IMMUNOVANT®

Targeted science, tailored solutions

for people with autoimmune disease

Corporate Presentation

September 2026

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 (imeroprubart); Immunovant's plan to develop IMVT-1402 across a broad range of indications; the registrational nature of Immunovant's clinical trials; 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; Immunovant is at various stages of clinical development for IMVT-1402; and Immunovant will require additional capital to fund its operations and advance 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 Annual Report on Form 10-K filed with the SEC on May 20, 2026, 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

5 US regulatory approvals, 9 positive Phase 3 trials and 17 positive Phase 2 trials

>$4.3 billion in global anti-FcRn sales in 2025[1,2]

IMVT-1402: Potential Best-in-Class Product Candidate

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, MG, CIDP, SjD & D2T RA

Significant Market Opportunity

Large total addressable market with 20+ indications announced or in development across the anti-FcRn class[3]

Current IMVT-1402 trials expected to potentially address ~600K patients in the US

Financial Strength

Cash balance of ~$798 million as of June 30, 2026[4]

Provides runway to Graves' disease commercial launch

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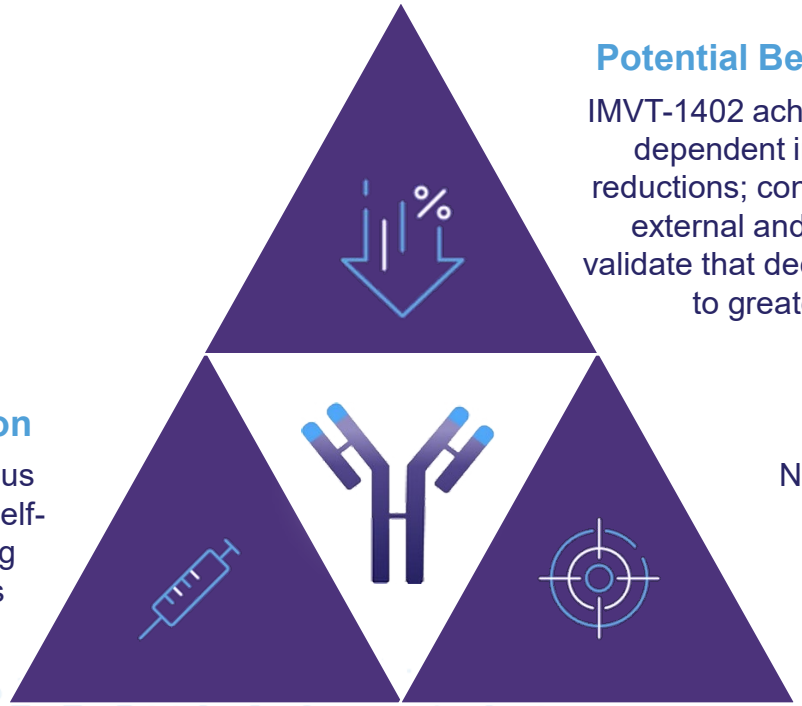
*Potentially registrational indicates registration is dependent on future alignment with FDA on clinical trial design, endpoints, statistical analysis plans, and overall development strategy

1. argenx Highlights 2026 Strategic Priorities
2. UCB HY 2025 Results Capital Markets Earnings Call
3. Indications announced or in development with anti-FcRn assets by Immunovant, argenx, Johnson & Johnson, and UCB
4. Includes cash and cash equivalents

Notes: GD: Graves' disease; SjD: Sjogren's disease; D2T RA: difficult-to-treat rheumatoid arthritis; CIDP: Chronic inflammatory demyelinating polyneuropathy; MG: Myasthenia gravis

IMVT-1402 has the potential to be a first- and best-in-class therapy in autoantibody-driven disease



Potential Best-in-Class Efficacy

IMVT-1402 achieves deep, rapid, dose-dependent immunoglobulin (IgG) reductions; consistent evidence across external and internal clinical trials validate that deeper IgG reductions lead to greater clinical benefit

Convenient Administration

YpsoMate® simple subcutaneous autoinjector with 5-10 second self-administration; currently being tested in all IMVT-1402 trials

Favorable Safety Profile

No significant expected safety issues based on data to-date

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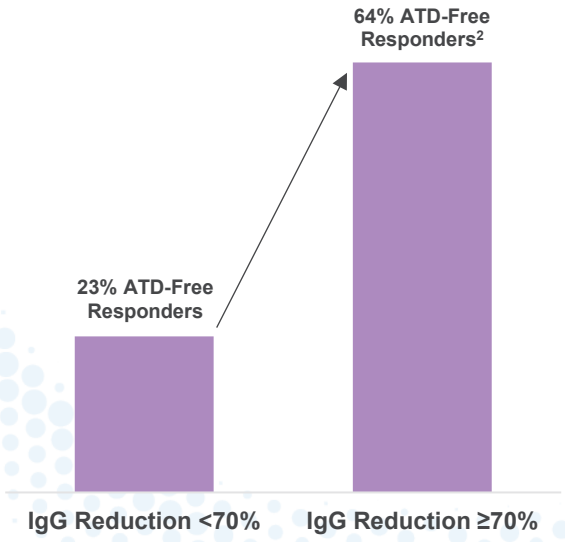
Note: Ypsomate® autoinjector used in ADBRY®, COSENTYX®, AJOVY®, NUCALA®, FASENRA®, TEZSPIRE®. Ypsomate® is a registered trademark of Ypsomed AG.

First generation batoclimab multi-indication clinical data demonstrated that deeper IgG reduction may lead to improved clinical outcomes

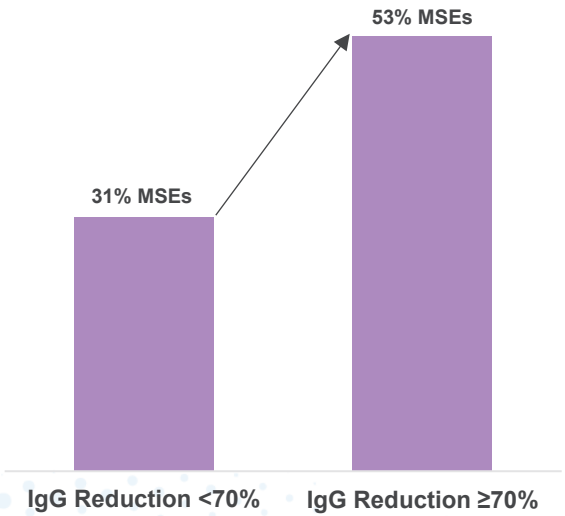
Graves' Phase 2a[1]

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



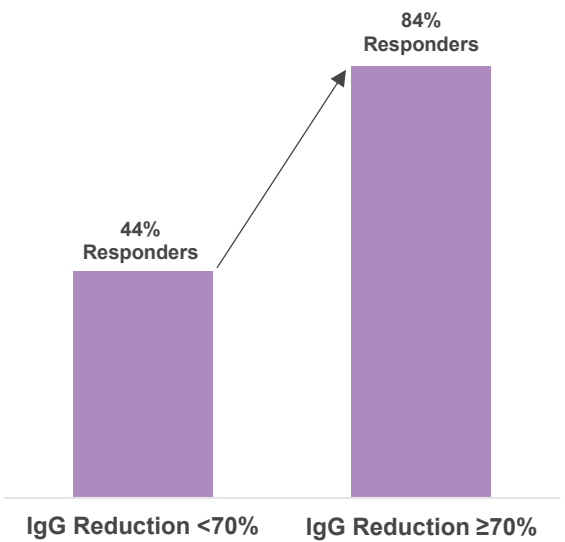
MG Phase 3[1]

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
2. Includes N=1 additional responder vs. September 2024 disclosure. Patient discontinued prior to Week 12 and was counted as a non-responder per protocol but was included by the PI in the ATA 2025 poster presentation, given they were a responder at time of discontinuation and continued through 6 month off-treatment follow-up period.

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

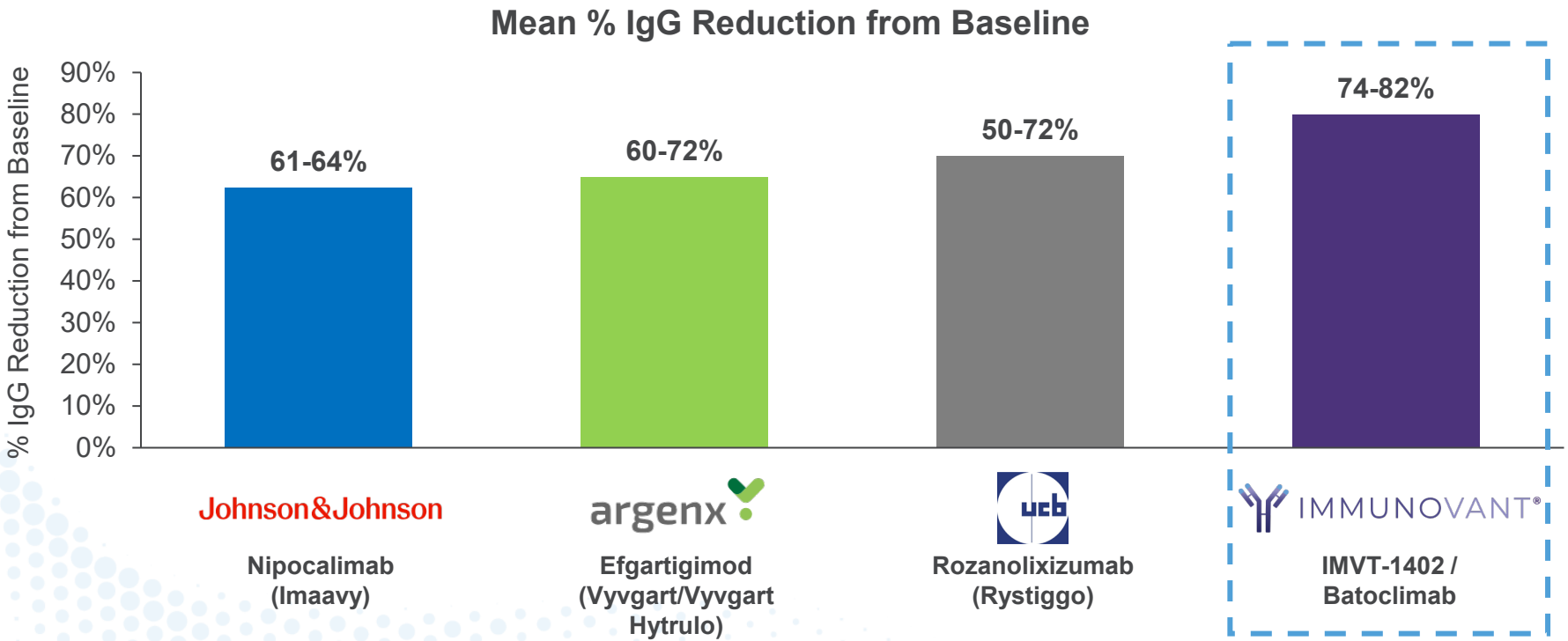

Figure reflects cross-trial comparisons and not data from head-to-head studies.
Differences exist between trial designs and participant characteristics and caution should be exercised when comparing data across trials.

