EX-99.2 4 f8k092919ex99-2_healthsci.htm INVESTOR PRESENTATION DATED OCTOBER 2, 2019

Exhibit 99.2

Corporate Overview

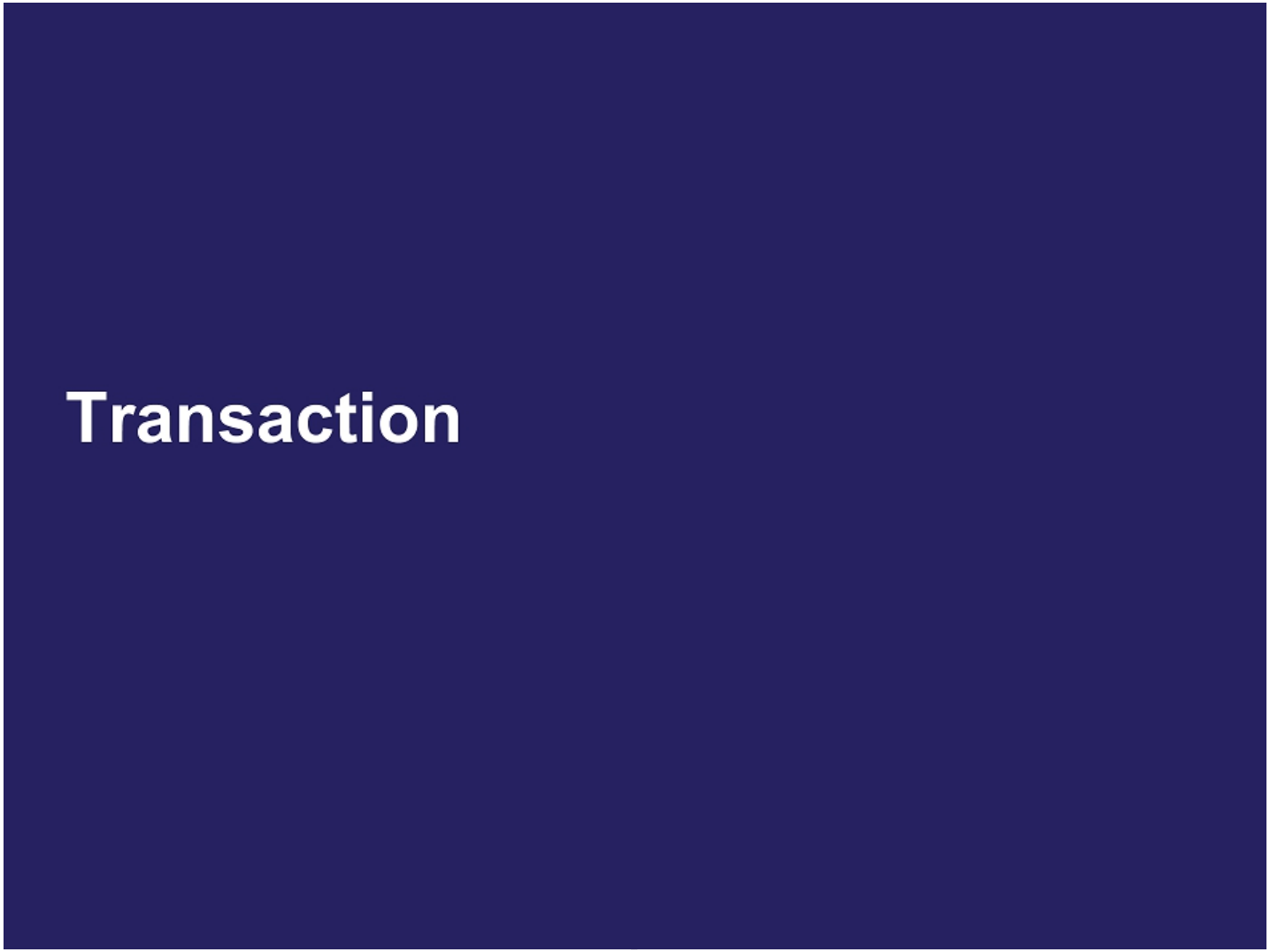
October 2019

This presentation has been prepared by Immunovant Sciences Ltd. ("we," "us," "our," "Immunovant" or the "Company") and contains forward-looking statements. In some cases, you can identify forward-looking statements by the words "anticipate," "believe," "continue," "could," "estimate," "expect," "intend," "may," "might," "objective," "ongoing," "plan," "potential," "predict," "project," "should," "will," and "would," or the negative of these terms, or other comparable terminology intended to identify statements about the future. These statements are based on management's current beliefs and expectations. These statements include but are not limited to statements regarding our business strategy, our plans to develop and commercialize our product candidates, our plans to enter into commercial transactions or strategic partnerships, the safety and efficacy of our product candidates, our expectations regarding timing, the design and results of clinical trials of our product candidates, our plans and expected timing with respect to regulatory filings and approvals, the size and growth potential of the markets for our product candidates, and our ability to serve those markets, and our plans and expected timing with respect to regulatory filings and approvals. These statements involve substantial known and unknown risks, uncertainties and other factors that may cause our actual results, performance or achievements to be materially different from the information expressed or implied by these forward-looking statements. We may not actually achieve the plans, intentions or expectations disclosed in our forward-looking statements, and you should not place undue reliance on our forward-looking statements. Actual results or events could differ materially from the plans, intentions and expectations disclosed in the forward-looking statements we make as a result of various important factors, including, without limitation, those inherent in the preclinical and clinical development process and the regulatory approval process, the risks and uncertainties in commercialization and gaining market acceptance, the risks associated with protecting and defending our intellectual property rights, our reliance on third-parties to conduct clinical and preclinical trials, our reliance on third-party suppliers to manufacture clinical, preclinical and any future commercial supplies of our product candidates, increased regulatory requirements, our ability to provide the financial support and resources necessary to develop our product candidate on the expected timeline, our ability to identify and acquire or in-license new product candidates and competition from others developing products for similar uses. These statements are subject to the risk that clinical study data are subject to differing interpretations, and regulatory agencies, medical and scientific experts and others may not share our view of the clinical study data. There can be no assurance that the clinical programs for our product candidate will be successful in demonstrating safety and/or efficacy, that we will not encounter problems or delays in clinical development, or that any of our product candidates will ever receive regulatory approval or be successfully commercialized. The forward-looking statements in this presentation represent our views as of the date of this presentation. We anticipate that subsequent events and developments will cause our views to change. While we may elect to update these forward-looking statements at some point in the future, we have no current intention of doing so except to the extent required by applicable law. You should, therefore, not rely on these forward-looking statements as representing our views as of any date subsequent to the date of this presentation.

HSAC, Immunovant, and their respective directors, executive officers and employees and other persons may be deemed to be participants in the solicitation of proxies from the holders of HSAC common stock in respect of the proposed transaction. Information about HSAC's directors and executive officers and their ownership of HSAC's common stock is set forth in HSAC's Registration Statement filed on Form S-1 filed with the SEC on May 3, 2019, as modified or supplemented by any Form 3 or Form 4 filed with the SEC since the date of such filing. Other information regarding the interests of the participants in the proxy solicitation will be included in the proxy statement pertaining to the proposed transaction when it becomes available. These documents can be obtained free of charge from the sources indicated below.

