Exhibit 99.1

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LifeSci Capital Fireside Chat

December 8, 2022

Forward-looking statements

This presentation contains forward-looking statements for the purposes of the safe harbor provisions under The Private Securities Litigation Reform Act of 1995 and other federal securities laws. The use of words such as "can," "may," "might," "will," "would," "should," "expect," "believe," "estimate," "design," "plan," "intend," and other similar expressions are intended to identify forward-looking statements. Such forward looking statements include Immunovant's expectations regarding patient enrollment, timing, design, and results of clinical trials of its product candidates and indication selections; Immunovant's plan to develop batoclimab and IMVT-1402 across a broad range of autoimmune indications; expectations with respect to the safety and monitoring plan and size of the safety database for these planned clinical trials; the timing of discussions with regulatory agencies; the size and growth of the potential markets for Immunovant's product candidates and indication selections; Immunovant's plan to explore in subsequent study periods follow-on treatment with alternative dosing regimens; Immunovant's expectations regarding its cash runway; Immunovant's beliefs regarding the potential benefits of batoclimab's and IMVT-1402's unique product attributes; and Immunovant's expectations regarding the issuance and term of any pending patents. All forward-looking statements are based on estimates and assumptions by Immunovant's management that, although Immunovant believes to be reasonable, are inherently uncertain. All forward-looking statements are subject to risks and uncertainties that may cause actual results to differ materially from those that Immunovant expected. Such risks and uncertainties include, among others: initial results or other preliminary analyses or results of early clinical trials may not be predictive of final trial results or of the results of later clinical trials; results of animal studies may not be predictive of results in humans; the timing and availability of data from clinical trials; the timing of discussions with regulatory agencies, as well as regulatory submissions and potential approvals; the continued development of Immunovant's product candidates, including the timing of the commencement of additional clinical trials and resumption of current trials; Immunovant's scientific approach, clinical trial design, indication selection, and general development progress; future clinical trials may not confirm any safety, potency, or other product characteristics described or assumed in this presentation; any product candidate that Immunovant develops may not progress through clinical development or receive required regulatory approvals within expected timelines or at all; Immunovant's pending composition of matter patent for IMVT-1402 may not be issued; Immunovant's product candidates may not be beneficial to patients, or even if approved by regulatory authorities, successfully commercialized; the potential impact of the ongoing COVID-19 pandemic, geopolitical tensions, and adverse macroeconomic conditions on Immunovant's clinical development plans and timelines; Immunovant's business is heavily dependent on the successful development, regulatory approval and commercialization of batoclimab and IMVT-1402; Immunovant is at an early stage in development for IMVT-1402 and in various stages of clinical development for batoclimab; and Immunovant will require additional capital to fund its operations and advance batoclimab and IMVT-1402 through clinical development. These and other risks and uncertainties are more fully described in Immunovant's periodic and other reports filed with the Securities and Exchange Commission (SEC), including in the section titled "Risk Factors" in Immunovant's most recent Annual Report on Form 10-K, its Form 10-Q filed with the SEC on November 4, 2022, and Immunovant's subsequent filings with the SEC. Any forward-looking statement speaks only as of the date on which it was made. Immunovant undertakes no obligation to publicly update or revise any forward-looking statement, whether as a result of new information, future events or otherwise.

 Dates used in this presentation refer to the applicable calendar year unless otherwise noted. No clinical head-to-head comparisons of Immunovant product candidates and third party anti-FcRn assets has been conducted.

Building a leading anti-FcRn franchise to address unmet need

Multiple paths to potential value creation with batoclimab and IMVT-1402

Compound	Target Indication / Therapeutic Area	Stage of Development
Batoclimab	Myasthenia Gravis (MG)	Pivotal Phase 3
	Thyroid Eye Disease (TED)	Pivotal Phase 3
	Chronic Inflammatory Demyelinating Polyneuropathy (CIDP)	Pivotal Phase 2b*
	Graves' Disease (GD)	Phase 2
	Warm Autoimmune Hemolytic Anemia (WAIHA)	Phase 2**
IMVT-1402	Rheumatology, Hematology***, and potentially Graves' Disease	Pre-clinical

Based on clinical development achievements to date in MG, we have incurred a $10M milestone payment obligation to HanAll in CYQ4 2022 which was already considered in our cash runway guidance

*Registrational package for CIDP may include 1 or 2 pivotal trials depending on a variety of factors

**WAIHA design to be finalized based on recent FDA interaction

***Including potentially WAIHA

Today's agenda/discussion topics

- Overview of the neonatal Fc receptor (FcRn)
- Anti-FcRn antibody basics – structure, affinity, etc.
- Translatability of albumin and LDL signals from non-human primates to humans
- Correlation of IgG reduction and clinical efficacy in autoimmune diseases
- CIDP trial design

FcRn Basics

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For some autoimmune diseases, IgG autoantibodies cause disease pathology directly or via immune complexes

	IgG autoantibodies can be directly pathogenic	IgG autoantibodies may also lead to pathogenic immune complexes
Characteristics	• In 'classic' auto-antibody mediated diseases, auto-antibodies tend to be highly specific to extracellular antigens • Most patients have these pathogenic auto-abs, and detection is an integral part of diagnosis	• In more complex autoimmune diseases, auto-antibodies are directed towards intracellular or circulating antigens • Auto-antibodies form immune complexes with their antigen (e.g. dsDNA) and may activate innate and adaptive immune system responses, driving aberrant inflammation and end-organ damage
Example Diseases	• Myasthenia gravis (MG) • Thyroid eye disease (TED) • Warm autoimmune hemolytic anemia (WAIHA) • Immune thrombocytopenic purpura (ITP)	• Rheumatoid arthritis (RA) • Primary Sjogren's Syndrome (pSS) • Inflammatory myositis • Certain forms of vasculitis

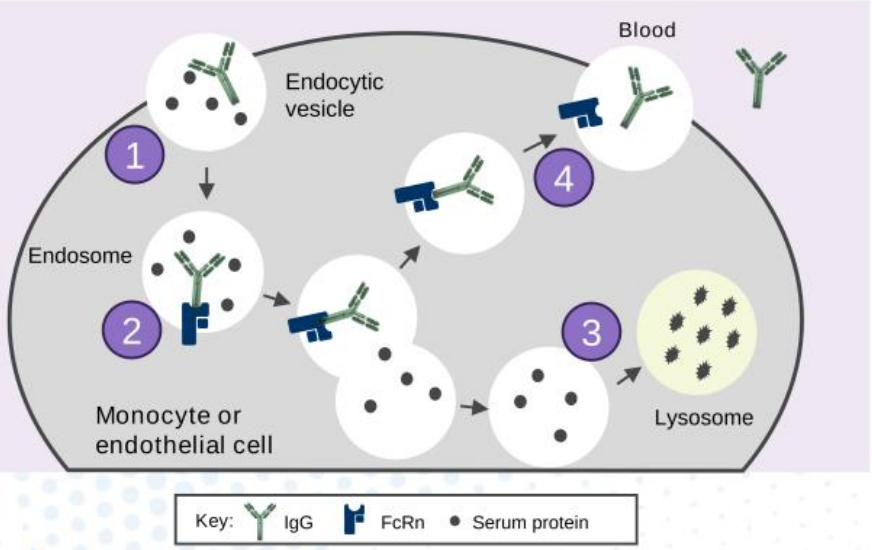
E.g., in TED, antibodies against thyroid stimulating hormone receptor (TSHR) activate orbital fibroblasts, causing proliferation and swelling around the eye

Hypothesis: in treating these diseases, greater IgG reduction may lead to less autoantibody-driven pathogenesis and therefore greater clinical efficacy

The neonatal Fc receptor (FcRn) promotes recycling of IgG antibodies

- FcRn extends the half-life of IgG autoantibodies in circulation exacerbating their autoimmune effects
- FcRn expressed in a variety of cells

