IMMUNOVANT®

Targeted science, tailored solutions

for people with autoimmune disease

Corporate Presentation

August 2025

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," "anticipate," and other similar expressions are intended to identify forward-looking statements. Such forward looking statements include statements regarding Immunovant’s expectations regarding the timing, design, and results of clinical trials of IMVT-1402; Immunovant’s plan to develop IMVT-1402 and batoclimab across a broad range of indications; the number and timing of potentially registrational programs and clinical trials Immunovant plans to initiate for IMVT-1402; and potential benefits of IMVT-1402’s unique product attributes and potential best-in-class and first-in-class profile. 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: Immunovant may not be able to protect or enforce its intellectual property rights; initial results or other preliminary analyses or results of early clinical trials may not be predictive final trial results or of the results of later clinical trials; 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 number and timing of the commencement of additional clinical 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 press release; 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 product candidates may not be beneficial to patients, or even if approved by regulatory authorities, successfully commercialized; the potential impact of global factors, such as international trade tariffs, geopolitical tensions, and adverse macroeconomic conditions on Immunovant’s business operations and supply chain, including its clinical development plans and timelines; Immunovant’s business is heavily dependent on the successful development, regulatory approval, and commercialization of IMVT-1402 or batoclimab; Immunovant is at various stages of clinical development for IMVT-1402 and batoclimab; and Immunovant will require additional capital to fund its operations and advance IMVT-1402 and batoclimab 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 Annual Report on Form 10-K filed with the SEC on May 29, 2025, 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.

Our Focus: Pursue a broad anti-FcRn strategy based on potential best-in-class profile of IMVT-1402 targeting autoantibody-driven diseases

Validated Target	IMVT-1402: Potential Best-in-Class Product Candidate	Significant Market Opportunity	Financial Strength
			
4 regulatory approvals, 8 positive Phase 3 trials and 17 positive Phase 2 trials with >2,300 patients treated to date across 4 compounds	Anti-FcRn with deepest, best-in-class IgG reduction delivered by simple autoinjector On track to be potential first-/best-in-class with potentially registrational trials in GD, SjD, ACPA+ D2T RA, CIDP, MG & proof-of-concept trial in CLE	Large total addressable market with 20+ indications announced or in development across the anti-FcRn class[1] Current IMVT-1402 trials expected to potentially address >600K patients in the US	Cash balance of ~$600 million as of June 30, 2025[2] Provides runway for announced indications through Graves' Disease readout expected in 2027

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1. Indications announced or in development with anti-FcRn assets by Immunovant, argenx, Johnson & Johnson, and UCB
2. Includes cash and cash equivalents

Note: GD: Graves' disease; SjD: Sjogren's disease; ACPA+ D2T RA: anti-citrullinated protein antibody positive (ACPA+), difficult-to-treat rheumatoid arthritis; CIDP: Chronic inflammatory demyelinating polyneuropathy; MG: Myasthenia gravis; CLE: Cutaneous lupus erythematosus

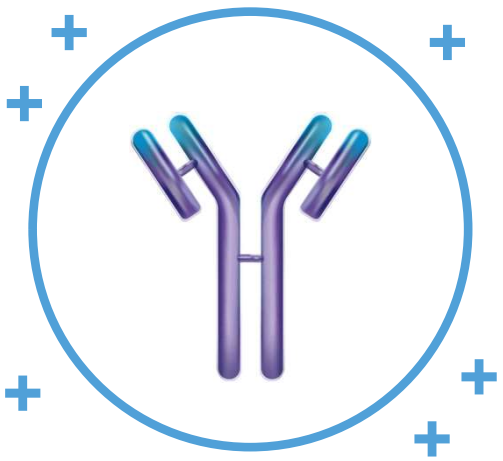
IMVT-1402 has potential to be first- and best-in-class across multiple indications

Robust IgG lowering and favorable safety profile drive optimism for differentiation vs. other FcRn inhibitors

Internal Data Validates Deeper is Better in multiple studies across GD, MG, and CIDP with notably improved clinical benefits for patients with IgG reduction ≥70%[1]

Convenient Administration
Delivered via market-proven, user-friendly auto-injector

IMVT-1402



Novel, fully human, monoclonal antibody inhibiting FcRn-mediated recycling of IgG

Deep IgG Lowering Phase 1 data suggests deep dose-dependent IgG lowering; expected to reach ~80% with continued weekly dosing of 600 mg

Ongoing Clinical Progress GD, D2T RA, MG, CIDP and SjD potentially registrational studies actively enrolling; CLE proof of concept also actively enrolling

Strong Patent Protection Issued patent covers composition of matter, method of use and methods for manufacturing to 2043[2]

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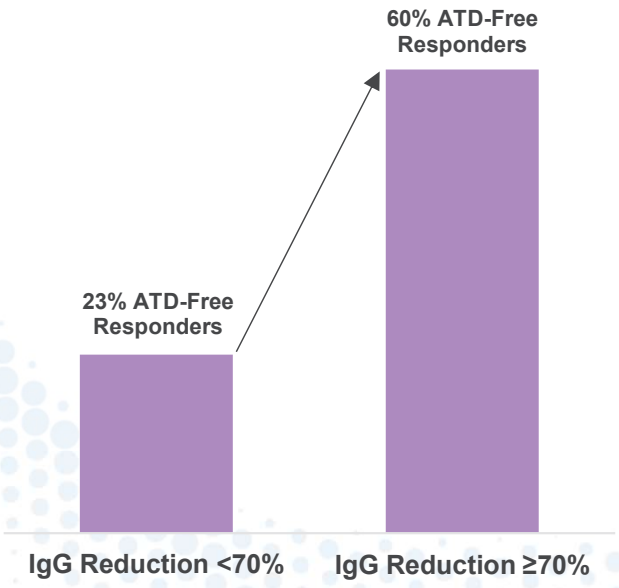
1. Compared to those with IgG reduction <70% in the same batoclimab studies
2. Not including any potential patent term extension

Note: GD: Graves' disease; D2T RA: Difficult-to-treat rheumatoid arthritis; MG: Myasthenia gravis; CIDP: Chronic inflammatory demyelinating polyneuropathy; CLE: Cutaneous lupus erythematosus; SjD: Sjogren's disease

First generation batoclimab clinical data across multiple indications shows that deeper IgG reduction leads to improved clinical outcomes

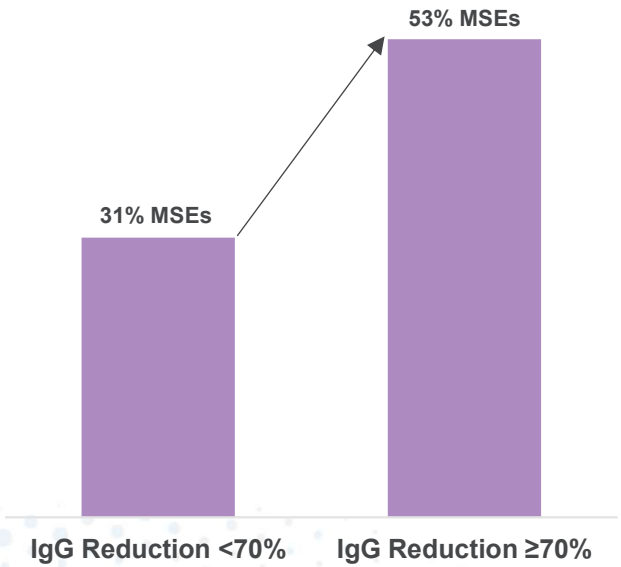
Graves' Phase 2a[1]

ATD-Free Response: % of participants who achieve normal T3 and T4 or have T3 or T4 below LLN, and ceased all ATD medications



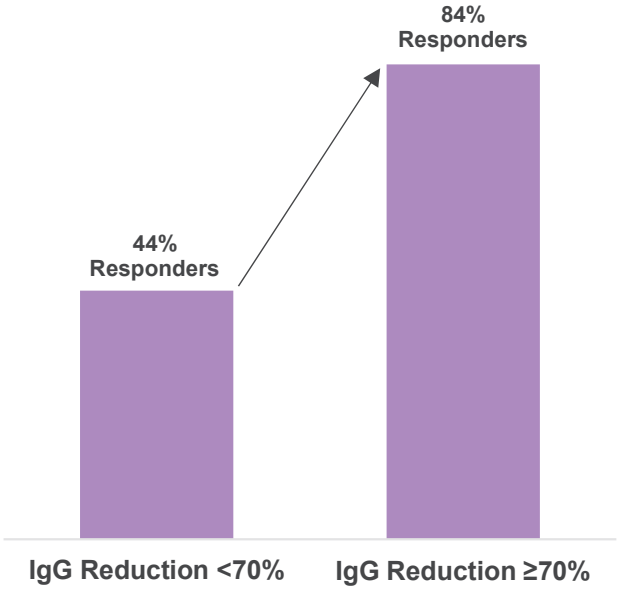
MG Phase 3[1]

Minimal Symptom Expression: % of participants who achieve MG-ADL score of 0 or 1 at Week 12



CIDP Phase 2b[1]

aINCAT Response: % of participants who achieve aINCAT improvement ≥1 at Week 12



Reflects data from multiple clinical trials in multiple indications. Differences exist between trial designs and participant characteristics and caution should be exercised when comparing data across trials.

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Notes: MG data presented for acetylcholine receptor antibody-positive patients; ATD: Antithyroid drug; aINCAT: Adjusted Inflammatory Neuropathy Cause and Treatment; IgG: Immunoglobulin G; MSE: Minimal Symptom Expression; LLN: Lower limit of normal. The data referenced here includes data from the ongoing batoclimab Phase 2 study in CIDP and is based on a preliminary analysis of key efficacy and safety data, and such data may change following completion of the clinical trial and may not accurately reflect the complete results of the study 1. Batoclimab clinical data

Best-in-class IgG reductions position Immunovant to drive best-in-class efficacy

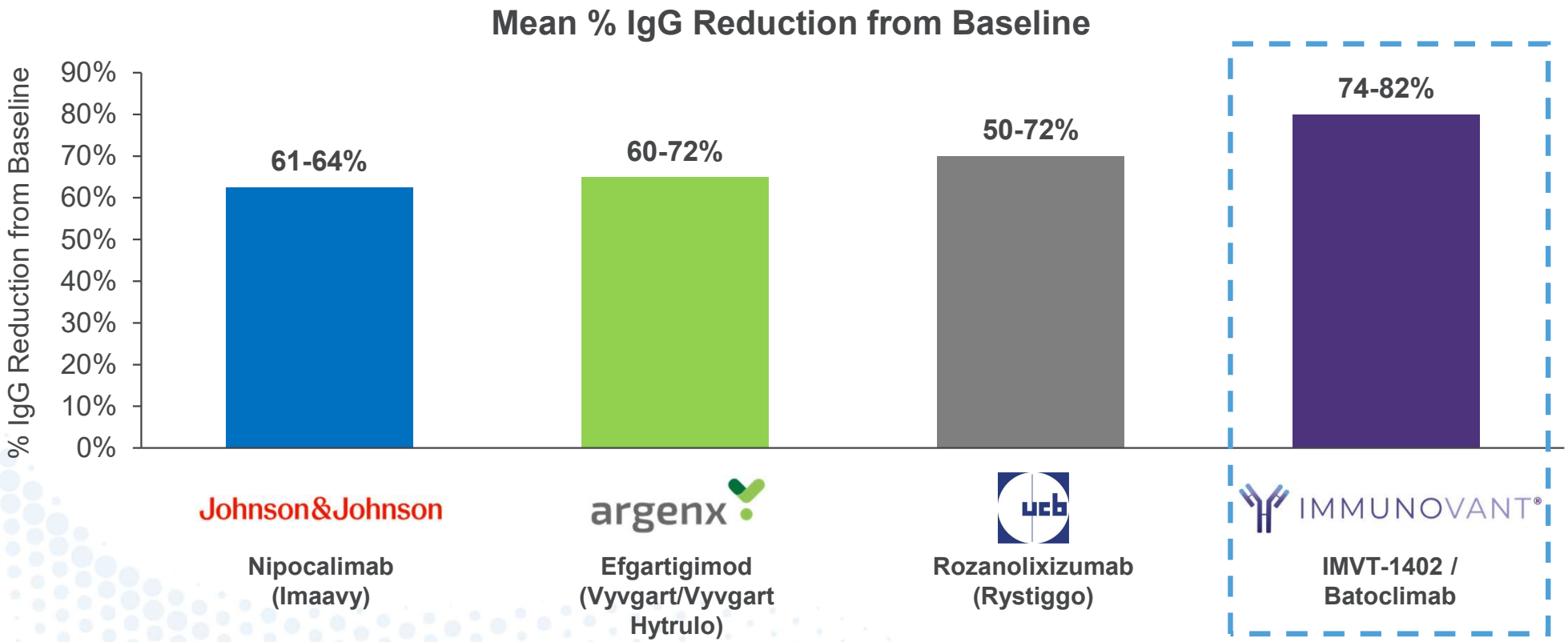

Figure reflects cross-trial comparisons and not data from head-to-head studies.
Differences exist between trial designs and participant characteristics and caution should be exercised when comparing data across trials.

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Notes: Mean IgG reductions only reflected for clinically-relevant/registrational doses for relevant indications. Immunovant data reflects batoclimab MG, Graves', TED studies, and IMVT-1402 Phase 1 study (IMVT Data on File). Ranges of reductions for competitors include mean reductions from the following trials: MG Phase 3 (Howard et al., 2022), CIDP Phase 2b (Allen et al., 2024), ITP Phase 3 (Broome et al., 2022), PV/PF Phase 2 (Goebeler et al., 2021) for ARGX, RA Phase 2 (Taylor et al., 2024), Sjogren's Phase 2 (Gottenberg et al., EULAR 2024), MG Phase 3 (Antozzi et al., 2025) for JNJ, and MG Phase 3 (Bril et al., 2023) and ITP Phase 3 (Cooper et al., 2024) for UCB. Some values are estimated from graphs where not reported.