In connection with the transaction described herein, HSAC has filed and will file relevant materials with the SEC, including a proxy statement on Schedule 14A. Promptly after filing its definitive proxy statement with the SEC, HSAC will mail the definitive proxy statement and a proxy card to each stockholder entitled to vote at the special meeting relating to the transaction. Investors and security holders of HSAC are urged to read these materials (including any amendments or supplements thereto) and any other relevant documents in connection with the transaction that HSAC will file with the SEC when they become available because they will contain important information about HSAC, Immunovant and the transaction. The preliminary proxy statement, the definitive proxy statement and other relevant materials in connection with the transaction (when they become available), and any other documents filed by HSAC with the SEC, may be obtained free of charge at the SEC's website (www.sec.gov) or by writing to HSAC at 412 West 15th Street, Floor 9. New York, NY 10011.

By attending or receiving this presentation you acknowledge that you will be solely responsible for your own assessment of the market and our market position and that you will conduct your own analysis and be solely responsible for forming your own view of the potential future performance of our business and the transactions. The information herein does not purport to be all-inclusive. The data and estimates contained herein was derived from various internal and external sources, and involves a number of assumptions and limitations. You are cautioned not to give undue weight to such data or estimates. Neither HSAC nor Immunovant nor any other person makes any representation as to the accuracy or completeness of such data or undertakes any obligation to update such data after the date of this presentation. This presentation does not constitute an offer to sell or the solicitation of an offer buy securities, nor is it a solicitation of any vote or approval of the transactions, nor will there be any sale of these securities in, any state or jurisdiction in which such offer, solicitation or sale would be unlawful. Any offering of securities or solicitation to vote regarding the proposed transactions described herein will be made only by means of a proxy statement on Schedule 14A filed with the SEC.


Transaction

Immunovant and HSAC to merge

TRANSACTION SUMMARY	• Immunovant Sciences Ltd. ("Immunovant") and Health Sciences Acquisitions Corporation ("HSAC," Nasdaq: HSAC) have entered into a definitive business combination agreement – Immunovant is a clinical-stage biopharmaceutical company focused on enabling normal lives for patients with autoimmune diseases – HSAC is a special purpose acquisition company sponsored by RTW Investments – Upon the closing of the transactions, HSAC will change its name to **Immunovant, Inc.** • Expected post transaction equity value of $556 million, assuming HSAC share price of $10/share and no redemptions from the HSAC shareholders • Transaction expected to close in December 2019
PREMIER INVESTORS AND ALIGNMENT OF INTEREST	• Provides Immunovant with a blue-chip investor base and cash resources to continue development of IMVT-1401, a compelling asset within the FcRn drug class • Shareholders of the combined company expected to include Roivant Sciences, RTW Investments, and leading biotech investors including BVF Partners, Adage Capital Management, Cormorant Asset Management, Eventide Asset Management, and Perceptive Advisors
USE OF PROCEEDS	• At the time of closing, the combined company is expected to have more than $100 million in cash and cash equivalents, including proceeds from the completed $35 million private bridge financing – Funding expected to enable completion of Phase 2 program in myasthenia gravis, Graves' ophthalmopathy, and warm autoimmune hemolytic anemia – Expected to provide runway into second half of 2021
KEY MANAGEMENT AND BOARD	• Combined company to be led by Immunovant Chief Executive Officer, Pete Salzmann, M.D., M.B.A. • Anticipated directors: Frank Torti (Chairperson), Andrew Fromkin, Douglas Hughes, George Migausky, Atul Pande, Myrtle Potter, Pete Salzmann

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At a glance: terms of transaction

Pro forma valuation	
Illustrative share price (per share)	$ 10.00
Non-redeemable shares outstanding[2]	55,575,000
Equity Value	**$ 555,750,000**
Cash provided by transaction	$ 115,341,558[1]

Sources of funds	
HSAC Cash in Trust	$115,341,558[1]
Immunovant Shareholder Equity Rollover	$395,000,000
Bridge Financing	$35,000,000
Sponsor Promote	$10,750,000
Total Sources	**$556,091,558**

Uses of funds	
Equity Issued to Immunovant Shareholders (inclusive of bridge financing)	$430,000,000
Cash to Balance Sheet	$106,989,301[1]
Estimated Transaction Costs	$8,352,257
Sponsor Promote	$10,750,000
Total Uses	**$556,091,558**

Pro forma ownership with earn out to Immunovant and % total ownership

	Pro forma share price, per share					
	$10.00		$17.50		$31.50	
Immunovant	Shares	%	Shares	%	Shares	%
• Immunovant Shareholders and Employees[2]	39,500,000		40,688,136		41,392,216	
• Bridge Financing Investors	3,500,000		3,500,000		3,500,000	
• Earnout Shares, cumulative[3]			10,000,000		20,000,000	
Immunovant Total	43,000,000	77%	54,188,136	78%	64,892,216	78%
HSAC Sponsors[4]	1,075,000	2%	1,975,000	3%	2,875,000	3%
HSAC Public Shareholders	11,500,000	21%	13,471,429[5]	19%	15,150,794[5]	18%
Pro Forma Diluted Shares Outstanding	55,575,000	100%	69,634,565	100%	82,918,010	100%

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1. Assuming no redemptions from the HSAC shareholders
2. Calculated using $395M pre-money equity value, on a fully-diluted basis. Includes imputed dilution from employee stock options and 10,000 shares of Series A Preferred Stock. Roivant Sciences will receive shares of Series A Preferred Stock that provides for certain proportional voting rights for the election of directors.
3. Bridge investors and Immunovant current shareholders, but not employees, are entitled to receive a pro rata portion of earnouts.
4. 1.8M sponsor shares are cancelled at $10.00; 900,000 shares are cancelled at $17.50; no shares are cancelled at $31.50
5. Giving effect to warrant exercisable at $11.50 per share, using treasury stock method to calculate fully diluted shares outstanding

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Immunovant

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Our vision: Normal lives for patients with autoimmune diseases

Our asset: IMVT-1401, a novel, fully human monoclonal antibody inhibiting FcRn-mediated recycling of IgG

Our strategy for IMVT-1401:

- **Be best-in-class** in target indications where anti-FcRn mechanism has already established clinical proof-of-concept
- **Be first-in-class** in target indications with clear biologic rationale and no known in-class competition

Our near-term value drivers: Four anticipated data readouts over the next 20 months

Immunovant Leadership

Management Team

Pete Salzmann, MD, MBA
Chief Executive Officer

Lilly
UCSF Medical Center

Robert Zeldin, MD
Chief Medical Officer

Ablynx NOVARTIS
MERCK

Sandeep Kulkarni, MD
Chief Operating Officer

QVT Financial LP BCG
IHME

Brad Middlekauff, JD
General Counsel

PDS Biotechnology
kolltan MEDAREX

Board

- **Frank Torti, MD, MBA** Immunovant Chairperson, Vant Investment Chair, Roivant Pharma
- **Myrtle Potter** Vant Operating Chair, Roivant Pharma
- **Andrew Fromkin**
- **Douglas Hughes**
- **George Migausky**
- **Atul Pande, MD**

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IMVT-1401: Program Highlights

IMVT-1401: A novel, fully human monoclonal antibody inhibiting FcRn

- Early evidence suggests that anti-FcRn agents could transform the treatment of autoimmune diseases mediated by pathogenic IgG antibodies

