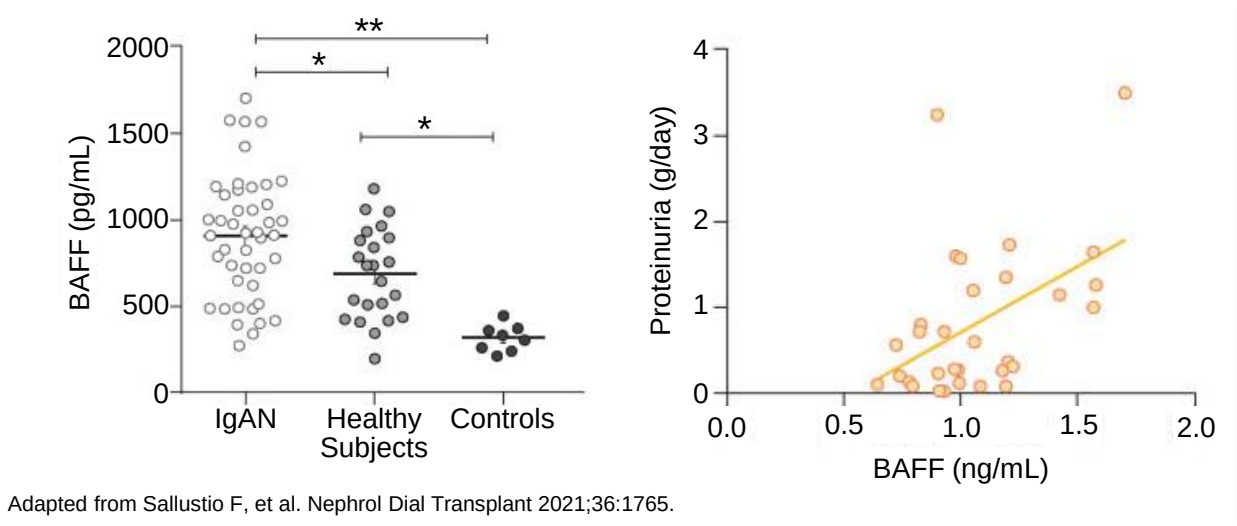
BP = blood pressure; BUN = blood urea nitrogen; eGFR = estimated glomerular filtration rate.
*p<0.05, **p<0.001.

Adapted from Xin G, et al. J Nephrol 2013;26:683-90.

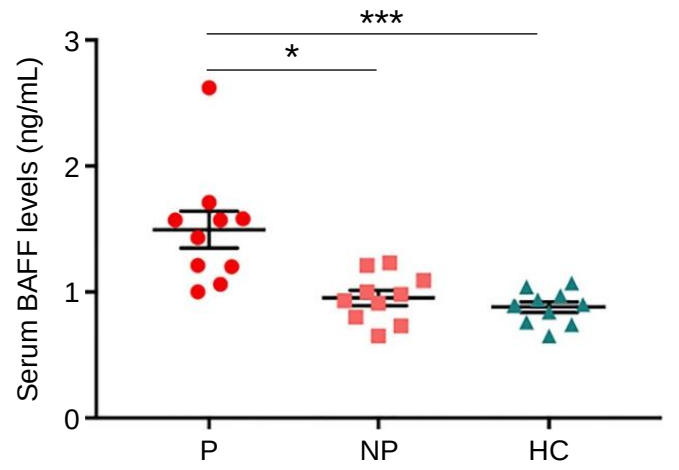
BAFF Levels are Elevated in IgAN and are Associated with Disease Severity



Adapted from Cao Y, et al. Mol Med Rep 2020;21:795-805.



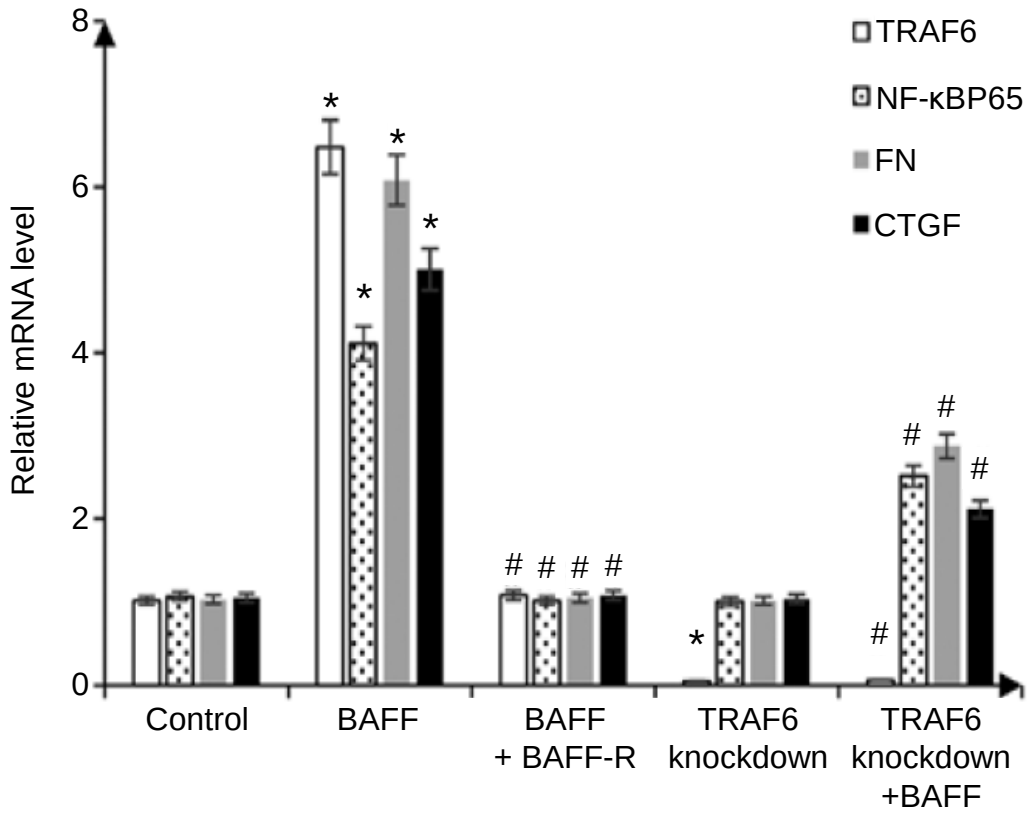
Adapted from Sallustio F, et al. Nephrol Dial Transplant 2021;36:1765.

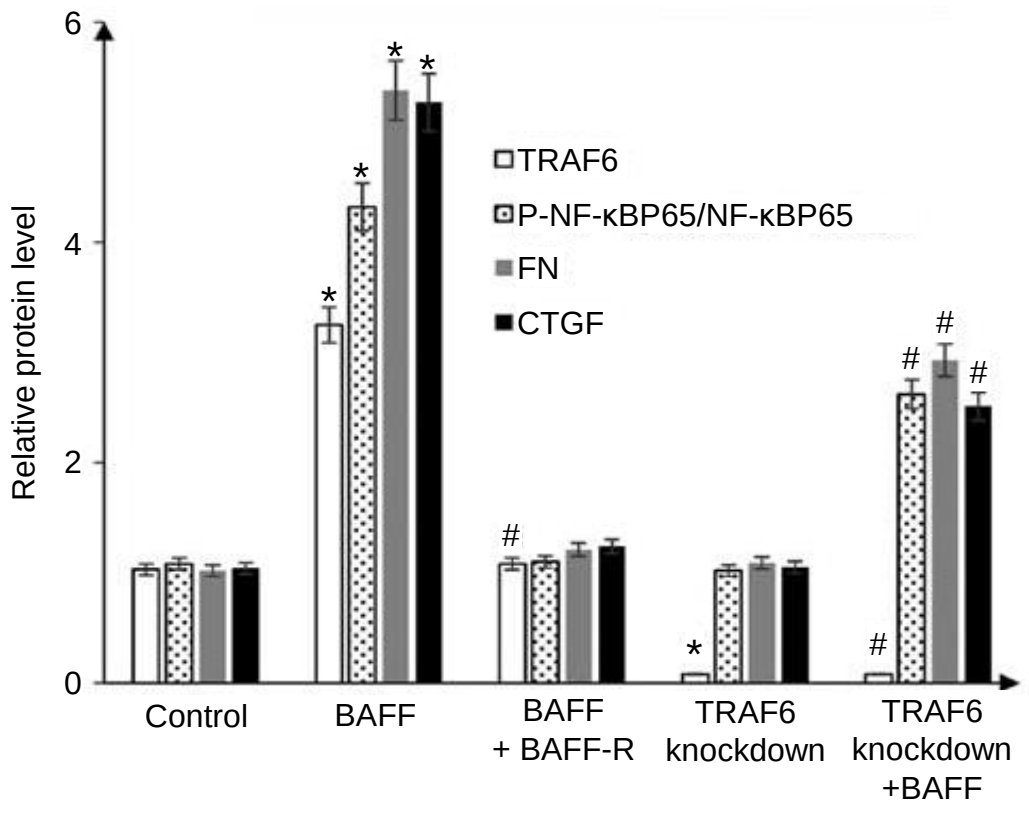


Adapted from Lauriero G, et al. Front Immunol 2021;12:694787.

HC = healthy control subjects; P = progressor patients; NP = non-progressor patients.

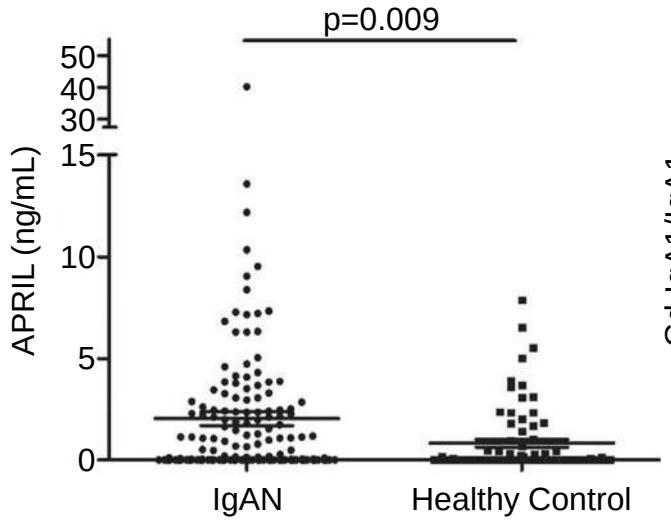
BAFF Can Directly Increase the Expression of Factors Associated with Fibrosis and Inflammation in Mesangial Cells

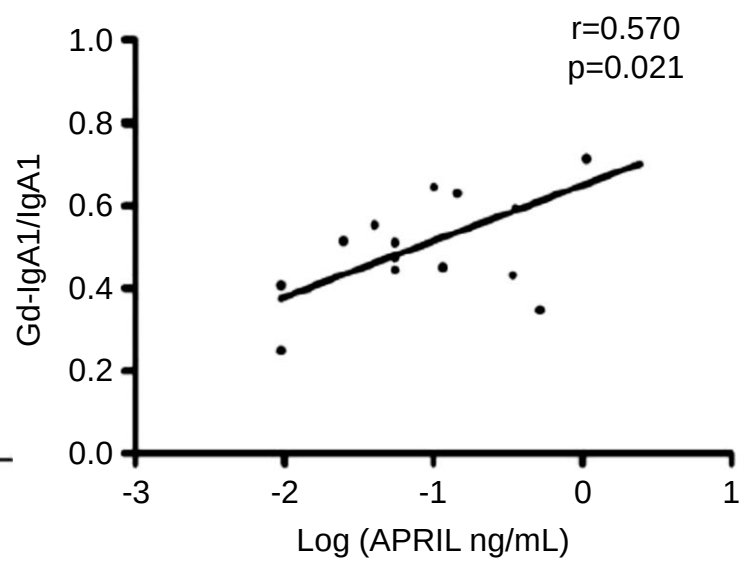




Adapted from Cao Y, et al. Mol Med Rep 2020;21:795-805. *p<0.05 vs control; #p<0.05 vs BAFF; †p>0.05 vs BAFF.
CTGF = connective tissue growth factor; FN = fibronectin; TRAF = tumor necrosis factor receptor-associated factor.