Our market: Autoimmune diseases driven by harmful IgG autoantibodies

Anti-FcRn mechanism potentially the leading therapeutic class with 20+ indications announced or in development[1]

NEUROLOGY

Chronic inflammatory demyelinating polyneuropathy (CIDP)
Generalized myasthenia gravis (MG)
Myelin oligodendrocyte glycoprotein antibody disorders (MOG-antibody disorder)
Ocular MG

ENDOCRINOLOGY

Graves' disease (GD)
Thyroid eye disease (TED)

HEMATOLOGY

Fetal neonatal alloimmune thrombocytopenia (FNAIT)
Hemolytic disease of the fetus and newborn (HDFN)
Idiopathic thrombocytopenic purpura
Warm autoimmune hemolytic anemia (WAIHA)

RHEUMATOLOGY

Rheumatoid arthritis (RA)
Sjögren's disease (SjD)
Myositis
Severe fibromyalgia syndrome
Systemic lupus erythematosus

DERMATOLOGY

Cutaneous lupus erythematosus (CLE)
Bullous pemphigoid
Pemphigus foliaceus/Pemphigus vulgaris
Systemic sclerosis

RENAL

Antibody-mediated rejection
Lupus nephritis
Membranous nephropathy

1. Indications announced or in development with anti-FcRn assets by Immunovant, argenx, Johnson & Johnson, and UCB.

Indication Strategy: Development strategy designed to maximize commercial potential, with IMVT-1402's potentially best-in-class profile

First-in-Class Best-in-Class	• Expanding use of FcRn inhibitors to benefit greater number of patients with several new indications, with a potential efficacy advantage driven by deeper IgG reduction • Example – GD, D2T RA, CLE
Nearly-First Best-in-Class	• Close from a timing perspective to in-class competition, while maintaining potential for a differentiated clinical profile driven by best-in-class IgG reductions • Example – SjD
Best-in-Class	• Well-established markets with multiple competitors; potential to differentiate on efficacy and convenience • Example – MG and CIDP

IMVT-1402's potentially differentiated product profile offers wide range of development opportunities

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Note: GD: Graves' disease; D2T RA: Difficult-to-treat rheumatoid arthritis; SjD: Sjogren's disease; CLE: Cutaneous lupus erythematosus; MG: Myasthenia gravis; CIDP: Chronic inflammatory demyelinating polyneuropathy

Broad development program for IMVT-1402 with trials underway, expected to potentially address >600K patient population

	Graves' Disease	Difficult-to-Treat Rheumatoid Arthritis	Cutaneous Lupus Erythematosus	Sjogren's Disease	Myasthenia Gravis	Chronic Inflammatory Demyelinating Polyneuropathy
Expected US Addressable Population [1]	**~330K**	**~70K**	**~75K**	**~90K**	**~20-35K**	**~16-58K**
Autoantibody Driven Pathology	Driven by autoantibodies to the thyroid-stimulating hormone receptor (TSHR-Ab)	Autoantibodies such as RF and ACPA are present in ~75% of RA patients	IgG autoantibodies (Ro/SSA, La/SSB) observed in majority of CLE patients	Autoantibodies detected in ~50-70% of patients with primary SjD	Driven by AChR autoantibodies disrupting signal transmission in nerve and muscle fibers	Driven by autoantibodies that demyelinate peripheral nerves and nerve roots
In-Class Data	Batoclimab data showed deeper IgG reduction correlated with improved clinical response	Response rate higher for patients with high baseline ACPA & deep IgG reduction	Proof of principle IMVT-1402 case study showed meaningful clinical response	Response rate higher for patients with deeper IgG reduction[2]	Batoclimab data showed deeper IgG reduction correlated with improved clinical response	Batoclimab data showed deeper IgG reduction correlated with improved clinical response
Stage of Development	Two Potentially Registrational Trials Enrolling	Potentially Registrational Trial Enrolling	Proof of Concept Enrolling	Potentially Registrational Trial Enrolling	Potentially Registrational Trial Enrolling	Potentially Registrational Trial Enrolling
Potential Best-in-Class	✓	✓	✓	✓	✓	✓
Potential First-in-Class[3]	✓	✓	✓	✓		

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1. IMVT data on file
2. Based on data generated by nipocalimab
3. Open check mark indicates nearly-first-in-class

Clear focus on execution to unlock value both near and long term

Indication	Study	Data Catalyst	2025	2026	2027	2028
GD	POC	Remission Data	■			
TED	Potentially Registrational	Top Line Results	■			
ACPA+ D2T RA	Potentially Registrational	Open-label Period 1 Initial Results		■		
CLE	POC	Initial Results		■		
ACPA+ D2T RA	Potentially Registrational	Top Line Results			■	
GD	Potentially Registrational	Top Line Results			■	
MG	Potentially Registrational	Top Line Results			■	
SjD	Potentially Registrational	Top Line Results				■
CIDP	Potentially Registrational	Top Line Results				■

IMVT-1402 **Batoclimab**

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Note: GD: Graves' disease; TED: Thyroid eye disease; APCA+ D2T RA: Anti-citrullinated peptide antibody positive difficult-to-treat rheumatoid arthritis; CLE: Cutaneous lupus erythematosus; MG: Myasthenia gravis; SjD: Sjogren's disease; CIDP: Chronic inflammatory demyelinating polyneuropathy

All references are to calendar years and are approximate and subject to change

IMVT-1402: Potentially first anti-FcRn to launch with simple autoinjector device

All current IMVT-1402 trials are being conducted with the YpsoMate® autoinjector – the intended commercial presentation

Established, user-friendly autoinjector with multiple approved products

- Automated, simple, subcutaneous injection
- Hidden needle shield
- Provides both visual and audio feedback
- <10 second at home self-administration or HCP administration

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*Ypsomate® autoinjector used in ADBRY®, COSENTYX®, AJOVY®, NUCALA®, FASENRA®, TEZSPIRE®
*Ypsomate® is a registered trademark of Ypsomed AG.
HCP: Health Care Provider

Graves' Disease

First-in-Class Opportunity

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IMVT-1402: potentially first- and best-in-class in Graves' disease (GD)

High Unmet Need	~25-30% of Graves' disease patients are challenging to manage on ATD therapy; these patients are either unable to complete initial treatment or unable to stay euthyroid despite treatment
Autoantibody Pathology	Role of TSH-R IgG autoantibodies well-recognized in Graves' disease; anti-FcRn directly targets the underlying disease pathophysiology, while ATDs do not
Lower is Better	Batoclimab POC demonstrated strong correlation between deep IgG lowering, normalization of thyroid hormone levels and reduced dependence on background ATD therapy
Optimized Study Design	IMVT-1402 trial designed to demonstrate thyroid hormone normalization and independence from ATD therapy at rates previously unattainable for challenging-to-manage Graves' patients
Potentially Registrational Trials Initiated	Two potentially registrational trials are actively enrolling, both with self-administration via market-proven autoinjector

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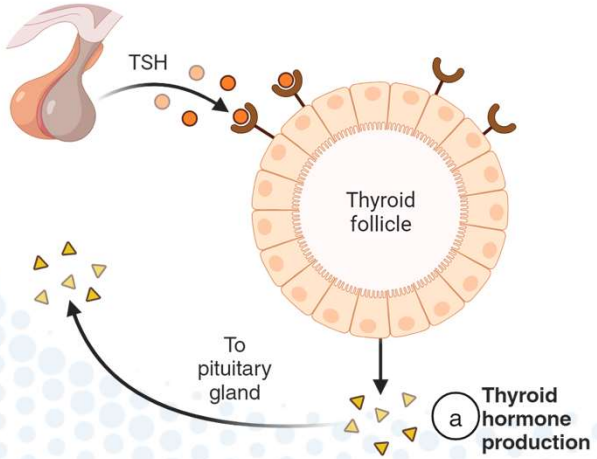
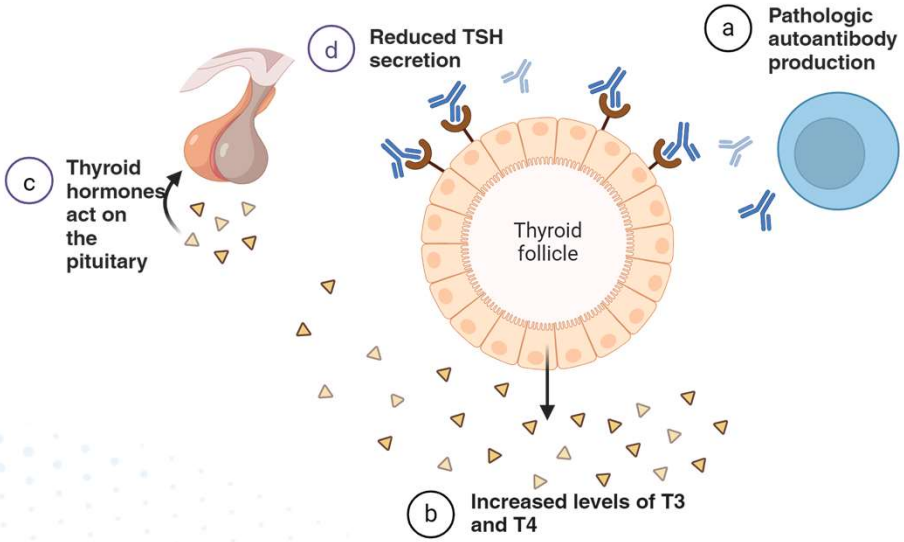
Notes: Thyroid Stimulating Hormone Receptor (TSRH), Anti-thyroid drug (ATD), Graves' disease (GD), Proof of concept (POC), Euthyroid = T3/T4 and TSH within normal limits

Graves' disease is a classic autoimmune condition driven by the presence of autoantibodies to the thyroid stimulating hormone receptor

Graves' Disease: Autoantibody-Driven Pathogenesis

Normal Function	Graves' Disease
TSH produced by the pituitary gland stimulates the thyroid gland to produce and release thyroid hormones (T3 & T4)	Autoantibodies to the thyroid stimulating hormone receptor (TSHR) stimulate thyroid hormone production and lead to excess thyroid hormone production (increased T3, T4)





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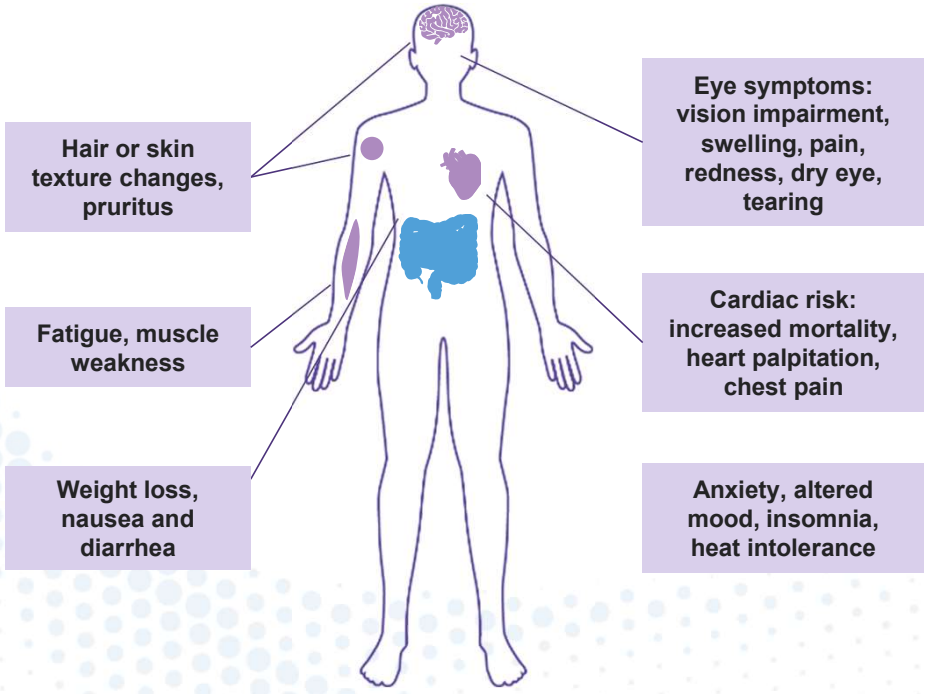
Sources: McIver, B. and Morris, J. The Pathogenesis of Graves' Disease (1996), Davies, T, Andersen, S, Latif, R. et al. Graves disease (2020). Image created using BioRender.

Graves' disease: high patient burden and significant morbidity

GD is the most common cause of hyperthyroidism; typically presents in adults aged 20–50[1]

Symptoms impact many organ systems and leave many patients with substantial burden[2,3]



Substantial morbidity and impaired quality of life if inadequately treated

Cardiovascular Complications

Graves' disease patients have a 23% increase in all cause mortality and more than double the risk of a major CV event[4]

Thyroid Eye Disease (TED)

TED affects ~40% of patients diagnosed with Graves' disease[5]

- ~10% of TED patients on novel therapies experience hearing-related events including hearing loss[6]

Pregnancy Complications[7]

Miscarriage, stillbirth, neuro-intellectual impairment in offspring, fetal thyroid disease

Other Significant Complications

Thyroid storm (~20% mortality rate[8]), thyroid cancer, psychiatric issues

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1. Davies TF, et al. Nat Rev Dis Primers. 2020;6(1):52 2. Smith et al. (2016), 3. Burch et al. (2015), 4. Okosieme et al. (2019), 5. Chin et al. (2020), 6. Smith et al. (2023), 7. Okosieme et al. (2020), 8. Bourcier et al. (2020)

Minimal innovation in GD treatment options over the past 70+ years

No existing pharmacologic therapy addresses underlying disease pathology

Standard-of-Care Treatments	Associated Challenges
Anti-Thyroid Drugs (ATDs) (e.g., Methimazole, Propylthiouracil)	• ~25-30% of patients are relapsed, uncontrolled or intolerant to ATDs[1] • Potential for serious adverse events, including hepatotoxicity (liver injury ~3%) and agranulocytosis (loss of white blood cells ~0.3%)[2,3]
Radioactive Iodine	• TED development and/or exacerbation in 15-33% of patients[4] • Dose dependent, long-term increased risk of death (5-12% increased risk per 100-mGy dose) from solid cancers[5] • Necessitates life-long thyroid replacement therapy
Thyroidectomy	• Recurrent laryngeal nerve damage risk in 1-4% of patients leading to dysphonia[3] • Permanent hypoparathyroidism observed in 2.6% of patients[4] • Necessitates life-long thyroid replacement therapy

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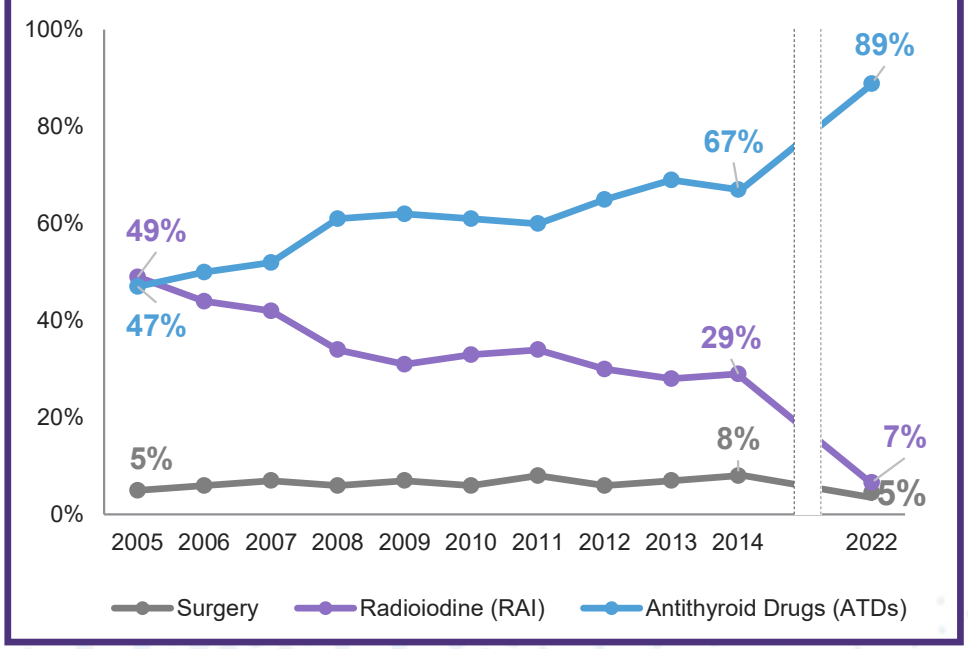
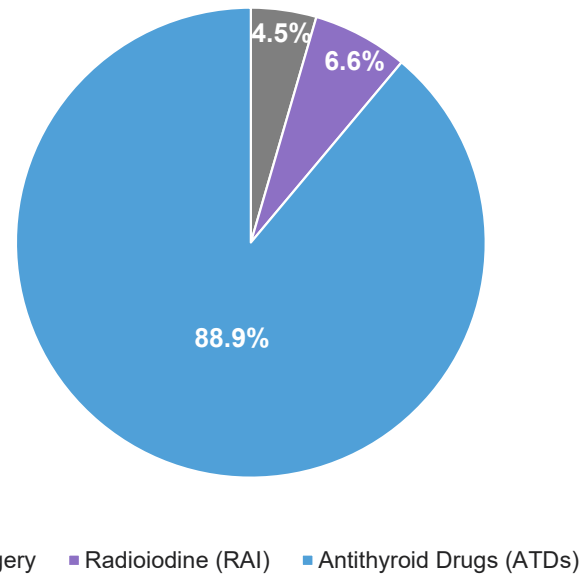
Sources: 1. Roivant / Inovalon Claims Analysis – 2021 incident patient population, first-line treatment is primary treatment in the first-year post diagnosis, claims review included a five-year lookback to define the incident population, IMVT Market Research 2020-2023; 2. Suzuki, N., et al. (2019), 3. Smith, T., & Hegedüs, L. (2016). 4. Sundaresh, V., et al. (2013). 5. Kitahara, C., et al. (2019).