In Phase 1, IMVT-1401 generated compelling pharmacodynamic activity

- Clinically meaningful IgG reductions observed (78% IgG reduction at 680mg dose level)
- No difference observed between intravenous and subcutaneous formulations at equivalent doses

IMVT-1401 has been well tolerated to date

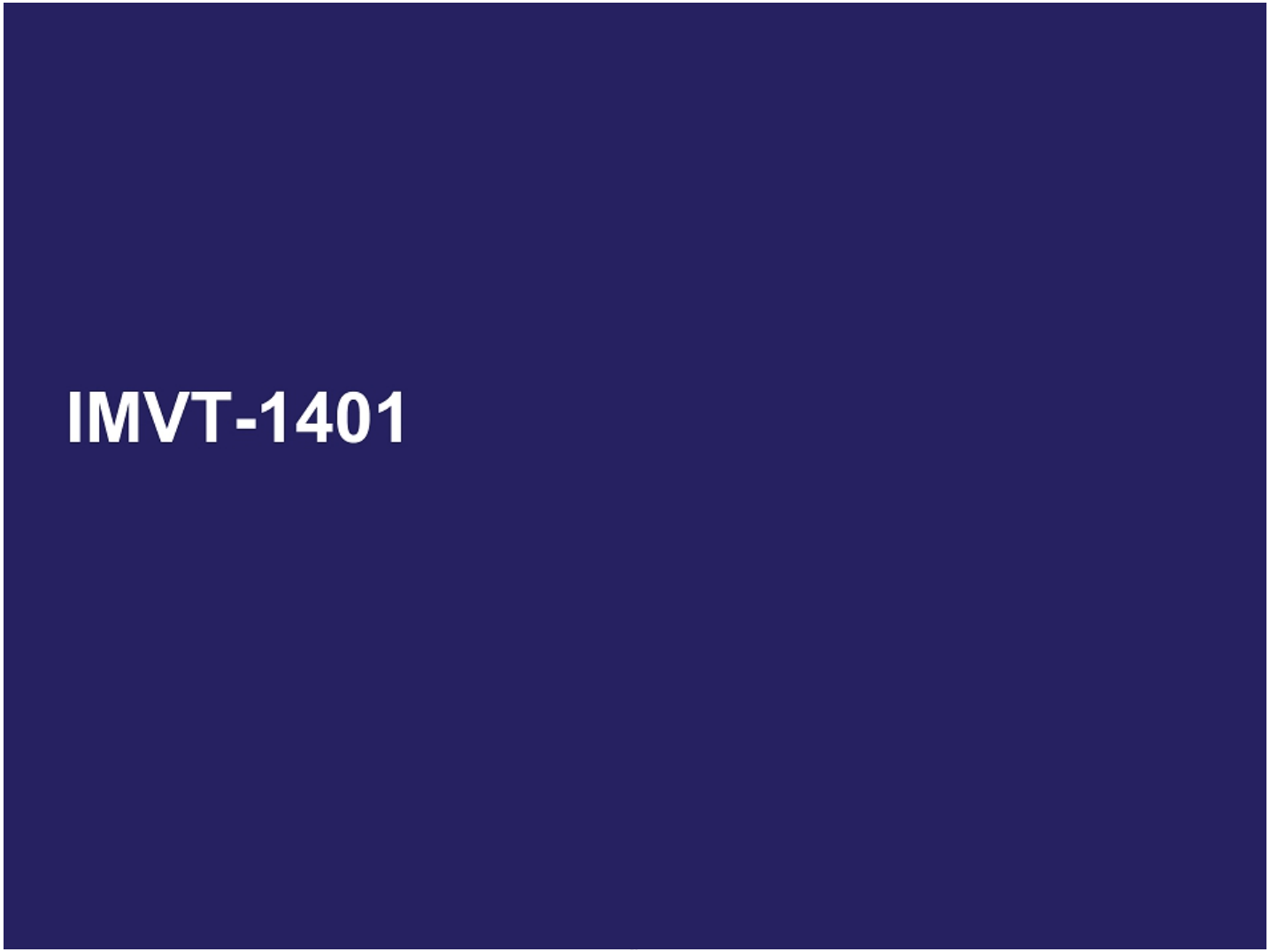
- No headaches reported in the highest dose multiple dose cohort tested
- No treatment-related serious adverse events (SAEs) or dose limiting toxicities reported
- No confirmed cases of anti-drug antibodies in any subject in multiple dose cohorts

IMVT-1401 was designed from inception for subcutaneous (SC) injection

- Requirement during development process
- Phase 1 data suggest every other week or less frequent dosing achievable for chronic use

IMVT-1401: A Pipeline in a Product

Target Indication	Pre-clinical	Phase 1	Phase 2	Phase 3	Status
Myasthenia Gravis (MG)	ASCEND-MG				• Phase 2a open for enrollment
Graves' Ophthalmopathy (GO)	ASCEND-GO				• Phase 2a open for enrollment • Phase 2b open for enrollment
Warm Autoimmune Hemolytic Anemia (WAIHA)	ASCEND-WAIHA				• IND submission expected in 2H 2019


IMVT-1401

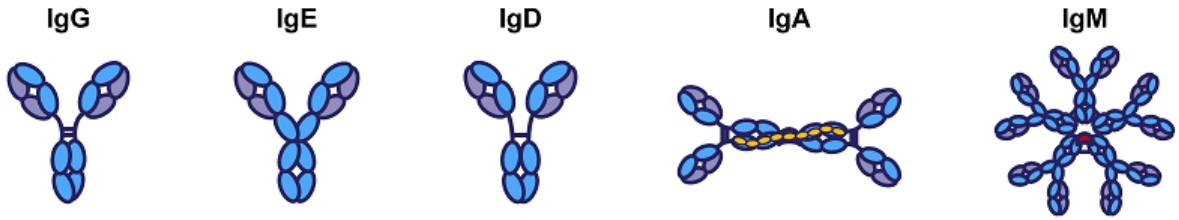
IgG antibodies implicated in certain autoimmune diseases

Antibodies in healthy individuals

- Antibodies play an important role in immune defense against pathogens[1]
  - Clearing bacteria, viruses, and other harmful organisms and substances
  - Eliciting an immune response that leads to inflammation
- IgG antibody subclass accounts for ~75% of antibodies in the plasma of healthy people[1]

Antibodies in autoimmune disease

- In many autoimmune diseases, IgG antibodies develop that can recognize and bind to normal tissues[2]
  - Targets may include cell-surface receptors or circulating proteins
  - Result is a harmful immune response that damages critical tissues and organs
- Predisposing factors may include genetic susceptibility, environmental triggers, and factors not yet known[3]



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1. Leusen J.H.W., The Role of IgG in Immune Responses. Molecular and Cellular Mechanisms of Antibody Activity, 2013
2. Isabela S., et al. The role of autoantibodies in health and disease. romanian journal of morphology and embryology, 2016
3. Mariani SM. Genes and autoimmune diseases - a complex inheritance. Med Gen Med, 2004