APRIL Levels are Elevated in IgAN and Correlate with Gd-IgA1 Levels

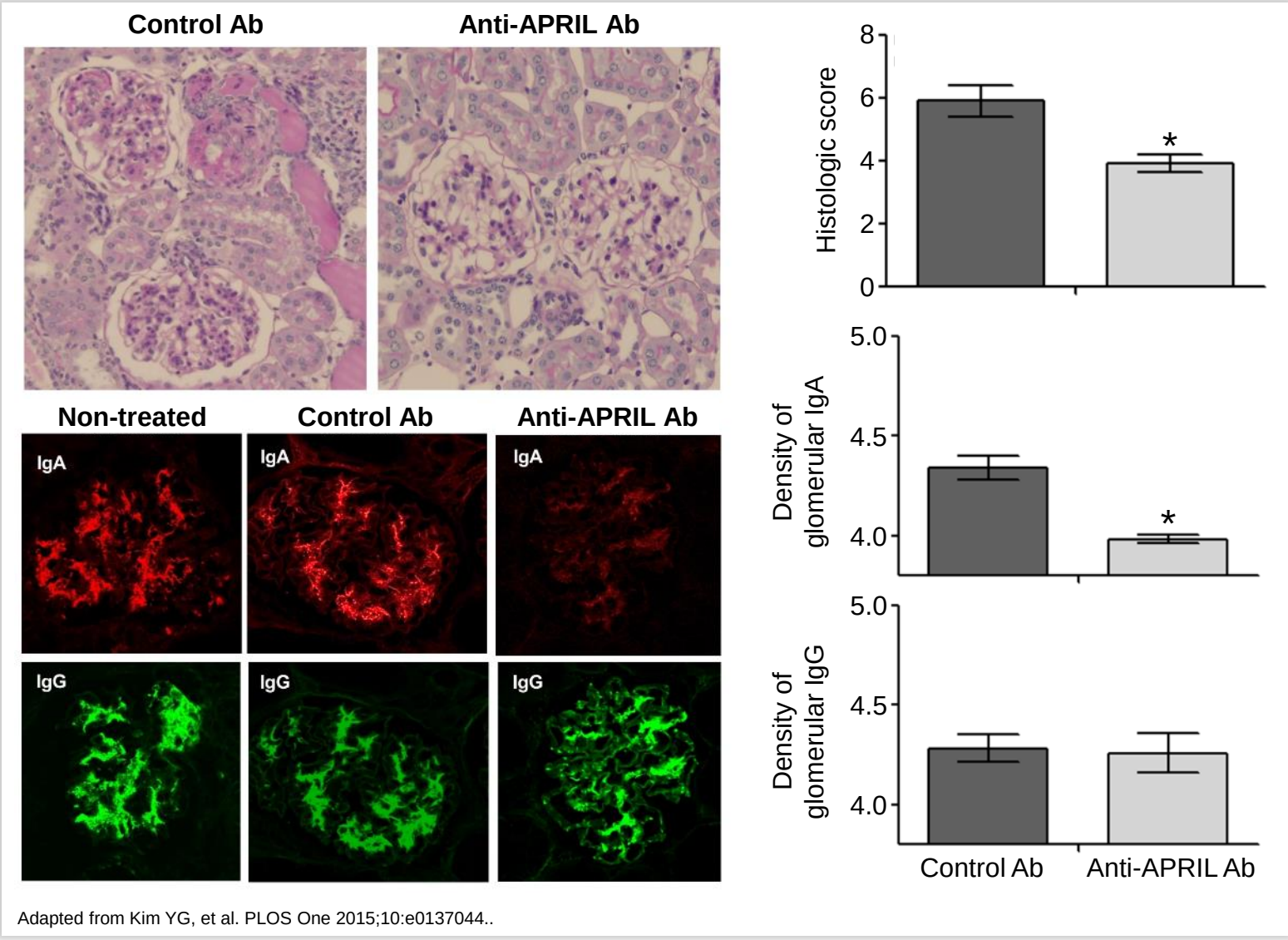




Adapted from Zhai Y-L, et al. Medicine 2016;95:e3099.

Adapted from Han SS, et al. J Am Soc Nephrol 2016;27:3430-9.

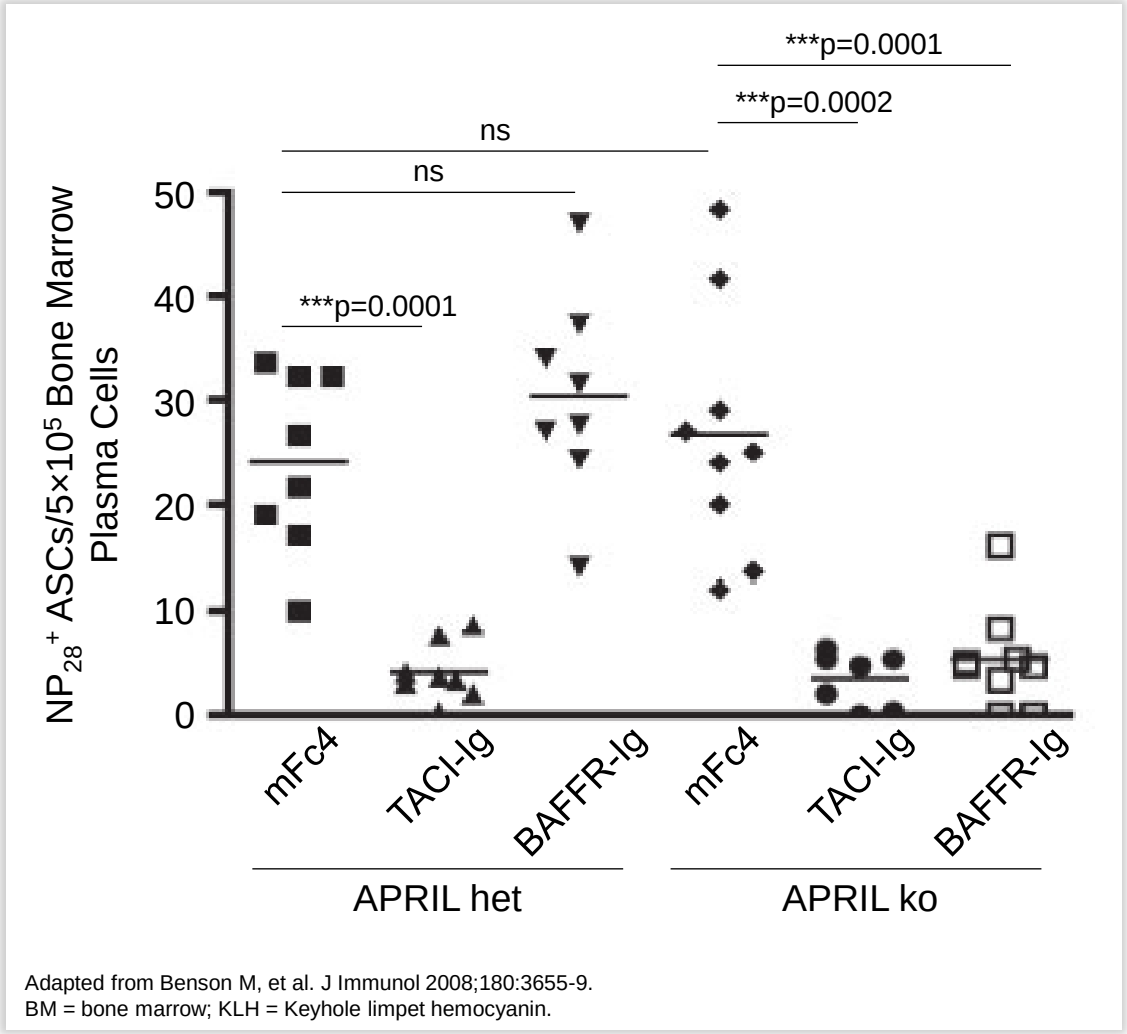
Anti-APRIL Antibodies Effective in Murine IgAN Model



Adapted from Kim YG, et al. PLOS One 2015;10:e0137044..

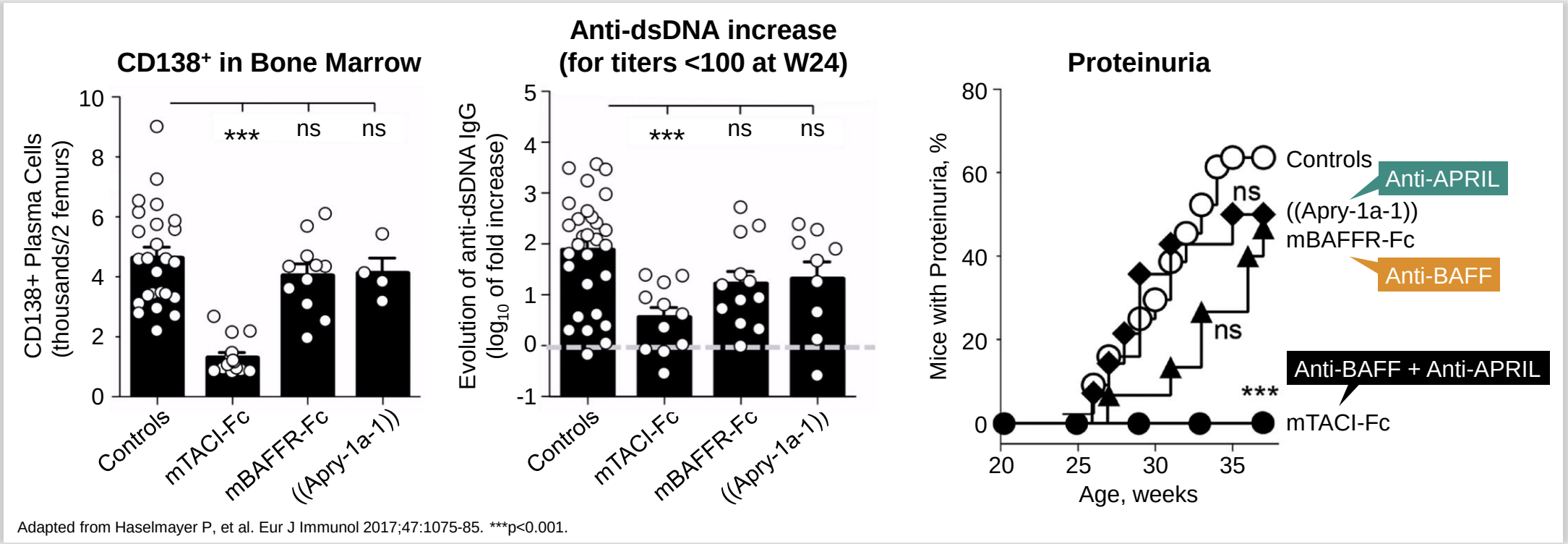
Dual BAFF and APRIL Blockade is Required for Plasma Cell Inhibition

- APRIL KO mice or heterozygous littermate controls were immunized with KLH antigen and treated with fusion proteins for 3 weeks, after which bone marrow was harvested and plasma cells were quantified
- Dual blockade better than either BAFF or APRIL alone to reduce bone marrow plasma cells



Adapted from Benson M, et al. J Immunol 2008;180:3655-9.
BM = bone marrow; KLH = Keyhole limpet hemocyanin.