In North America, the treatment paradigm for Graves' disease continues to shift away from radioactive iodine and surgery

US Claims Data (2005-2022)[1,2]



Real-World US Claims Analysis (2021-2022)[2]



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1. Villagelin D. et al., Journal of Clincial Endocrinology & Metabolism, 2024; 00, 1-11; North American Respondents – n=263 Endocrinologist
2. Roivant Claims Analysis – 2021 incident patient population, first-line treatment is primary treatment in the first-year post diagnosis, claims review included a five-year lookback to define the incident population.

Substantial unmet need in Graves' disease

Prevalent Market Opportunity

880K

US prevalence of adult Graves' disease[1]

X

85%

Patients treated or previously treated and off ATD therapy[2,3]

X

45%

ATD treatment relapses[2,3]

=

330K

US addressable population choosing not to undergo ablation[4]

Incident Market Opportunity

65K

Annual diagnosed & treated US adult Graves' population[5]

X

85-90%

patients on 1st line ATD[5]

X

35%

ATD treatment relapses[2,4,6]

=

20K

US addressable incident population

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Note: All estimates are approximate

1. Roivant Claims Analysis – 2022 prevalent patient population based on a two-year lookback for diagnosis. 2. Azizi et al. (2019): Note, the relapse rate was calculated as a weighted average considering relapse rate in patients on ATDs <18months is 53% compared to patients on ATDs >18months is 15%. Of the 570K patients treated with ATDs, ~470K are on ATDs <18months and ~100K are on ATDs for >18months. Rates have been applied proportionally. 3. Bandai et al. (2019): Of the ~190K patients previously treated with ATDs and currently monitored off-therapy, ~40% experience relapse, which is 75K. 4. Grove-Laugesen et al. (2023): 3.4% of ATD relapse patients will pursue ablation. 3.4% applied to the ~340K ATD treatment relapse patients is ~10K . 5. Roivant / Inovalon Claims Analysis – 2021 incident patient population, first-line treatment is primary treatment in the first-year post diagnosis, claims review included a five-year lookback to define the incident population, IMVT Market Research 2020-2023. 6. Stokland et al. (2023).

Myasthenia Gravis

Best-in-Class Opportunity

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IMVT-1402 has the potential to improve Myasthenia Gravis treatment outcomes as a best-in-class therapy, leveraging batoclimab learnings

High Unmet Need	95% of Neurologists agree there is an opportunity for greater disease control (e.g., deeper responses)[1]
Autoantibody Pathology	Classic IgG mediated disease, with proven anti-FcRn mechanistic response[2]
Lower is Better	First-gen anti-FcRn batcolimab demonstrated deeper IgG suppression is consistently associated with deeper clinical effect[2]
Optimized Study Design	Simple parallel continuous dose trial design with two dose options, designed to demonstrate a clear difference of effect between doses
Potentially Registrational Trial Initiated	Potentially registrational trial enrolling with self-administration via market-proven autoinjector

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1. IMVT Market Research HCP MG Unmet Need: Part II (n=85), 2025 Neurologists/Neuromuscular Specialists treating ~28 gMG patients/month, reporting T3B percentages; 2. IMVT Investor Presentation March 19, 2025

MG patients and providers indicate a need for deeper and more durable disease control

Deeper Disease Control	Durable Response	Continuous Control	Dose Flexibility
95% Neurologists agree that despite recent advancements with FcRn inhibitors, there is an opportunity for **greater disease control** (e.g., deeper responses)[1]	**95%** Neurologists indicate that their existing MG patients could benefit from a new therapy that offers **greater durability**[2]	**84%** Neurologists report that their patients experience **breakthrough symptoms** with currently available FcRn inhibitors[1]	**92%** Neurologists agree a treatment with high-dosage and standard **dosage strength options is attractive** because it allows **flexibility** to address residual disease as needed[2]

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Notes: 1. IMVT Market Research HCP MG Unmet Need: Part II (n=85), 2025 Neurologists/Neuromuscular Specialists treating ~28 gMG patients/month, reporting T3B percentages; 2: gMG treatment preferences survey 2024 (n=77), Neurologists/Neuromuscular Specialists treating ~38 gMG patients/year.

Phase 3 batoclimab MG data[1] positions IMVT-1402 as the potentially best-in-class anti-FcRn

01 680 mg batoclimab outperformed other anti-FcRn, complement, and CD19 inhibitors, demonstrating highest MG-ADL reduction from baseline (-5.6 points) observed in any global Phase 3 MG trial to-date

02 Highest rate of patients with minimal symptom expression (42%) observed in MG patients across any FcRn in a Phase 3 trial

03 93% of patients achieve clinical response (MG-ADL reduction of 2 or more points), representing highest response rate achieved in a global Phase 3 trial

04 75% of patients who achieved Minimal Symptom Expression (MG-ADL = 0 or 1) on 680 mg dose by Week 6 maintained MSE status for ≥6 weeks

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Notes: Statements are based on cross-trial comparisons and not data from head-to-head studies. Caution should be exercised when evaluating data across trials due to differences in trial designs and participant characteristics.

MG data presented for acetylcholine receptor antibody-positive patients; MSE: Minimal Symptom Expression; MG-ADL: Myasthenia Gravis Activities of Daily Living scale

1. IMVT Investor Presentation March 19, 2025

MG: IgG-mediated autoimmune disease with growing enthusiasm for the anti-FcRn class

Market Opportunity

59K – 116K
Total MG patients in the US[1,2]

x

85%
anti-acetylcholine receptor (AChR) antibody positive[3]

x

35%
who are not well-controlled on standard of care[4,5]

=

20K – 35K
US addressable population

Despite innovation, patients report residual and breakthrough symptoms on anti-FcRn therapy

70% of patients currently on an anti-FcRn report having very or extremely bothersome symptoms

- **97%** experiencing fatigue and muscle weakness
- **~3 in 4** report drooping eyelids walking/coordination issues, blurred/double vision
- **~1 in 2** report difficulty chewing, speech difficulty, weakness of eye muscles

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Note: All estimates are approximate

1. Phillips LH 2nd, et al. (1992) The epidemiology of myasthenia gravis in central and western Virginia. Neurology. 42(10):1888-93; 2. Mina-Osorio P, et al. Incidence and prevalence of myasthenia gravis: analysis of a US commercial insurance claims database. Presented at American Association of Neuromuscular and Electrodiagnostic Medicine; 1-4 November 2023. Phoenix, Arizona. 3. Lazaridis K, Tzartos SJ. Autoantibody Specificities in Myasthenia Gravis; Implications for Improved Diagnostics and Therapeutics. Front Immunol. 2020. 4. Wang L, Zhang Y, He M. Clinical predictors for the prognosis of myasthenia gravis. BMC Neurol. 2017. 5. IMVT Market Research HCP Unmet Need 2025

Chronic Inflammatory Demyelinating Polyneuropathy

Best-in-Class Opportunity

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IMVT-1402 has the potential to deliver best-in-class efficacy in chronic inflammatory demyelinating polyneuropathy (CIDP)

High Unmet Need	30-50% of CIDP patients are inadequately controlled with existing therapies[1]
Lower is Better	First-gen anti-FcRn batcolimab demonstrated deeper IgG suppression delivered greatest in-class mean change from baseline in aINCAT score in CIDP patients[2]
Optimized, Patient-Centric Study Design	Simplified study design leveraging prior batoclimab experience to eliminate need for patient worsening via washout prior to treatment
Potentially Registrational Trial Initiated	Potentially registrational trial enrolling with self-administration via market-proven autoinjector

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1. Internal Market Research Market Dynamics 2024
2. IMVT batcolimab initial Period 1 Data Investor Presentation March 19, 2025

Batoclimab CIDP Phase 2b proof-of-concept data[1] positions IMVT-1402 to potentially be best-in-class

Best-in-class efficacy observed across multiple efficacy measures: aINCAT, I-RODS, MRC-SS, and grip strength[2]

Demonstrated that deeper IgG reductions translate to improved response with 84% aINCAT response rate in patients achieving ≥70% IgG reduction

Generated learnings to inform IMVT-1402 trial design optimization

Opportunity to accelerate registrational program for IMVT-1402 in CIDP

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1. IMVT batcolimab initial Period 1 Data Investor Presentation March 19, 2025
2. Based on data pooled for 340 mg and 680 mg batoclimab dose groups

CIDP patients and providers are seeking a new treatment option that reduces symptom and treatment burden[1]

75% of HCPs prefer to treat CIDP patients as early and aggressively as possible

Sizable Market Opportunity

58K

Total CIDP Patients in the US[2]

X

30%

who are inadequately controlled on treatment[4]

=

16K

US addressable population

Substantial Unmet Need

Lower Relapse Rates	30-50% of CIDP patients are inadequately controlled with existing therapies[5]
Improved Response and Durability	60% of physicians report a need for better response to treatment and more durable CIDP treatments
More Convenient Dosing Options	~90% of physicians noted a high need for treatments with improved ROA (e.g., at home administration)
Improved safety & tolerability	71% of US physicians report a need for treatment options with fewer side effects[6]

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Note: All estimates are approximate.

1. Internal Market Research Market Dynamics 2024 2. Broers M, et al (2019) Incidence and prevalence of CIDP: a systematic review and meta-analysis. Neuroepidemiology 52(3–4):161–172; 3. Querol, L., et al. Systematic literature review of burden of illness in chronic inflammatory demyelinating polyneuropathy (CIDP). J Neurol 268, 3706–3716 (2021).; 4. 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.; 5. Internal Market Research HCP Survey and KOL advising 2023 6. Internal Market Research CIDP Patient Journey 2022

ACPA+ Difficult-to-Treat Rheumatoid Arthritis

First- and Best-in-Class Opportunity

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IMVT-1402 has the potential to achieve a first- and best-in-class profile for people with ACPA+ difficult-to-treat rheumatoid arthritis (D2T RA)

High Unmet Need Subgroup	5-20% of RA patients are difficult-to-treat (D2T), with inadequate or loss of response to multiple classes of advanced therapies[1]
Autoantibody Pathology	Autoantibodies such as ACPA play a key role in pathophysiology, and ACPA-positive RA is associated with severe disease and poor outcomes
Lower is Better	Phase 2 FcRn RA data demonstrated that greater IgG reduction led to greater autoantibody reductions, which correlated with greater clinical response[2]
Potentially Registrational Trial Initiated	Potentially registrational trial enrolling with self-administration via market-proven autoinjector

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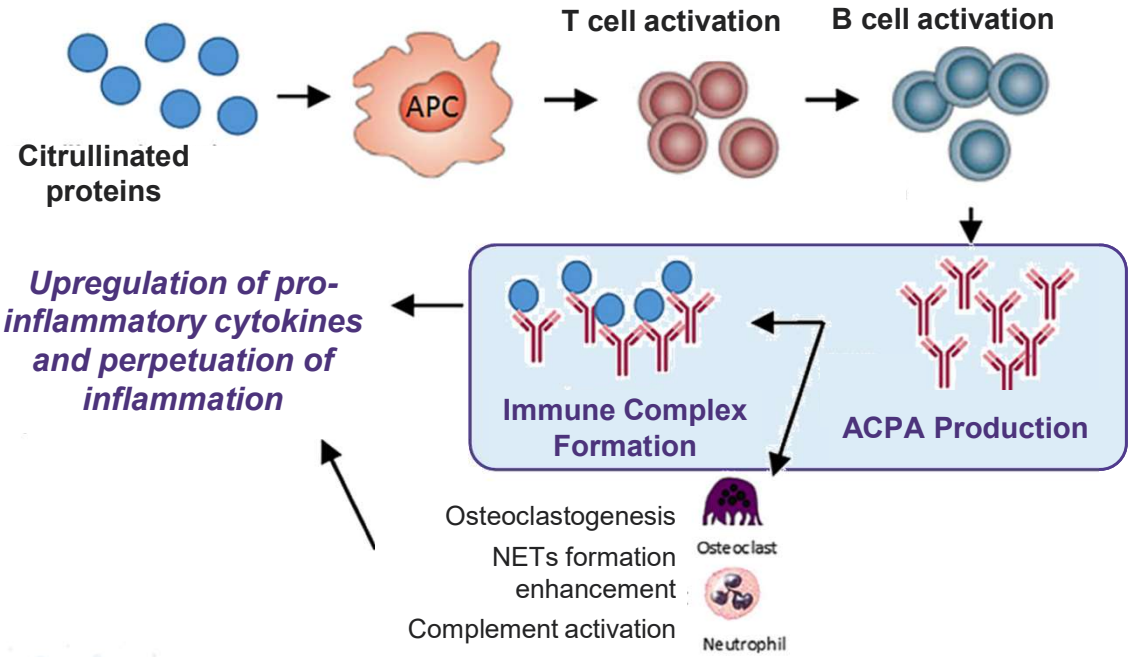
1. Takanashi S, et al. Rheumatology. 2021;60:5247-56 2. Taylor PC et al. "Efficacy and Safety of Nipocalimab in Patients with Moderate to Severe Active Rheumatoid Arthritis (RA): The Multicenter, Randomized, Double-blinded, Placebo-controlled Phase 2a IRIS-RA Study *Presented at ACR, Nov 10-15, 2023.*
ACPA: anti-citrullinated protein antibody

In addition to cellular autoimmunity and cytokine dysregulation, autoantibodies like ACPA play a key role in the pathophysiology of RA

Autoantibodies such as Rheumatoid Factor (RF) and ACPA are present in ~75% of RA patients[1]

Role of ACPA in RA pathophysiology

1. Antigen presenting cells (APCs) process and present citrullinated peptides to T cells
2. T cells activate B cells to generate autoantibodies
3. Immune complex formation upregulates pro-inflammatory cytokines
4. ACPA may bind to osteoclasts and thereby promote bone erosion



Anti-FcRn has the potential to directly target underlying disease biology by lowering pathogenic autoantibodies (i.e., ACPA) and immune complexes