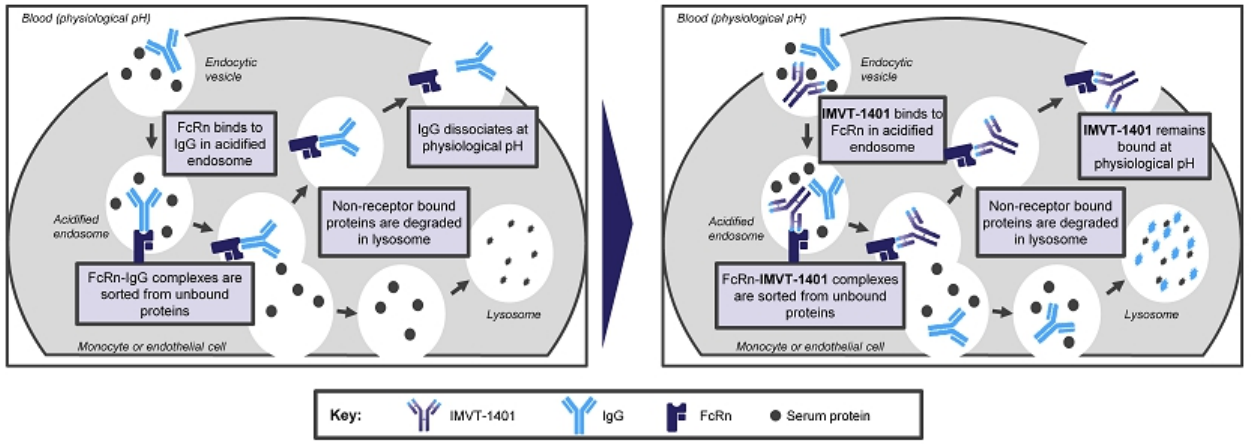
IMVT-1401's mechanism shown to promote IgG degradation[1]

FcRn prolongs the half-life of IgG[2]

- **FcRn intercepts IgG**, which would otherwise be degraded in lysosomes
- The FcRn–IgG complex is then recycled to the cell surface and free IgG is **released back into circulation**

Inhibiting FcRn promotes IgG degradation[2]

- **IMVT-1401 binds to FcRn**, thereby preventing it from recycling IgG antibodies back to circulation
- As a result, IgG is **increasingly delivered to lysosomes** for degradation



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1. See Phase 1 MAD/SAD data on slide 16
2. Derry C., et al. FcRn: the neonatal Fc receptor comes of age. Nat Rev Immunol, 2007

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Broad range of potential applications for anti-FcRn mechanism

IgG-mediated autoimmune diseases where FcRn mechanism may be relevant:

- Myasthenia Gravis
- Graves' Ophthalmopathy
- Warm Autoimmune Hemolytic Anemia
- Chronic Inflammatory Demyelinating Polyneuropathy
- Neuromyelitis Optica
- Idiopathic Thrombocytopenic Purpura
- Guillain-Barré Syndrome
- PLA2R+ Membranous Nephropathy
- Pemphigus Vulgaris

Additional IgG-mediated autoimmune diseases

Note: List of diseases is illustrative only and does not necessarily represent our targeted indications

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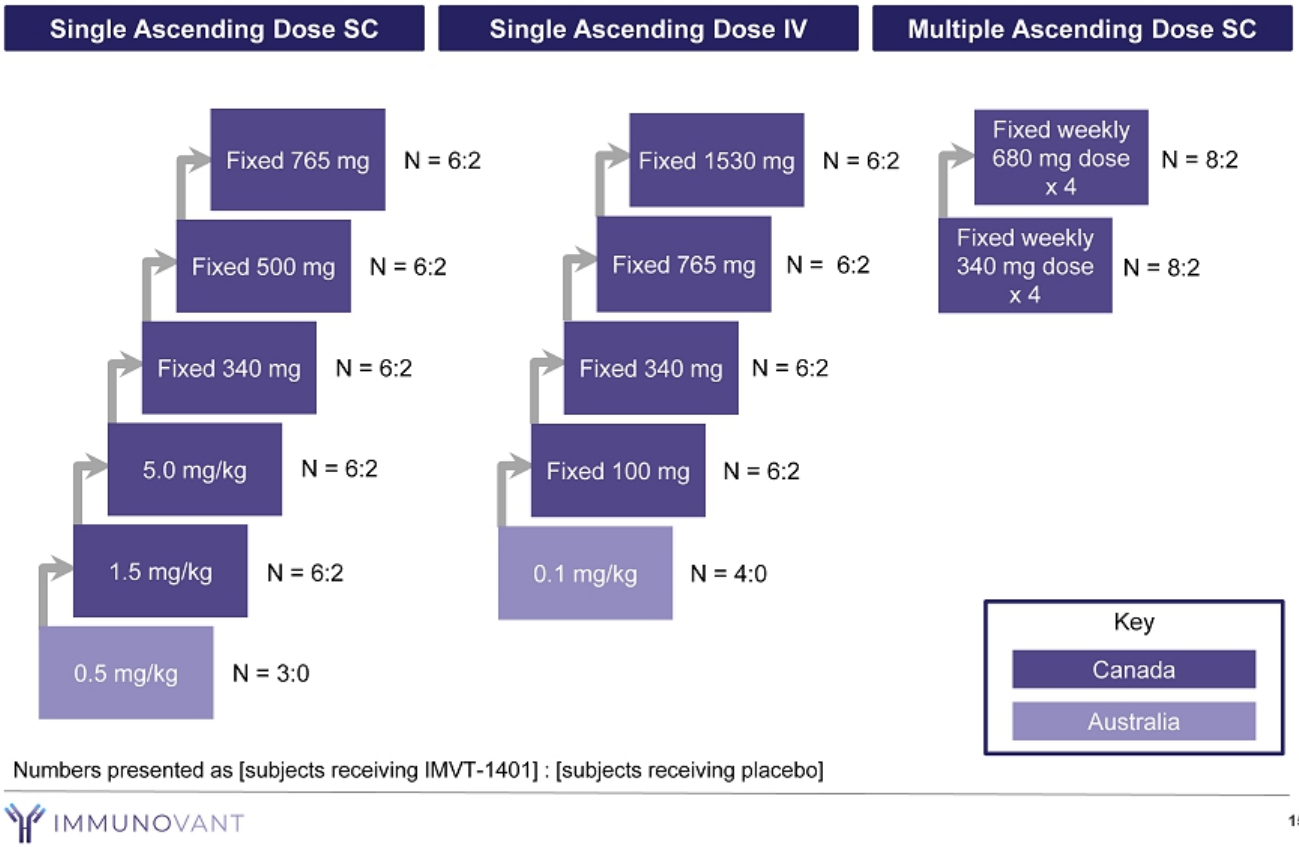
Phase 1 SAD/MAD study design



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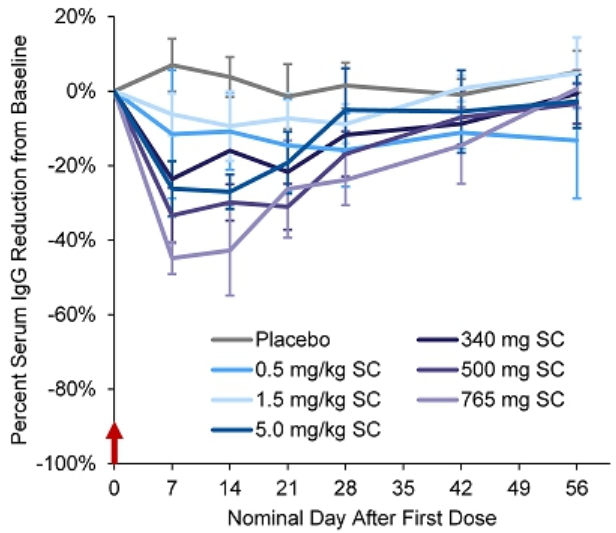
IMVT-1401 produced clinically meaningful IgG reductions in Phase 1 study