Dual BAFF/APRIL Inhibition Prevented Proteinuria in Mouse Lupus Model Compared to BAFF or APRIL-Only Inhibitors

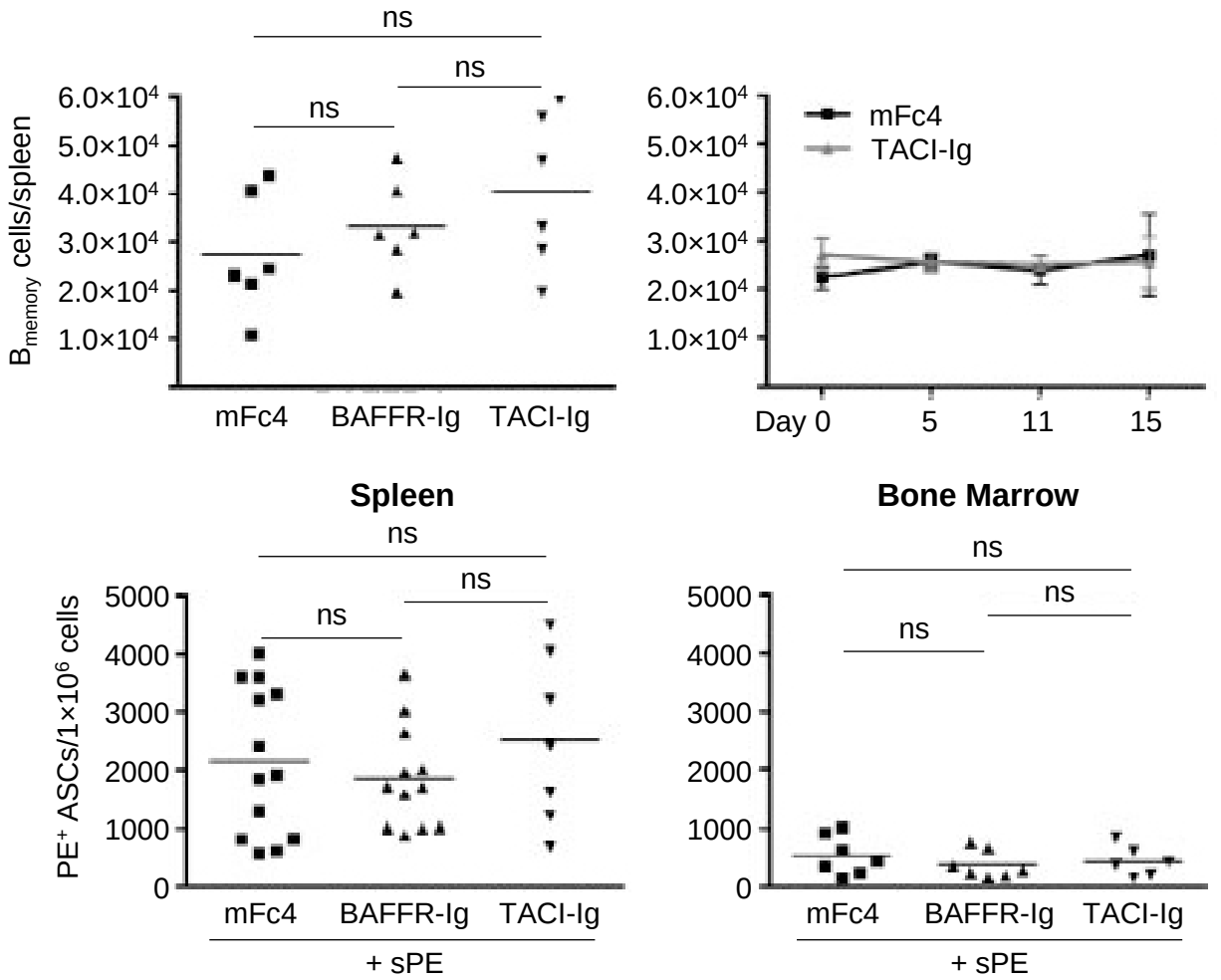


Adapted from Haselmayer P, et al. Eur J Immunol 2017;47:1075-85. ***p<0.001.

- A study using TACI-Fc to block both BAFF and APRIL in NZB/NZW F1 mouse model of SLE found that the neutralization of both BAFF and APRIL always lead to a decrease in bone marrow plasma cells and slowed formation of autoantibodies
- In a mouse model of lupus nephritis, only a dual BAFF/APRIL inhibitor (atacicept analogue) effectively prevented proteinuria compared to inhibitors of BAFF or APRIL alone

Dual BAFF and APRIL Blockade Does Not Impact Long-lived Ag-specific B_{memory} Cell Survival and Function

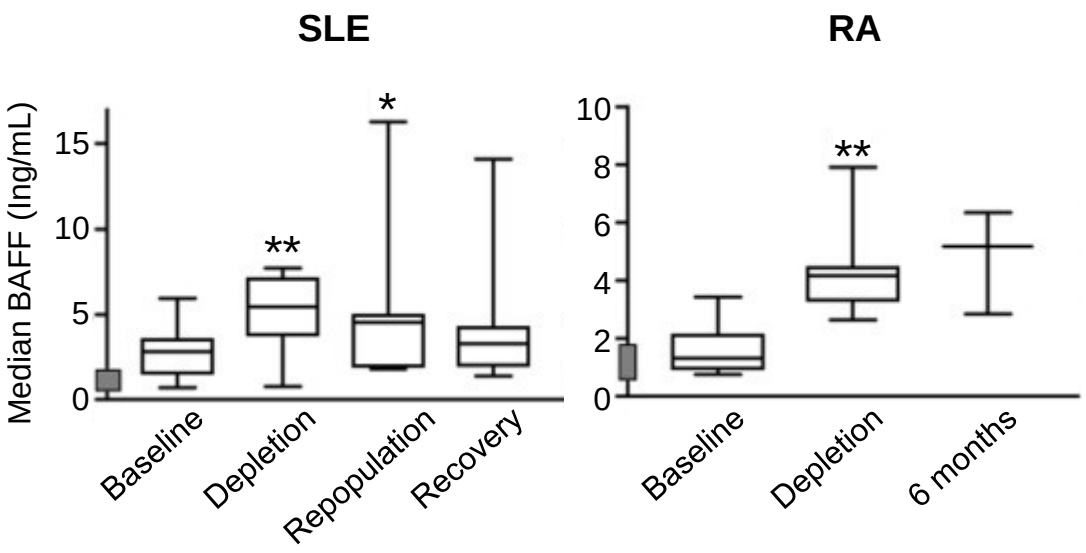
- PE-immunized mice treated with TACI-Ig showed no difference in PE^+ B_{memory} cell count vs control
- After antigen recall, TACI-Ig treated mice showed no difference in the number of PE+IgG+ plasma cells



Adapted from Benson M, et al. J Immunol 2008;180:3655-9. PE = phycoerythrin.

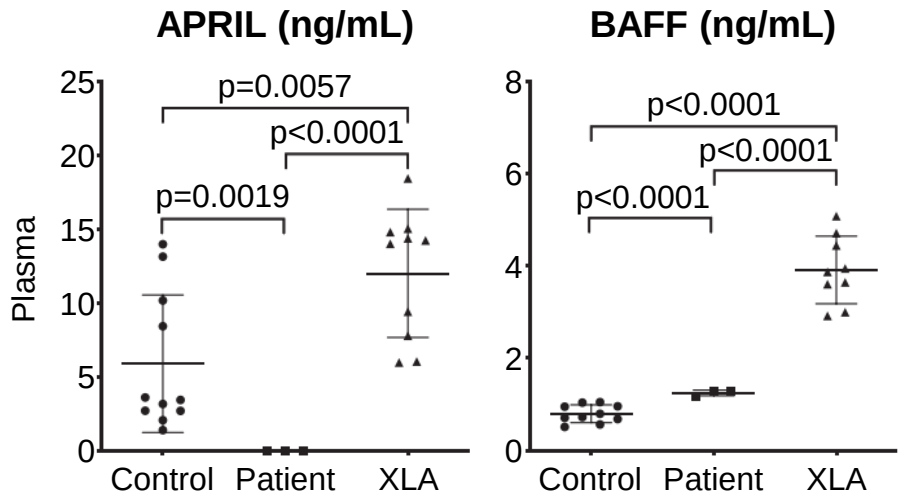
Blocking APRIL Alone May Lead to Upregulation of BAFF Signaling with Potential Consequences on Efficacy

BAFF Elevation in Patients with SLE and RA After B Cell Depletion with Rituxan Treatment[1]



Adapted from Vallerskog T, et al. Arthritis Res Ther 2006;8:R167.

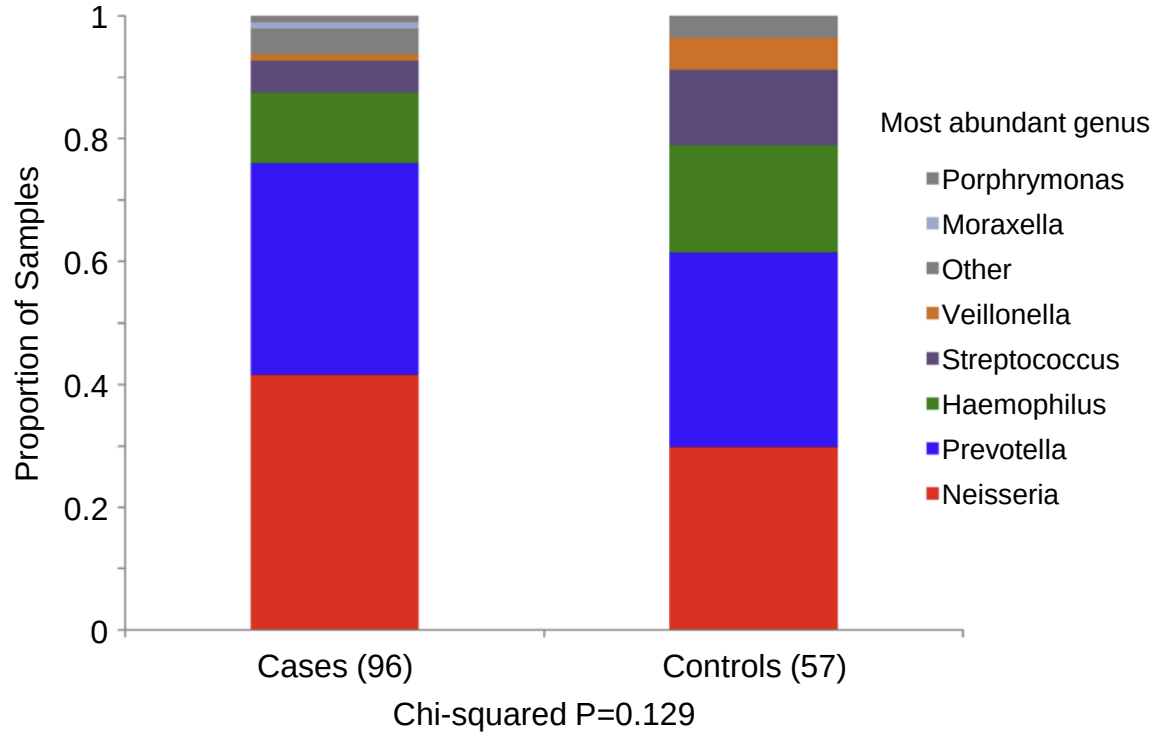
BAFF Elevation in a Patient with APRIL Deficiency (CVID)[2]

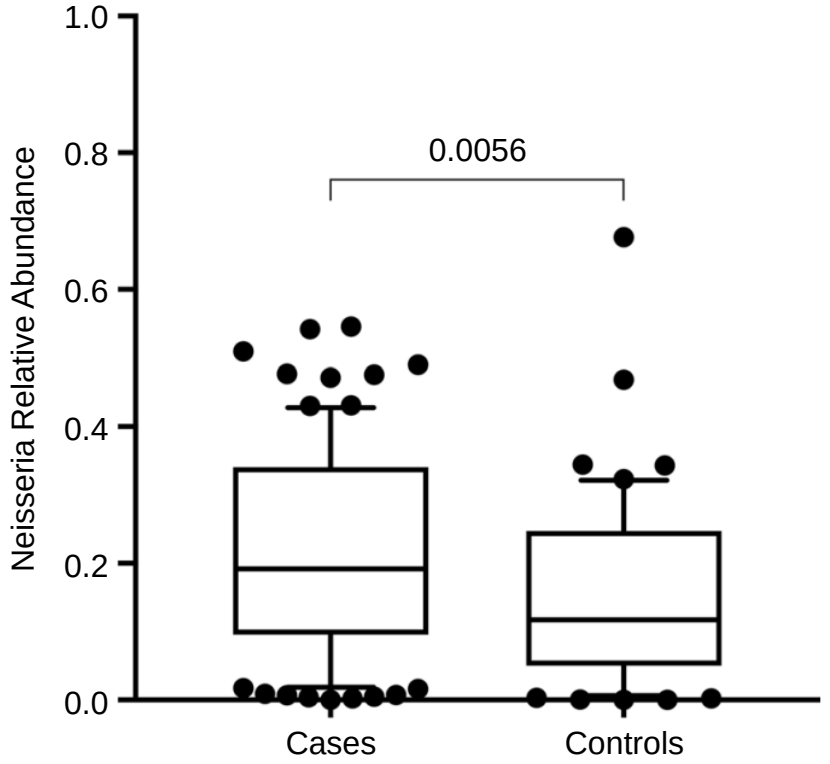


Adapted from Yeh T-W, et al. J Allergy Clin Immunol 2020;146:1109-20.e4.