FcRn maintains levels of IgG in circulation by preventing IgG degradation



FcRn Mechanism of Action

1. IgG is taken up into cells in endocytic vesicle
2. FcRn-IgG complexes are sorted from unbound proteins
3. Unbound proteins are trafficked to lysosome for degradation
4. IgG is recycled back into circulation

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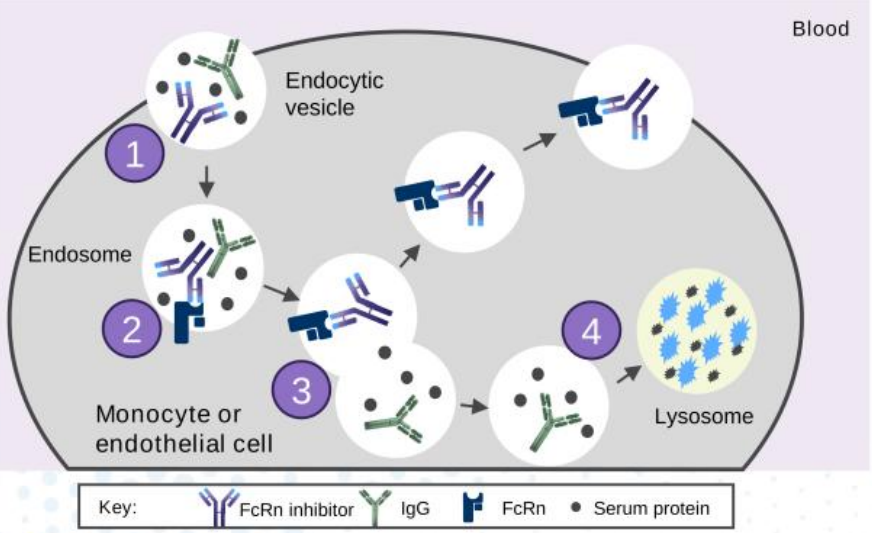
FcRn inhibition promotes IgG degradation

- FcRn inhibition reduces the recycling of IgG antibodies
- As a result, IgG is increasingly delivered to lysosomes for degradation
- Relative to older, broad-spectrum immunosuppressants, FcRn inhibitors deliver a more targeted approach to immunomodulation

FcRn inhibitor blocks binding of pathogenic antibodies to FcRn and promotes their removal and degradation



Mechanism of Action of FcRn Inhibition

1. IgG and FcRn inhibitor are taken up into cells in endocytic vesicles
2. FcRn inhibitor binds to FcRn in endosomes
3. IgGs are blocked from forming complexes with FcRn
4. Non-receptor bound IgGs are degraded in lysosomes

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FcRn Inhibitor

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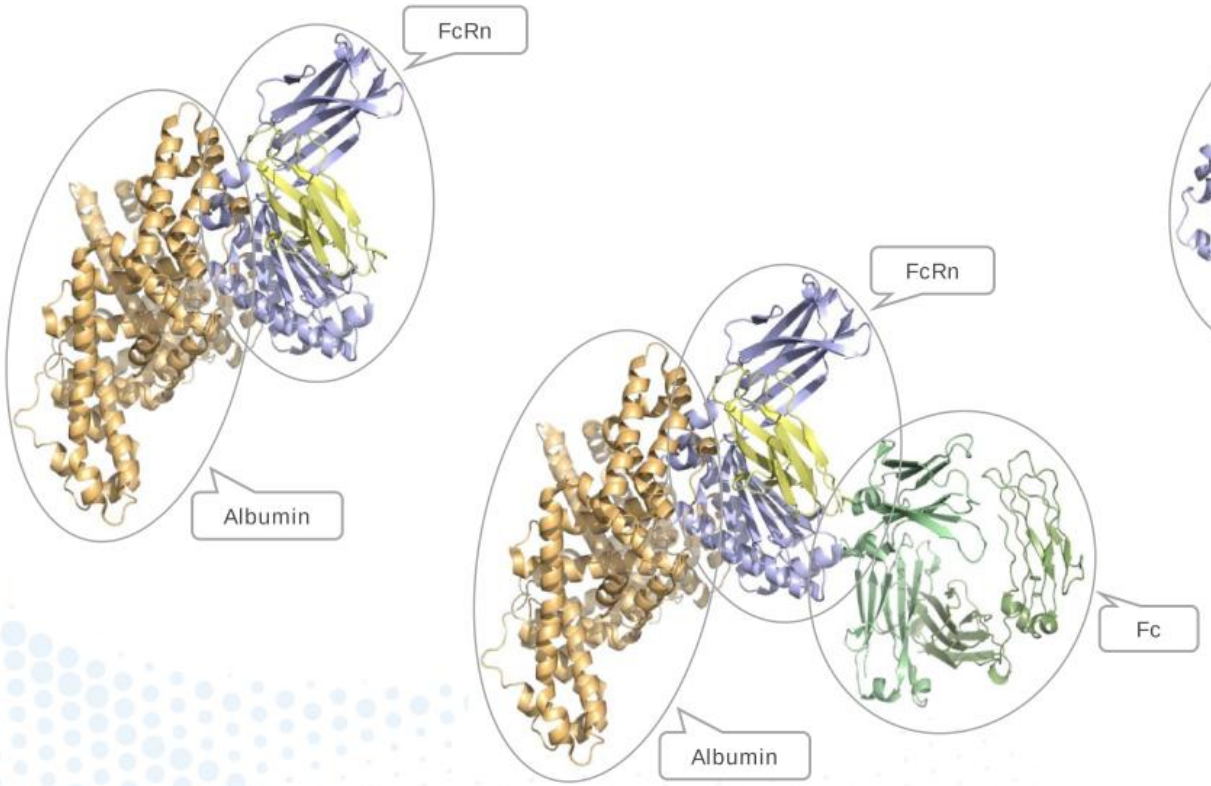
Anti-FcRn assets have unique characteristics