Preliminary results from Phase 1 SAD/MAD cohorts

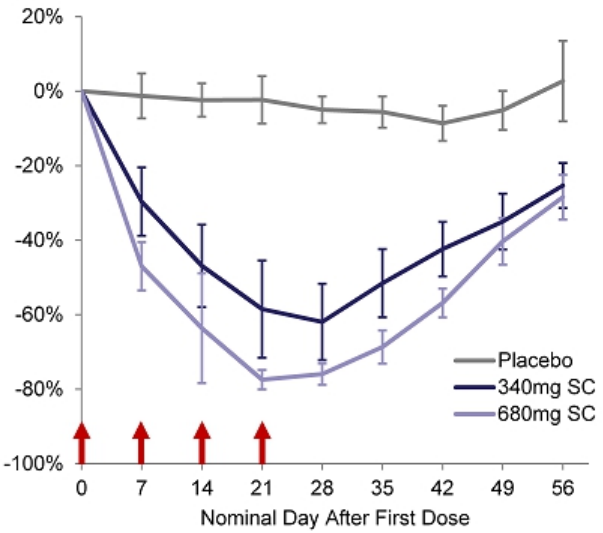
Single-dose administration produced dose-dependent IgG reductions

Repeat dosing at 680mg SC resulted in a 78% IgG reduction without the need for IV induction

Mean total IgG reduction after single dose in healthy volunteers



Mean total IgG reduction after 4 weekly doses in healthy volunteers



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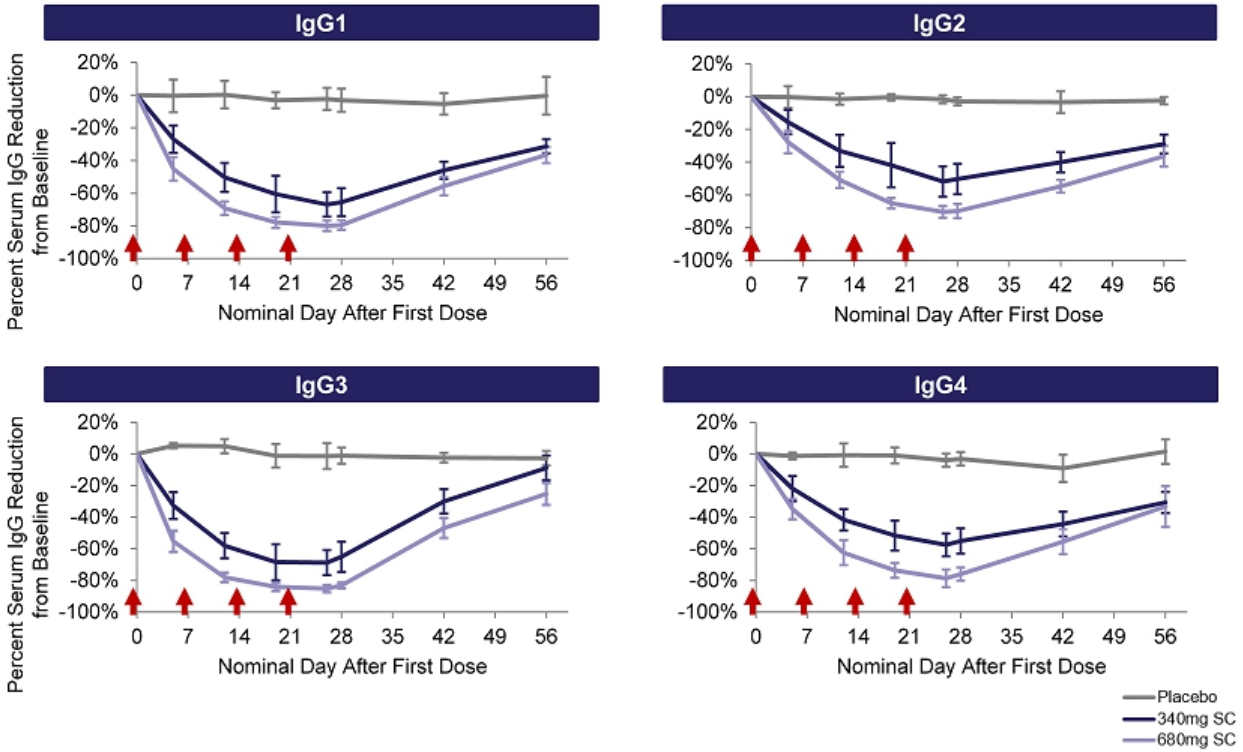
IMVT-1401 reduced levels of all four IgG subtypes

Preliminary results from Phase 1 MAD cohorts



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Generally well-tolerated in Phase 1 study

Preliminary results from Phase 1 SAD/MAD cohorts

- 99 subjects dosed to date through SAD and MAD portions of Phase 1
  - IMVT-1401: 77 subjects
  - Placebo: 22 subjects
- Most common AEs were mild erythema and swelling at injection site
  - Injection site reactions were not dose or frequency related
  - Occurred at similar incidence for drug and placebo treated subjects
- No headaches observed in 680mg SC MAD cohort
- Albumin changes:
  - Dose-dependent, reversible, and asymptomatic albumin reductions observed
  - At day 28, mean albumin levels were 37.5 g/L in the 340 mg cohort, and 32.4 g/L in 680mg cohort (normal range 36-51 g/L)
- 2 SAEs observed in two separate SAD cohorts, both ruled unrelated to treatment by study investigator (cancer, appendicitis)
- Treatment-emergent ADA confirmed in 8% of IMVT-1401-treated subjects and 6% of placebo-treated subjects
  - No subject in MAD cohorts has developed a confirmed ADA response to IMVT-1401

Adverse events reported in Phase 1

Preliminary results from Phase 1 SAD/MAD cohorts

	Single-Ascending Dose												Multiple-Ascending Dose		
	Intravenous Infusion						Subcutaneous Injection						Subcutaneous Injection		
	0.1 mg/kg n=4	100 mg n=6	340 mg n=6	765 mg n=6	1530 mg n=6	Placebo n=8	0.5 mg/kg n=3	1.5 mg/kg n=6	5 mg/kg n=6	340 mg n=6	500 mg n=6	765 mg n=6	Placebo n=10	340 mg n=8	680 mg n=8	Placebo n=4
MedDRA Preferred Term																
Abdominal pain									1					1		
Abdominal pain upper													2	1		
Abnormal sensation in eye					1					1						
Back pain						2					1		1	1		
Constipation						1								1		
Cough											1		2			
Diarrhea														2		
Dizziness						1							1			1
Dry skin													1		1	
Erythema							1								1	
Fatigue	1			1	1	1	1			1			1			
Headache	1	1	1	1	1			1	1	4	1		1	2		
Injection site erythema									5	1	5	6	7	8	7	4
Injection site pain											1			2		1
Injection site swelling									3		2	4	3	7	6	2
Insomnia									1					4		
Myalgia														1	1	
Nasal congestion									1		1		1	1		
Nausea									1	1			1		1	1
Ocular hyperaemia															2	
Oropharyngeal pain	1			1	2				1		1		1	2		
Pain in extremity						1							1			
Procedural complication								1		1						
Procedural dizziness					2						1					
Pyrexia			1	1					1							
Rash					2				2				2		1	
Rhinorrhea									1				2			
Sinusitis			1										1			
Somnolence		1							1							
Upper respiratory tract infection	1	1	1				3		1	1				1		
Vision blurred					1					1						