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Source: 1. Brito Rocha et al. Advances in Rheumatology (2019) 59:2
ACPA: anti-citrullinated protein antibody

Difficult-to-treat RA is estimated to comprise 5-20% of RA patients whose disease cannot be managed by available therapies

Need for More Options

- Estimated 5-20% of patients remain symptomatic despite multiple treatment rounds[1]
  - These patients need new therapies and approaches, according to a global survey of 410 rheumatologists
- Difficult-to-treat (D2T) RA defined by EULAR as[2]:
  - Multiple DMARD failures
  - Signs suggestive of active/progressive disease
  - Symptom management viewed as problematic to doctor and/or patient

D2T RA Criteria

- At least moderate disease activity as defined by composite endpoints which include tender and swollen joint counts
- Progressive joint damage on imaging
- Inability to decrease chronic glucocorticoid therapy below 7.5mg/day
- Ongoing RA symptoms and QoL impact despite therapy

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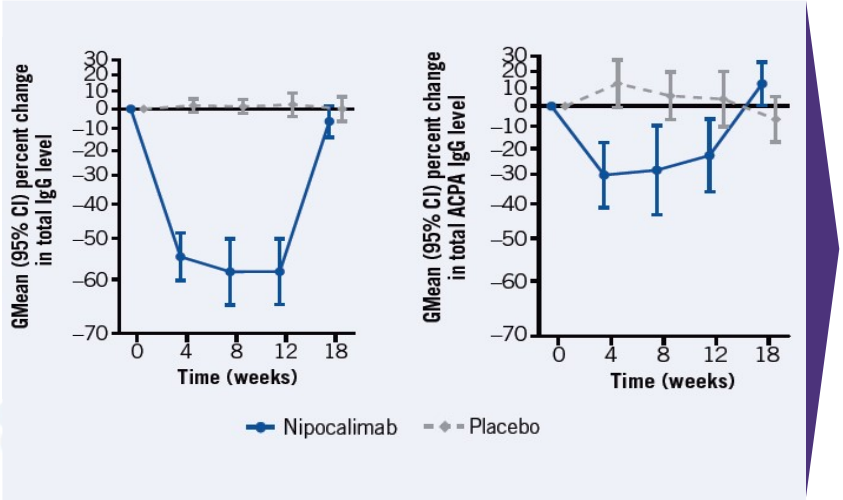
1. Roodenrijs NMT et al. *Ann Rheum Dis* 2018;1705 -09, 2. Nagy G, et al. *Ann Rheum Dis* 2021; 80: 31-35
EULAR: European League Against Rheumatism Collaborative Initiative; DMARD: disease-modifying antirheumatic drug; QoL: Quality of Life

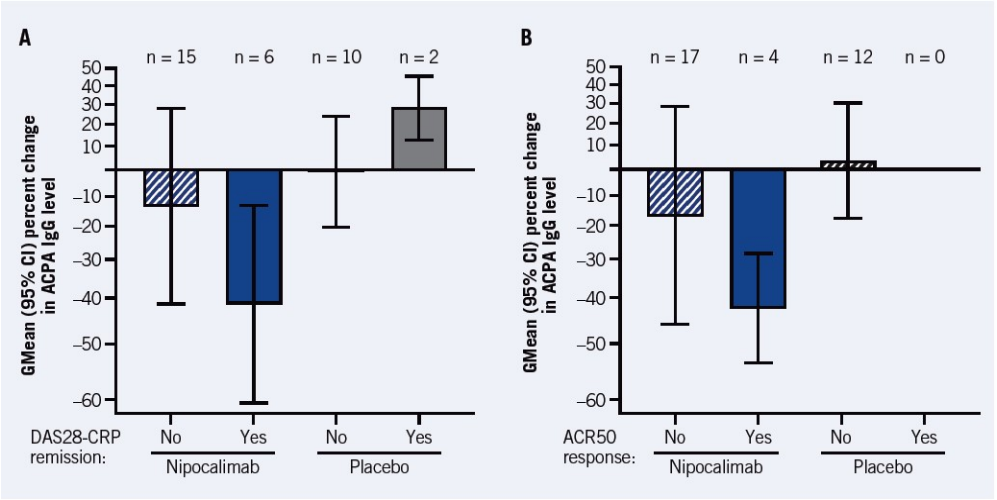
Publicly available nipocalimab data in RA showed that deeper autoantibody (ACPA) reduction correlated with clinical response[1]

Select results from a study of FcRn inhibition vs. placebo in biologic-experienced RA patients

~60% Total IgG And ~30% Pathogenic Auto-Ab (ACPA) Reductions In JNJ Phase 2 RA Study



Correlation Between Auto-Ab Reductions and Clinical Response using (A) DAS28-CRP Remission and (B) ACR50 Response at Week 12



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Notes: 1. Pharmacodynamic effects of nipocalimab in patients with moderate to severe active rheumatoid arthritis (RA): Results from the multicenter, randomized, double-blinded, placebo-controlled Phase 2A IRIS-RA study. Janssen Research & Development, ACR poster, November 2023. Auto-Ab: Autoantibody; ACPA: Anti-citrullinated protein autoantibody; DAS28-CRP: Disease Activity Score 28 using C-reactive protein; GMean: Geometric mean.

Of the 1.5M US RA patients[1], a subset progresses to D2T status in a relatively short period of time and requires new therapeutic options

Market Opportunity

490K

US prevalence of severe RA[2]

X

15%

autoantibody positive with inadequate response to prior b/tsDMARD[2,3]

=

70K

US addressable population

Patient Journey Learnings

Fewer than 50% of RA patients remain on first therapy	~50% of patients fail their first b/tsDMARD therapy within the first year of treatment[4,5]
D2T emerges for some in ~4 years	In a large US registry, the median time to meeting D2T criteria was 4 years in those who were D2T[6]
5%-20% of RA patients are D2T	5%-20% of all RA patients meet the criteria for D2T in the US[6]

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Note: All estimates are approximate

1. Aletaha D, Smolen JS. *JAMA.* 2018;320(13):1360. 2. GlobalData Analysis and Forecast, 2023. 3. Okada et al. Ann Rheum Dis 2019;78; 446-453. 4.. Murray K et al. *Arthritis Res Ther* 2021; 23(1):25. 5. Rosenberg V et al. *Adv Ther* 2023; 40(10):4504-4522. 6. *Takanashi S, et al. Rheumatology. 2021;60:5247-56*

b/tsDMARD: biologic (b) or targeted synthetic (ts) disease-modifying antirheumatic drug

Sjögren's Disease

Best-in-Class Opportunity

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Sjögren's disease (SjD) is a potentially best-in-class indication for IMVT-1402

High Unmet Need Disease	No therapies are approved for the treatment of primary SjD
Autoantibody Pathology	Autoantibodies detected in ~50-70% of patients with primary SjD; anti-FcRn proof of mechanism established
Lower is Better	Nipocalimab data demonstrated that deeper IgG reduction leads to better clinical response across all primary and secondary endpoints
Potentially Registrational Trial Initiated	Potentially registrational trial enrolling with self-administration via market-proven autoinjector

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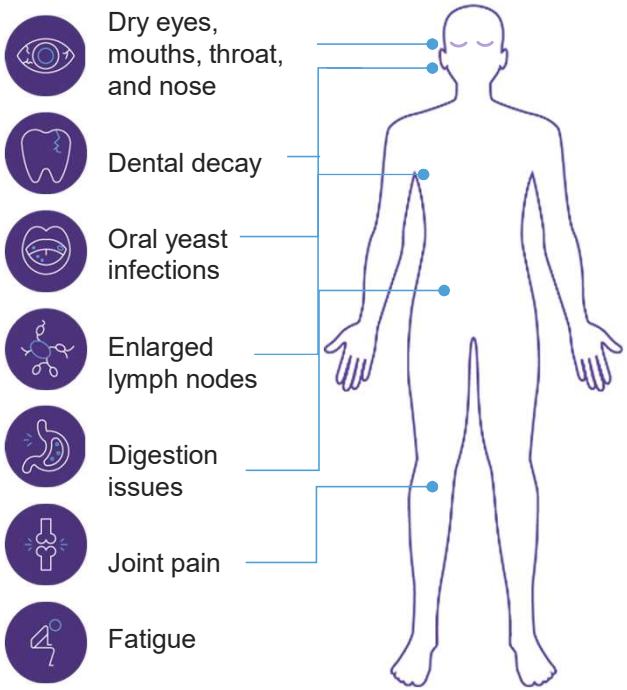
SjD is an autoimmune disease associated with a myriad of clinical manifestations

Disease Overview

- SjD is a chronic autoimmune disease characterized by lymphocytic infiltration of the salivary and lacrimal glands
- Symptoms include severe dryness of the eyes and mouth; the latter frequently associated with difficulty swallowing or speaking, tooth decay, gum disease, and impaired QoL[1,2]
- May occur in isolation (primary SjD) or in association with another systemic autoimmune disease such as RA (secondary SjD)
- SjD can be challenging to diagnose due to the heterogeneity of presentation[3]
- ACR/EULAR classification criteria are now widely endorsed for diagnosing primary SjD

Common symptoms



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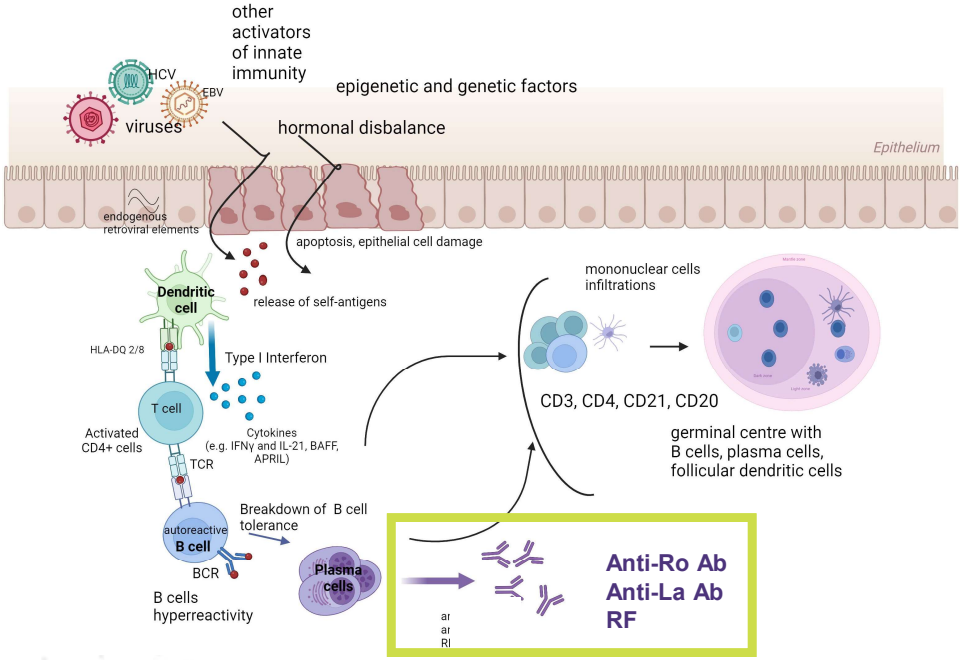
1. Mariette X, et al. *N. Engl J Med.* 2018; 378:931-9
2. Brito-Zeron P et al. *Nature Reviews.* 2016; 2:1-20
3. GlobalData Epi Analysis and Forecast, Oct. 2020

Autoantibodies play crucial roles in both the diagnosis and prognosis of SjD

Autoantibody Involvement

- Serological abnormalities are common in SjD and include autoantibodies, hypergammaglobulinemia, and hypocomplementemia[1]
- Identification of disease-precipitating antibodies were discovered back in 1975. Anti-Ro/SSA and anti-La/SSB antibodies were detected in patients with SjD in 1982[2]
- Present day, autoantibodies are detected in ~50-70% of patients with primary SjD

Disease Pathogenesis[3]

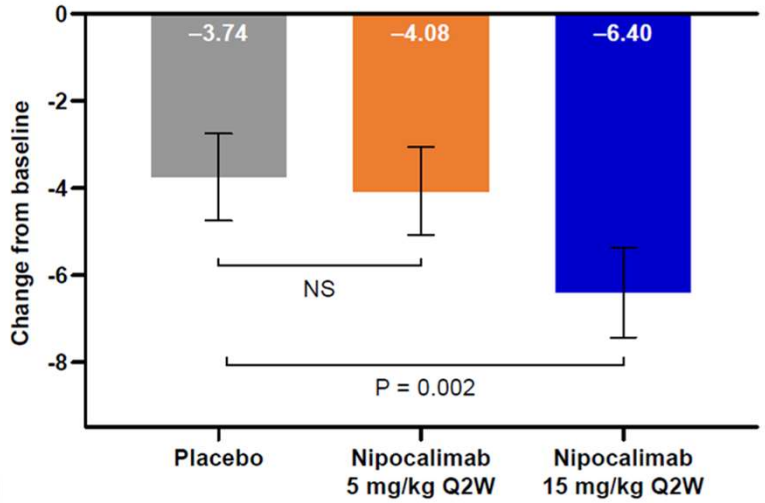


1. Baer AN, et al. Elsevier; 2023. Chapter 45, Clinical aspects of Sjögren's disease; p. 637-647
2. Brito-Zeron P et al. *Nature Reviews.* 2016; 2:1-20
3. Figure reprinted from Maslinska M, Kostyra-Grabczak K. *Front Immunol.* 2024 Sep 19:15:1376723 under the terms of the Creative Commons Attribution License (CC BY)

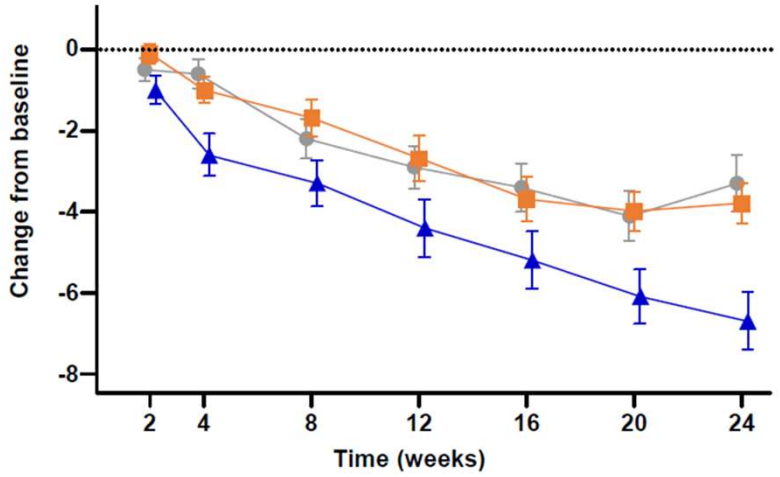
Publicly available nipocalimab data support anti-FcRn proof of mechanism and dose response in SjD

Select results from a study of FcRn inhibition vs. placebo in primary SjD

LS mean (90%) change in ClinESSDAI score at Week 24



Mean (SE) change in ClinESSDAI score



Placebo ■ Nipocalimab 5mg/kg Q2W ▲ Nipocalimab 15mg/kg Q2W

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CI: confidence interval; ClinESSDAI: clinical European League Against Rheumatism Sjögren's Syndrome Disease Activity Index; LS: least squares; NS: not significant; Q2W: every 2 weeks; SE: standard error. 1. Gottenberg JE et al. Efficacy and Safety of Nipocalimab, an Anti-FcRn Monoclonal Antibody, in Primary Sjogren's Disease: Results from a Phase 2, Multicenter, Randomized, Placebo-Controlled, Double-Blind Study (DAHLIAS). ACR Convergence 2024, November 16-19, 2024