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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
Ocular MG
Autoimmune encephalitis

ENDOCRINOLOGY

Graves' disease (GD)

HEMATOLOGY

Fetal neonatal alloimmune thrombocytopenia
Hemolytic disease of the fetus and newborn
Idiopathic thrombocytopenic purpura
Warm autoimmune hemolytic anemia

RHEUMATOLOGY

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

DERMATOLOGY

Bullous pemphigoid
Pemphigus foliaceus/Pemphigus vulgaris
Systemic sclerosis

RENAL

Antibody-mediated rejection
Lupus nephritis
Membranous nephropathy

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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 blockers to benefit greater number of patients with several new indications, with a potential efficacy advantage driven by deeper IgG reduction • Example – GD and D2T RA
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; 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	ACPA+ Difficult-to-Treat RA	Sjögren's Disease	Myasthenia Gravis	Chronic Inflammatory Demyelinating Polyneuropathy
Expected US Addressable Population[1]	**~330K**	**~85K**	**~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 ~70% of RA 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	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 Fully Enrolled	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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*Potentially registrational indicates registration is dependent on future alignment with FDA on clinical trial design, endpoints, statistical analysis plans, and overall development strategy

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	2H 2026	2027	2028
ACPA+ D2T RA	Potentially Registrational*	Further Updates	■		
GD	Potentially Registrational	Top Line Results		■	
MG	Potentially Registrational	Top Line Results		▢	
SjD	Potentially Registrational	Top Line Results			■
CIDP	Potentially Registrational	Top Line Results			▢

■ Rheumatology ■ Endocrinology ▢ Neurology

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*The regulatory path for IMVT-1402, including whether any ongoing or future clinical trials may be considered registrational or registration-enabling, depends on future alignment with FDA on clinical trial design, selection of endpoints, statistical analysis plans, and overall development strategy

Note: GD: Graves' disease; APCA+: Anti-citrullinated peptide antibody positive; D2T RA: difficult-to-treat rheumatoid arthritis; 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

IMVT-1402

2.25 mL automated disposable injection device

Dose: 150 mg/mL
Injection volume: 2 mL

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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Note: 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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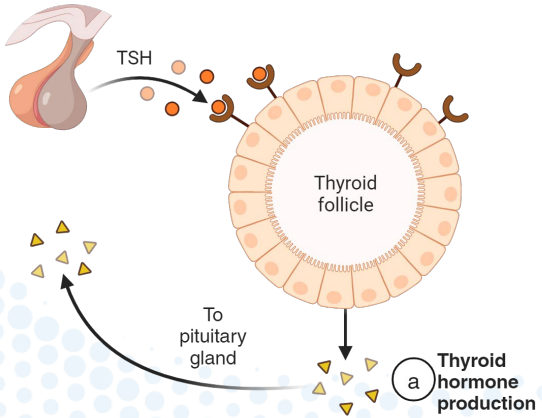
*Potentially registrational indicates registration is dependent on future alignment with FDA on clinical trial design, endpoints, statistical analysis plans, and overall development strategy
Notes: Thyroid Stimulating Hormone Receptor (TSRH), Anti-thyroid drug (ATD), Graves' disease (GD), Proof of concept (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

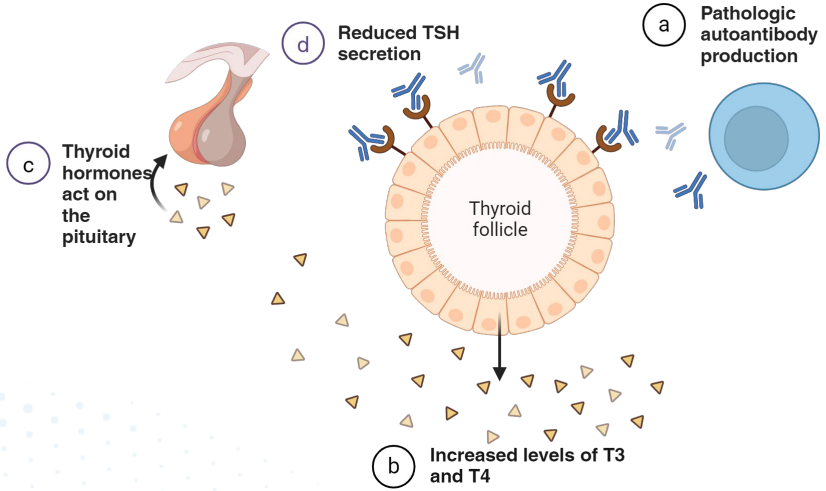
Normal Function

TSH produced by the pituitary gland stimulates the thyroid gland to produce and release thyroid hormones (T3 & T4)



Graves' Disease

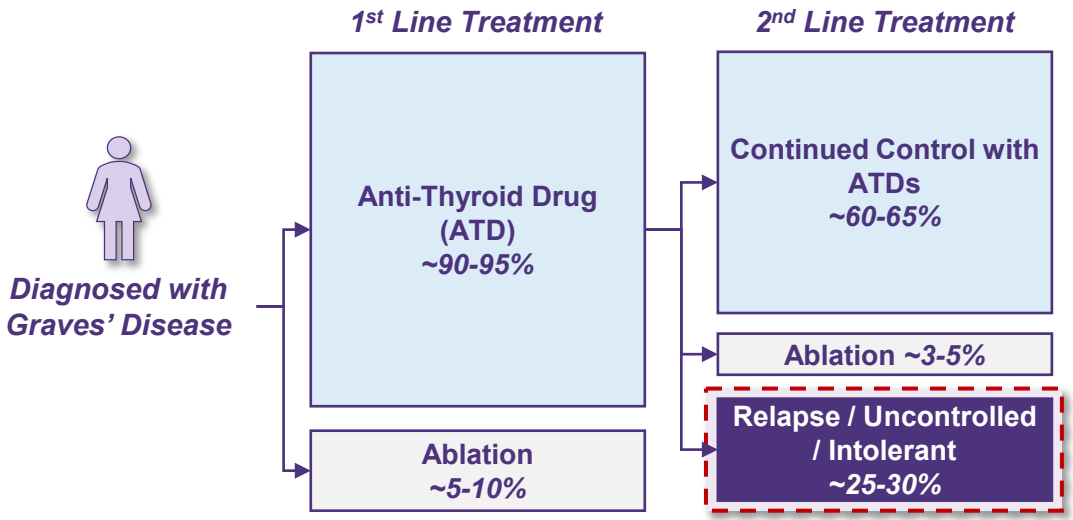
Autoantibodies to the thyroid stimulating hormone receptor (TSHR) stimulate thyroid hormone production and lead to excess thyroid hormone production (increased T3, T4)



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.

Shift away from ablation and lack of new medical therapies leaves 25-30% of patients who are relapsed, uncontrolled on, or intolerant to ATDs

Graves' Disease Patient Journey:



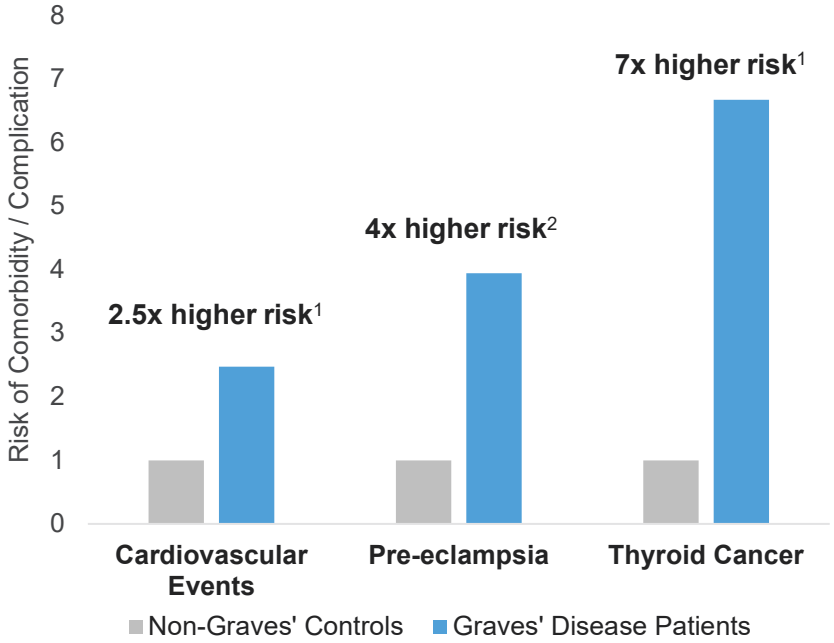
Unmet Need

- 25-30% of patients are relapsed, uncontrolled on or intolerant to ATDs
- US data on ablation rates indicate that patients with ATD-refractory disease are choosing not to undergo ablation
- Patients and healthcare providers seek therapeutic options that address underlying disease pathology

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 55.9K 1st line ATD patients, a total of ~75% are either in remission (56.0%: 31.3K) or continued ATDs (18.8%: 10.5K)
3. Azizi et al. (2019): ATD remission for patients on long-term ATDs is 85%. Of the 10.5K patients who continued ATDs, 15% relapse (1.6K) and 85% go into remission (8.9K). These 8.9K patients in remission will have a 15% rate of relapse resulting in 1.3K relapses. From the original 10.5K patients who continued on ATDs, there will be a total of 3K (1.3K +1.6K) relapses
4. Stokland et al. (2023): Relapse post remission 15%. Of the 31.3K patients who are in remission, 15% will relapse (4.7K). In total, the late relapses from remission and continued ATDs will be ~7.6K, resulting in a weighted average relapse rate of ~18% (4.7K relapses from the 31.3K patients in remission averaged with the 2.9K relapses from the 10.5K patients who continued on ATDs).

Scientific literature indicates that Graves' disease patients are at a higher risk of a sequelae of severe comorbidities

Relative to Healthy Controls, Graves' Patients Are at Increased Risk of Developing Several Severe Comorbidities



Untreated Or Insufficiently Treated Graves' Patients Experience Substantial Morbidity And Loss Of Quality Of Life

Thyroid Eye Disease (TED)

- TED affects ~40% of patients diagnosed with Graves' Disease[3]
  - Up to 8% of TED patients experience dysthyroid optic neuropathy (impairment of visual function, leading to permanent sight loss)[4]

Other Significant Complications

- In patients hospitalized for Graves' Disease, ~16% are diagnosed with thyroid storm[5], which has a ~20% mortality rate[6]
- Graves' Disease patients who develop thyroid cancer are at a >3x risk of recurrent disease / progressive distant metastases relative to euthyroid controls[7]

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1. Okosieme et al., 2019
2. Aggarwal et al., 2014
3. Chin et al., 2020
4. Potvin et al., 2023
5. Galindo et al., 2019
6. Bourcier et al., 2020
7. Pellgriti et al., 1998

Graves' patients uncontrolled on ATDs experience significant disease burden and risk of adverse events with limited treatment options

RAI and surgery are associated with **significant complications** including increased risk of death from solid cancers; patients are often hypothyroid and require **lifelong thyroid hormone replacement**

Chronic ATD use can be associated with risk of severe adverse events, such as **hepatotoxicity**, **pancreatitis**, and **agranulocytosis** (loss of white blood cells)

Uncontrolled Graves' patients are at risk for a sequelae of **severe comorbidities** (e.g., **cardiovascular events**, **thyroid cancer**) and experience significant **anxiety** and **impact to quality of life**