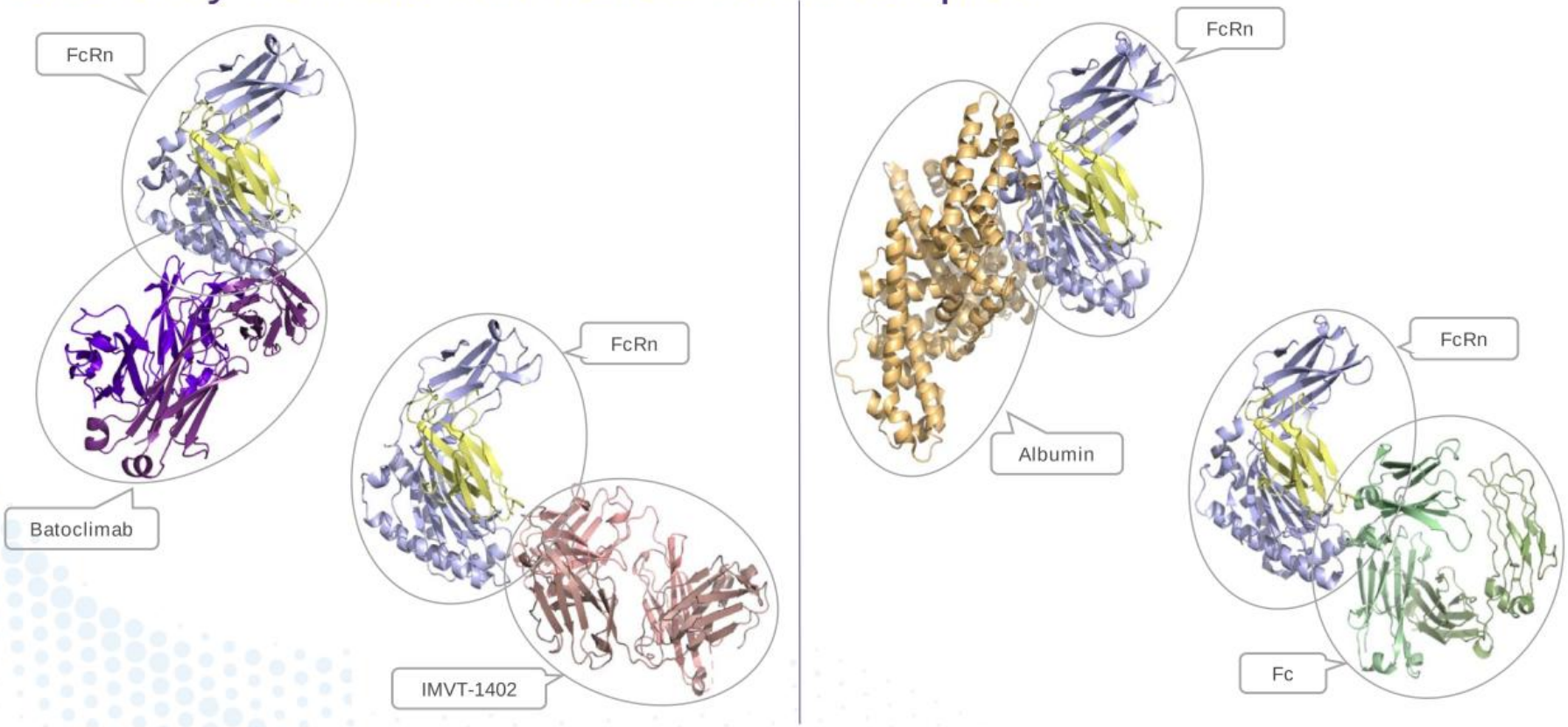
		Batoclimab (IMVT-1401)[1]	IMVT-1402[1]	Efgartigimod[2]	Nipocalimab (M281)[3]	Rozanolixizumab (UCB7665)[4]	ALXN1830/ SYNT001[5]
Company		Immunovant	Immunovant	Argenx	Janssen	UCB	Alexion/AstraZeneca
Structure		Human IgG1	Human IgG1	Human IgG1 frag, Fc mutations	Human IgG1	Humanized IgG4	Humanized IgG4
Fc Effector Potential		No	No	No	No	Low	Low
FcRN-IgG Binding- pH 7.4	Affinity (KD)	1.6 nM +++	0.122 nM +++	320 nM +	0.029 nM ++++	0.023 nM ++++	0.87 nM +++
FcRN-IgG Binding- pH 6.0-6.3	Affinity (KD)	0.56 nM +++	0.129nM +++	14.2 nM ++	0.044 nM ++++	0.034 nM ++++	1.19 nM +++
Human Half-life		10-38 hours	Ph1 study planned for 2023	85-104 hours for 2-50 mg/kg	7.82-33.7 hours		0.636-7.779 hours

No clinical head-to-head comparisons of Immunovant product candidates and third party anti-FcRn assets has been conducted.
Binding affinities are SPR
Sources: 1. On file at Immunovant; 2. Ulrichts 2018; 3.Ling, 2019 (ASH 2015 poster); 4.Smith, 2018; Kiessling, 2017; 5. Blumberg, 2017 (ASH 2017 poster)

The Fc portion of endogenous IgG (Fc) and albumin have different binding sites on the FcRn



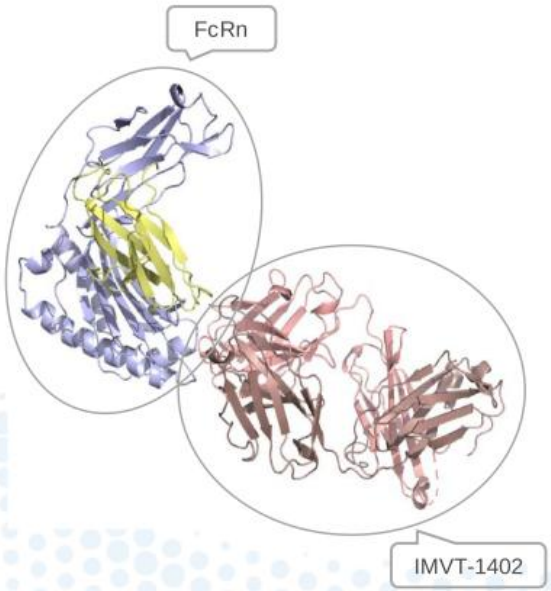
Co-crystallization shows that the batoclimab-FcRn complex orients differently from the IMVT 1402-FcRn complex

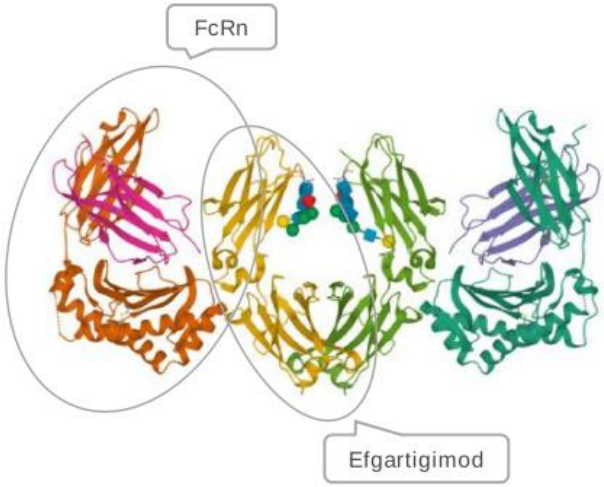


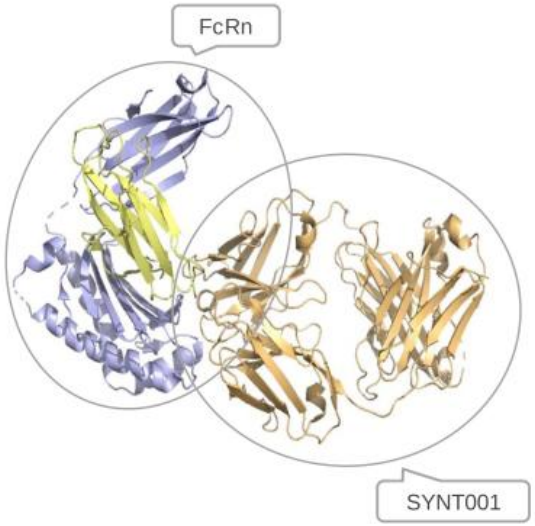
Note: Ribbon representations generated from X-Ray co-crystal structure. Batoclimab solved at 2.4Å resolution. IMVT-1402 solved at 2.6Å resolution.

Co-crystal structures for IMVT-1402, for efgartigimod and for SYNT001 all with FcRn

IMVT-1402	Efgartigimod*	SYNT001**







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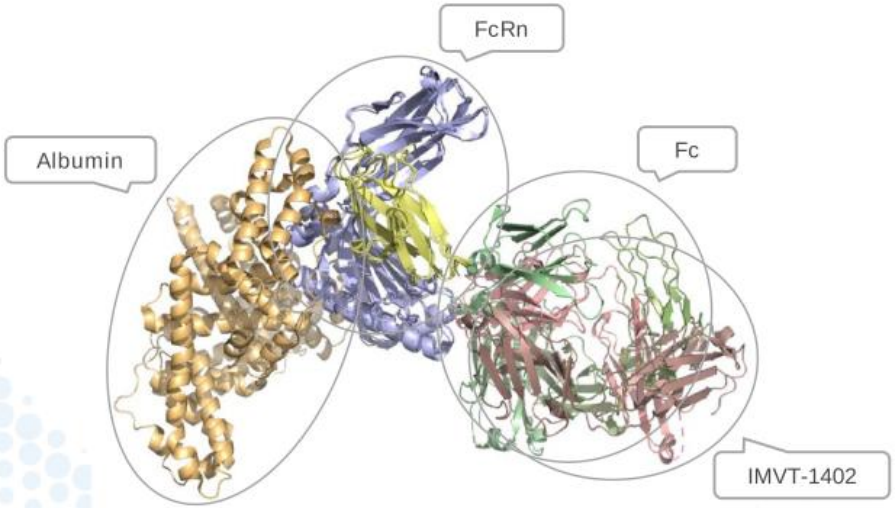
*https://www.rcsb.org/structure/7Q15; **Blumberg et al., Sci. Adv. 2019 Dec 18;5(12):eaax9586.