Sizable patient group with unmet need for an approved treatment option in SjD

Market Opportunity

290K

US prevalence of primary Sjögren's disease[1]

X

Up to 30%

moderate-to-severe with anti-Ro/SSA antibodies[2,3]

=

90K

US addressable population

Expansion Opportunities

Secondary Sjögren's	Potential to impact conditions with shared autoimmune pathology
Glandular Disease	Unmet need to improve glandular manifestations beyond symptom relief
Less Severe Disease	Disease impact on patient QoL varies widely; so-called "nuisance" symptoms can become debilitating if inadequately managed

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Note: All estimates are approximate

1. GlobalData Analysis and Forecast, January 2025
2. Brito-Zeron P et al. Nature Reviews 2016; 2:1-20
3. Decision Resources Group

Cutaneous Lupus Erythematosus

First-in-Class Opportunity

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IMVT-1402 is potentially first-/best-in-class in Cutaneous Lupus Erythematosus (CLE)

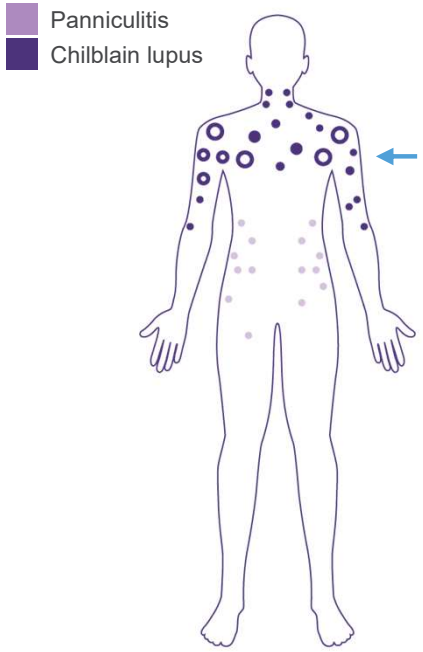
Untapped Market Opportunity	IMVT-1402 has potential to be the first novel targeted therapy for CLE in >50 years[1]
IgG and Immune Complex Driven	Biologic, translational, and mechanistic evidence support the critical role of IgG autoantibodies and immune complexes in the pathogenesis of CLE
Upstream Targeting	Disruption of CLE pathology by upstream targeted approach supported by IMVT-1402 patient case studies
IMVT-1402 Trial Initiated	Proof-of-concept trial enrolling with self-administration via market-proven autoinjector

1. Presto JK, Werth VP: Cutaneous Lupus Erythematosus: Current Treatment Options. Curr Treat Option Rheumatol. 2016; 2(1): 36–48

CLE is a rare, chronic autoimmune disease affecting the skin, with limited available treatment options and high unmet need

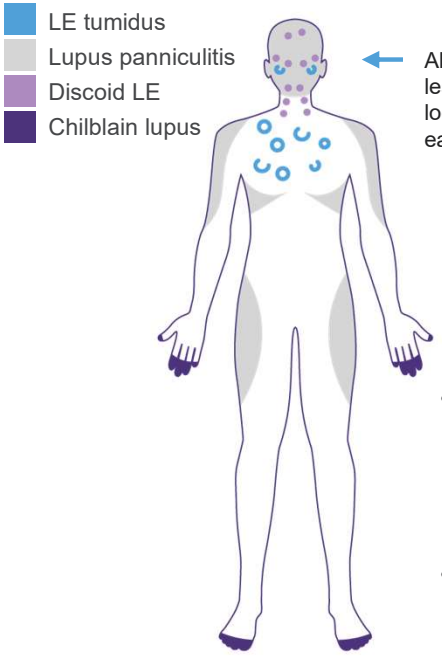
- CLE is a rare, chronic skin disease characterized by skin-specific disease-activity, inflammation and eventually damage[1,2]
- Symptoms include painful skin lesions, itching, burning, and alopecia[3]
- Limited innovation and no novel therapies in >50 years[4]

Subacute Chronic Lupus Erythematosus (SCLE)



- Annular or papulosquamous, psoriasis-like scaling erythematous plaques
- Estimated 59K prevalence (25%)[5]

Chronic Cutaneous Lupus Erythematosus (CCLE)



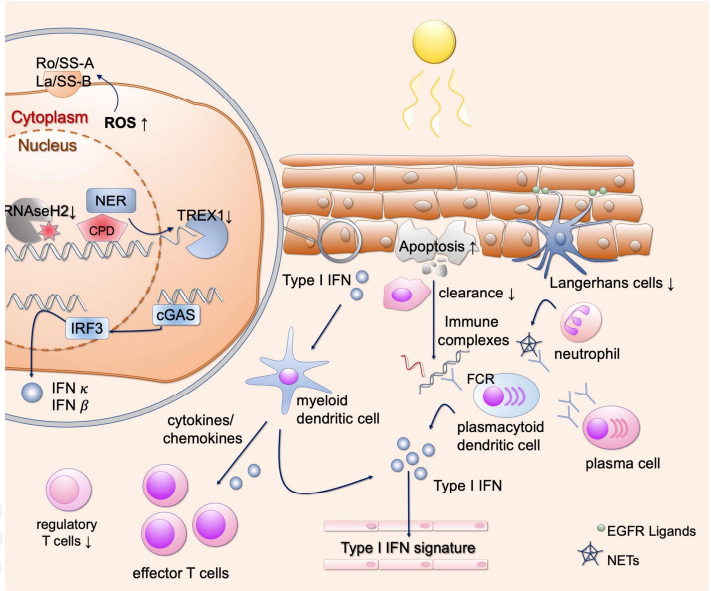
- Scaling, erythematous, typically scarring, disc-shaped plaques, alopecia
- Estimated 94K prevalence (40%)[5]

For the purposes of this presentation, reference to CLE is focused on SCLE and CCLE subtypes.

1. Vale ECSD and Garcia LC. An Bras Dermatol. 2023;98(3):355-372. 2. Presto JK, Werth VP: Cutaneous Lupus Erythematosus: Current Treatment Options. Curr Treat Option Rheumatol. 2016; 2(1): 36–48 Stull, et. al. The Journal of Rheumatology 2023;50:27–35; doi:10.3899/jrheum.220089 3. Klein R, et al. J Am Acad Dermatol. 2011;64(5):849-858, 4. Wahie S, Meggitt SJ. Long-term response to hydroxychloroquine in patients with discoid lupus erythematosus. Br J Dermatol. 2013 Sep;169(3):653-9. doi: 10.1111/bjd.12378. PMID: 23581274 5. Internal market research Spherix 2024

CLE: IgG autoantibodies and immune complexes mediate a cycle of self-amplifying skin inflammation and tissue damage in the skin

Pathogenesis of CLE Disease



UV light triggers enhanced cell death, IgG autoantibody immune response, and produces immune complex formation, leading to skin tissue damage and increased inflammation[1]

Autoantibody Involvement[2]

CLE specific IgG autoantibodies produced (i.e., Ro/SSA, La/SSB)

IgG Autoantibodies:

- Induce skin cell death
- Trigger recruitment of inflammatory cells that form immune complexes

Immune Complex Involvement[2]

Immune complexes can activate receptors of the innate immune system that drive:

- Inflammation
- Tissue damage
- Skin cell death
- Recruit other immune cells

FcRn inhibition has the potential to disrupt CLE pathology

IMVT-1402's deep suppression of IgG autoantibodies and immune complexes has the potential to dampen multiple downstream inflammatory cascades by providing upstream inhibition of inflammatory cascade

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1. Klein Benjamin, Kunz Manfred. Current concepts of photosensitivity in cutaneous lupus erythematosus. Frontiers in Medicine, 2022. 10.3389/fmed.2022.939594.
2. Achtman, J.C., Werth, V.P. Pathophysiology of cutaneous lupus erythematosus. Arthritis Res Ther 17, 182 (2015). https://doi.org/10.1186/s13075-015-0706-2

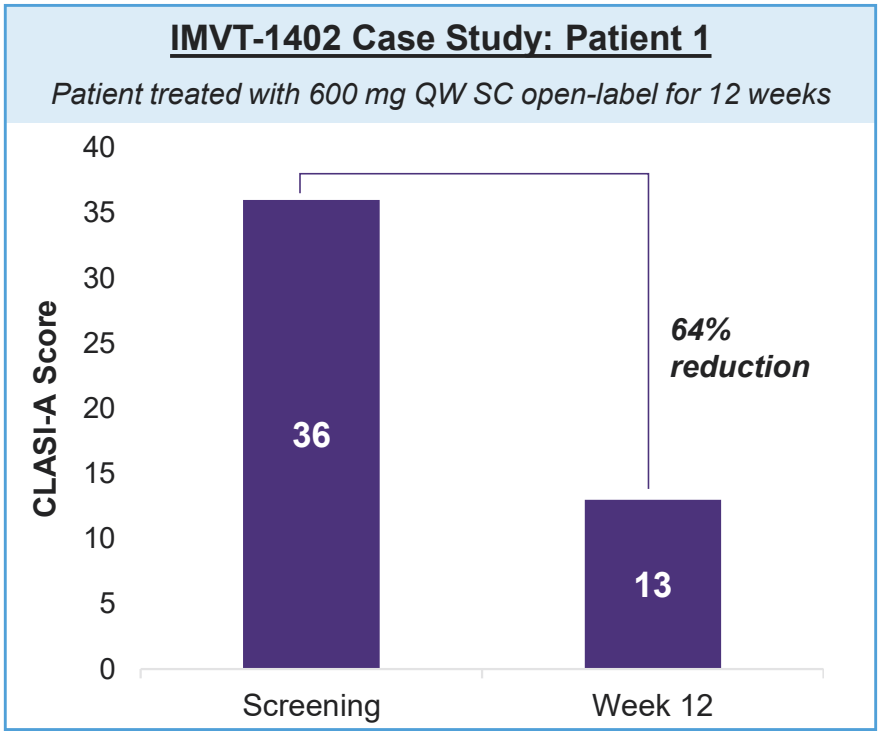
Case Study: 12-Week Treatment with IMVT-1402 in CLE

Baseline Demographics

- Female, 57
- Subacute CLE and alopecia
- Multiple skin locations affected
- CLASI-A score at screening = 36
- Background medications: hydroxy-chloroquine, methotrexate, leflunomide

Treatment Outcomes

- **>60% reduction in CLASI-A** score to 13 by week 12
- Significant clinical **improvement in both skin lesions and alopecia**
- **78% total IgG reduction** from baseline achieved by week 12



Second patient dosed also showed >50% improvement in CLASI-A score by week 12 (CLASI-A at screening of 18 reduced to 8 by week 12)

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CLASI-A: Cutaneous Lupus Area and Severity Score Index – Activity; QW: once weekly; SC: subcutaneous
IMVT Data on File 2025

Dermatologists desire a skin-focused, targeted biologic that addresses CLE unmet needs[1]

IMVT-1402 has potential to be the first novel dermatology therapy for CLE in >50 years[2]

Considerable Market Opportunity

153K

US prevalence of SCLE and CCLE[3]

X

Up to 50%

Non-responders to antimalarials or topicals[4]

=

75K

Target addressable US population

Potential Differentiated Profile

Targeted Biologic	Dermatologists are frustrated by the skin-specific therapies currently available
Quick control	Speed of action is critical to disease control and QoL- prevention of scarring and potential disfigurement[1]
Sustained remission	90% of dermatologists cite sustained remission and reduced severity of flares as top unmet needs[1]
Improved safety & tolerability	80% of HCPs report lack of long-term efficacy, tolerability and toxicity risks with current CLE treatments[2]

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Note: All estimates are approximate

1. Internal Market Research CLE Dermatologist Unmet Need 2023, Internal Market Research CLE Patient Journey 2024 2. Presto JK, Werth VP: Cutaneous Lupus Erythematosus: Current Treatment Options. Curr Treat Option Rheumatol. 2016; 2(1): 36–48 3. Jarukitsopa et al 2015; IMVT Spherix Internal Market Research 4. Wahie S, Meggitt SJ. Long-term response to hydroxychloroquine in patients with discoid lupus erythematosus. Br J Dermatol. 2013 Sep;169(3):653-9. doi: 10.1111/bjd.12378. PMID: 23581274

Appendix

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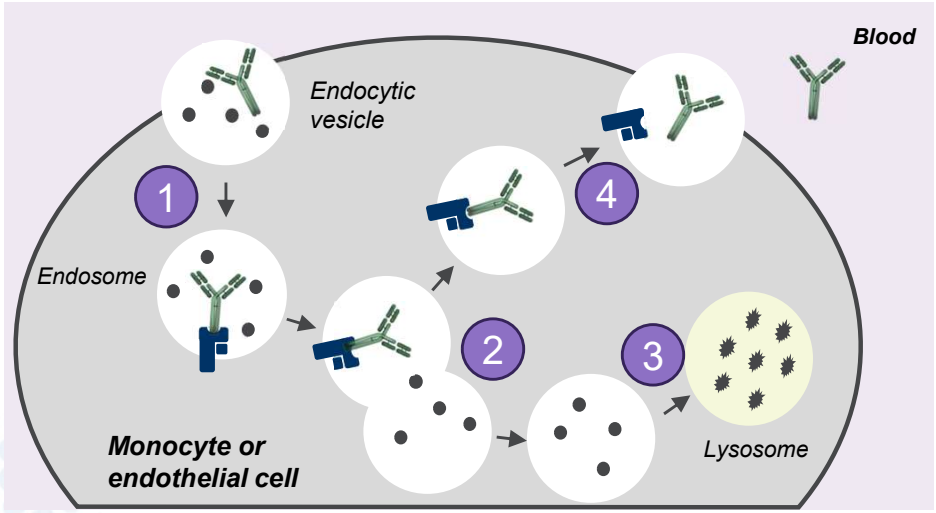
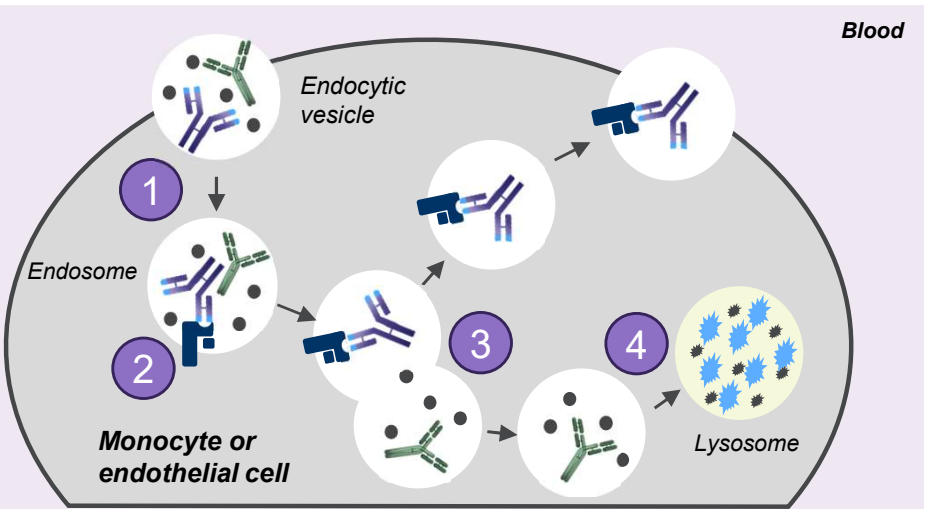
Our target: Neonatal Fc receptor (FcRn)