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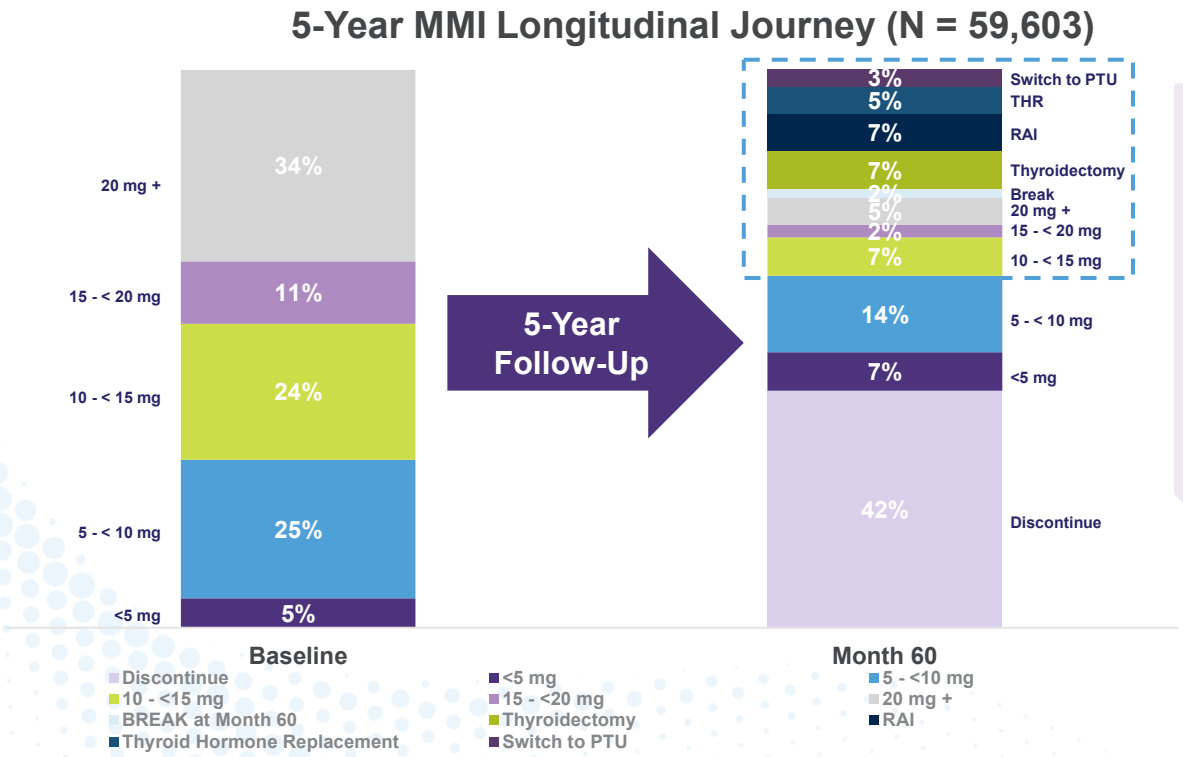
1. Sundaresh et al., J Clin Endocrinol Metab, 2013
2. Kitahara, et al., JAMA Intern Med, 2019
3. Suzuki et al., Thyroid, 2019
4. Smith and Hegedüs, N Engl J Med, 2016
5. Brix et al., ETA, 2019
6. Okosieme et al., Lancet Diabetes Endocrinol, 2019
7. Cramon et al., Thyroid, 2016

Follow-up of Graves' disease patient methimazole dosing shows significant percent of patients remaining on ATDs after 5-years



- **In a 5-year follow-up period, only 42% of patients were controlled on ATDs alone**
- **~37% of patients were on ≥10 mg MMIs, break, switched to PTU, received thyroid hormone replacement or ablation**

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Source: Roivant and Immunovant claims analysis
Patients continuously enrolled from 2016 - 2024, newly diagnosed in 2019, 5 years of follow up data from initial MMI script

Treatment Period: 24 weeks		Follow-up: 24 weeks
680 mg batoclimab QW SC (Week 0-12)	340 mg batoclimab QW SC (Week 12-24)	Off-Treatment (Week 24-48)

Potential for disease modification with batoclimab responders demonstrating strong durability of response through six months off-treatment at end of follow-up

Baseline

Week 12
Pts receive 12 weeks of 680 mg QW batoclimab[a]

Dose step-down

Week 24
Pts receive 12 weeks of 340 mg QW batoclimab[a]

Week 48
Patients off-drug for 24 weeks[a,b]

25
Uncontrolled Graves' disease patients

20/25
T3/T4 ≤ULN; ATD dose ≤Baseline

18/25
T3/T4 ≤ULN; ATD dose ≤Baseline

17/21
T3/T4 ≤ULN; ATD dose ≤Baseline[c]

Strong durability of response despite being off-batoclimab for six months

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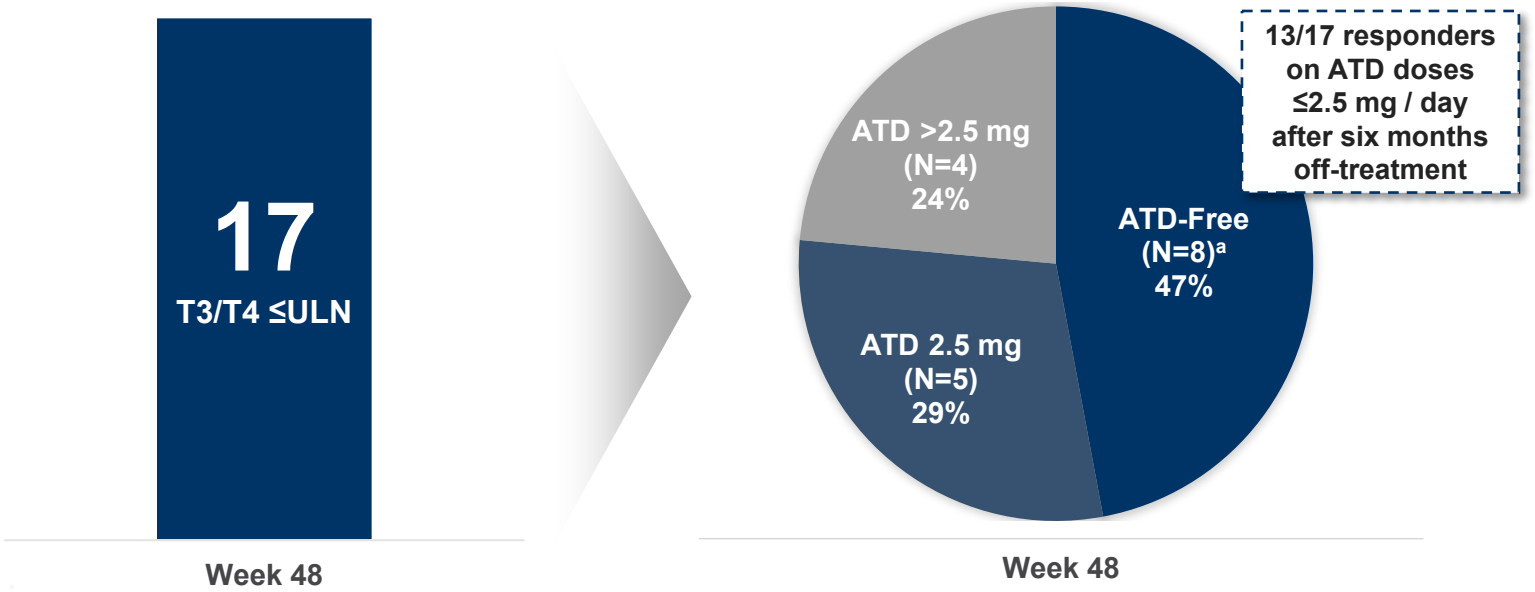
Notes: Responders: Patients who have T3 and T4 values ≤ULN and no increase in ATD dose from baseline. Pts: Patients; T3: Triiodothyronine; T4: Thyroxine; ULN: Upper limit of normal; ATD: Anti-thyroid drug. a. Includes N=1 patient who discontinued prior to Week 12 but remained in off-drug follow-up. b. Includes N=21 patients who entered follow-up period and could be assessed for remission. c. N=1 patient had T3/T4 ≤ULN, and one day following Week 48 visit had ATD dose equivalent to baseline.

Treatment Period: 24 weeks		Follow-up: 24 weeks
680 mg batoclimab QW SC (Week 0-12)	340 mg batoclimab QW SC (Week 12-24)	Off-Treatment (Week 24-48)

~50% of responders at Week 48 achieved ATD-free remission, demonstrating strong potential for disease modification by a high-dose FcRn



8 of 17 patients with normal T3/T4 at Week 48 were in ATD-free remission

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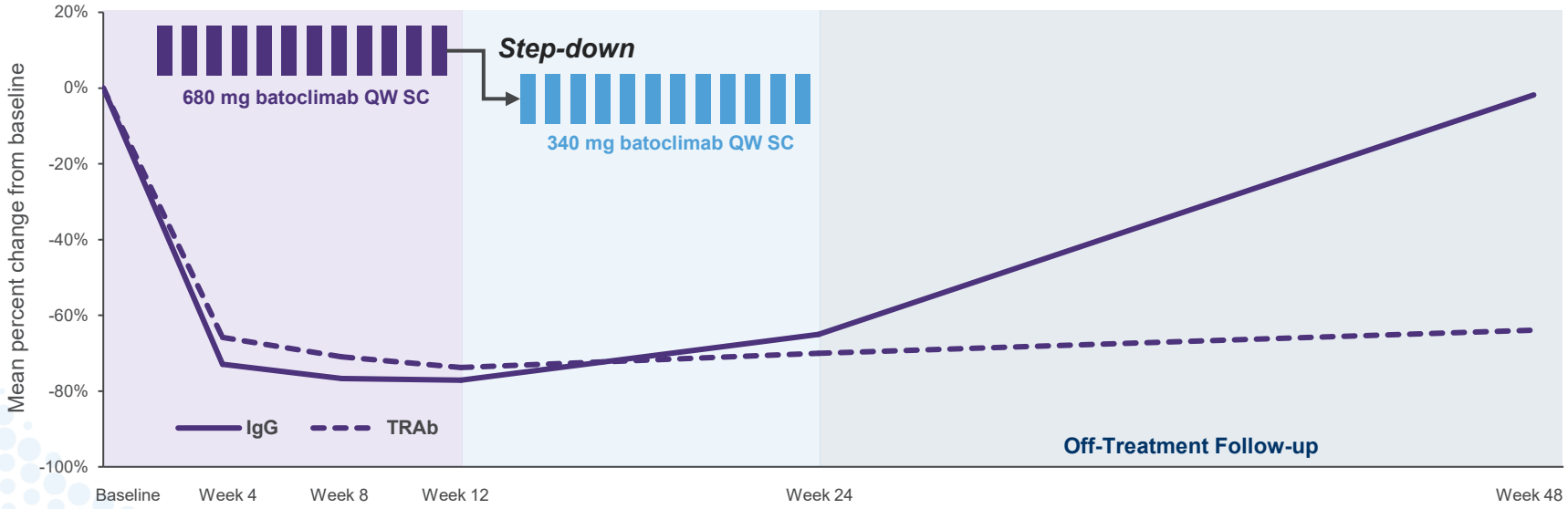
Notes: Responders: Patients who have T3 and T4 values ≤ULN and no increase in ATD dose from baseline. T3: Triiodothyronine; T4: Thyroxine; ULN: Upper limit of normal; ATD: Anti-thyroid drug; FcRn: Neonatal fragment crystallizable receptor inhibitor. a. Includes N=1 patient who discontinued prior to Week 12 but remained in off-drug follow-up.

Treatment Period: 24 weeks		Follow-up: 24 weeks
680 mg batoclimab QW SC (Week 0-12)	340 mg batoclimab QW SC (Week 12-24)	Off-Treatment (Week 24-48)

Sustained TRAb reductions post-batoclimab treatment further demonstrate the potential for disease modification



Notes: Data includes up to last measurement available for patients who discontinued. IgG: Immunoglobulin G; TRAb: Thyroid Stimulating Hormone Receptor Antibody; QW: Once weekly; SC: Subcutaneous. Patient counts at each time include Baseline (N=25), Week 12 (N=24), Week 24 (N=23), Week 48 (N=19).