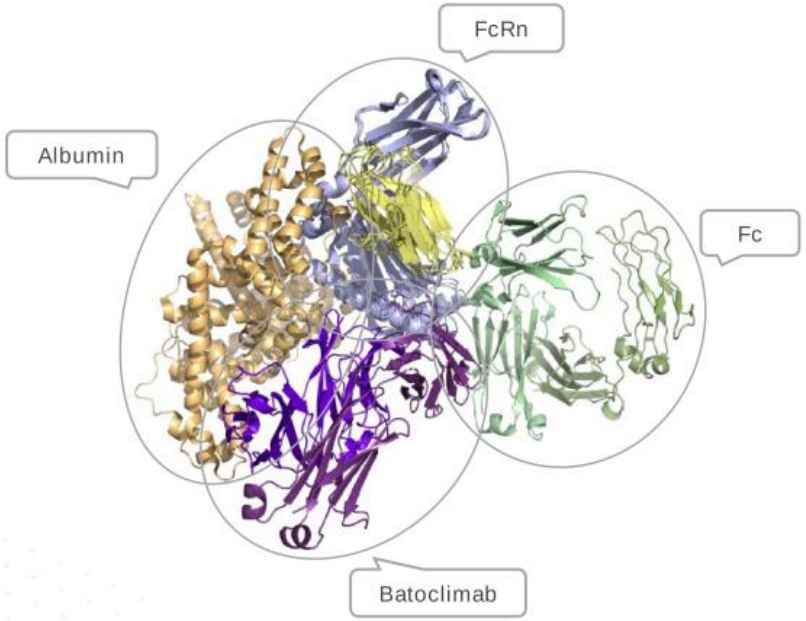
Note that orientation of FcRn is shown a bit differently (based on publicly available data) for efgartigimod vs 1402 and SYNT001

IMVT-1402 was selected to deliver maximum IgG reduction while minimizing interference with the albumin recycling

IMVT-1402: overlay with albumin and Fc



Batoclimab: overlay with albumin and Fc



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Note: Ribbon representations generated from X-Ray crystal structure. Batoclimab solved at 2.4Å resolution. IMVT-1402 solved at 2.6Å resolution.

Translatability of data from cynomolgus monkey to humans

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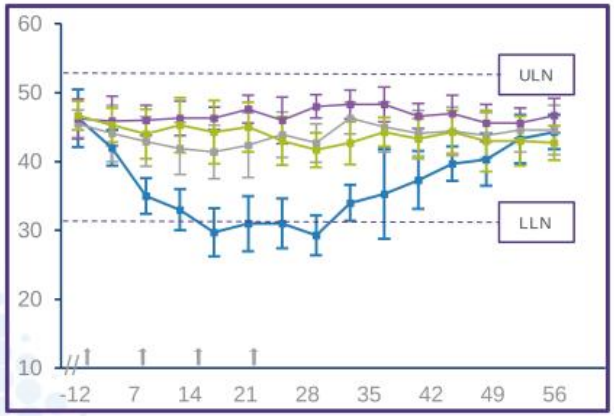
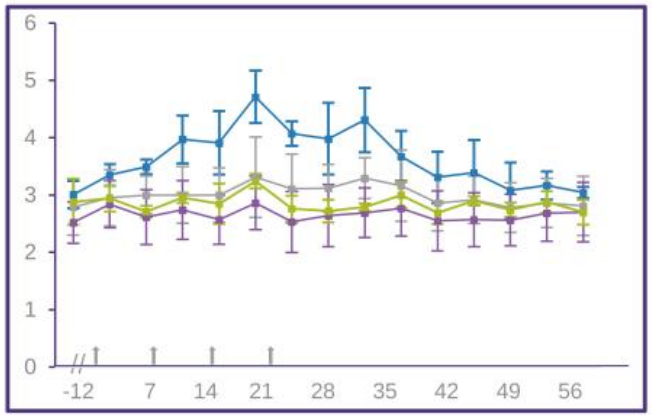
IMVT-1402 and placebo demonstrated similar albumin and LDL

Head-to-Head Monkey Study

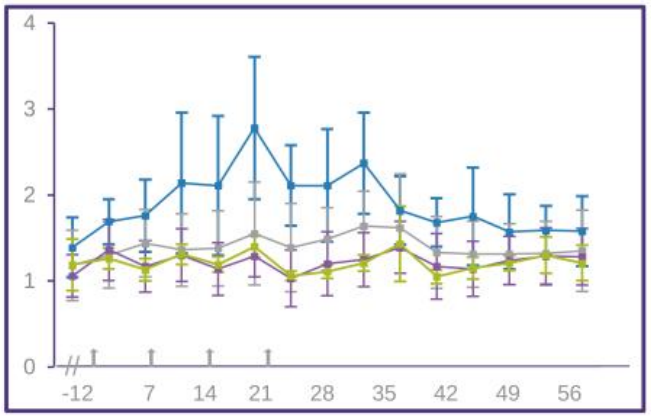
Albumin concentration (g/L), mean ± SD



Cholesterol concentration (mmol/L), mean ± SD



LDL concentration (mmol/L), mean ± SD



- Batoclimab 50 mg/kg (n=3)
- IMVT-1402 50 mg/kg (n=7)
- IMVT-1402 5 mg/kg (n=7)
- Placebo (n=3)

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SD, standard deviation; ULN, upper limit of normal; LLN, lower limit of normal; Arrows indicate time of dosing.
Data on file at Immunovant

Impact on albumin observed in non-human primates has been highly translatable to humans

Evidence of translatability observed across multiple anti-FcRn agents

Product (Company)	Impact on Albumin Levels from Baseline	
	Cynomolgus Monkeys	Clinical Data
Efgartigimod (Argenx)	• Reported no impact on albumin homeostasis[1] • EMA public assessment report indicates that there was no impact on albumin levels across doses[2]	• Phase 1 reported multiple doses had no impact on albumin levels in humans[1] • Phase 3 pivotal trials did not identify a safety signal of hypoalbuminemia[3]
SYNT-001 (Syntimmune)	• Reported no difference in albumin levels from baseline for vehicle, 10, 30, or 100mg/kg[4]	• Phase 1 data showed no difference in albumin levels from baseline following a single dose of vehicle, 1, 3, 10, or 30mg/kg[4]
Nipocalimab (Momenta / J&J)	• Data not published • Momenta management's public commentary indicated that albumin reductions were seen in MAD studies in cynomolgus monkeys[5]	• Phase 1 reported reductions in albumin levels from baseline at 15 and 30mg/kg doses[6] • Phase 2 showed reductions in albumin levels from baseline at 30 and 60mg/kg[7]
Rozanolixizumab (UCB)	• Reported small reductions (1-13%) in albumin levels from baseline[8]	• Phase 1 reported a small decrease in albumin levels from baseline for both IV and SC (1-5%)[9]
Batoclimab (Immunovant)	• Observed consistent reduction in albumin levels from baseline	• Observed dose dependent decreases in albumin levels from baseline
IMVT-1402 (Immunovant)	• No or mininal impact on albumin levels observed from baseline (variability same as placebo)	• Initial Phase 1 data available in mid-2023