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IMVT-1401 has been given as a convenient SC injection

Subcutaneous Injection

<10 seconds

Subcutaneous Infusion

30-60 minutes

Intravenous Infusion

Potentially Hours

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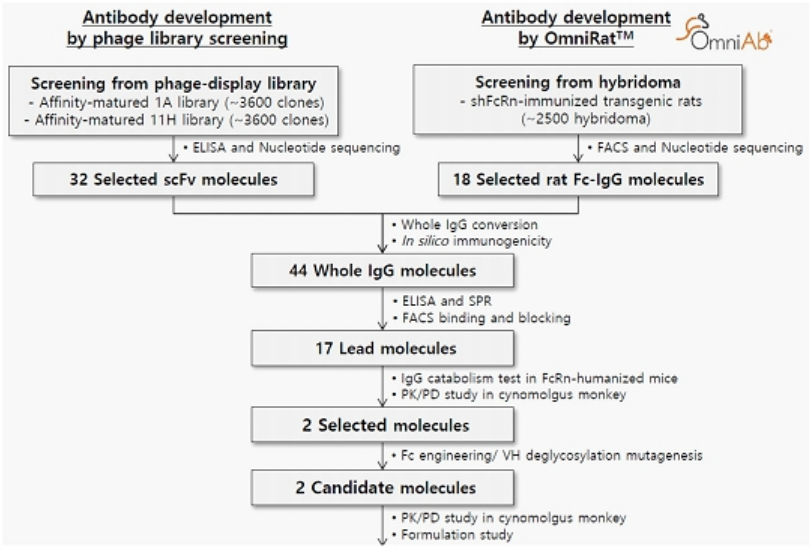
IMVT-1401 designed from inception to be a potentially class-leading SC injection

- Fully human monoclonal antibody
- Generated from Ligand/OMT's OmniAb transgenic rat platform
  - >400 antibody campaigns ongoing that use OmniAb technology[1]
  - 12 clinical-stage antibodies in development[1]
- IgG1 backbone Fc-engineered to reduce effector function
- Optimized for SC delivery
  - Current clinic formulation is 170mg/mL
  - Delivered by 27-gauge needle



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1. Ligand 2019 Analyst Day presentation, presented March 12, 2019

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IMVT-1401 has the potential to deliver a class-leading profile

IMVT-1401 attribute	Potential patient benefit
Clinically meaningful IgG reductions	• 680mg SC weekly: 78% reduction after four doses • 340mg SC weekly: 63% reduction after four doses
SC injection	• Fast and minimally invasive
Simple dosing schedule	• No requirement for IV induction doses or lengthy SC infusions • Provides option for at-home administration • Fixed dosing, vs. weight-based, reduces potential for dose miscalculations
Fully human antibody	• Low risk of immunogenicity
Fc-engineered to reduce effector function	• Low potential for unintended immune responses

IMVT-1401 and competitors' programs with subcutaneous (SC) injection or infusion

Company	IMMUNOVANT	argenx	ucb	ALEXION AFFIBODY	MOMENTA	ALEXION syntimmune
Anti-FcRn candidate	IMVT-1401	Efgartigimod (ARGX-113)	Rozanolixizumab (UCB7665)	ABY-039	M281	ALXN1830 (SYNT001)
SC administration regimen	SC injection	IV induction, followed by SC injection[1]	SC infusion given over 30-60 minutes[3,4]	SC injection	Not in clinic with SC formulation	Not in clinic with SC formulation
IV induction dosing	N/A	20mg/kg IV x 2 doses[1]	N/A	N/A		
SC dose	340mg SC weekly 680mg SC weekly	300mg SC weekly[1]	7mg/kg SC weekly[5]	200mg SC single dose[6]		
Mean IgG reduction observed	63-78% after 4 doses	"Approximately" 50% after two IV induction doses followed by 8 SC doses[2]	56% after 3 doses[5] 68% after 6 doses[5]	~45%		

Note: data as of 9/24/2019 and is not based on head to head comparison studies

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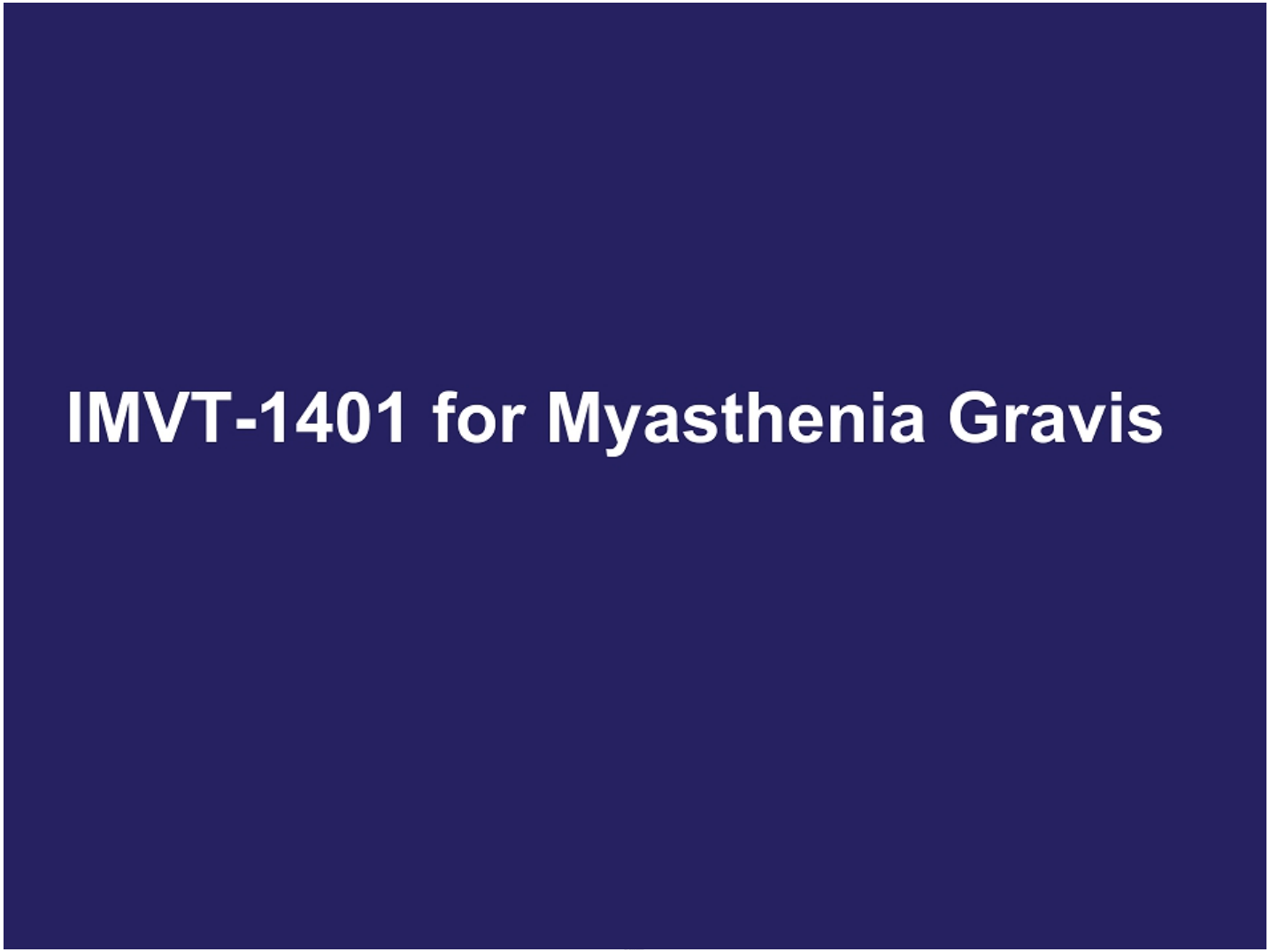
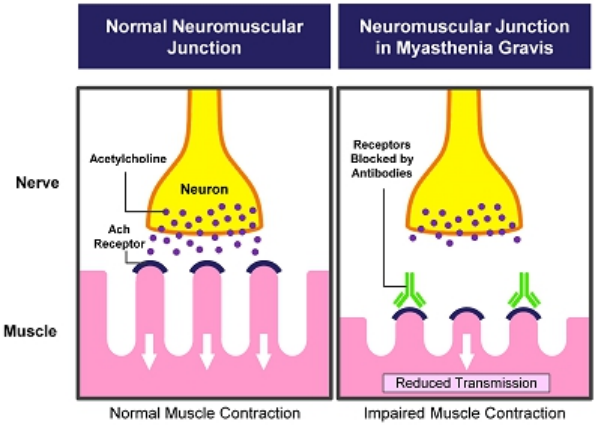
1. Argenx, corporate presentation, August 2018
2. Argenx, press release, issued June 14, 2018
3. Kiessling P. et al, The FcRn inhibitor rozanolixizumab reduces human serum IgG concentration: A randomized phase 1 study Science Translational Medicine, 2017
4. UCB, ASH presentation, December 2017
5. UCB, press release, issued October 18, 2018
6. Affibody, PEGS conference presentation, April 2019