IMVT-1402 could potentially be the first-in-class disease-modifying therapy in Graves' disease

01 Significant effect seen in uncontrolled Graves' disease patients: 18 of 25 patients treated with batoclimab were responders* at Week 24

02 Durable off-drug response observed: Of the 21 patients who entered the off-drug follow-up period, 17 remained responders* six months following batoclimab treatment

03 First-ever observed ATD-free remission in uncontrolled patients: 8 of 17 responders* remained off all medications six months following batoclimab treatment demonstrating potential for disease modification

04 IMVT-1402 pivotal trial design could potentially generate improved efficacy data due to continuous 600 mg QW dosing vs. batoclimab's step-down dosing design

05 Two potentially registrational** trials for IMVT-1402 in Graves' disease are currently enrolling

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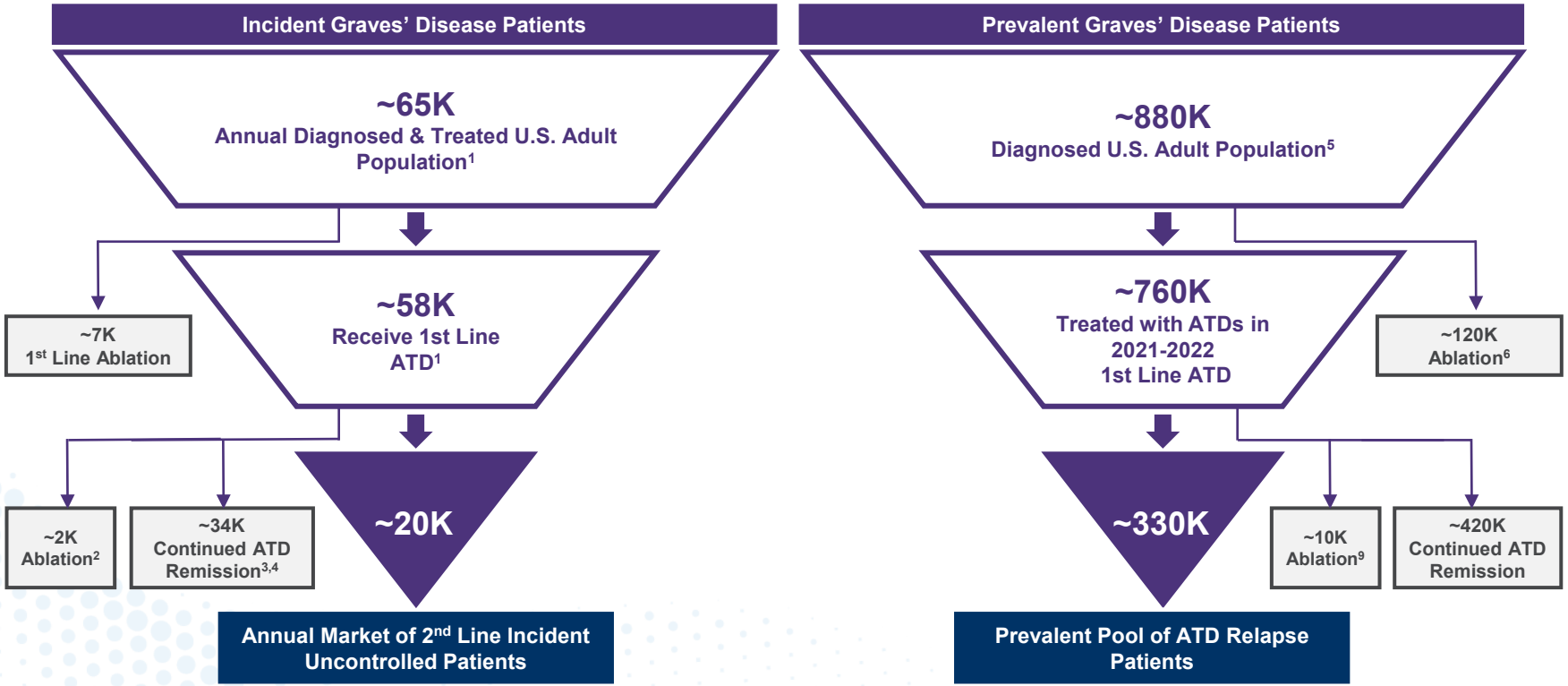
Notes: ATD: Anti-thyroid drug; QW: Once weekly. Data includes N=1 patient who discontinued prior to Week 12 but remained in off-drug follow-up. *Responders: Patients who have T3 and T4 values ≤ULN and no increase in ATD dose from baseline. **Potentially registrational indicates registration is dependent on future alignment with FDA on clinical trial design, endpoints, statistical analysis plans, and overall development strategy

Graves' disease market opportunity includes annual incident opportunity and a significant untapped prevalent patient pool



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).
5. Roivant Claims Analysis – 2022 prevalent patient population based on a two-year lookback for diagnosis
6. Of the 120K patients ablated, ~60K were ablated prior to 2021 and ~40K were ablated in 2021/2022
7. Azizi et al. (2019): 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.
8. Bandai et al. (2019): Of the ~190K patients previously treated with ATDs and currently monitored off-therapy, ~40% experience relapse, which is 75K
9. 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

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 improved 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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*Potentially registrational indicates registration is dependent on future alignment with FDA on clinical trial design, endpoints, statistical analysis plans, and overall development strategy
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. Data on file

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 blockers, 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 blockers[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. Data on file

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

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. 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

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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 batoclimab 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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*Potentially registrational indicates registration is dependent on future alignment with FDA on clinical trial design, endpoints, statistical analysis plans, and overall development strategy

1. Internal Market Research Market Dynamics 2024
2. Data on file

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. Data on file
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
5. Internal Market Research HCP Survey and KOL advising 2023 6. Internal Market Research CIDP Patient Journey 2022

ACPA+ Difficult-to-Treat RA

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+ D2T RA, who have failed two or more prior advanced mechanisms[1]

High Unmet Need Subgroup	5-20% of RA patients have inadequate or loss of response to two or more mechanisms of advanced therapies[2]
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 increased IgG reduction led to increased autoantibody reductions, which correlated with improved clinical response[3]
Potentially Registrational* Trial Fully Enrolled	Clinically meaningful response rates were observed at Week 16 in the open label period of the trial[4]; Immunovant expects to provide further updates in 2H 2026

1. Advanced mechanisms include biologics (e.g., TNFα inhibitors, IL-6R inhibitors, CD80/86 inhibitors, CD20 inhibitors) and targeted synthetic DMARDs (e.g., JAK inhibitors). For patients failing ≥2 prior advanced mechanisms in addition to conventional DMARDs, IMVT-1402 would be the fourth line or later line of therapy
2. Takanashi S, et al. Rheumatology. 2021;60:5247-56
3. 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*
4. Data on file

*Potentially registrational indicates registration is dependent on future alignment with FDA on clinical trial design, endpoints, statistical analysis plans, and overall development strategy

ACPA: anti-citrullinated protein antibody; TNF: tumor necrosis factor; IL-6R: interleukin-6 receptor; DMARD: disease-modifying anti-rheumatic drug: JAK: Janus kinase

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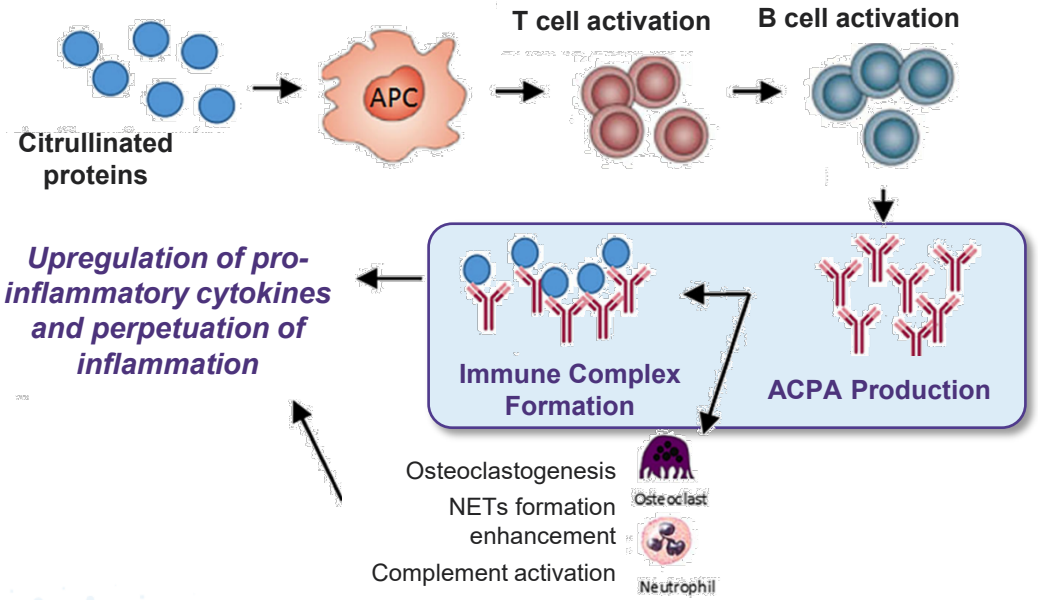
In addition to cellular autoimmunity and cytokine dysregulation, autoantibodies like ACPA play a key role in the pathophysiology of RA

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

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

The D2T population of multi-advanced mechanism failures has no proven treatment options

Need for More Options

- Estimated 5-20% of patients remain symptomatic despite multiple treatment rounds with advanced mechanisms[1,2]
  - These patients need new therapies and approaches, according to a global survey of 410 rheumatologists
- Difficult-to-treat (D2T) RA defined by EULAR as[3]:
  - Failure on conventional DMARDs and 2 or more advanced therapy mechanisms (i.e., TNF inhibitors, JAK inhibitors and/or other biologics[4])
  - 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. Advanced mechanisms include biologic and targeted synthetic DMARDs
2. Roodenrijs NMT et al. *Ann Rheum Dis* 2018;1705 -09
3. Nagy G, et al. *Ann Rheum Dis* 2021; 80: 31-35
4. Other biologics include IL-6R inhibitors, CD80/86 inhibitors, CD20 inhibitors, etc.; EULAR: European League Against Rheumatism Collaborative Initiative; DMARD: disease-modifying antirheumatic drug; QoL: Quality of Life

Trial enrolled a difficult-to-treat population: patients were 4L+, having failed conventional therapy and ≥2 mechanisms of advanced therapies

Baseline Characteristics *Mean unless otherwise noted*	**IMVT-1402 (Period 1) (N=165[2])**
Age, years	58.7
Sex, % female, n (%)	116 (70.3%)
Time since RA diagnosis, years	12.8
Tender joint count (0-68)	24.2
Swollen joint count (0-66)	16.7
Clinical Disease Activity Index (CDAI)	44.6
Simplified Disease Activity Index (SDAI)	46.3
DAS28-CRP score	6.1
Number of Prior Advanced Therapy Mechanisms[1]	
1 Advanced Therapy Mechanism, n (%)	1 (0.6%)
2 Advanced Therapy Mechanisms, n (%)	143 (86.7%)
≥3 Advanced Therapy Mechanisms, n (%)	19 (11.5%)

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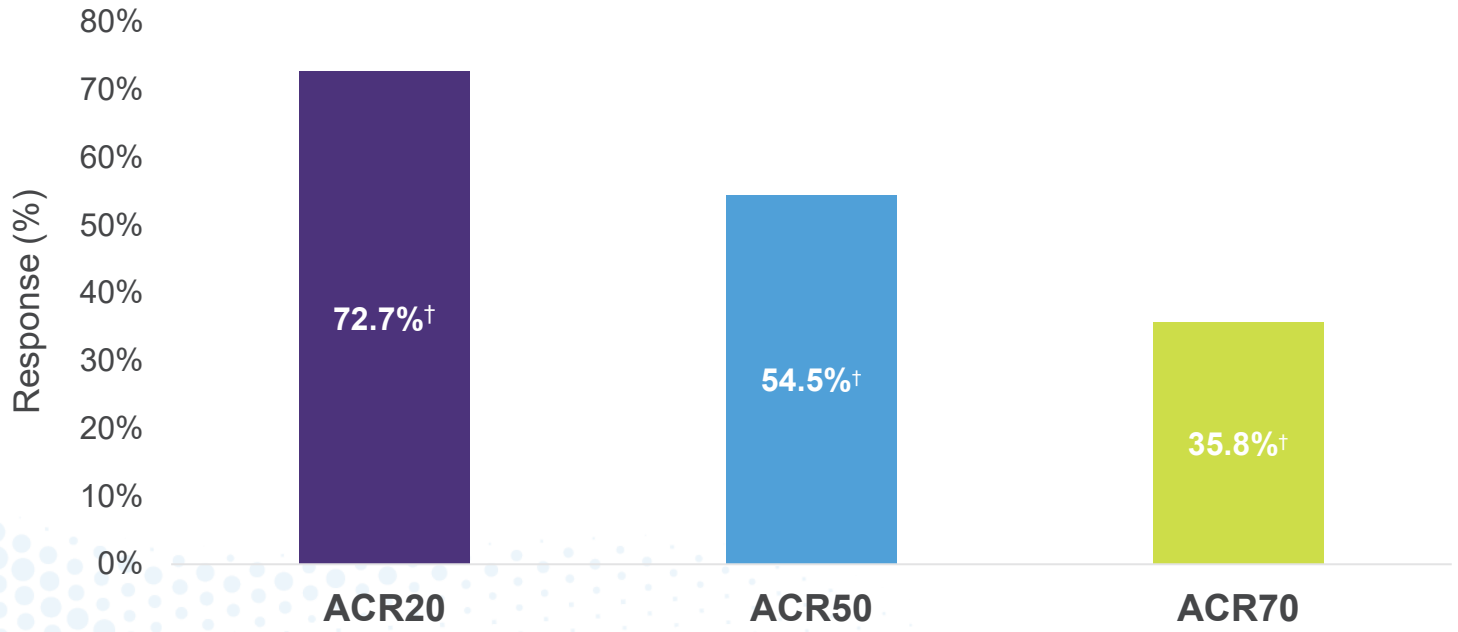
Notes: Period 1 data is preliminary – minor changes are likely as data is finalized. 1. N=2 patients missing information. Advanced therapies include biologic (e.g., anti-TNFs, IL-6R inhibitors, CD80/86 inhibitors, CD20 inhibitors) and targeted synthetic (e.g., JAK inhibitors) DMARDs. 2. Efficacy analysis population (N=165) excludes N=5 patients from one site due to protocol non-compliance. DAS28-CRP: Disease Activity Score 28 with C reactive protein; 4L+: Fourth line or later; DMARD: Disease-modifying anti-rheumatic drug.