XLA = X-linked agammaglobulinemia.

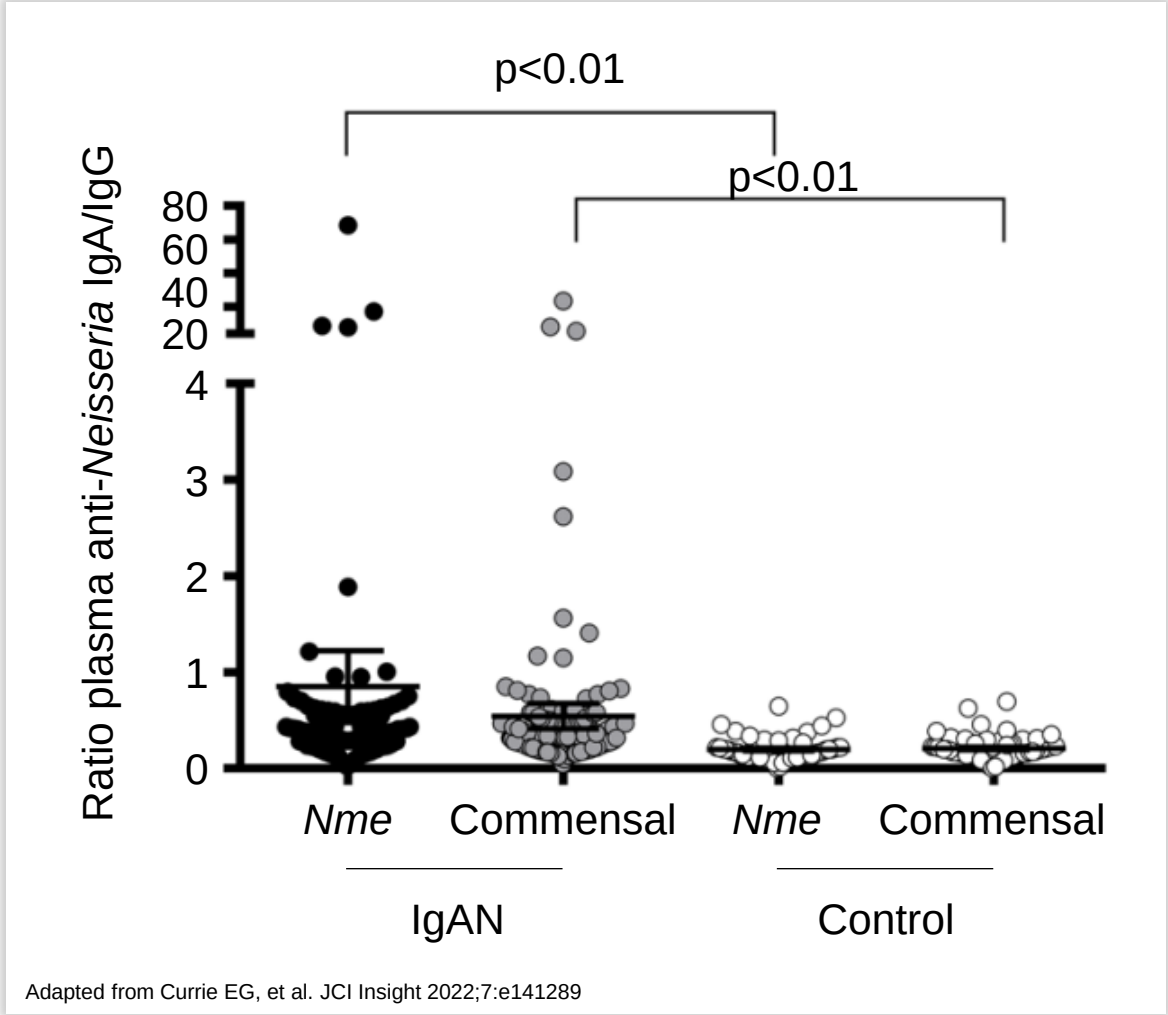
Alterations in Tonsillar Microbiome of IgAN Patients





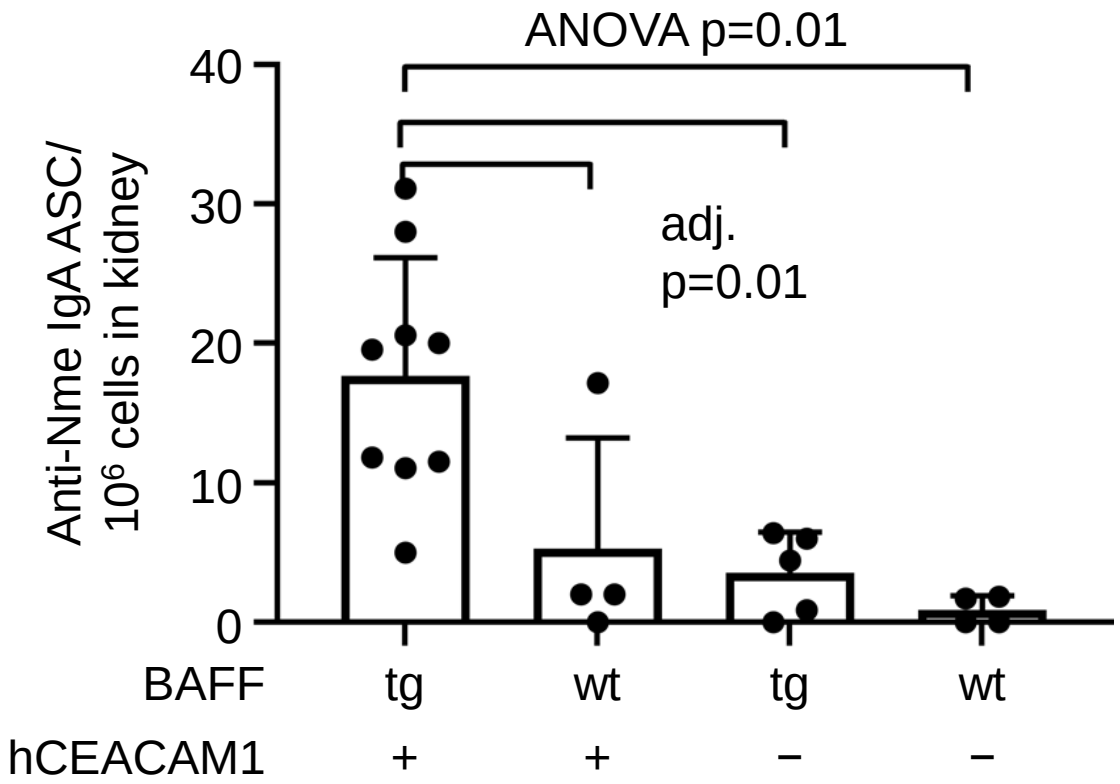
Adapted from Currie EG, et al. JCI Insight 2022;7:e141289

Enhanced Anti-commensal IgA Response in IgAN Patients



Adapted from Currie EG, et al. JCI Insight 2022;7:e141289

N. Meningitidis-reactive IgA Cells in Kidneys of BAFF Transgenic/Nme Infected Mice



Adapted from Currie EG, et al. JCI Insight 2022 Mar 8;7(5):e141289.
ASC = antibody-secreting cell; hCEACAM = human carcinoembryonic antigen-related cell adhesion molecules; Nme = *Neisseria meningitidis*.

Conclusions: Dual Blockade of BAFF and APRIL May Play an Important Role in the Treatment of IgAN

- BAFF and APRIL levels are both elevated in patients with IgAN and are each associated with clinical severity[1-3]
- In preclinical studies, overexpression of BAFF alone can lead to the development of kidney IgA deposits and IgA-like nephropathy[4] and can directly increase the expression of factors associated with fibrosis and inflammation in mesangial cells[2]
- Dual blockade of BAFF and APRIL decreased renal damage in an immunologic animal model more than individual blockade of either pathway alone[5]
- BAFF or APRIL alone are each capable of independently supporting plasma cell survival, indicating dual blockade may be necessary for maximal and sustained clinical efficacy[5,6]
- Blocking both biologic targets (BAFF and APRIL) may avoid compensatory increase in parallel signal[7,8]

BAFF and APRIL are key factors in the production of Gd-IgA1, autoantibodies and thus immune complexes; dual blockade of these cytokines offer promise as a potential treatment for IgAN

1. Xin G, et al. J Nephrol 2013;26:683-90; 2. Cao Y, et al. Mol Med Rep 2020;21:795-805; 3. Zhai Y-L, et al. Medicine 2016;95:e3099; 4. McCarthy D, et al. J Clin Invest 2011;121:3991-4002;
5. Haselmayer P, et al. Eur J Immunol 2017;47:1075-85; 6. Benson M, et al. J Immunol 2008;180:3655-9; 7. Yeh T-W, et al. J Allergy Clin Immunol 2020;146:1109-20.e4; 8. Vallerskog T, et al. Arthritis Res Ther 2006;8:R167.