IMVT-1401 for Myasthenia Gravis

Myasthenia Gravis overview

- Rare autoimmune disorder affecting an estimated 65,000 people in the US[1]
- Characterized by weakness of voluntary muscles including ocular, facial, oropharyngeal, limb, and respiratory muscles[1]
- 15-20% of MG patients will experience at least one myasthenic crisis over their lifetimes, a potentially life threatening acute complication[2]
- Disease caused by autoantibodies targeting the neuromuscular junction (NMJ)[1]
- ~93% of patients have an identified autoantibody[1]
  - Anti-acetylcholine receptor (AChR) antibodies (~85%)
  - Anti-muscle-specific tyrosine kinase (MuSK) antibodies (~8%)



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1. Meriggioli M.N. & Sanders D.B. Muscle autoantibodies in myasthenia gravis: beyond diagnosis? Expert Rev Clin Immunol, 2012
2. Sudulagunta S.R. et al. Refractory myasthenia gravis – clinical profile, comorbidities and response to rituximab. Ger Med Sci., 2016

Unmet need persists despite availability of treatment options

Current treatment paradigm[1]

1st Line	2nd Line	3rd Line	4th Line
• Acetylcholinesterase inhibitors • Corticosteroids	• Immunosuppressive agents • Thymectomy	• IVIg • Plasma exchange • Immunoadsorption • Rituximab (off-label)	• Eculizumab

Unmet need

- ~10% of MG patients refractory to current treatments, while 80% fail to achieve complete stable remission[1]
- Existing therapies associated with significant side effects
  - Early line agents can lead to disease exacerbation and do not always prevent disease progression
  - Treatment for more advanced disease often requires invasive and burdensome infusions
- Patients with anti-MuSK antibodies more likely to become refractory[1]
  - ~50% of the refractory MG population, despite comprising <10% of the overall MG population
  - Newest treatment option, eculizumab, only indicated for anti-AChR positive patients

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1. Mantegazza R & Antozzi C. When myasthenia gravis is deemed refractory: clinical signposts and treatment strategies. Ther Adv Neurol Disord., 2018

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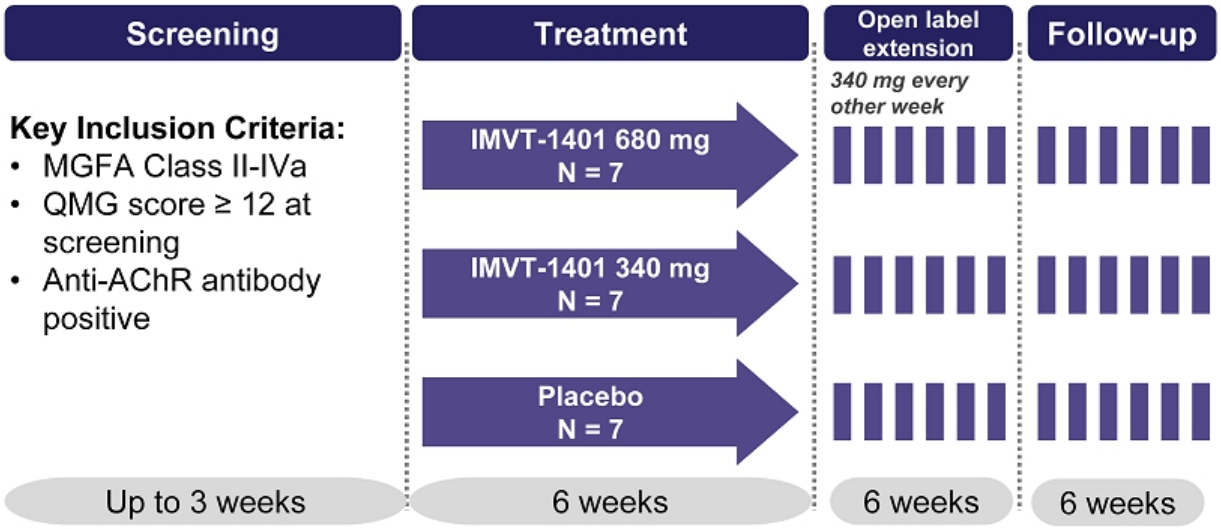
ASCEND-MG: Phase 2a study design



Primary Endpoint:

- Safety & tolerability
- Change from baseline levels of anti-AChR antibodies, total IgG, and IgG by subclasses

Secondary Endpoints:

- IMVT-1401 pharmacokinetics
- Change from baseline in QMG, MG-ADL, quality of life measures

Exploratory Endpoints:

- Biomarkers (gene expression, serum pro-inflammatory markers, receptor occupancy)