Period 1 preliminary data

Trial enrolled a difficult-to-treat population: patients had to fail at least one conventional DMARD therapy and at least 2 additional mechanisms of advanced therapies (biologic and targeted synthetic DMARDs)

ACR20, ACR50, ACR70 Response Rates at Week 16 (N=165[1])



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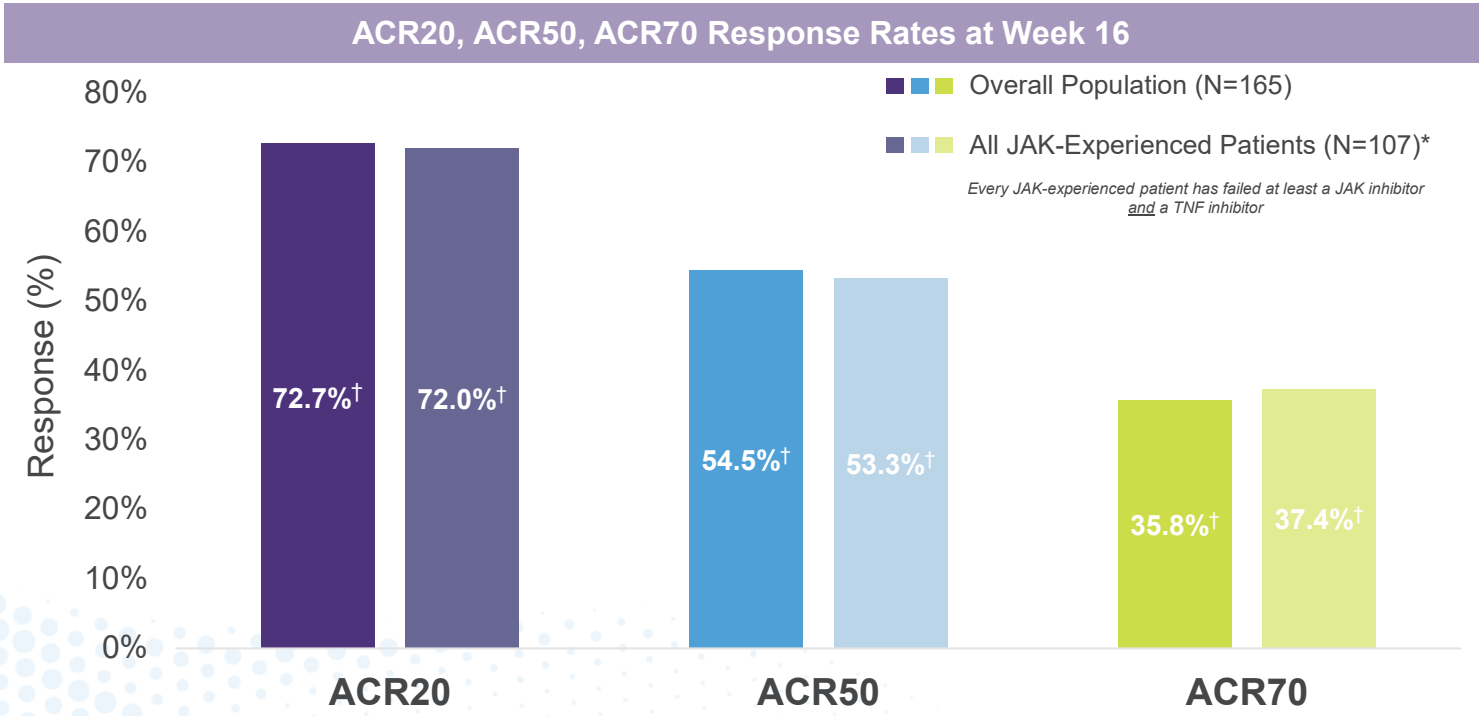
Notes: †Period 1 data is preliminary – minor changes are likely as data is finalized. 1. Efficacy analysis population (N=165) excludes N=5 patients from one site due to protocol non-compliance. Patients who discontinued treatment during Period 1 are included as non-responders. DMARD: Disease-Modifying Anti-Rheumatic Drug. ACR: American College of Rheumatology. ACR20, ACR50, and ACR70 refer to standardized criteria established by the American College of Rheumatology to measure change in rheumatoid arthritis (RA) symptoms.

Period 1 preliminary data: consistent & meaningful effect in JAK-class failures

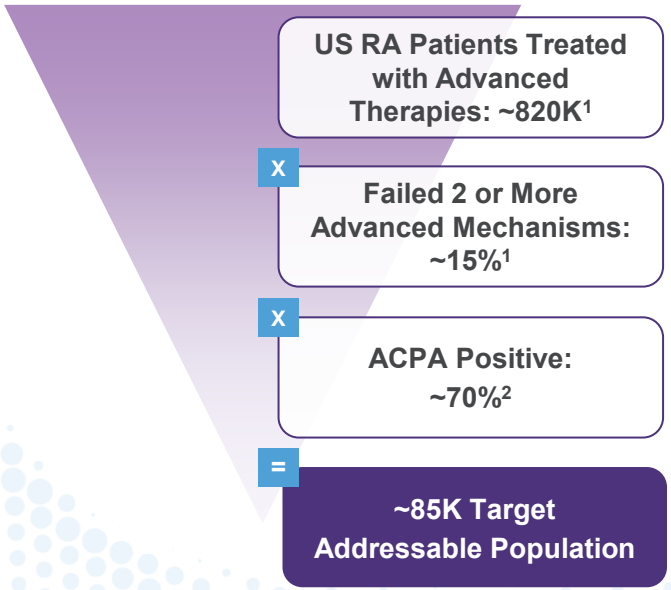
IMVT-1402 response rates were consistent between the overall difficult-to-treat population and patients whose treatment history included failure on at least a TNF inhibitor and a JAK inhibitor



Notes: †Period 1 data is preliminary – minor changes are likely as data is finalized. *N=107 includes the entire subset of patients enrolled who had previously failed on a JAK inhibitor in addition to a TNF inhibitor +/- other biologics. Patients who discontinued treatment during Period 1 are included as non-responders. ACR: American College of Rheumatology; DMARD: Disease-Modifying Anti-Rheumatic Drug; JAK: Janus Kinase Inhibitor; TNF: Tumor Necrosis Factor.

~820K US RA patients are treated with advanced mechanisms: a subset progresses on multiple mechanisms and requires new therapeutic options

Market Opportunity



Unmet Needs in Difficult-to-Treat RA

Fewer than 50% of RA patients remain on first advanced therapy	~50% of patients fail their first advanced therapy within the first year of treatment[3,4]
Patients resistant to multiple therapies have more severe disease activity	Multi-drug-resistant patients have significantly higher tender joint count and global evaluator activity scores, despite therapy[5]
Currently approved advanced mechanisms have significant contraindications	Severe RA patients are highly comorbid, which significantly impacts drug selection given contraindications for approved therapies[6]

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Note: All estimates are approximate; ACPA: Anticitrullinated Protein Autoantibody. Advanced mechanisms include biologic (e.g., anti-TNFs, IL-6R inhibitors, CD80/86 inhibitors, CD20 inhibitors) and targeted synthetic (e.g., JAK inhibitors) DMARDs.

1. Roivant RWE RA Claims Analysis (2026)
2. Okada et al., Ann Rheum Dis (2019)
3. Murray et al., Arthritis Res Ther (2021)
4. Rosenberg et al., Adv Ther (2023)
5. Takanashi et al., Rheumatology (2021)
6. UpToDate, April 2026

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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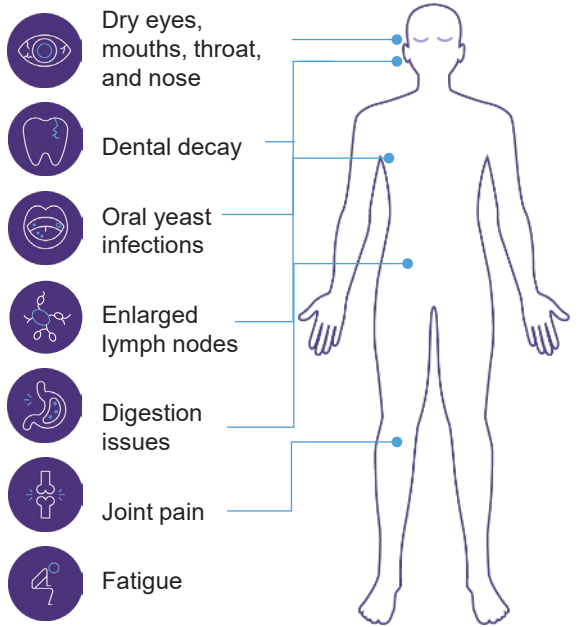
*Potentially registrational indicates registration is dependent on future alignment with FDA on clinical trial design, endpoints, statistical analysis plans, and overall development strategy.