FcRn maintains levels of antibodies (IgG) in circulation by preventing their degradation



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

FcRn inhibitor blocks binding of IgG to FcRn and promotes their removal and degradation



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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Key: IgG | FcRn | FcRn inhibitor | Serum protein

Totality of FcRn clinical evidence demonstrates that deeper IgG reductions result in better clinical outcomes across multiple indications

	Company	Evidence of Greater IgG Reductions Translating to Clinical Benefit
GD	IMMUNOVANT	Phase 2: Greater IgG reduction across treatment cohorts ➔ higher rates of anti-TSHR antibody reduction and numerically higher responses for ATD dose tapering and ATD discontinuation
CIDP	IMMUNOVANT	Phase 2b: Greater IgG reduction across treatment cohorts ➔ higher aINCAT response rates
MG	IMMUNOVANT	Phase 2: Deeper IgG across treatment arms ➔ AChR autoantibody reductions and enhanced clinical activity Phase 3: 680 mg dose with greater IgG reduction out-performs 340 mg dose across endpoints
	janssen	Phase 2: Patient-level scatter plot demonstrating greater IgG declines ➔ greater MG-ADL improvements[2]
	argenx	Phase 3: Patient-level scatter plot demonstrating greater IgG declines ➔ greater MG-ADL improvements[3]
TED	IMMUNOVANT	Phase 2s: Greater IgG reduction across arms ➔ higher rates of anti-TSHR antibody reduction and greater clinical response rates
SjD	janssen	Phase 2: Dose-dependent efficacy ➔ deeper IgG reduction (same dose regimen used in RA trial) led to better clinical response[4]
RA	janssen	Phase 2: In those patients with greater IgG reduction ➔ correlation with greater autoAb reduction ➔ correlation with greater clinical response[5]
ITP	ucb	Phase 2: Greater IgG reduction across arms ➔ greater platelet responses[6]
PV/PF	argenx	Phase 2: More intensive dosing regimens across arms led to deeper IgG lowering ➔ deeper skin responses and lower rates of relapse[7]

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1. Many of the analyses above were post-hoc and not all were statistically significant. Cross trial and post-hoc analyses are inherently limited and are presented for hypothesis generating purposes only, nevertheless consistent and numerically positive increases in efficacy were observed as noted above; 2. Momenta Vivacity-MG Interim Phase 2 Investor Presentation, 2020; 3. argenx JP Morgan Healthcare Conference Presentation January 2021; 4. EULAR 2024 Abstract. 5. Janssen Research & Development, ACR poster, November 2023. 6. IgG reduction at day 8 estimated by WebPlotDigitizer for 4mg/kg, 7mg/kg and 10mg/kg doses. 7. Argenx phase 2 PV/PF publication, Br J Dermatol. 2022 Mar;186(3):429-439; MG: Myasthenia gravis, TED: Thyroid eye disease, GD: Graves' disease, ITP: Immune thrombocytopenic purpura, RA: Rheumatoid arthritis;

Innovation over time has historically raised the bar for clinical outcomes

	Pre-targeted therapies	1st Gen Innovation	Next Gen Innovation
PSO	Physician Global Assessment	PASI 75	PASI 100
MS	Annual Relapse Rate Reduction 30%	Expanded Disability Status Scale (EDSS)	No Evidence of Disease Activity (NEDA)
MG	MG-ADL Response	Minimum Symptom Expression (MSE)	**Deep and Durable Response**

Innovation over Time

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PSO: psoriasis; MS: multiple sclerosis; PASI: psoriasis area severity index

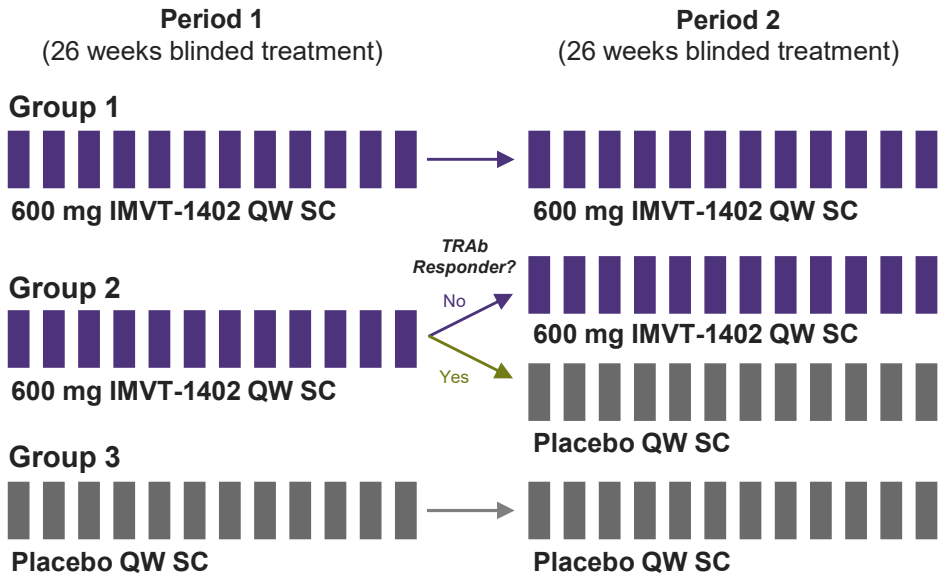
IMVT-1402 potentially registrational trial in Graves' disease

Inclusion[a]

- Adults with active Graves' disease as documented by presence of TSH-R binding autoantibodies (TRAb)
- Subjects on an ATD for ≥12 weeks before the Screening Visit
- Subjects who are hyperthyroid based on suppressed TSH despite ATD treatment

Randomization (1:1:1)

Treatment Period: 52 weeks
N = 240



Off-Treatment Follow-up (52 weeks)

ATD titration to lowest effective dose (including 0 mg/day) to maintain euthyroidism

Primary Endpoint at Week 26: Proportion of participants who become euthyroid[b] and stop ATD

Key Secondary Endpoint at Week 52: Proportion of participants who become euthyroid[b] and stop ATD

Design enables study of remission as upside

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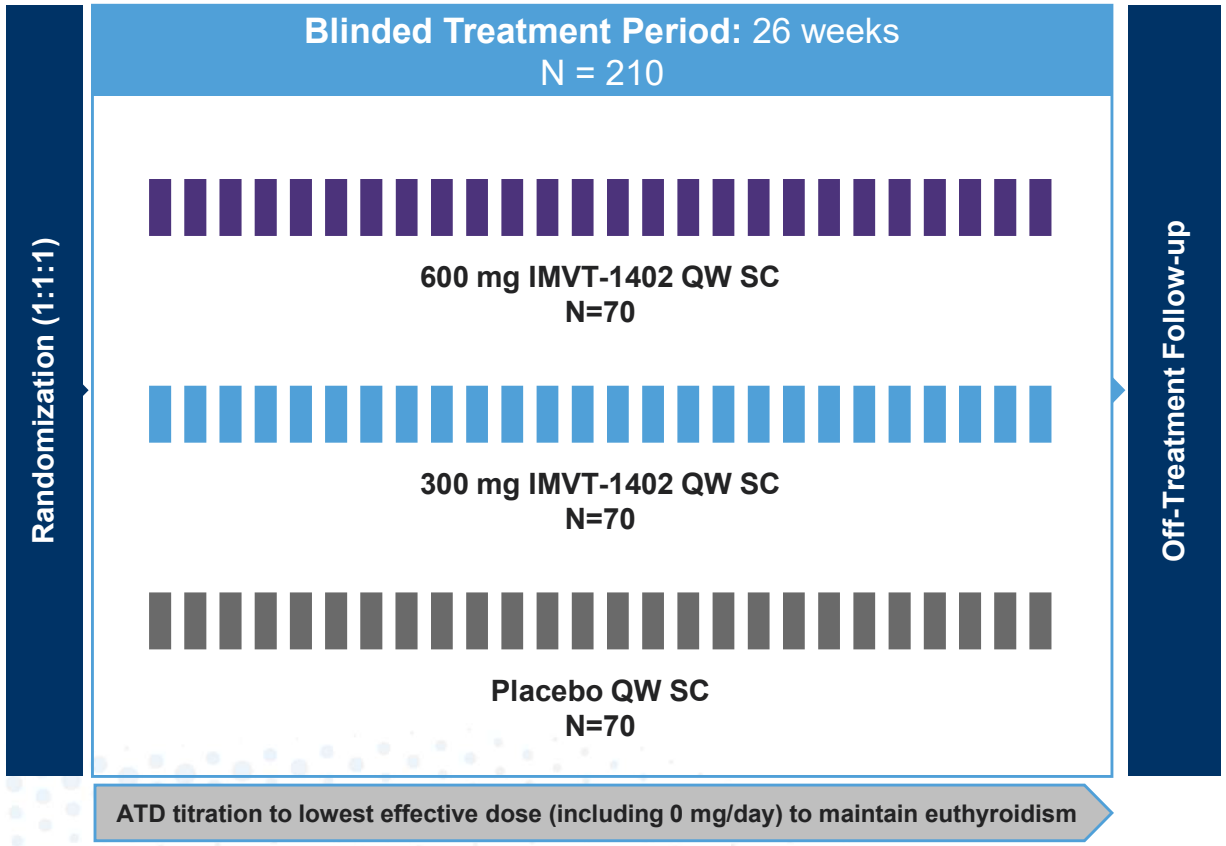
a: Additional inclusion and exclusion criteria not listed on slide
b. Euthyroid = T3/T4 and TSH within normal limits
TSH: Thyroid-stimulating hormone; ATD: Antithyroid drugs; QW: Weekly; SC: Subcutaneous; T3 = triiodothyronine; FT3: free triiodothyronine; FT4: free thyroxine; ULN: upper limit of normal

IMVT-1402 second potentially registrational trial in Graves' disease

Inclusion[a]

- Adults with active Graves' disease who are hyperthyroid based on suppressed TSH despite ATD treatment



Primary Endpoint at Week 26: Proportion of participants on 600 mg who become euthyroid[b] and off ATD versus placebo

Secondary Endpoint at Week 26: Proportion of participants on 600 mg who have T3 (Total T3 or FT3) and FT4 ≤ ULN and off ATD

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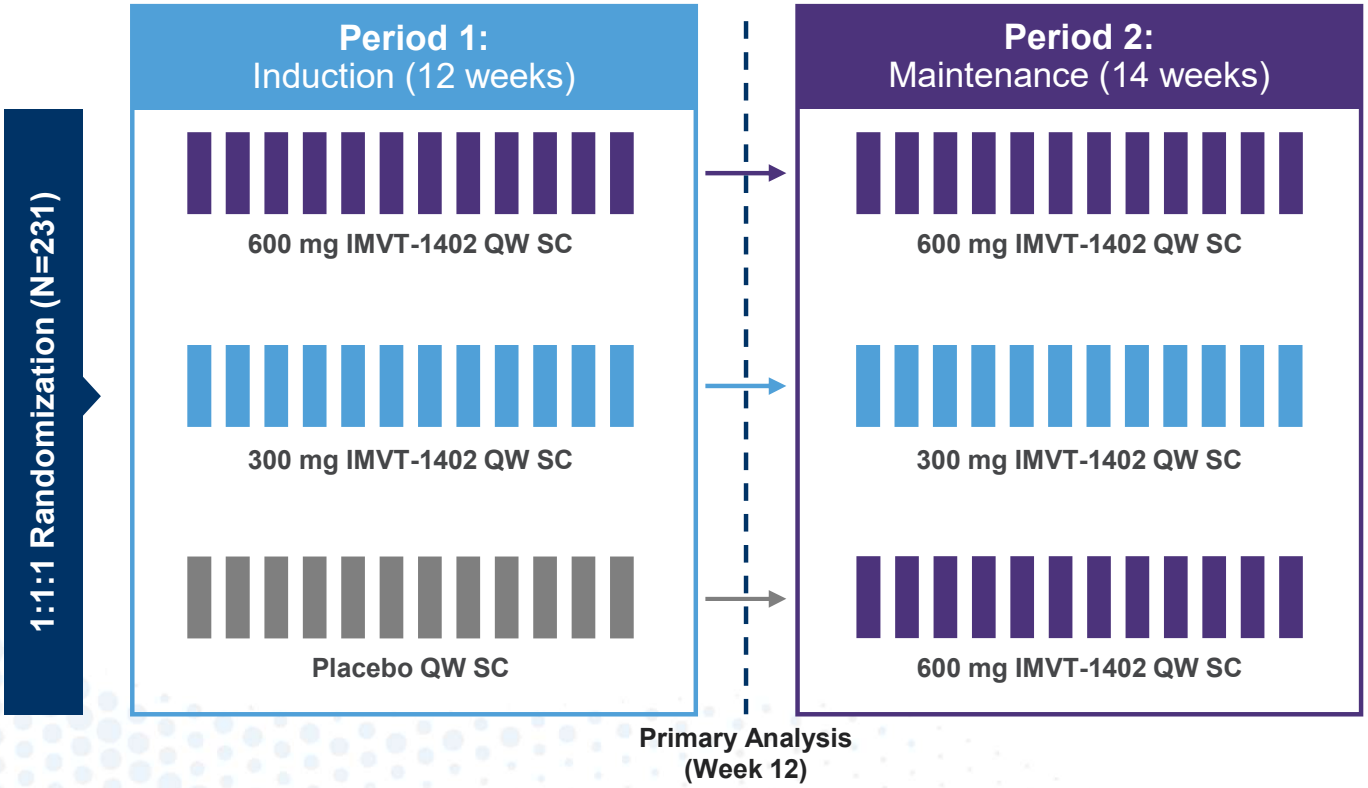
a: Additional inclusion and exclusion criteria not listed on slide
b. Euthyroid = T3/T4 and TSH within normal limits
TSH: Thyroid-stimulating hormone; ATD: Antithyroid drugs; QW: Weekly; SC: Subcutaneous; T3 = triiodothyronine; FT3: free triiodothyronine; FT4: free thyroxine; ULN: upper limit of normal

IMVT-1402 potentially registrational trial in MG

Trial designed to enable demonstration of deep, durable responses



Primary Analysis Population: AChR Ab+, MuSK+, LRP4+

Primary Endpoint: Change in MG-ADL from baseline through 12 weeks

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QW: Weekly; SC: Subcutaneous injection; AChR Ab+: Acetylcholine receptor antibody-positive; MuSK+: Muscle-specific tyrosine kinase antibody-positive; LRP4: Low-density lipoprotein receptor-related protein 4 antibody-positive; MG-ADL: Myasthenia Gravis Activities of Daily Living scale; QMG: Quantitative Myasthenia Graves scale.