Emerging Therapies in IgA Nephropathy: Targeted Mechanisms and Clinical Implications

Richard Lafayette, MD, FACP

Professor, Medicine (Nephrology), Director,
Glomerular Disease Center,
Stanford University Medical Center

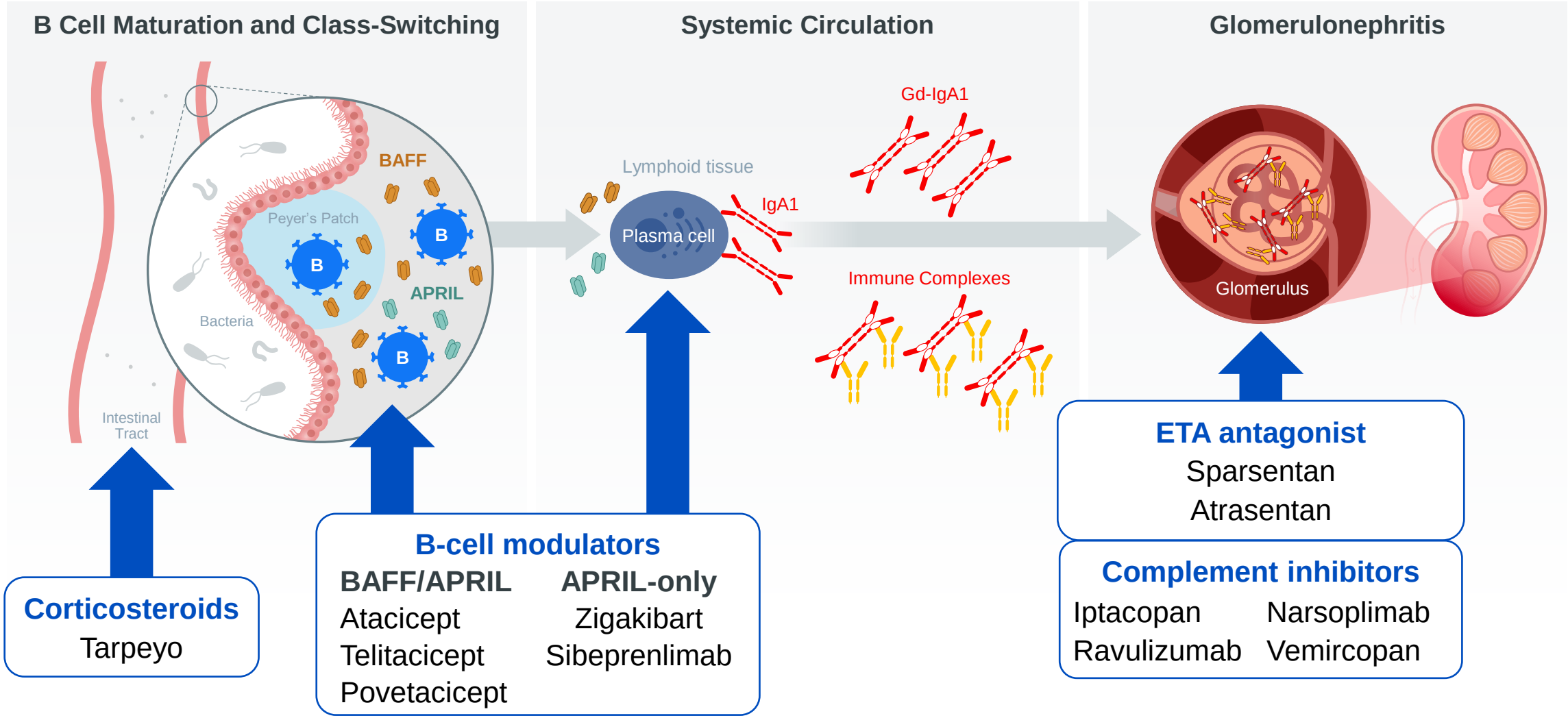
vera therapeutics

COI Disclosure

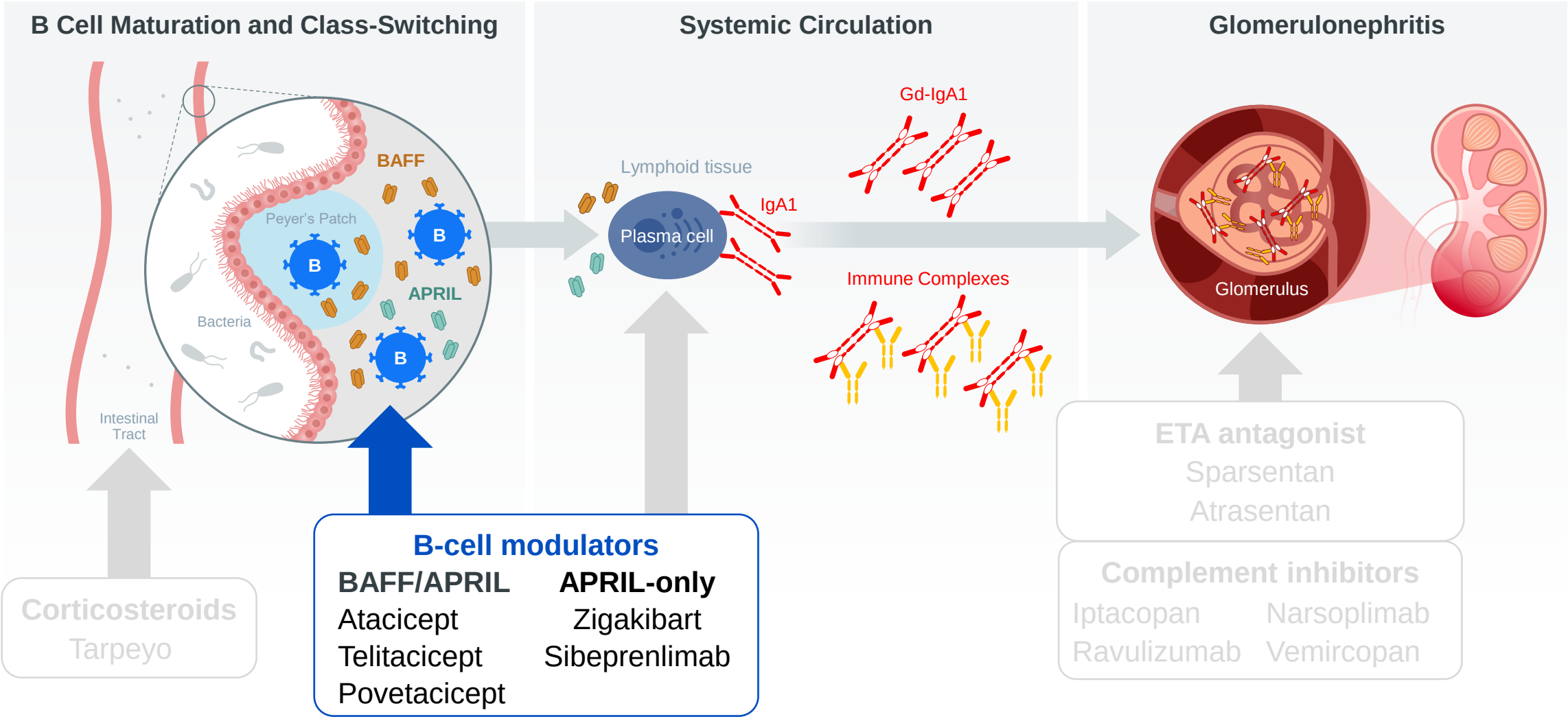
I have the following relationships to disclose any COI for this presentation within the period of 36 months.

- Consultant for Vera, Omeros, Calliditas, Chinook, Alexion, Otsuka, Novartis, GSK, Alnylam
- Employee of Stanford University Medical Center, which has received research funding from Vera, Omeros, ChemoCentryx, Chinook, Alexion, Otsuka, Calliditas, Roche, NIH, and University of Michigan

IgAN Therapies in Clinical Development



IgAN Therapies in Clinical Development

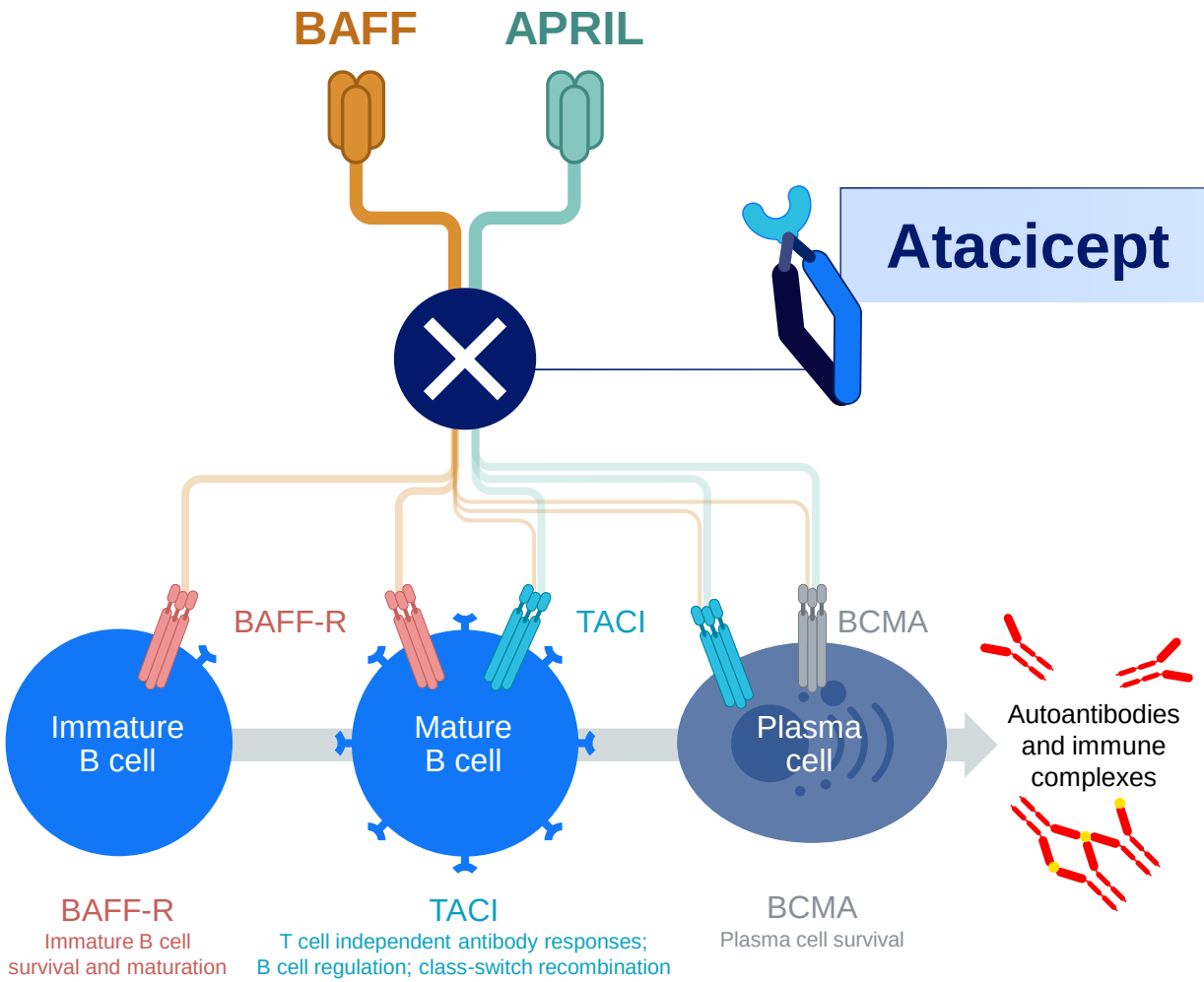


B-cell Modulators in Development for IgAN Treatment

Investigational Agent	Mechanism of Action	Development Phase	Primary Endpoint
Atacicept	Dual BAFF/APRIL inhibitor	Phase 3: ORIGIN 3	UPCR change from baseline at week 36
Telitacicept	Dual BAFF/APRIL inhibitor	Phase 3 (China only)	UPCR change from baseline at week 39; Annualized change in eGFR slope at week 104
Povetacicept	Dual BAFF/APRIL inhibitor	Phase 1b: RUBY-3	AEs through 30 days of last dose of study drug
Zigakibart	APRIL inhibitor	Phase 3: BEYOND	UPCR change from baseline at week 40
Sibeprenlimab	APRIL inhibitor	Phase 3: Visionary	UPCR change from baseline at 9 months

Clinicaltrials.gov

Atacicept is a Dual Inhibitor (BAFF and APRIL) of Plasma Cells and B Cells with Potential to Address Multiple Autoimmune Diseases