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1. Ulrichts P.J Clin Invest. 2018 Oct 1;128(10):4372-4386
2. Efgartigimod EMA assessment report - EMA/641081/2022
3. Efgartigimod FDA integrated review - 761195Orig1s000
4. Blumberg LJ. Sci Adv. 2019 Dec 18;5(12):eaax9586
5. Stifel research note – Momenta Pharmaceuticals, December 18, 2018
6. Ling et.al, Clin Pharmacol Ther. 2019 Apr;105(4):1031-1039.
7. Momenta Investor Presentation – June 15, 2020
8. Smith B, MAbs. 2018 Oct;10(7):1111-1130
9. Kiessling P. Sci Transl Med. 2017 Nov 1;9(414):eaan1208

IgG Reduction and Clinical Efficacy

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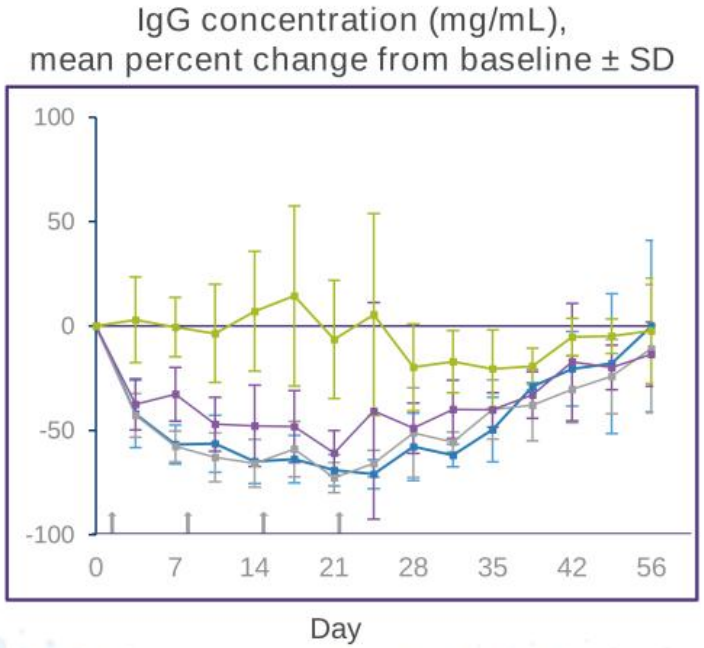
IMVT-1402 and batoclimab demonstrated similar, maximum IgG reduction

Head-to-Head Monkey Study



- 20 monkeys, dosed IV in head-to-head study across four groups
- At comparable doses, IgG lowering is similar for both batoclimab and IMVT-1402
- Cynomolgus monkeys observed to be reliable pharmacodynamic proxy for anti-FcRn mediated impacts on IgG[1,2]

We believe that deeper IgG suppression correlates with the clinical benefits across several anti-FcRn data sets

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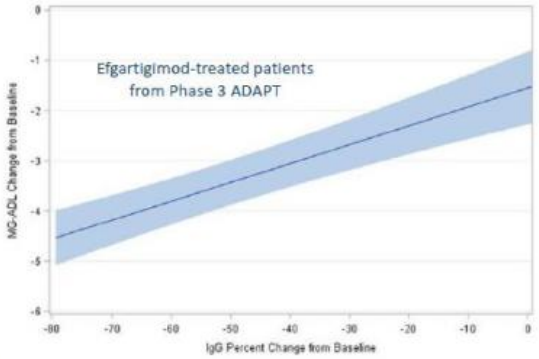
1. Source: Lledo-Garcia, et al, Pharmacokinetic-pharmacodynamic modelling of the anti-FcRn monoclonal antibody rozanolixizumab: Translation from preclinical stages to the clinic, UCB Pharma, 2022.
2. Data on file at Immunovant

Clinical data in MG across anti-FcRn assets indicate strong correlation between deep IgG reduction and increased clinical efficacy

The ADAPT Phase 3 trial of IV efgartigimod demonstrated that patients with deeper IgG reductions saw greater improvements in their disease activity (MG-ADL) compared to patients with lesser IgG suppression



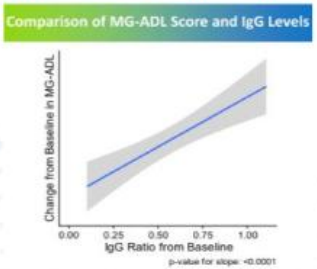
Patient-level data from Efgartigimod (n=84) arm in P3 study

In Batoclimab's (IMVT) Phase 2 trial in MG, we observed deeper IgG and AChR autoantibody reductions correlated with bigger MG-ADL changes

Data at week 7	Placebo (N=6)	Batoclimab 340 mg / week (N=5)	Batoclimab 680 mg / week (N=6)
% Change in total IgG from baseline	-3%	-59%	-76%
% Change in Anti-AChR-IgG from baseline	2%	-54%	-87%
% Change in MG-ADL from baseline	3%	-23%	-38%

While a small n, deeper IgG and anti-AChR autoantibody reductions achieved greater % improvements in MG-ADL

Nipocalimab Phase 2 trial in MG showed a correlation between IgG reductions and clinical activity



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Note: No clinical head-to-head comparisons of Immunovant product candidates and third party anti-FcRn assets has been conducted.

Clinical data in multiple other autoantibody-driven indications suggest strong correlation between IgG reduction and clinical efficacy

Immunovant's Phase 2 trial in TED indicated that reduction in IgG led to greater restoration of normal levels of pathogenic Abs and greater proptosis response rates

	Placebo	Batoclimab 255 mg	Batoclimab 340 mg	Batoclimab 680 mg
Median Max % IgG Reduction Through Week 6*	3%	54%	63%	79%
% Subjects with Stimulatory anti-TSHR Antibody below 140 at Week 6	0%	0%	12%	57%
Proptosis Response Rate at week 6**	0%	11%	29%	43%

*Week 6 data selected as it represents the latest time point at which the largest amount of patient data is available prior to the voluntary pause. **Post-hoc analysis of proptosis response at week 6. Proptosis response defined as proptosis reduction ≥2 mm in study eye, without ≥2 mm increase in non-study eye at same visit.

In UCB's Phase 2 trial in ITP, higher doses and greater IgG reductions were associated with better platelet responses

Single Dose of Rozanolixizumab	Est. IgG Reduction	Mean platelet count ($x10^9$/L)	% change platelet count ($x10^9$/L)
Day 8			
4 mg/kg	27%*	27	53%
7 mg/kg	27%*	21	53%
10 mg/kg	47%*	41	122%
15 mg/kg	52%	108	409%
20 mg/kg	60%	145	706%

*IgG reduction at day 8 estimated by WebPlotDigitizer for 4mg/kg, 7mg/kg and 10mg/kg doses

In efgartigimod Phase 2 in Pemphigus Vulgaris (PV), more intensive dosing regimens led to deeper skin responses

	Cohort 1	Cohort 2	Cohort 3	Cohort 4
Dosing				
Dose	10mg/kg	10mg/kg	10mg/kg	25mg/kg
Induction Dose Regimen	QW, 4 weeks	QW, 4 weeks	QW, 4 weeks	QW, until EoC
Maintenance Dose Regimen	Week 2, Week 6	Q2W, 8 weeks	Q2W, 12 weeks	Q2W, up to 34 weeks
IgG Reduction*				
Est. Max IgG Reduction (Day 28)	-56%	-69%	-62%	-67%
Est. IgG Reduction Day 120	11%	-33%	-52%	-54%
Efficacy†				
Complete Response	0%	0%	71%	60%
Relapse	50%	67%	43%	29%

Highest doses → highest sustained IgG reduction → higher CRs & lower relapse rates

Note: No clinical head-to-head comparisons of Immunovant product candidates and third party anti-FcRn assets has been conducted.