IMVT-1402 potentially registrational trial in CIDP

Trial designed to maximize dose response with IMVT-1402 600 mg

Inclusion[a]

- Clinical criteria for typical CIDP
- Having evidence of active disease

2:1 Randomization

Blinded Treatment Period: 24 Weeks
N = 162

600 mg IMVT-1402 QW SC
N = 108

Placebo QW SC
N = 54

Primary Endpoint at Week 24:

Proportion of participants remaining relapse-free (aINCAT)

Simplified study design without washout period and flare requirement prior to randomization based on experience in the batoclimab CIDP study in identifying patients with active disease

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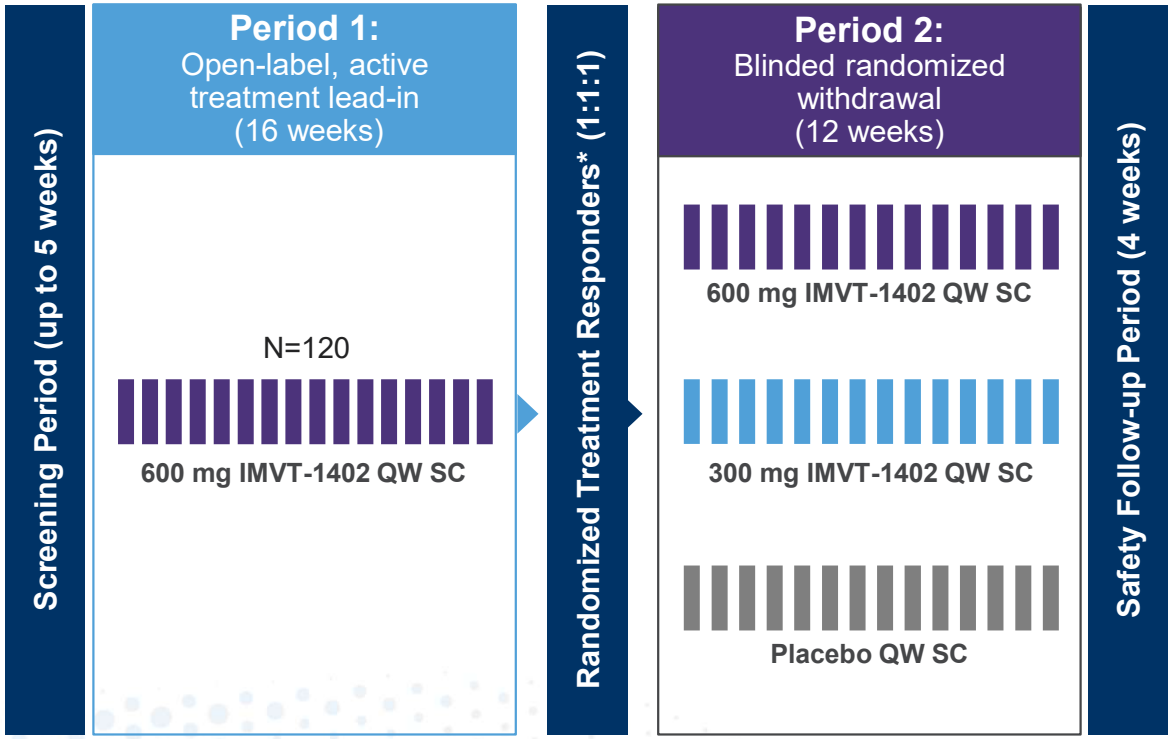
a. Additional inclusion and exclusion criteria not listed on slide. QW: Once weekly; SC: Subcutaneous; aINCAT: Adjusted Inflammatory Neuropathy Cause and Treatment disability score

IMVT-1402 potentially registrational trial in ACPA+ D2T RA

Trial designed as open label lead-in with randomized withdrawal D2T population enriched for higher baseline ACPA levels

Inclusion[a]

- CRP > upper limit of normal
- Active RA defined as ≥ 6/68 tender/painful joints, ≥ 6/66 swollen joints (SJC), and DAS28-CRP > 4.1
- Anti-citrullinated protein antibody positive (ACPA+)
- Inadequate response to 2 or 3, but not more than 3, classes of b/tsDMARDs
- On stable treatment with csDMARD



Endpoints

Primary endpoint:

For participants achieving ACR20 response at Weeks 14 and 16, proportion of participants who achieve ACR20 response at Week 28

Secondary endpoint:

Change from baseline in CDAI and SDAI from Weeks 16 to Week 28

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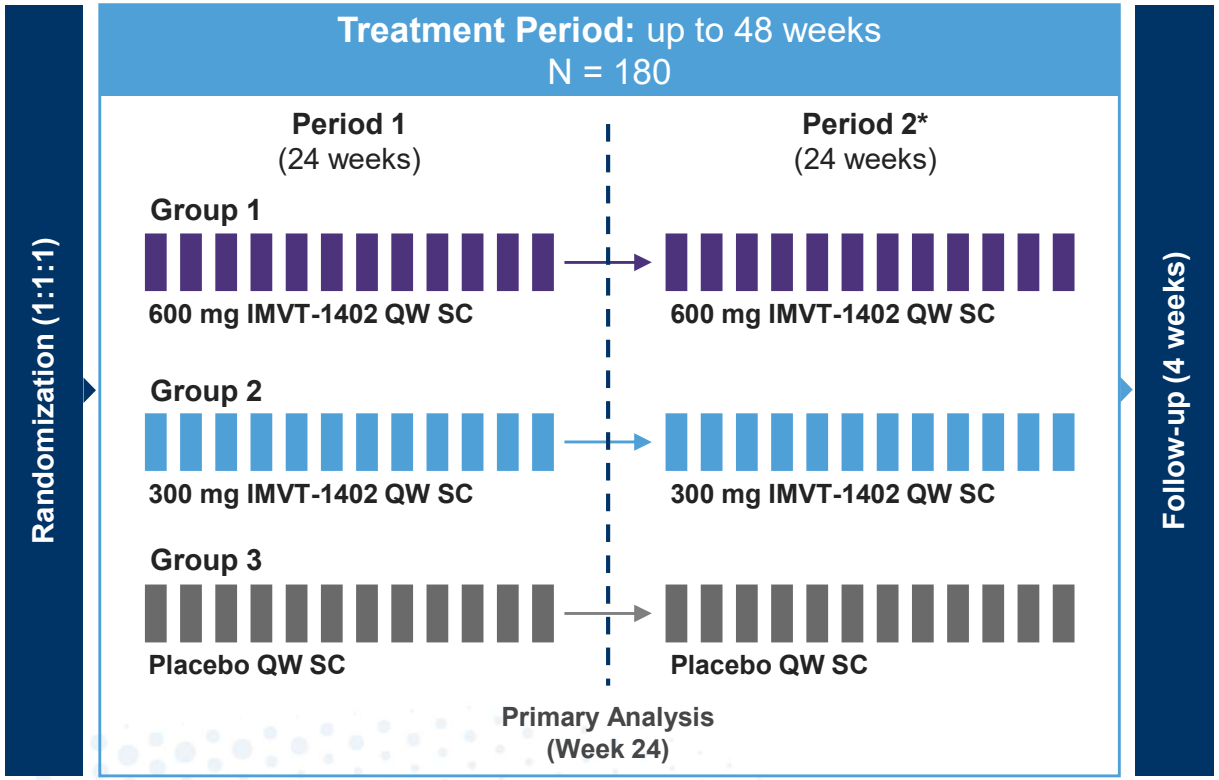
*Meets ACR20 criteria at Week 14 & Week 16

a. Additional inclusion and exclusion criteria not listed on slide. C-reactive protein (CRP; Disease Activity Score-28 (DAS28); Clinical Disease Activity Index (CDAI); Simplified Disease Activity Index (SDAI); Disease-modifying antirheumatic drugs (DMARDs); American College of Rheumatology (ACR)

IMVT-1402 potentially registrational trial in SjD enables comparison of high dose to low dose FcRn inhibition

Inclusion[a]

- Primary SjD
- Moderate to severe systemic disease activity (clinESSDAI total score ≥ 5)
- Anti-SSA/Ro antibody positive
- Residual unstimulated salivary flow
- On stable background medication(s) for primary SjD, if applicable



*Only ClinESSDAI responders (improvement of ≥ 4 points from baseline) continue through period 2

Endpoints

Primary Endpoint at Week 24: Change from baseline in clinESSDAI score

Key Secondary Endpoint at Week 48: Change from baseline in clinESSDAI score

Design enables comparison of high dose (600 mg) to standard FcRn inhibition (300 mg)

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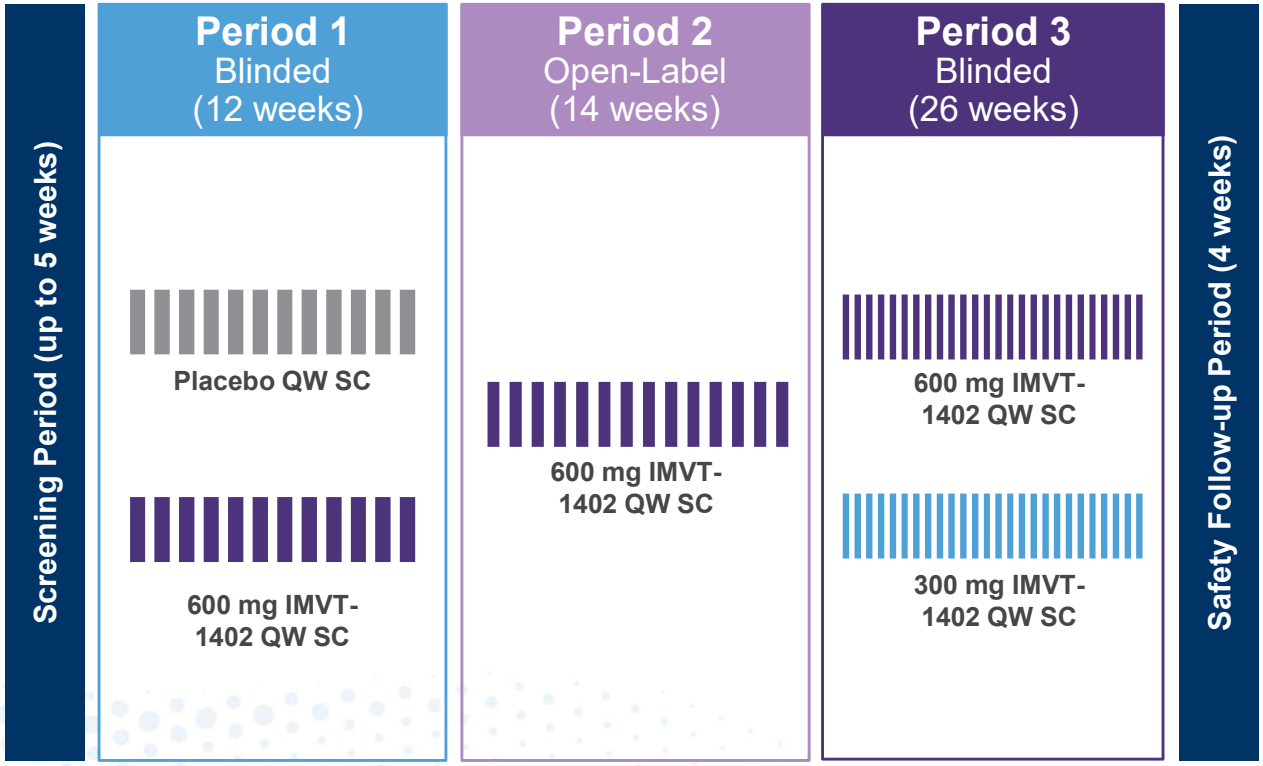
a. Additional inclusion and exclusion criteria not listed on slide
QW: Weekly; SC: Subcutaneous; ClinESSDAI: clinical European League Against Rheumatism Sjögren's Syndrome Disease Activity Index

CLE proof-of-concept study designed to demonstrate short-term and long-term efficacy with IMVT-1402

Global trial with N=56 participants

Inclusion[a]

- SCLE or CCLE, with or without SLE
- Autoantibody positive
- CLASI-A score ≥ 10 at Screening and Day 1
- Inadequate response to conventional therapies (steroids or antimalarial agents)



Endpoints

Primary endpoint: Percent change from baseline in CLASI-A score at Week 12

Secondary endpoints: % of participants who have disease improvement as defined by a reduction in CLASI-A at Week 12 of:

- ≥ 5 points
- ≥ 50%
- ≥ 70%

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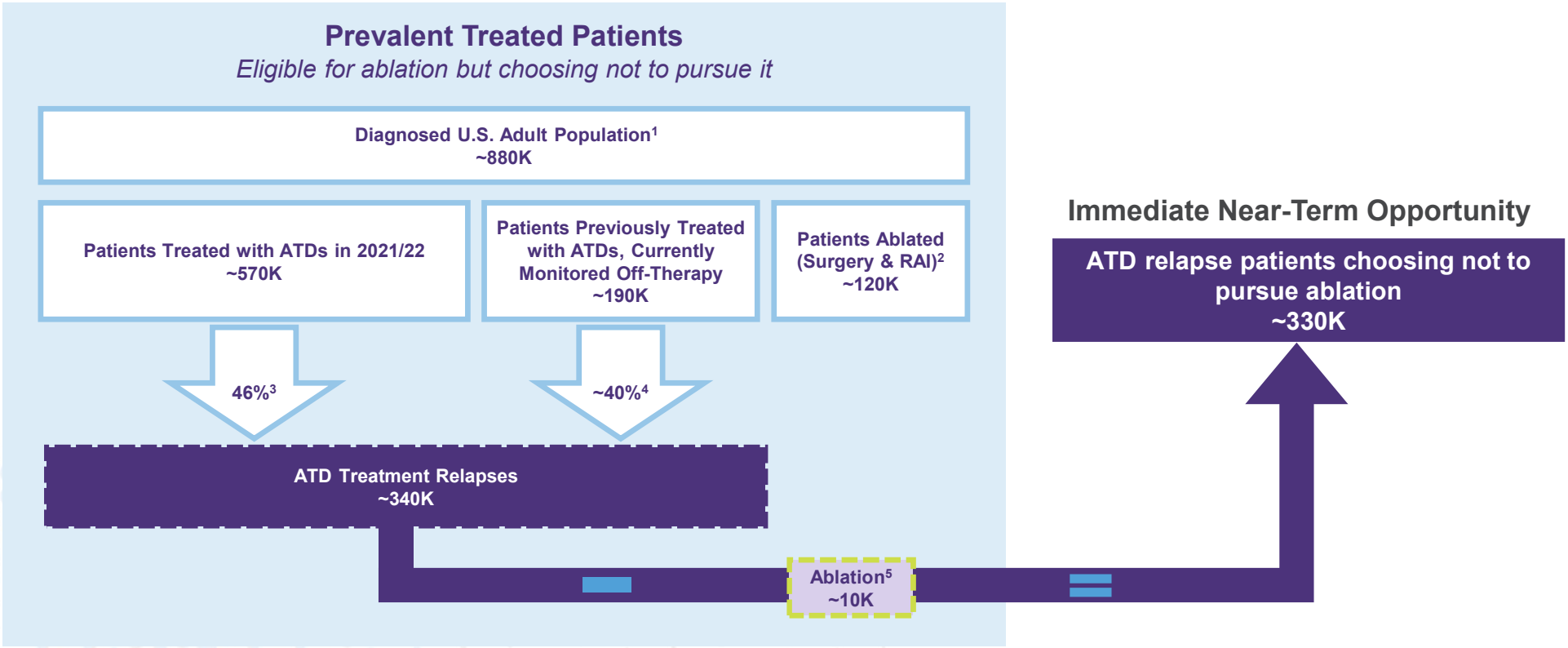
a. Additional inclusion and exclusion criteria not listed on slide
QW: Weekly; SC: Subcutaneous; CLASI-A: Cutaneous Lupus Area and Severity Score Index - Activity