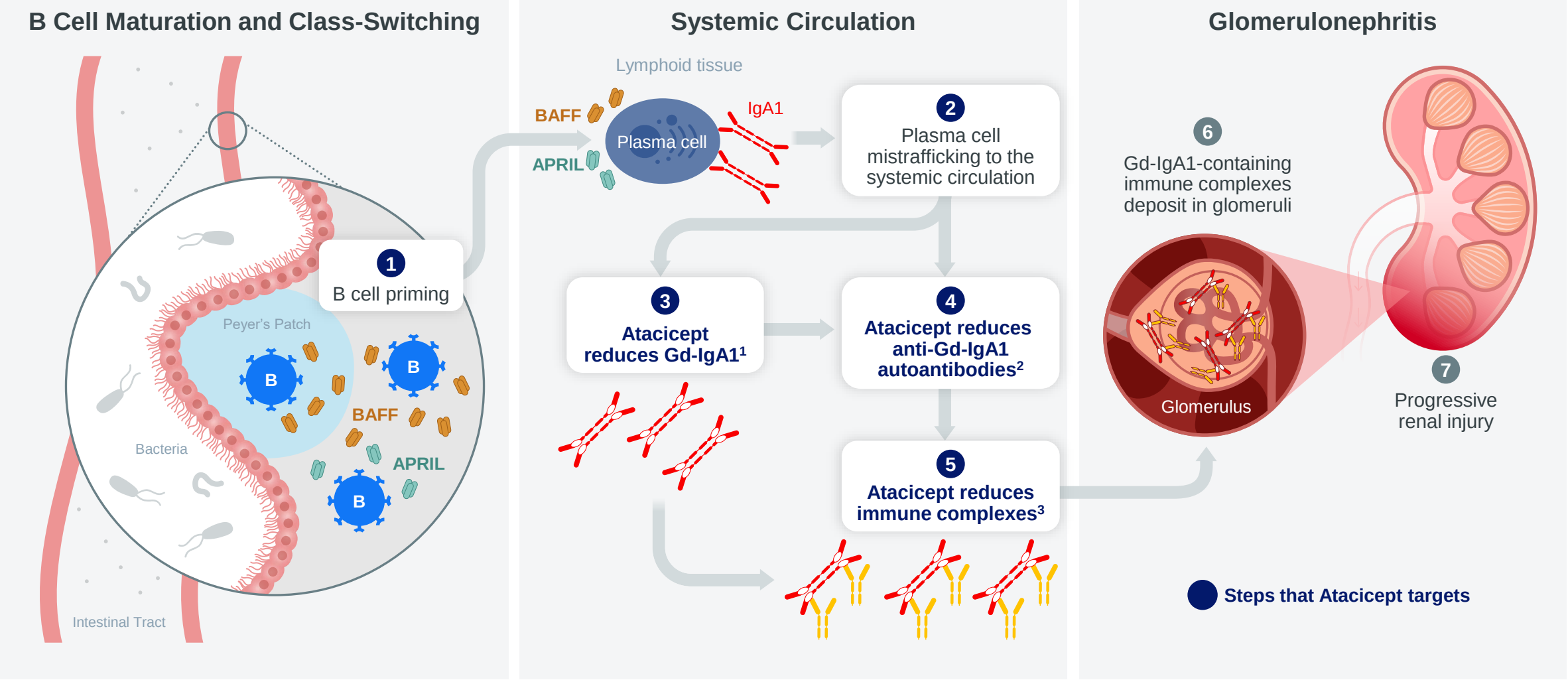


Atacicept

- Fully humanized TACI-Ig fusion protein, subcutaneously administered
- Low nanomolar potency vs BAFF (Kd 1.45 nM) and APRIL (Kd 0.672 nM)
- Atacicept targets long-lived plasma cells[1], in addition to B cells, thus reducing autoantibody production[2]
- Well-characterized safety profile with exposure in >1500 patients across different indications[3]
- Self-administration of small volume (1 mL) injection

1. Hiepe F, et al. Nat Rev Rheumatol 2011;3:170-178. 2. Gordon C, et al. Arthritis Rheumatol 2017;69:122-30; Gordon C, et al. Rheumatol Adv Pract 2019;0:1-12.
APRIL = A Proliferation-Inducing Ligand; BAFF = B-cell Activating Factor.

Atacicept Targets Upstream Hits of IgAN Pathogenesis

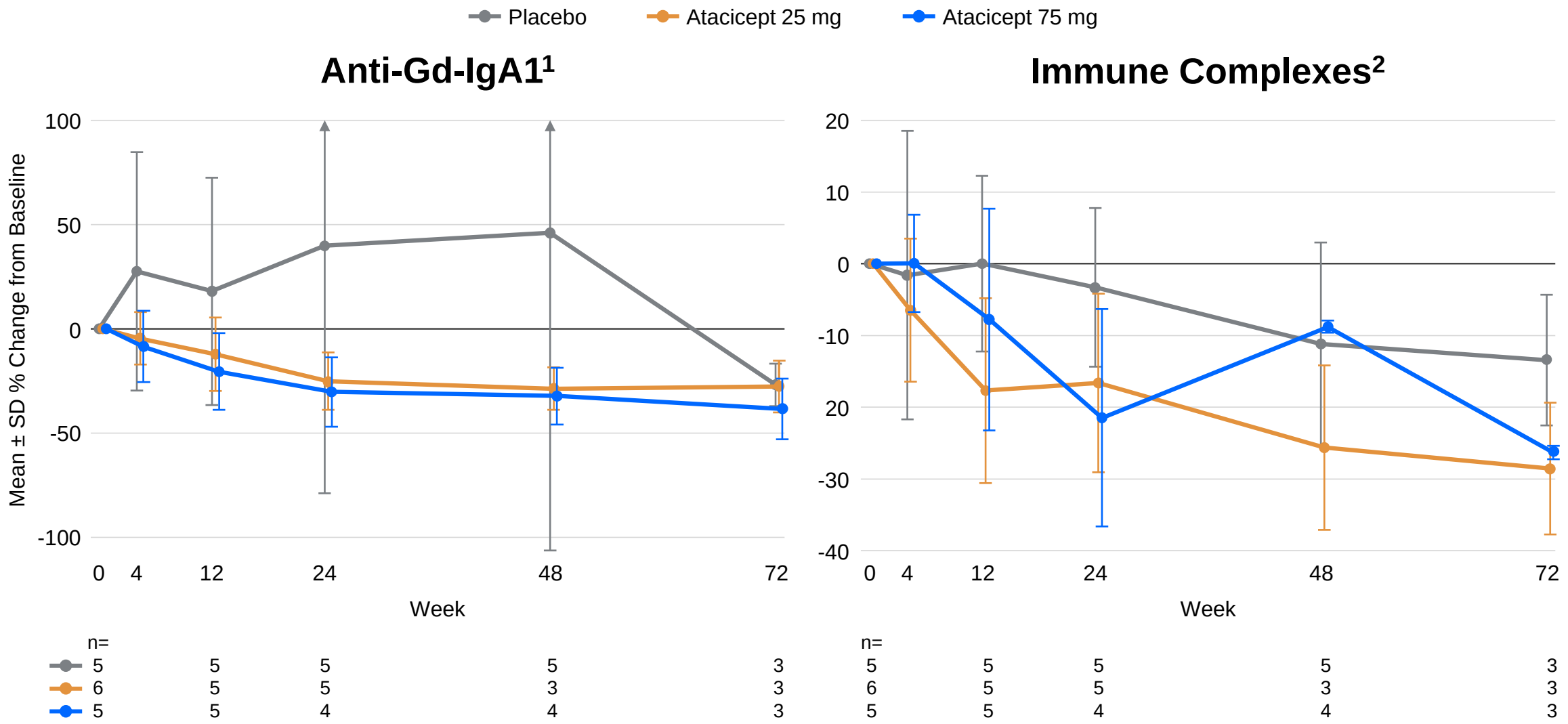


Gd-IgA1 = galactose-deficient immunoglobulin A1.

1. Vera Therapeutics Jan 30 2023 press release; 2. Barratt J, et al. Nephrol Dial Transplant 2022;3 suppl 3, abstr FC051; 3. Barratt J, et al. ASN Kidney Week 2022, abstr SA-PO655.

Phase 2a JANUS Study

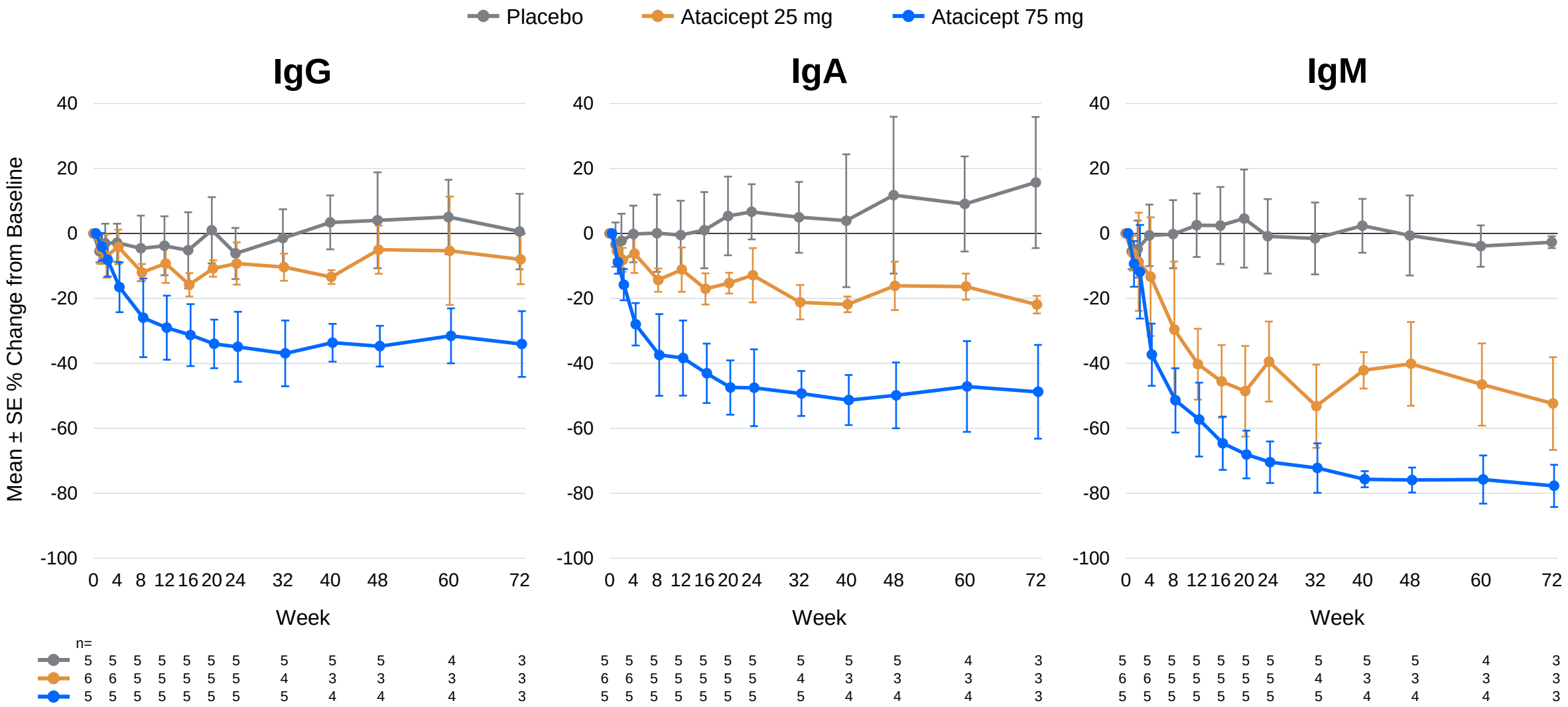
Percent Change from Baseline in Anti-Gd-IgA1 by Visit



1. Barratt J, et al. Nephrol Dial Transplant 2022;3 suppl 3, abstr FC051; 2. Barratt J, et al. ASN Kidney Week 2022, abstr SA-PO655.