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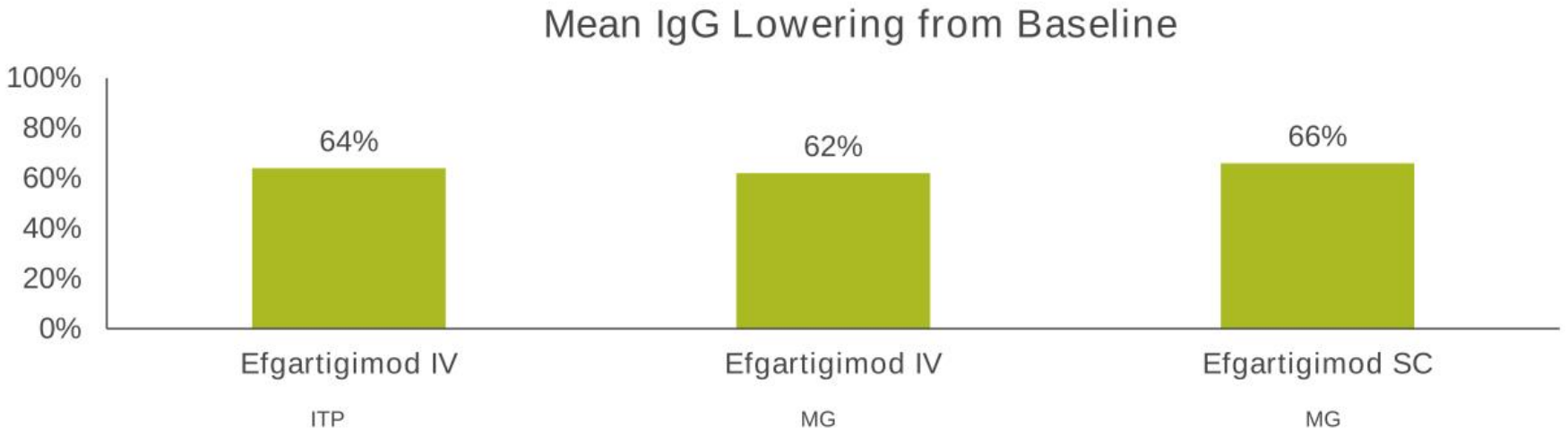
Argenx phase 2 PV/PF publication, Br J Dermatol. 2022 Mar;186(3):429-439; * Estimated by WebPlotDigitizer
† End of Consolidation (EoC): the time at which no new lesions had developed for min. 2 weeks and ~80% of lesions had healed; Disease control (DC): no new lesions and established lesions starting to heal; Complete response (CR): no new lesions and established lesions completely healed; Relapse: Appearance of three or more new lesions per month that do not heal spontaneously in 1 week, or extension of established lesions, evaluated after DC

In order to achieve IgG reductions beyond the 65% range, we believe efgartigimod dose would need to be increased beyond what is currently under study in clinical trials



Efgartigimod IgG Reduction in Pemphigus Vulgaris

Efgartigimod Dosage	Median IgG Reduction (Day 29)
10 mg / kg (approved dose)	62%
25 mg / kg	66%

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ARGX IV: 10 mg/kg recommended for MG
ARGX SC: 1,000 mg dose studied in ADAPT-SC bridging study

Consistent evidence across all programs and all indicators that greater IgG reduction leads to greater efficacy

	Company	Evidence of Greater IgG Reductions Translating to Clinical Benefit
MG	IMMUNOVANT	Greater IgG reductions across arms ➔ greater anti-AChR autoantibody reductions and greater MG-ADL improvements
MG	argenx / janssen	Patient-level scatter plot showed that greater IgG declines ➔ greater MG-ADL improvements
TED	IMMUNOVANT	Greater IgG reduction across arms ➔ higher rates of anti-TSHR antibody reduction and higher proptosis response rates
PV	argenx	Greater sustained IgG reduction across arms ➔ higher complete response and lower relapse rates
ITP	ucb	Greater IgG reduction across arms ➔ greater platelet responses

Batoclimab and IMVT-1402's unique product attributes amongst anti-FcRn assets provide significant potential impact to patient experience

Product Company	Impact to Albumin and/or LDL	IgG Reduction	RoA	Dosage/Dosing Regimen	Injection / Infusion Time
Efgartigimod Argenx	None observed	~65%[1]	Weekly IV cycles x4 approved; Halozyme SC in development[2]	1,000 mg QW	1 minute
Nipocalimab-MG JnJ	Likely minimal at dose in MG program	~60s% with variation due to Q2W dosing[3]	IV	15 mg/kg[4] Q2W	Possibly as low as 7.5 minutes
Rozanolixizumab UCB	None or minimal at doses studied	~70's%	SC infusion	10 mg/kg or 7 mg/kg[5] QW	30 – 90 minutes[6]
Batoclimab Immunovant	Dose-dependent and reversible	~80%[7] or ~65%	SC	680 or 340 mg[5] QW[8]	~5-10 seconds per dose
IMVT-1402 Immunovant	None or minimal observed in cynos	Likely similar to batoclimab	SC	Likely similar to batoclimab[5]	~5-10 seconds per dose

Batoclimab + IMVT-1402 Advantages	Deeper IgG reductions with higher dose	More convenient formulation	Offers dosing flexibility	Fast administration

No clinical head-to-head comparisons of Immunovant product candidates and third party anti-FcRn assets has been conducted.

1. ARGX MG PH3 IV, SC
2. Except as initial approvals in MG and ITP, which will be IV 10mg/kg QW
3. Estimated as pivotal trial dose of 30mg/kg IV loading dose followed by 15mg/kg Q2W IV was not studied in the proof-of-concept trial
4. Dosing amount disclosed for MG Phase 3 MG trial after loading dose of 30 mg/kg IV
5. Specific dosing TBD
6. Reported infusion times of 30-90mins for doses ranging from 4mg/kg to 20mg/kg in ITP as reported by Robak et. Al in Blood Nov 2019; http://doi.org/10.1182/blood-2019-129839
7. Based on TED P2 data
8. Potential Q2W dosing option also being explored

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De-risking CIDP Trial with Unique Design

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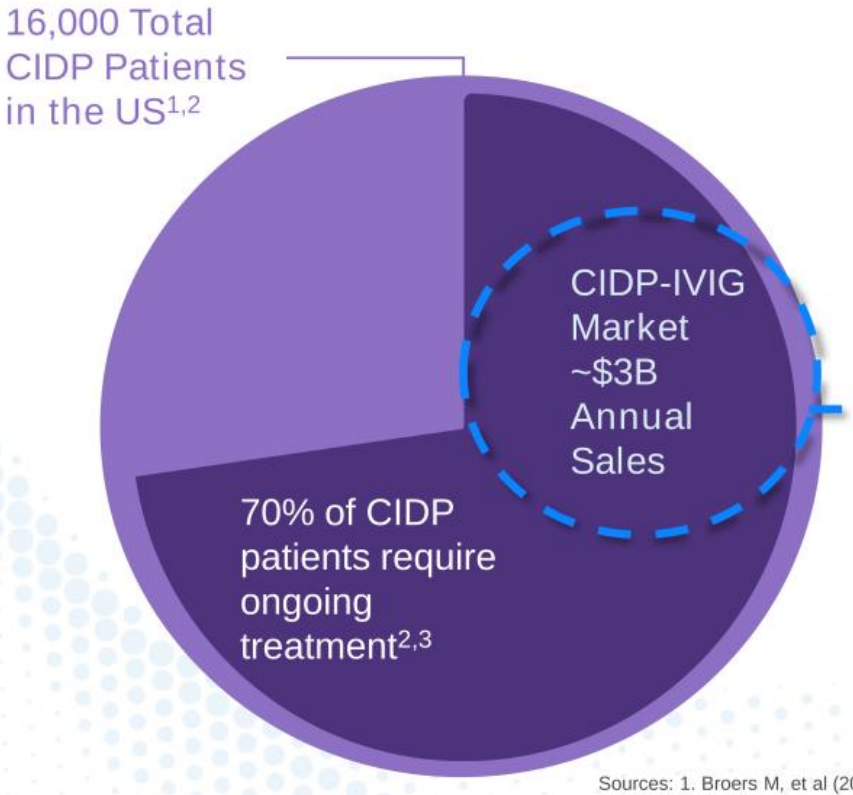
CIDP represents an exciting opportunity