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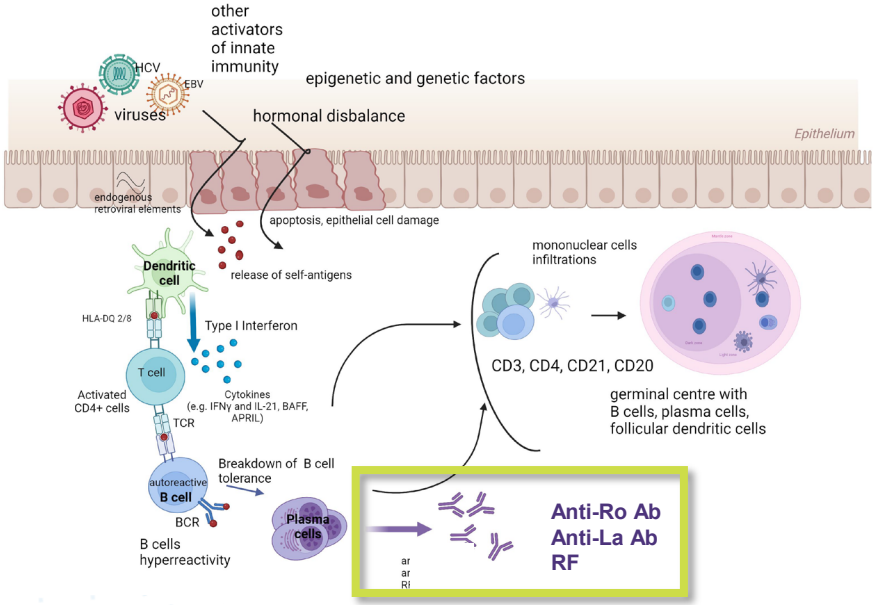
1. Mariette X, et al. *N. Engl J Med.* 2018; 378:931-9
2. Brito-Zeron P et al. *Nature Reviews.* 2016; 2:1-20
3. GlobalData Epi Analysis and Forecast, Oct. 2020

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]

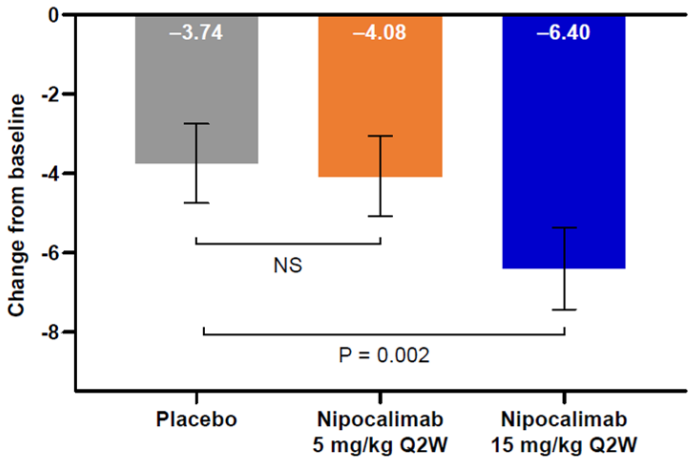


1. Baer AN, et al. Elsevier; 2023. Chapter 45, Clinical aspects of Sjögren's disease; p. 637-647
2. Brito-Zeron P et al. *Nature Reviews.* 2016; 2:1-20
3. Figure reprinted from Maslinska M, Kostyra-Grabczak K. *Front Immunol.* 2024 Sep 19:15:1376723 under the terms of the Creative Commons Attribution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

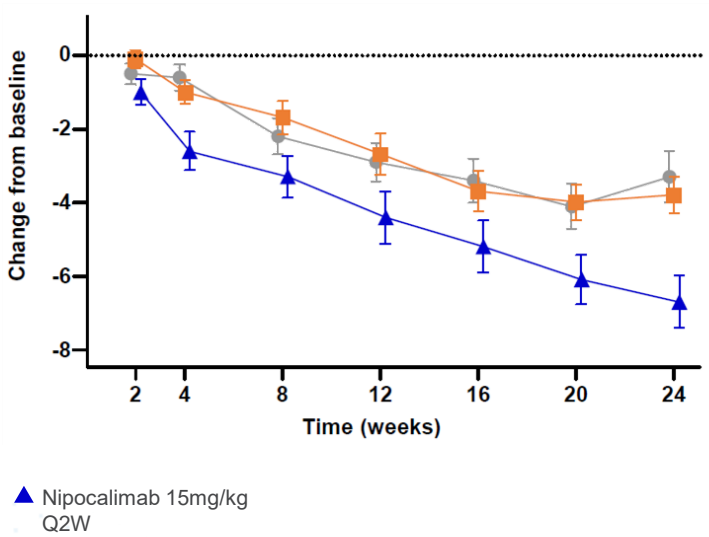
Publicly available nipocalimab data support anti-FcRn proof of mechanism and dose response in SjD

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

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



Mean (SE) change in ClinESSDAI score



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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

Notes: 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.

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

Appendix

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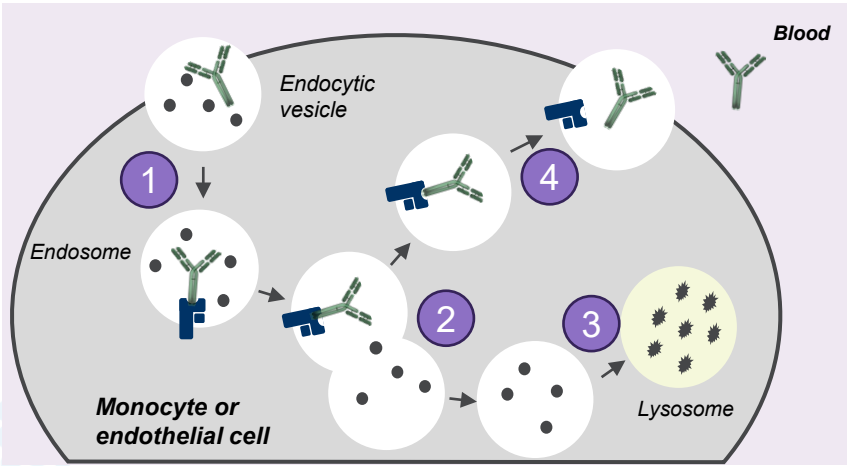
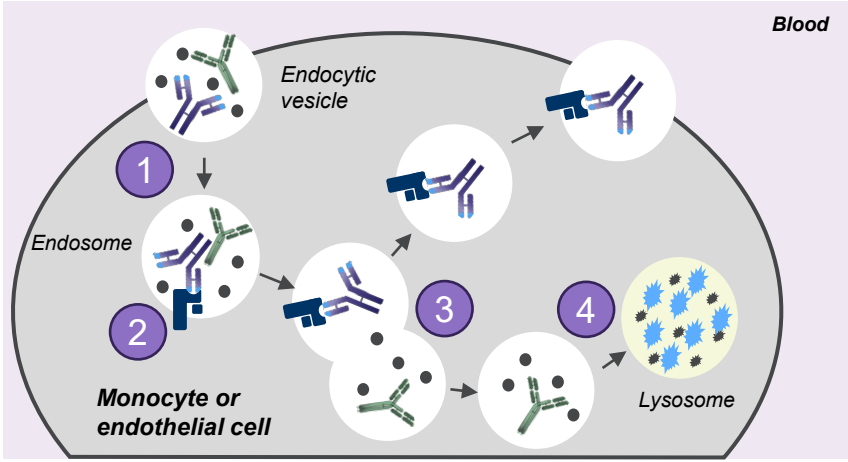
Our target: Neonatal Fc receptor (FcRn)

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

Anti-FcRn blocks binding of IgG to FcRn and promotes their removal and degradation



1. IgG and FcRn blocker are taken up into cells in endocytic vesicles
2. FcRn blocker 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 blocker | 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[1]
	argenx	Phase 3: Patient-level scatter plot demonstrating greater IgG declines ➔ greater MG-ADL improvements[2]
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[3]
RA	janssen	Phase 2: In those patients with greater IgG reduction ➔ correlation with greater autoAb reduction ➔ correlation with greater clinical response[4]
ITP	ucb	Phase 2: Greater IgG reduction across arms ➔ greater platelet responses[5]
PV/PF	argenx	Phase 2: More intensive dosing regimens across arms led to deeper IgG lowering ➔ deeper skin responses and lower rates of relapse[6]

Note: 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; MG: Myasthenia gravis, TED: Thyroid eye disease, GD: Graves' disease, ITP: Immune thrombocytopenic purpura, RA: Rheumatoid arthritis

1. Momenta Vivacity-MG Interim Phase 2 Investor Presentation, 2020
2. argenx JP Morgan Healthcare Conference Presentation January 2021
3. EULAR 2024 Abstract.
4. Janssen Research & Development, ACR poster, November 2023
5. IgG reduction at day 8 estimated by WebPlotDigitizer for 4mg/kg, 7mg/kg and 10mg/kg doses.
6. Argenx phase 2 PV/PF publication, Br J Dermatol. 2022 Mar;186(3):429-439

IMVT-1402 first 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

	Period 1 (26 weeks blinded treatment)	Period 2 (26 weeks blinded treatment)
Group 1	600 mg IMVT-1402 QW SC	600 mg IMVT-1402 QW SC
Group 2	600 mg IMVT-1402 QW SC	TRAb Responder? No: 600 mg IMVT-1402 QW SC; Yes: Placebo QW SC
Group 3	Placebo QW SC	Placebo QW SC

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 off ATD

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

Design enables study of remission

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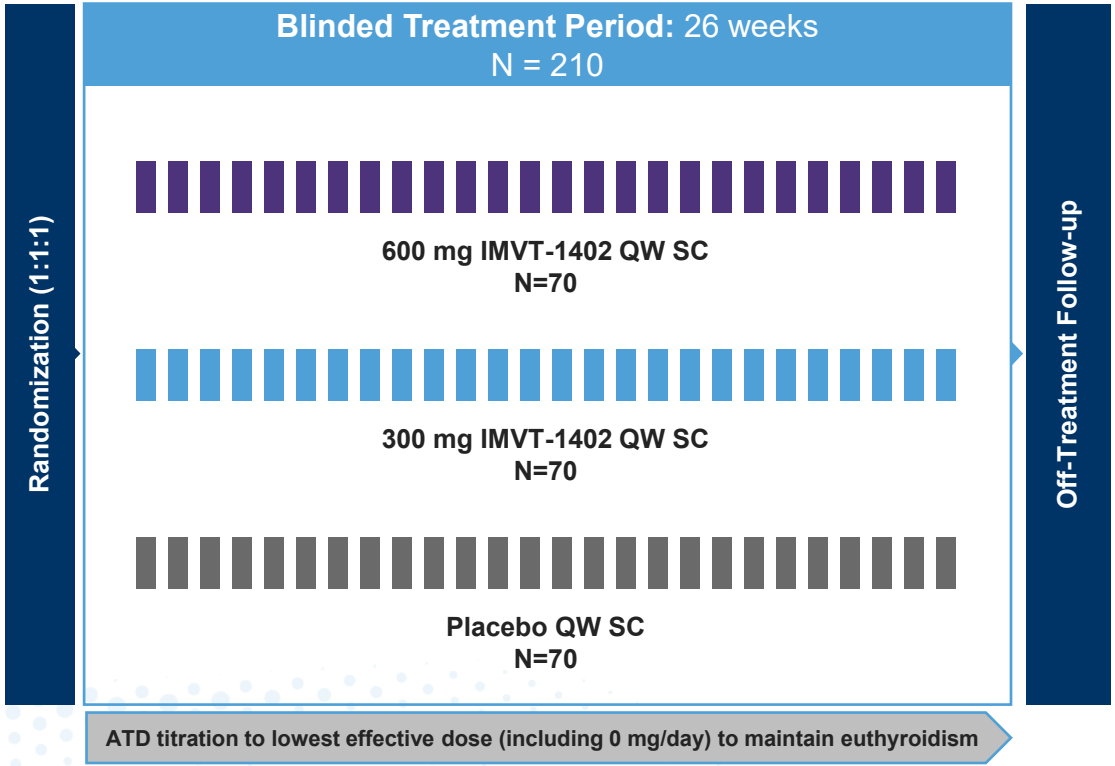
*Potentially registrational indicates registration is dependent on future alignment with FDA on clinical trial design, endpoints, statistical analysis plans, and overall development strategy
a: Additional inclusion and exclusion criteria not listed on slide
b. Euthyroid = T3/T4 and TSH within normal 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 who become euthyroid[b] and off ATD