Phase 2a JANUS Study

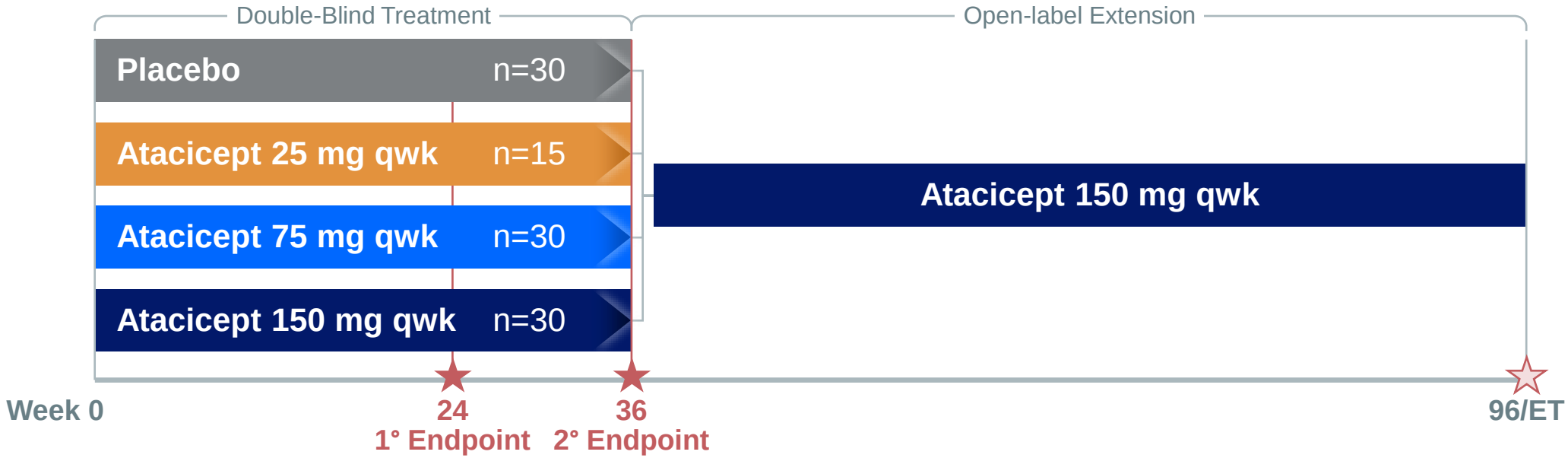
Serum IgG, IgA, and IgM % Change Through Week 72



Barratt J, et al. Kidney Int Rep 2022;7:1831-41.

ORIGIN Phase 2b IgAN Trial: Study Design and Objectives

Multinational, randomized, placebo-controlled trial



Inclusion Criteria

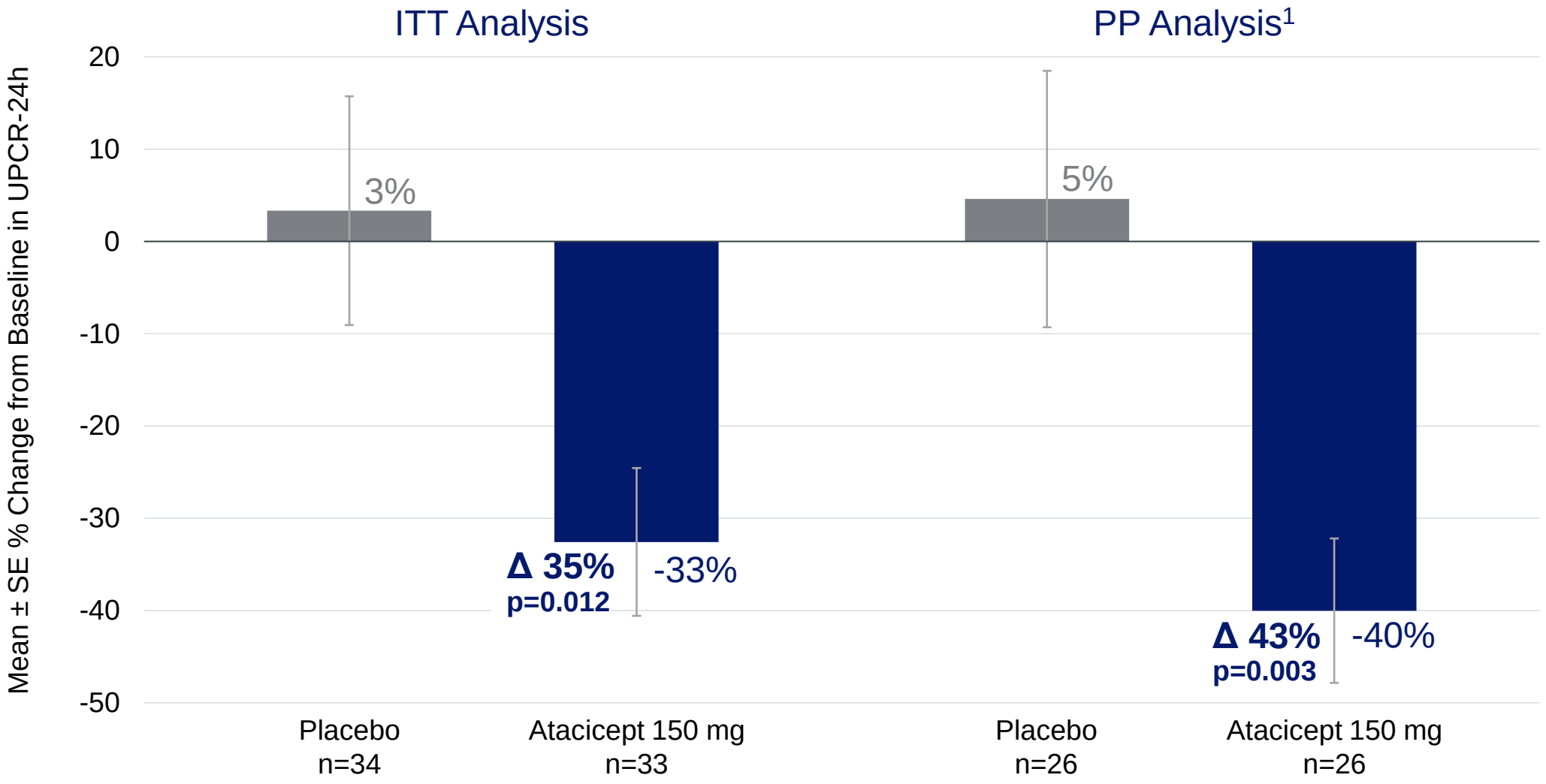
- Patients ≥18 years old with IgAN on renal biopsy and high risk of disease progression
- Stable and optimized RAASi for 12 weeks
- Use of SGLT2i allowed
- UPCR-24h >0.75 g/g or UP >0.75 g per 24h
- eGFR ≥30 mL/min/1.73 m^2
- Blood pressure ≤150/90 mmHg

Endpoints

- Primary efficacy: UPCR-24h at week 24 ★
- Key secondary: UPCR-24h at week 36 ★
- eGFR change up to week 96 ☆
- Gd-IgA1 change
- Safety

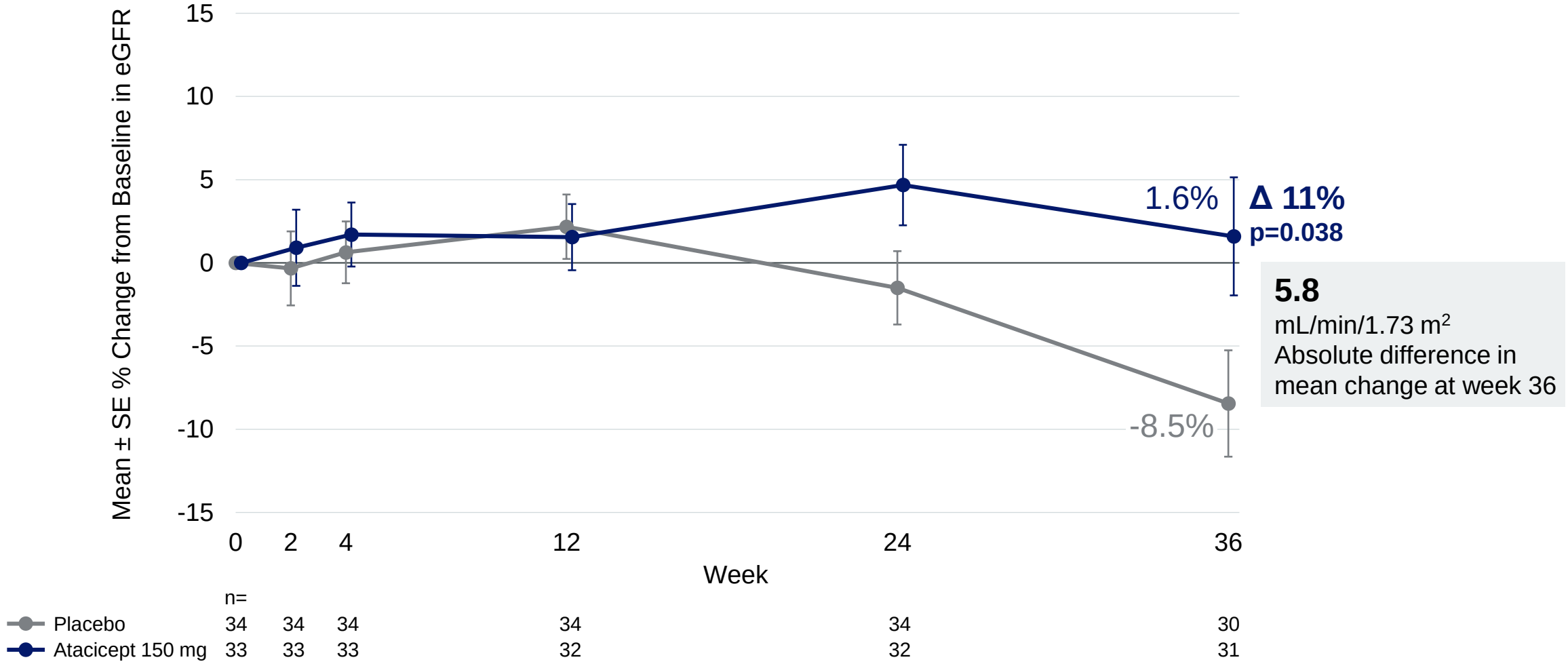
ET = end of treatment; RAASi = renin-angiotensin-aldosterone system inhibitor; SGLT2i = sodium-glucose cotransporter-2 inhibitor.

UPCR % Change with Atacicept 150 mg at Week 36



p-values, percentage changes from baseline, and treatment differences were computed using FDA-endorsed mixed-effects modeling which takes into account the effects of baseline UPCR and eGFR.
1. PP analysis excludes patients with protocol violations identified at week 36 data-cut prior to unblinding.