- CIDP represents 22% of total IVIG market by volume: ~$3B in global annual sales for IVIG in CIDP[4]
- Existing CIDP treatments can produce side effects and have logistical challenges
- Target population: Active CIDP patients

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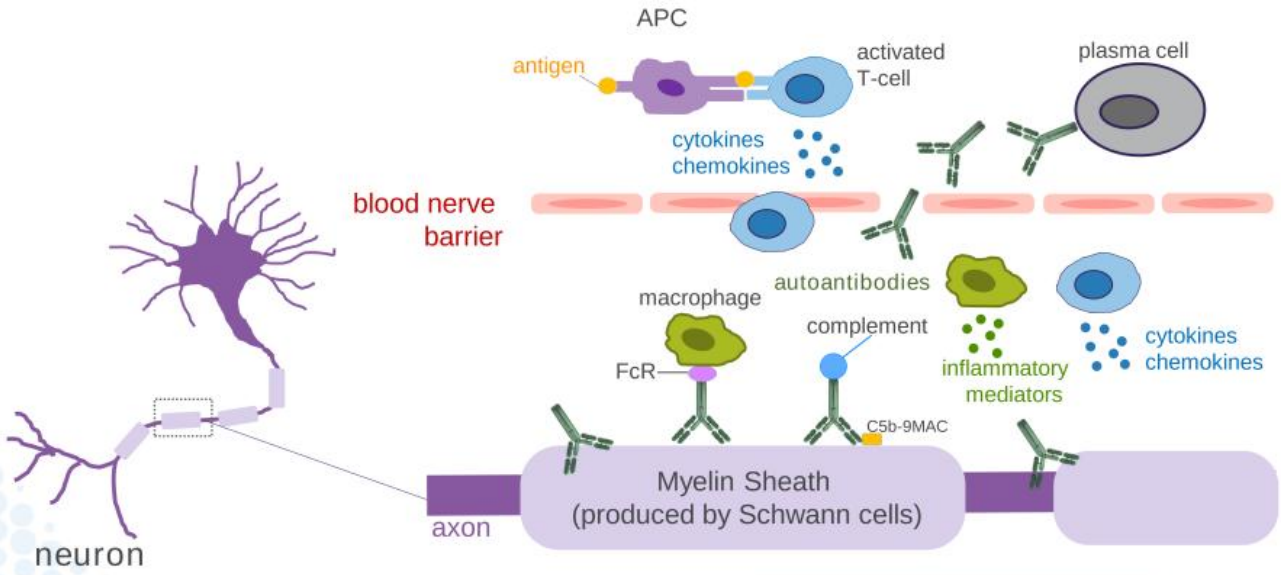
Sources: 1. Broers M, et al (2019) Incidence and prevalence of CIDP: a systematic review and meta-analysis. Neuroepidemiology 52(3–4):161–172; 2. Querol, L., et al. Systematic literature review of burden of illness in chronic inflammatory demyelinating polyneuropathy (CIDP). J Neurol 268, 3706–3716 (2021).; 3. Kuitwaard K, Bos-Eyssen ME, Blomkwist-Markens PH et al (2009) Recurrences, vaccinations and long-term symptoms in GBS and CIDP. J Periph Nerv Syst 14(4):310–315. https://doi. org/10.1111/j.1529-8027.2009.00243.; 4. CSL Behring R&D Investor Briefting, 2021.

Response to current treatments of IVIG and Plasma Exchange creates strong rationale for potential benefit of anti-FcRn mechanism



anti-myelinated peripheral nerve IgG in 30-40% patients[1]

CIDP is characterized by predominant demyelination of motor and sensory nerves. Although the cause of CIDP is unknown, significant evidence suggests that the disorder(s) are immunologically mediated.[1]

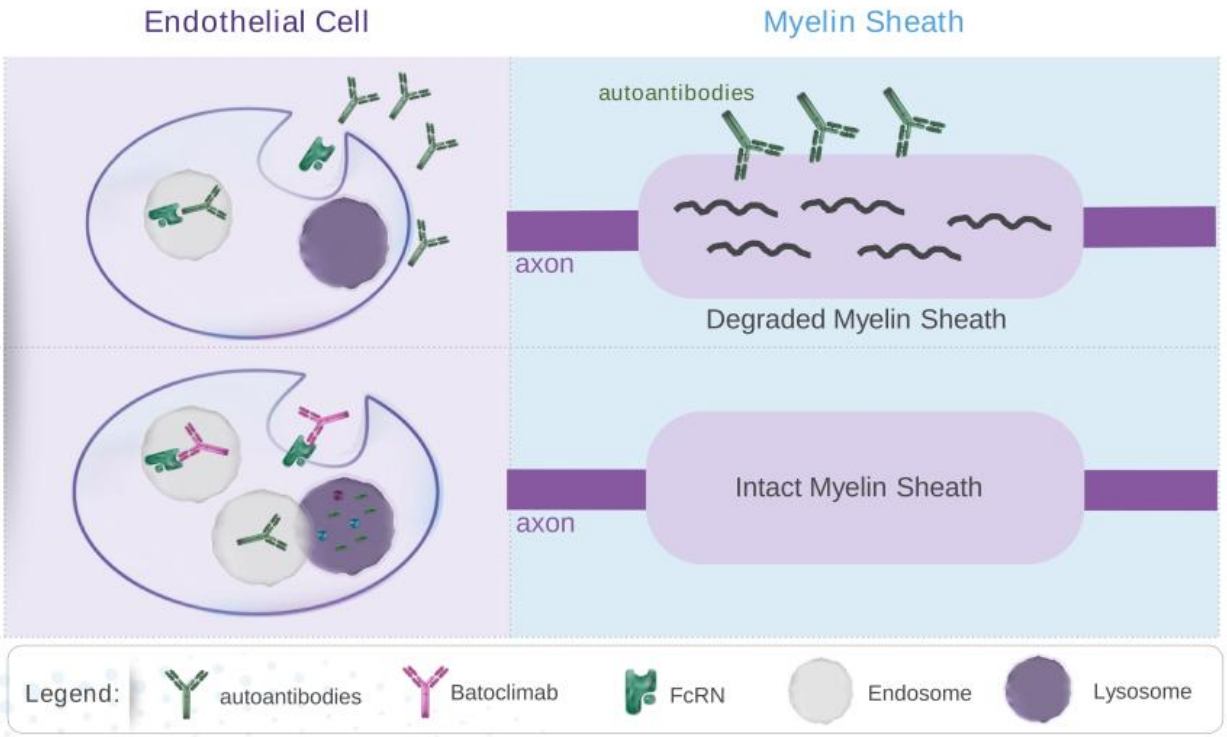
Multiple immune mechanisms including cellular (macrophages), humoral and complement pathways contribute to the pathogenesis of CIDP.[2,3]

Sources: 1. Mathey EK, Park SB, Hughes RAC, et al Chronic inflammatory demyelinating polyradiculoneuropathy: from pathology to phenotype Journal of Neurology, Neurosurgery & Psychiatry 2015; 2. Koike H, Katsuno M. Pathophysiology of Chronic Inflammatory Demyelinating Polyneuropathy: Insights into Classification and Therapeutic Strategy. Neurol Ther. 2020 Dec;9(2):213-227. doi: 10.1007/s40120-020-00190-8. Epub 2020 May 14; 3. Querol LA, Hartung HP, Lewis RA, van Doorn PA, Hammond TR, Atassi N, Alonso-Alonso M, Dalakas MC. The Role of the Complement System in Chronic Inflammatory Demyelinating Polyneuropathy: Implications for Complement-Targeted Therapies. Neurotherapeutics. Apr 2022.