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

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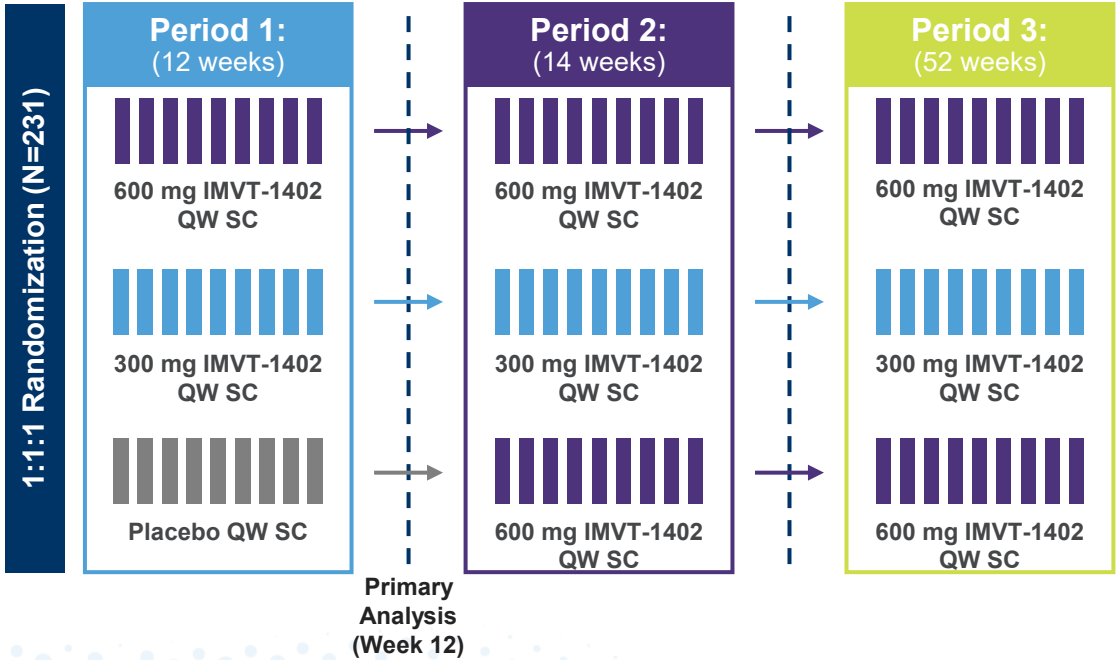
a: Additional inclusion and exclusion criteria not listed on slide
b. Euthyroid = T3/T4 and TSH within normal limits
TSH: Thyroid-stimulating hormone; ATD: Antithyroid drugs; QW: Weekly; SC: Subcutaneous; T3 = triiodothyronine; FT3: free triiodothyronine; FT4: free thyroxine; ULN: upper limit of normal

IMVT-1402 potentially registrational* trial in MG

Global trial with N=231 participants; designed to enable demonstration of deep, durable responses

Inclusion[a]

- Mild to severe gMG
- Positive diagnostic autoantibody confirmation (see note[b])



Primary Analysis Population:
anti-AChR+, anti-MuSK+, anti-LRP4+ (antibody positive)

Primary Endpoint:
Change from baseline in MG-ADL to Week 12 for antibody-positive participants

*Potentially registrational indicates registration is dependent on future alignment with FDA on clinical trial design, endpoints, statistical analysis plans, and overall development strategy
a. Additional inclusion and exclusion criteria not listed on slide.
b. Anti-AChR+, anti-MuSK+, or anti-LRP4+, with subset of antibody negative patients allowable by protocol. QW: Once weekly; SC: Subcutaneous; 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

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IMVT-1402 potentially registrational* trial in CIDP

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

Inclusion[a]

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

2:1 Randomization

Blinded Treatment Period[b]: 24 Weeks
N = 162

600 mg IMVT-1402 QW SC
N = 108

Placebo QW SC
N = 54

Open-label Extension: 52 Weeks

600 mg IMVT-1402 QW SC

600 mg IMVT-1402 QW SC

Primary Endpoint:

Proportion of participants remaining relapse-free (aINCAT) by Week 24

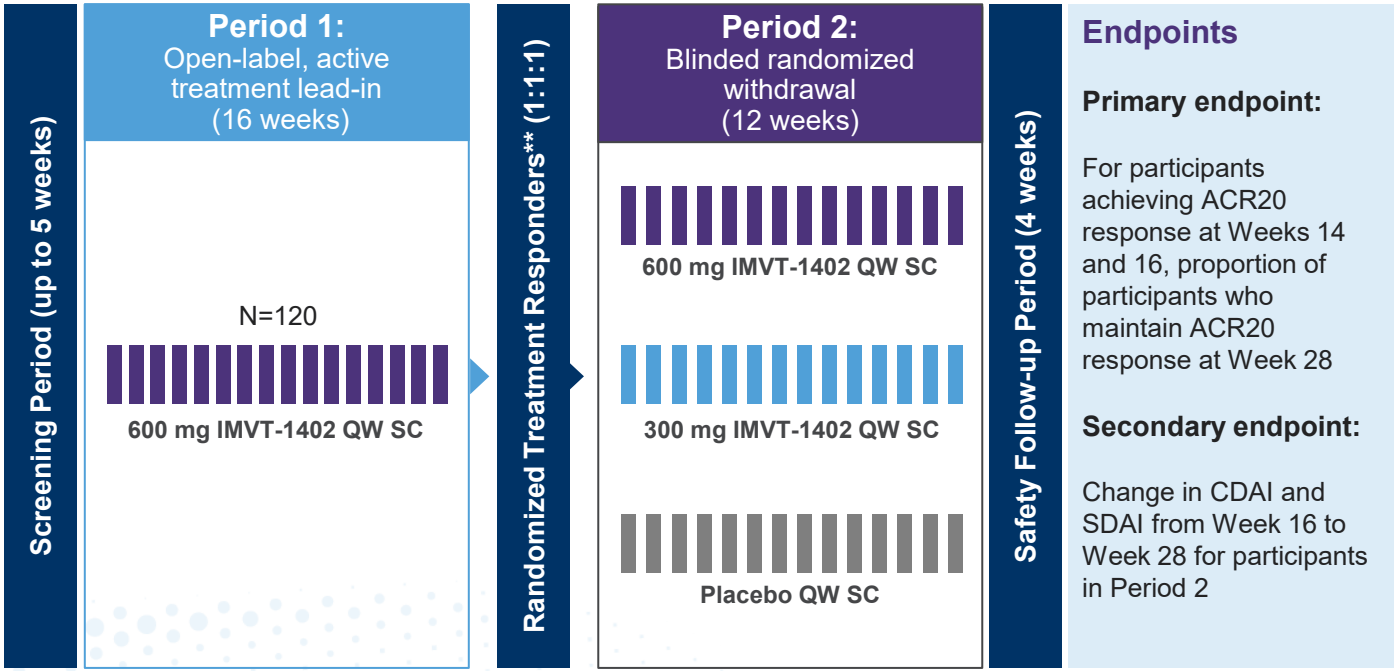
*Potentially registrational indicates registration is dependent on future alignment with FDA on clinical trial design, endpoints, statistical analysis plans, and overall development strategy
a. Additional inclusion and exclusion criteria not listed on slide.
b. Period 1: An additional dose of IMVT-1402 600 mg SC or placebo SC will be administered on Day 4 (± 1 day).
QW: Once weekly; SC: Subcutaneous; aINCAT: Adjusted Inflammatory Neuropathy Cause and Treatment disability score

IMVT-1402 potentially registrational* trial in ACPA+ D2T RA[1]

Trial designed as open label lead-in followed by randomized withdrawal in multi-mechanism failure population enriched for higher baseline ACPA levels

Inclusion[a]

- hsCRP ≥ upper limit of normal
- Active RA defined as ≥ 6/68 tender/painful joints, ≥ 6/66 swollen joints (SJC), and DAS28-CRP > 4.1
- IgG+ anti-citrullinated protein antibody (ACPA+)
- Inadequate response to 2, but not more than 4, 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 maintain ACR20 response at Week 28

Secondary endpoint:

Change in CDAI and SDAI from Week 16 to Week 28 for participants in Period 2

*Potentially registrational indicates registration is dependent on future alignment with FDA on clinical trial design, endpoints, statistical analysis plans, and overall development strategy
**Meets ACR20 criteria at Week 14 & Week 16
1. Defined as patients who have failed ≥2 advanced mechanisms (biologic and/or targeted synthetic DMARDs) in addition to conventional DMARDs
a. Additional inclusion and exclusion criteria not listed on slide.
Notes: 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)

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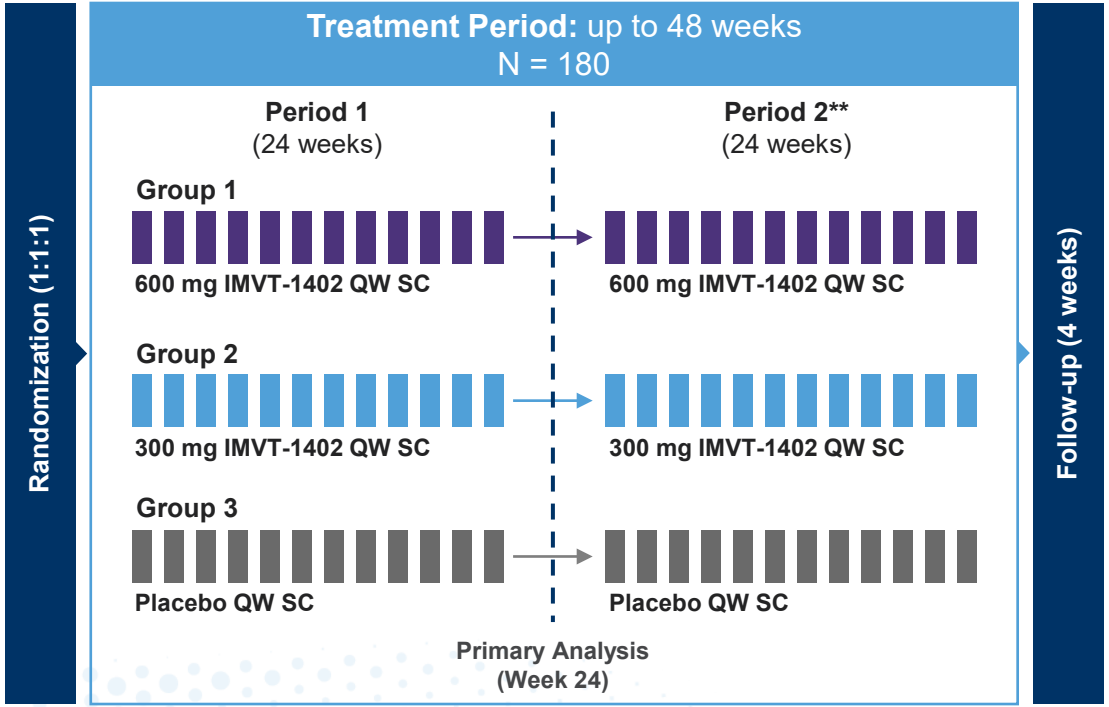
IMVT-1402 potentially registrational* trial in SjD

Trial enables comparison of high dose to low dose FcRn blockade

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



Endpoints

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

Select Secondary Endpoints:

- Proportion of clinESSDAI responders at Week 24
- Change from baseline in clinESSDAI score at Week 48