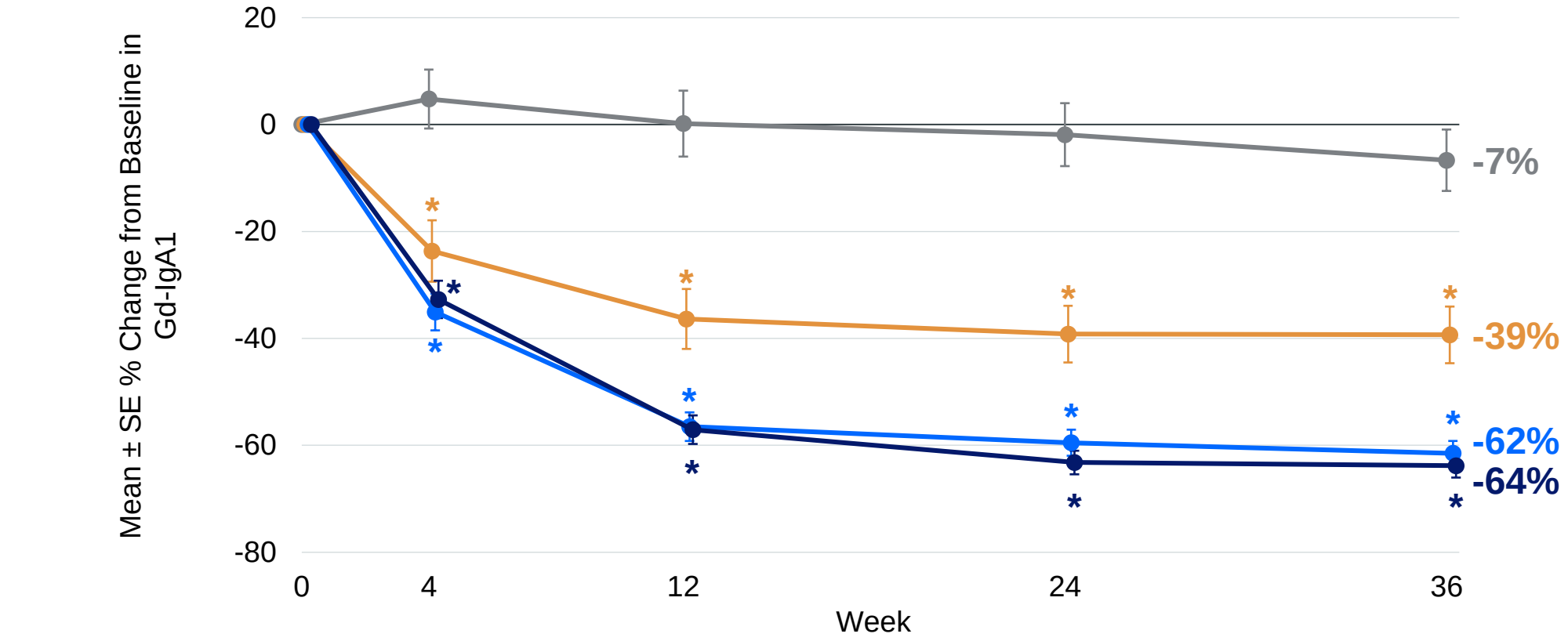
eGFR Change with Atacicept 150 mg Through Week 36



5.8
mL/min/1.73 m^2
Absolute difference in mean change at week 36

ITT analysis; p-values, percentage changes from baseline, and treatment differences were computed using FDA-endorsed mixed-effects modeling.

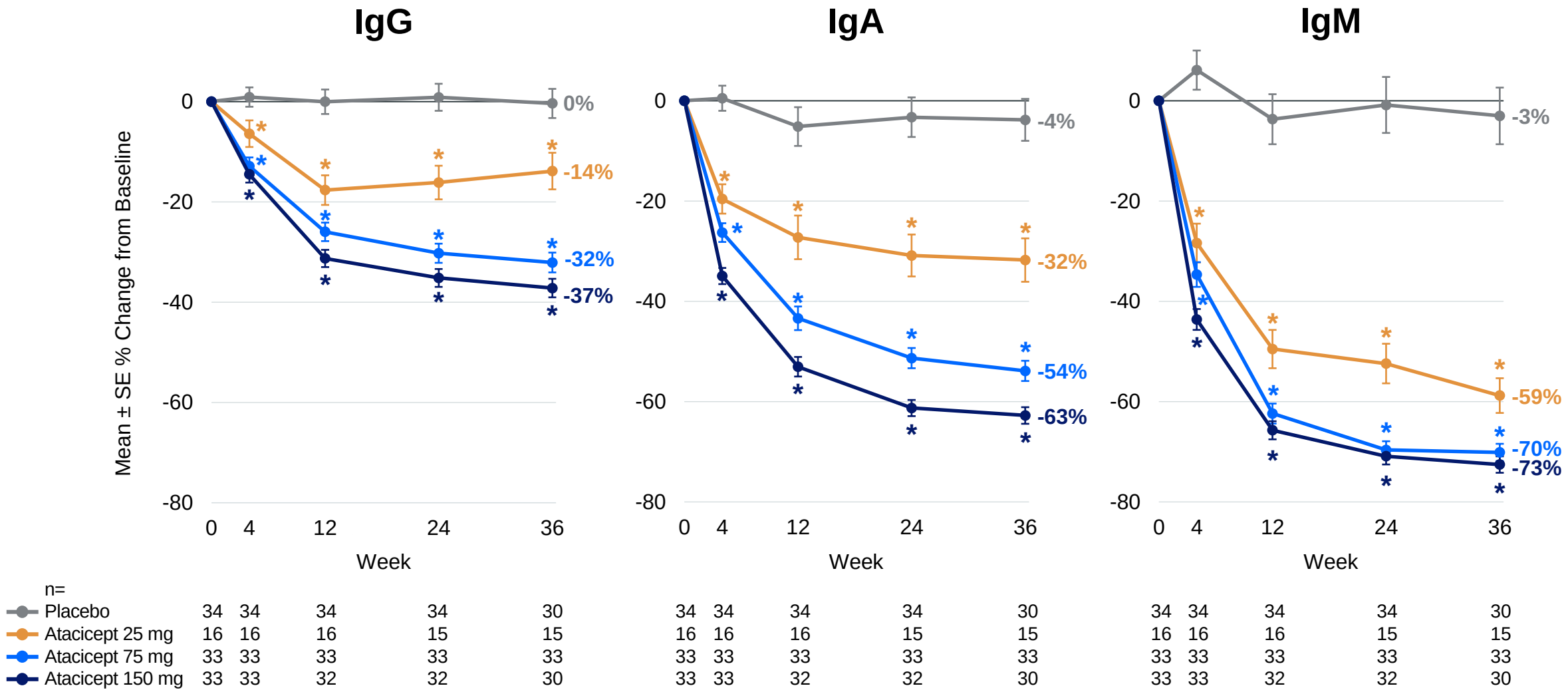
Gd-IgA1 % Change Through Week 36



n=					
Placebo	33	33	33	33	29
Atacicept 25 mg	16	16	16	15	15
Atacicept 75 mg	33	33	33	33	33
Atacicept 150 mg	32	32	30	30	30

ITT analysis; *p<0.05 vs placebo. p-values, percentage changes from baseline, and treatment differences were computed using FDA-endorsed mixed-effects modeling.

Dose-dependent Reductions Observed in Serum IgG, IgA, and IgM Through Week 36



ITT analysis; *p<0.05. p-values, percentage changes from baseline, and treatment differences were computed using FDA-endorsed mixed-effects modeling.

Treatment-Emergent Adverse Events Through Week 36

Patients, n (%)	Atacicept 25 mg n=16	Atacicept 75 mg n=33	Atacicept 150 mg n=33	Placebo n=34
TEAEs	11 (69)	24 (73)	25 (76)	27 (79)
Study drug-related TEAEs[1]	6 (38)	17 (52)	19 (58)	14 (41)
Serious TEAEs	0	1 (3)[2]	1 (3)[3]	3 (9)[4]
TEAEs leading to study drug discontinuation	0	0	1 (3)[5]	1 (3)[6]
Deaths	0	0	0	0

1. Majority of study drug-related TEAEs were injection site reactions; one contributed to drug discontinuation.
2. Multiple fractures, resolved, unrelated to study treatment.
3. Gastroenteritis norovirus, resolved, unrelated to study treatment.
4. Anaphylactic reaction resolved (n=1); forearm fracture resolved (n=1); flank pain not resolved and ulnar nerve paralysis resolved with sequelae (n=1); all unrelated to study treatment.
5. Discontinued after 3 injections due to injection site reaction and positive hepatitis B DNA that resolved without treatment 3 weeks later, normal liver function throughout.
6. Discontinued after 31 injections due to worsening flank pain that was not resolved; unrelated to study treatment.

Summary of Clinical Safety Data Through Week 36

- Atacicept was generally well tolerated in IgAN patients, with no reported deaths, low rate (2%) of serious AEs overall, and 1 patient (1%) discontinued atacicept due to AE
- Infections were balanced between atacicept and placebo
- No study drug-related serious AE in atacicept 150 mg group
- No patient had study drug discontinuation or interruption due to hypogammaglobulinemia

Prior Integrated Safety Analysis

Atacicept Safety Tolerability Profile in Over 1,000 Patients from Prior Trial Experience in Other Indications

Summary of AEs >5% in any arm, by dose in the double-blind placebo-controlled set

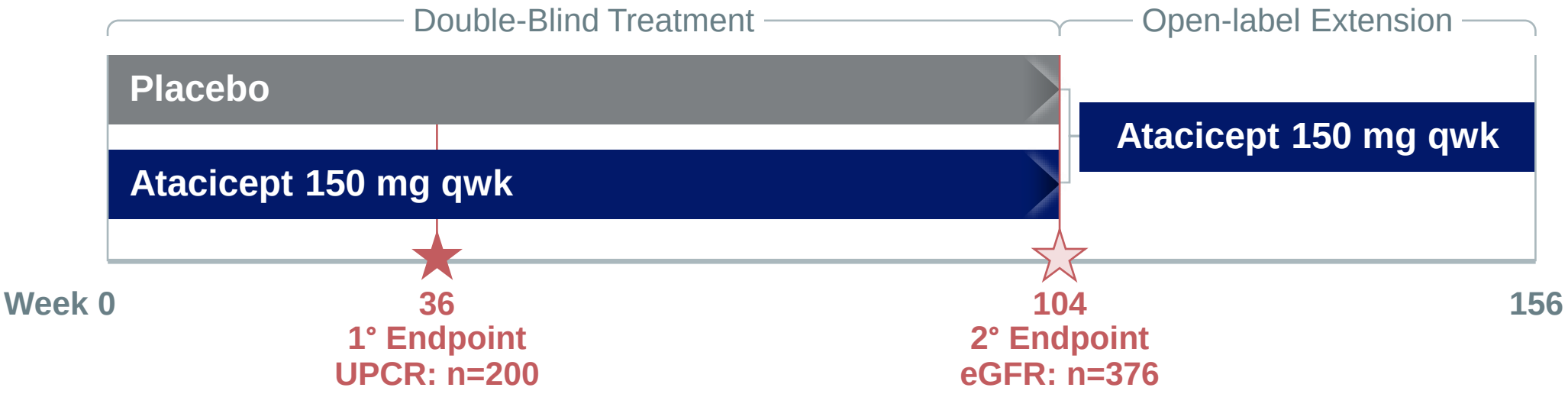
Patients, %	Overall n=1568	Atacicept 25 mg n=129	Atacicept 75 mg n=384	Atacicept 150 mg n=572	Placebo n=483
Discontinuation due to AE	8	11	8	8	6
Serious AE	11	12	13	11	11
Severe AE	9	8	12	10	6
Infections	46	33	47	49	44
Serious infections	4	1	6	4	4
Hypersensitivity	9	6	10	10	8
Injection site reactions	22	21	28	27	11
Cardiac arrhythmias	5	9	6	4	4
Vestibular disorders	4	4	5	5	4

- A total of 1,000+ patients have received at least 1 dose of atacicept across different indications including two large SLE studies and a long-term extension study (as of April 2023)
- Exposure-adjusted incidence rates of serious infection and serious treatment emergent adverse event rates were similar between atacicept and placebo
- No association between risk of infection and magnitude of pharmacodynamic effects with atacicept
- Clinical trials require standard risk mitigation including up-to-date vaccinations, eligibility review by medical monitor, and education on early detection of signs/symptoms of infection

Adapted from Gordon C, et al. Rheumatol Adv Pract 2019;0:1-12.

Initiated Phase 3 Pivotal Trial in June 2023

Atacicept Once Weekly SC Injection Formulation in Phase 3, Same as Phase 2b



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- eGFR ≥30 mL/min/1.73 m^2
- Blood pressure ≤150/90 mmHg

Endpoints

- Primary efficacy: UPCR-24h at week 36 ★ to support potential accelerated approval
- Key secondary: eGFR change up to week 104 ☆
- Safety

Summary

- Treatment landscape is changing rapidly in IgAN
- In IgAN, overactive B-cells lead to elevated serum levels of Gd-IgA1, autoantibodies, and immune complexes which progressively damage the kidneys
- New therapies in development with dual inhibition of BAFF and APRIL may provide additional disease modifying therapeutic options

Thank you

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