Market Opportunity in Graves' Disease

Analysis #1: Real world claims analysis indicates a substantial untapped opportunity in the prevalent treated Graves' Disease market



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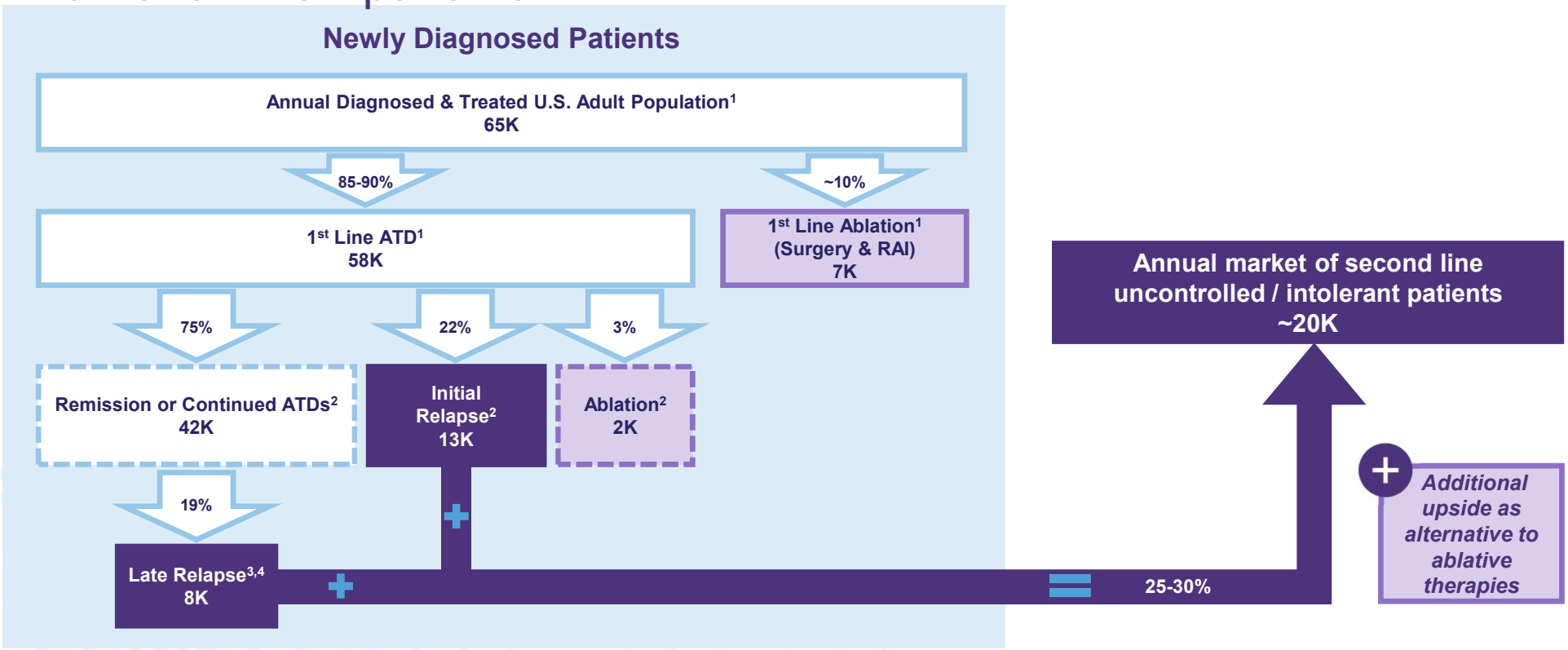
Sources: 1. Roivant Claims Analysis – 2022 prevalent patient population based on a two-year lookback for diagnosis, 2. Of the 120K patients ablated, ~80K were ablated prior to 2021 and ~40K were ablated in 2021/2022. 3. Azizi et al. (2019): Note, the relapse rate was calculated as a weighted average considering relapse rate in patients on ATDs <18months is 53% compared to patients on ATDs >18months is 15%. Of the 570K patients treated with ATDs, ~470K are on ATDs <18months and ~100K are on ATDs for >18months. Rates have been applied proportionally. 4. Bandai et al. (2019): Of the ~190K patients previously treated with ATDs and currently monitored off-therapy, ~40% experience relapse, which is 75K. 5. Grove-Laugesen et al. (2023): 3.4% of ATD relapse patients will pursue ablation. 3.4% applied to the ~340K ATD treatment relapse patients is ~10K.

Analysis #2: Real world claims analysis conservatively estimates an incident US population of ~65K leading to an annual second line market of ~20K patients



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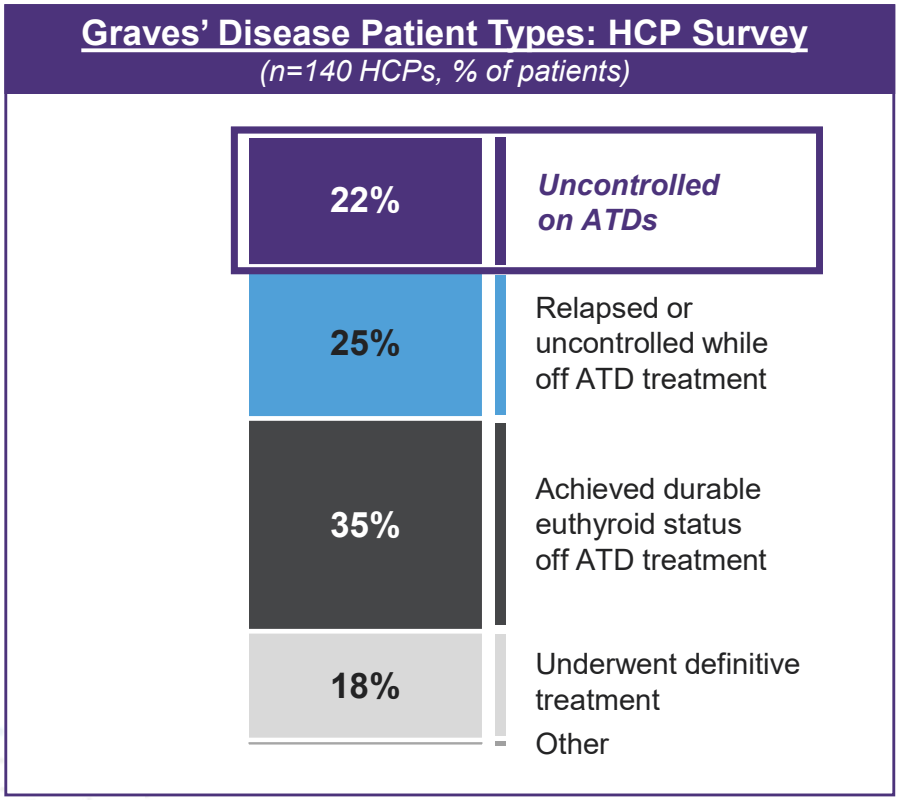
Sources: 1. Roivant Claims Analysis – 2021 incident patient population, first-line treatment is primary treatment in the first-year post diagnosis, claims review included a five-year lookback to define the incident population. 2. Grove-Laugesen et al. (2023): Completer rates for combined arms: ATD remission 56.0%, continuing ATD 18.8%, ATD relapse of 21.8%, ablation of 3.4%. Of the 58K 1st line ATD patients, a total of ~75% are either in remission (56.0%: 32.5K) or continued ATDs (18.8%: 10.9K), 3. Azizi et al. (2019): ATD remission for patients on long-term ATDs is 85%. Of the 10.9K patients who continued ATDs, 15% relapse (1.6K) and 85% go into remission (9.3K). These 9.3K patients in remission will have a 15% rate of relapse resulting in 1.4K relapses. From the original 10.9K patients who continued on ATDs, there will be a total of 3K (1.4K +1.6K) relapses, 4. Stokland et al. (2023): Relapse post remission 15%. Of the 42K patients who are in remission, 15% will relapse (6.3K). In total, the late relapses from remission and continued ATDs will be ~9.3K, resulting in a weighted average relapse rate of ~19% (6.3K relapses from the 32.5K patients in remission averaged with the 3K relapses from the 10.5K patients who continued on ATDs).

Analysis #3: Surveyed endocrinologists indicate that ~25% of their patients remain uncontrolled on ATDs

Endocrinologist Survey Methodology

1. Board-certified endocrinologists (N=140) were screened based on Graves' disease patient volume (10+ patients in the past 3 months) and time in practice (2-40 years in practice with ≥50% of time spent in direct patient care)
2. The N=140 endocrinologists completed a double-blinded online quantitative survey regarding their treatment experience



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Sources: 1) Graves' Disease Physician Survey (n=140) by Immunovant

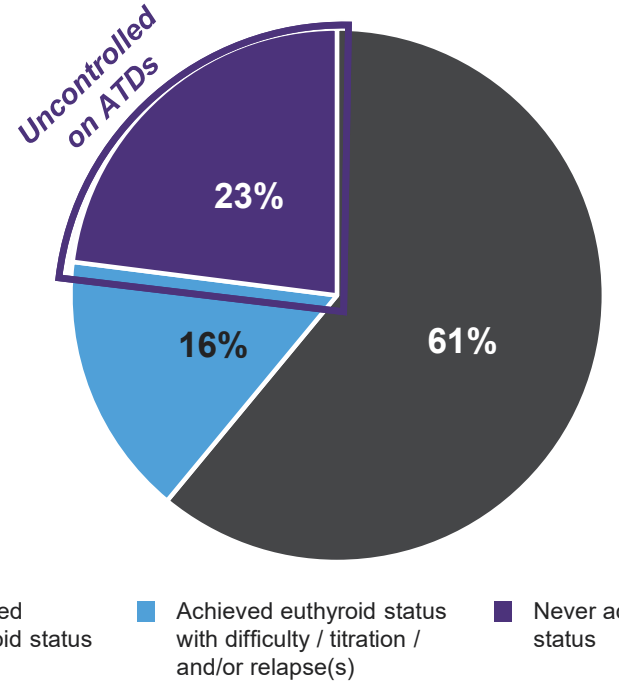
Analysis #4: Real-world in-depth chart review of 1,000+ patient records from 140 endocrinologists indicates ~25% have never achieved euthyroid status on ATDs

Real World Chart Audit Methodology

1. As part of the endocrinologist survey, each healthcare provider was asked to complete N=8 Graves' disease patient charts for a total of 1,120 charts collected via randomized selection to minimize bias
2. Chart selection followed various qualifications:
    1. Diagnosed with Graves' disease
    2. Seen by the healthcare provider in the past 3 months
    3. Under the healthcare provider's care for at least 6 months
    4. First visit in the past 3 years
    5. Either on ATD therapy currently or previously

Characterization of Thyroid Control with ATD Therapy

(n=998 Patient Charts, % of patients)*



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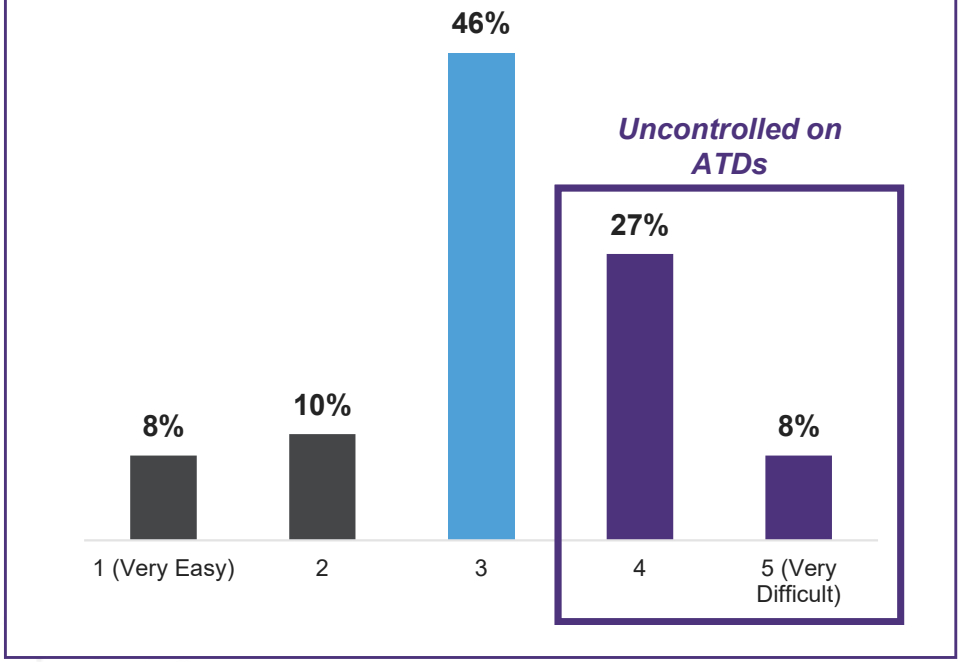
*Excludes patients who have received definitive treatment; Sources: Patient Chart Audit analysis (n=988 on ATD, n=1120 total) by Immunovant

Analysis #5: ~35% of Graves' disease patients report that they have found it difficult or very difficult to achieve stable thyroid disease while on ATDs

Patient Survey Methodology

1. A double-blinded online survey was conducted with N=100 patients who reported being diagnosed by a healthcare provider with Graves' disease
2. Screening criteria included patients who were diagnosed in the past 3 years OR diagnosed in the past 5 years with a recurrence in the past year
3. Excluded patients who had received radioactive iodine or thyroidectomy

Ability to Achieve Stable Thyroid Disease
(n=100 Graves' disease patients, % of patients)



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Sources: Graves' Disease Patient Quantitative Survey (n=100) by Immunovant

Graves' US market-sizing analyses confirm high unmet need with ~330K prevalent patients relapsed, uncontrolled, or intolerant to ATDs

1. Conservative Inovalon claims analysis[1] yields ~880K prevalent Graves' disease patients, including ~330K prevalent ATD relapsed patients choosing not to pursue ablation
2. Conservative Inovalon claims analysis[2] yields ~65K annual incident Graves' disease patients, including ~20K annual incident second line uncontrolled / intolerant patients
3. Deep dive endocrinologist survey of 140 healthcare providers treating Graves' disease patients indicates ~25-30% of patients are relapsed, uncontrolled, or intolerant to ATDs
4. Real-world chart audit of 1,120 Graves' disease patients treated by surveyed endocrinologists indicates ~25-30% of patients are relapsed, uncontrolled, or intolerant to ATDs
5. Patient survey of 100 diagnosed Graves' disease patients indicates ~25-30% of patients are relapsed, uncontrolled, or intolerant to ATDs

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1. Roivant Claims Analysis – 2022 prevalent patient population based on a two-year lookback for diagnosis
2. Roivant Claims Analysis – 2021 incident patient population based on a five-year lookback to define the incident population

Note: See Immunovant, Inc. Graves' Disease Program Update Deck dated September 9, 2024, available at Immunovant.com