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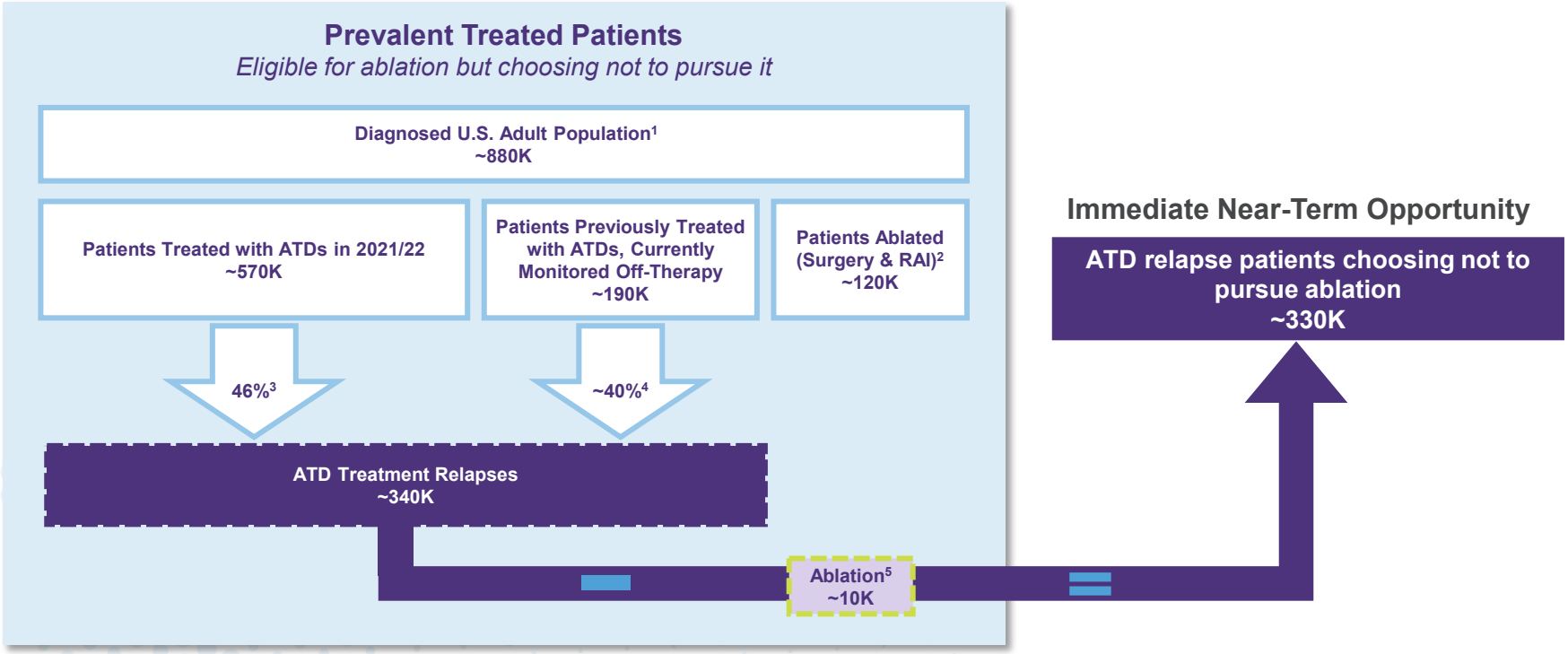
*Potentially registrational indicates registration is dependent on future alignment with FDA on clinical trial design, endpoints, statistical analysis plans, and overall development strategy
**Only ClinESSDAI responders (improvement of ≥ 4 points from baseline) continue through period 2
a. Additional inclusion and exclusion criteria not listed on slide
QW: Weekly; SC: Subcutaneous; ClinESSDAI: clinical European League Against Rheumatism Sjögren's Syndrome Disease Activity Index

Market Opportunity in Graves' Disease

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



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.

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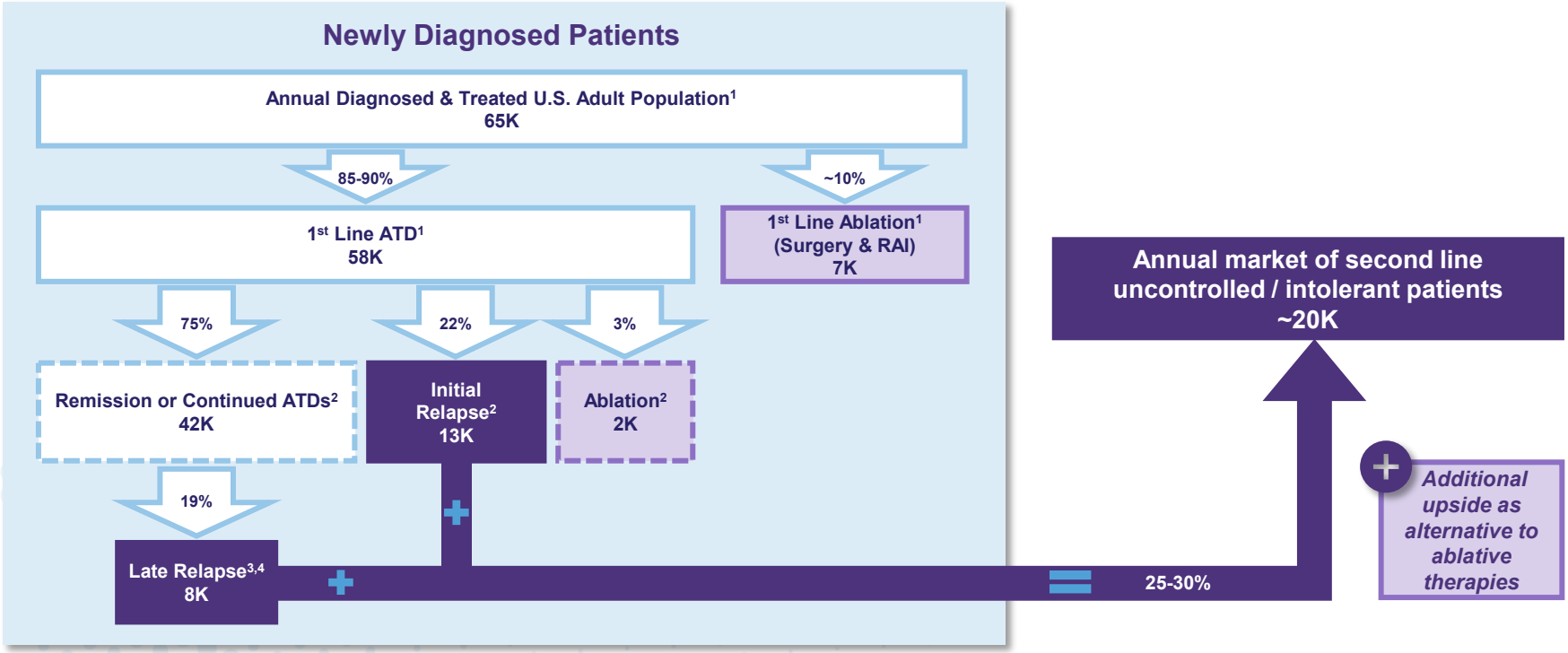
Analysis #2: Real world claims analysis conservatively estimates an incident US population of ~65K leading to an annual second line market of ~20K patients



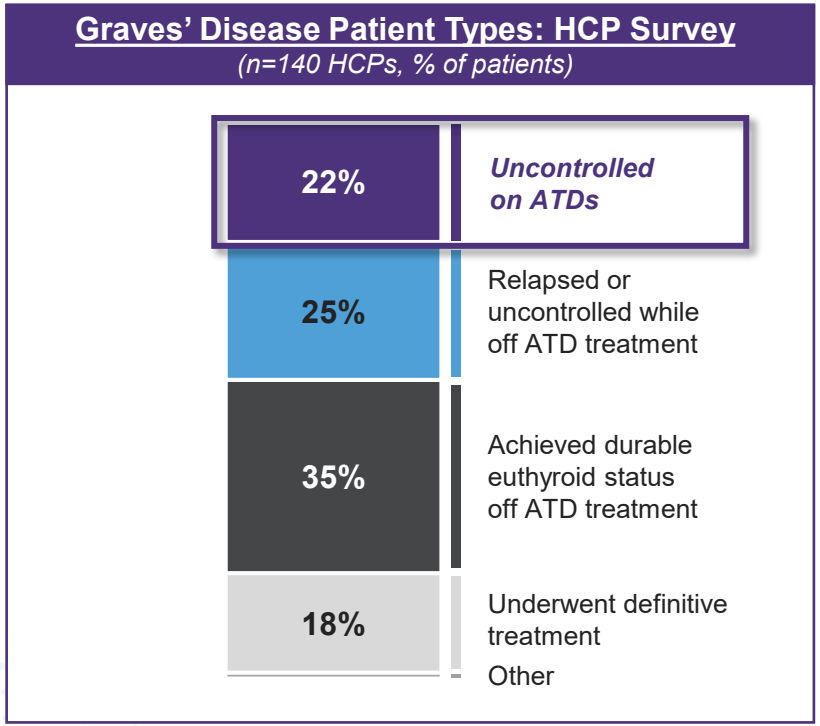
1. Roivant Claims Analysis – 2021 incident patient population, first-line treatment is primary treatment in the first-year post diagnosis, claims review included a five-year lookback to define the incident population
2. Grove-Laugesen et al. (2023): Completer rates for combined arms: ATD remission 56.0%, 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



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

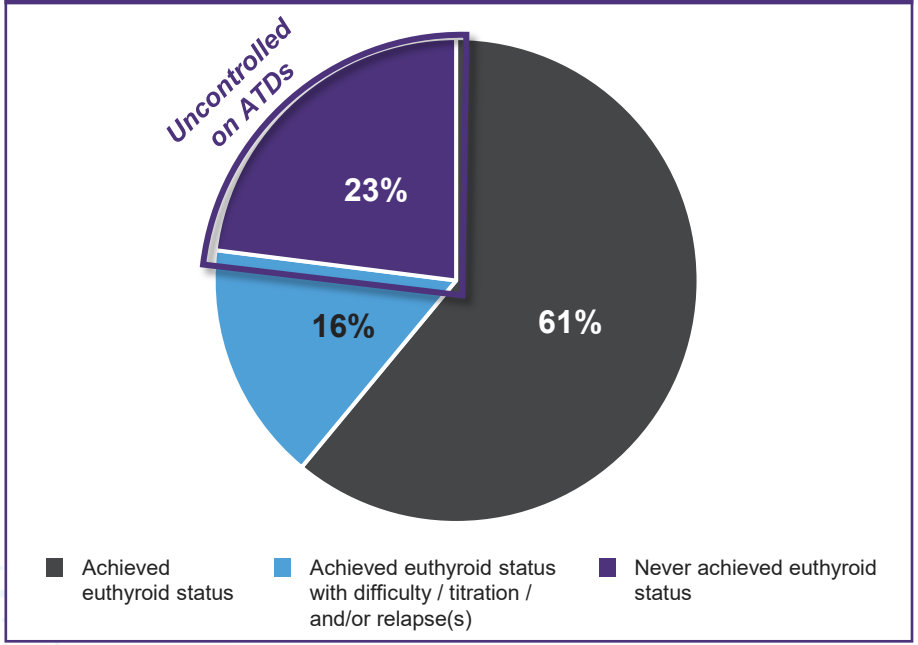
Analysis #4: Real-world in-depth chart review of 1,000+ patient records from 140 endocrinologists indicates ~25% have never achieved euthyroid status on ATDs

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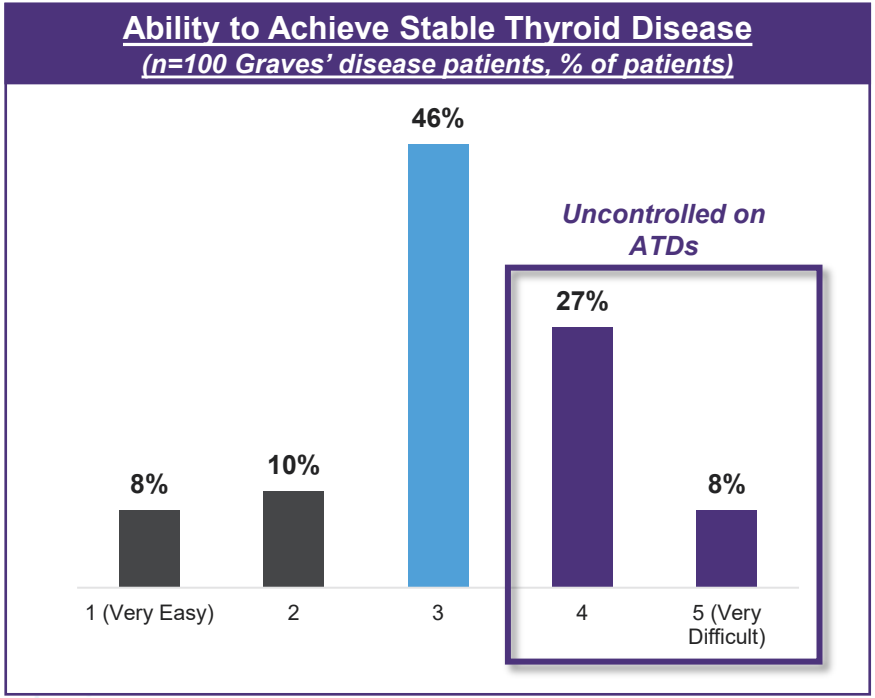
*Excludes patients who have received definitive treatment; Sources: Patient Chart Audit analysis (n=988 on ATD, n=1120 total) by Immunovant

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



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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