Anti-FcRn mechanism of action degrades IgG, potentially protecting the myelin sheath from pathogenic IgG autoantibody attack in CIDP

In the absence of batoclimab, FcRn binds to the IgG autoantibodies, inhibiting their degradation and returning them into circulation. IgGs may attack the myelin resulting in myelin degradation and neuropathy

With batoclimab, FcRn is blocked from binding to IgG autoantibodies, which are then transported to the lysosome for degradation, decreasing their levels in circulation and potentially reducing pathogenesis



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Source: Koike H, Katsuno M. Pathophysiology of Chronic Inflammatory Demyelinating Polyneuropathy: Insights into Classification and Therapeutic Strategy. Neurol Ther. 2020 Dec;9(2):213-227. doi: 10.1007/s40120-020-00190-8. Epub 2020 May 14.

Our development approach applies key learnings from historical and ongoing CIDP trials to address challenges unique to CIDP

Trial risks	Mitigations	Mitigation included in other anti-FcRn Trials*	Mitigation included in IMVT trial
Disease heterogeneity and challenging diagnosis	Diagnostic algorithm	X	✓
Mix of active and inactive disease among those enrolled creates risk of low relapse rate in the placebo arm, thereby shrinking potential effect size for the investigational product	Double enrichment: 1.Subjects must worsen after withdrawal / taper of standard of care (e.g. IVIG/SCIG, PLEX, or steroids) on study entry, AND 2.Subjects must then improve on open label investigational product	Not All**	✓
Patients enrolled in placebo arm of trial may not have demonstrated initial response to investigational product		Not All**	✓
Steroids are a common standard of care outside the US and often can't be fully tapered, weakening double enrichment	Third enrichment: Primary endpoint on IVIG/SCIG/Plex cohort only to maximize the potential effect size	X	✓
Lack of dose exploration	Data on multiple doses in “Period 1” of trial will inform future development strategy	X	✓
Single large trial limits flexibility to optimize product label and differentiation	Smaller, initial pivotal trial that can be adjusted or complemented with a second smaller pivotal trial to optimize product label and differentiation based on new data	X	✓

Two-stage approach (if additional trial is required for registration) has the potential to deliver a differentiated product label with a larger effect size

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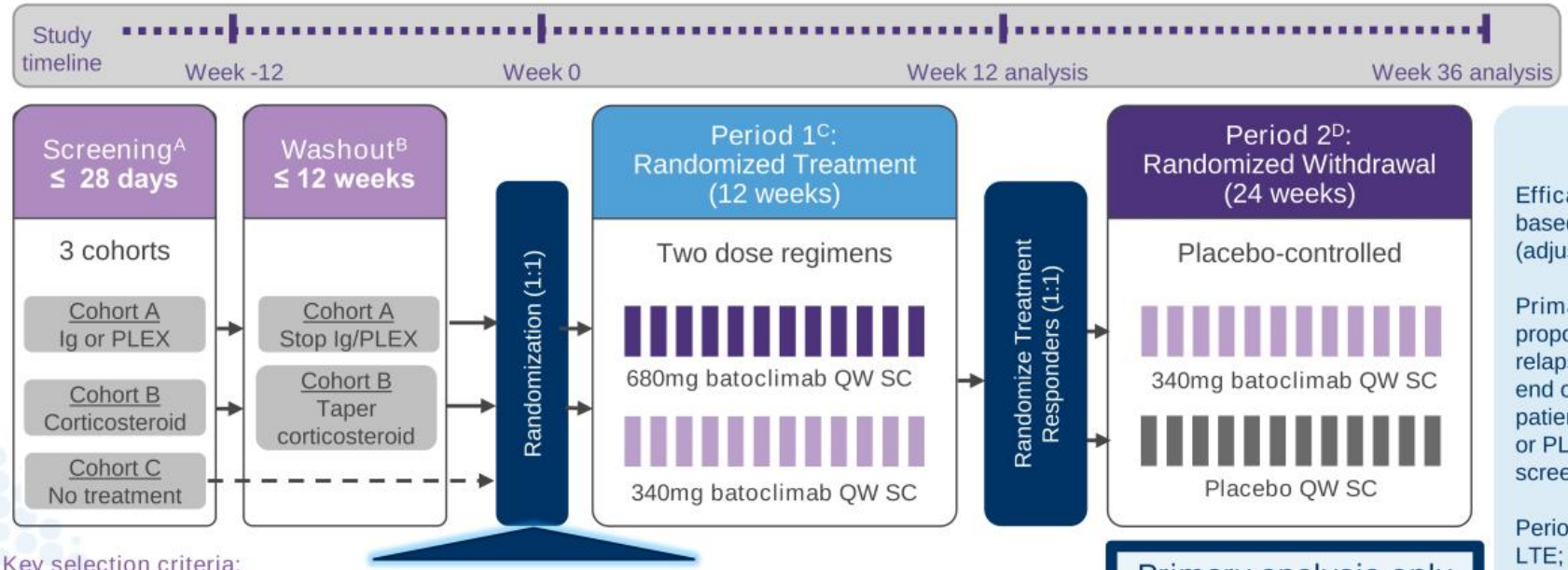
Notes: *Other anti-FcRn trials in CIDP include efgartigimod, nipocalimab, and rozanolixizumab. **clinical trial designs for efgartigimod in CIDP and nipocalimab in CIDP include double enrichment in trial design. Rozanolixizumab ph2 trial in CIDP did not include double enrichment.

CIDP pivotal Phase 2b trial design intended to enable development of potentially best-in-anti-FcRn-class chronic therapy for CIDP



Key selection criteria:

Adult participants diagnosed per EAN/PNS CIDP guidelines, 2021 revision

Cohorts A (n=100): Randomize participants who worsen

Cohort B: Same as A

Cohort C: Randomize all

Primary analysis only on Cohort A (IG/PLEX)

Efficacy analysis based on relapse (adjusted INCAT)

Primary endpoint: proportion remaining relapse free by the end of period 2 for patients receiving Ig or PLEX at time of screening (Cohort A)

Period 2 followed by LTE; 680mg QW x 4 for period 2 relapsers

A: Cohorts are defined by CIDP treatment at Screening., B: Participants who fail to worsen by Week 0 will be withdrawn from the study at Week 0., C: Period 1 Non-Responders who complete Period 1 will be withdrawn from the study after completing Week 12 and the subsequent 4-week Follow-Up visit. Period 1 Non-Responders who require protocol-prohibited rescue therapy prior to Week 12 will discontinue IMP and may return to standard of care; these participants will be encouraged to remain in the study for Safety Follow-Up through Week 12 and the Follow-Up Visit., D: Participants that relapse in Period 2 or complete Period 2 without relapse will be eligible for participation in the Long-Term Extension study.

Acronyms: CIDP= Chronic Inflammatory Demyelinating Polyneuropathy; EAN/PNS = European Academy of Neurology/Peripheral Nerve Society; Ig = immunoglobulin (IVIG and SCIG) therapy; IMP = investigational medicinal product; LTE = Long-term Extension; PLEX = plasma exchange; QW = every week; Wk = weekly; SC = subcutaneously; INCAT = Inflammatory Neuropathy Cause and Treatment

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Immunovant pursuing a differentiated approach to developing batoclimab as a chronic treatment for CIDP

1

CIDP is an exciting indication that is ripe for disruption

Given disease complexity, trial design is critical

2

Our pivotal study is optimized versus historical and current studies

To improve probability of success and effect size, and include multiple doses for optimal differentiation

3

Batoclimab has potential best-in-class efficacy and could be first simple SC

Representing meaningful innovation for patients with this chronic disease

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Note: SC = Subcutaneous administration

Thank You

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