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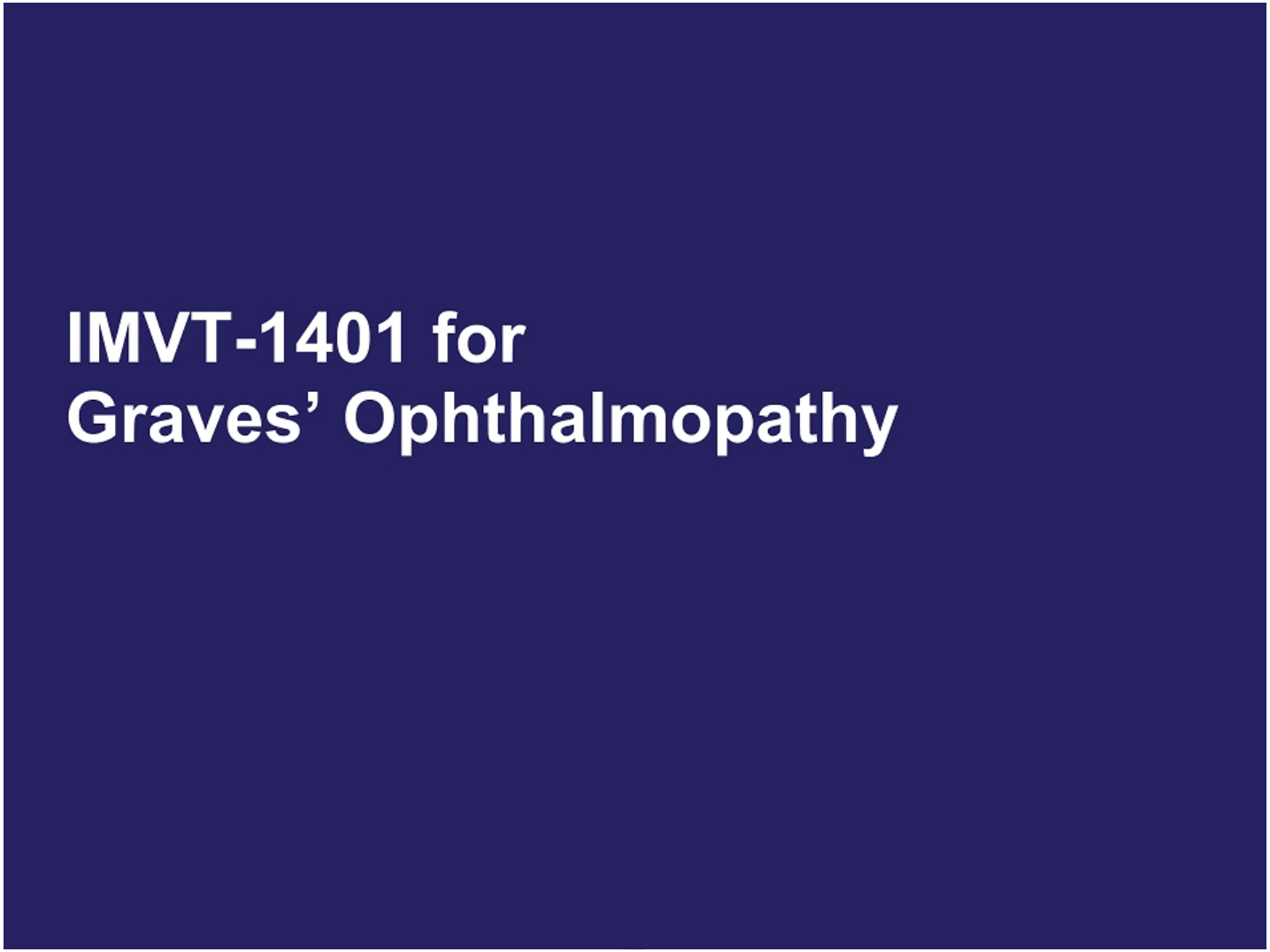
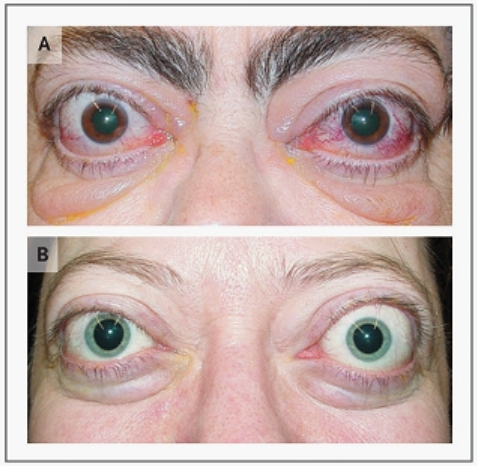

IMVT-1401 for
Graves' Ophthalmopathy

Graves' Ophthalmopathy overview

- Also called Graves' orbitopathy or thyroid eye disease (TED)
- 15,000-20,000 patients with active GO in the United States per year
- Clinical features[1]:
  - Proptosis
  - Eye pain
  - Double vision
  - Light sensitivity
- Can be sight-threatening[2]
- Caused by autoantibodies that activate cell types present in tissues surrounding the eye[2]
- Close temporal and pathobiologic relationship with Graves' disease



Bahn, 2010
Figure 1. Patients with Graves' Ophthalmopathy
Panel A shows a 59-year-old woman with excess proptosis, moderate eyelid edema, and erythema with moderate eyelid retraction affecting all four eyelids. Conjunctival chemosis (edema) and erythema with bilateral edema of the caruncles, with prolapse of the right caruncle, are evident. Panel B shows a 40-year old woman with excess proptosis, minimal bilateral injection, and chemosis with slight erythema of the eyelids. She also had evidence, on slit-lamp examination, of moderate superior limbic keratoconjunctivitis.

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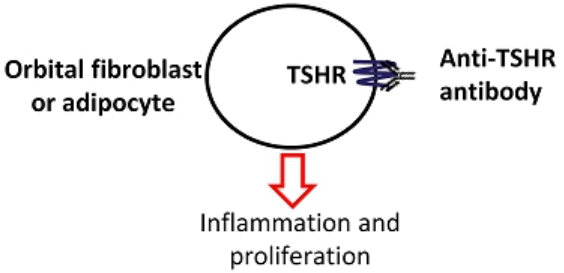
1. Davies T & Burch HB. Clinical features and diagnosis of Graves' orbitopathy (ophthalmopathy), UpToDate, 2018
2. McAlinden C. An overview of thyroid eye disease. Eye and Vision, 2014

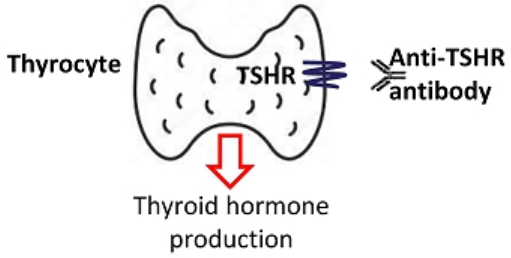
Anti-TSHR autoantibodies drive progression of GO and Graves' disease

Graves' ophthalmopathy



- Thyroid-stimulating hormone receptor (TSHR) highly expressed on ocular fibroblasts and adipocytes[1]
- Activation leads to inflammation and proliferation
- Autoantibodies against Insulin-like growth factor-1 receptor (IGF-1R) also identified[2]

Graves' disease



- TSHR highly expressed on thyrocytes (cells that make up the thyroid gland)[3]
- Activation leads to increased production of thyroid hormone[3]

IMVT-1401 could address GO and Graves' disease caused by any IgG autoantibody, whether against TSHR or IGF-1R

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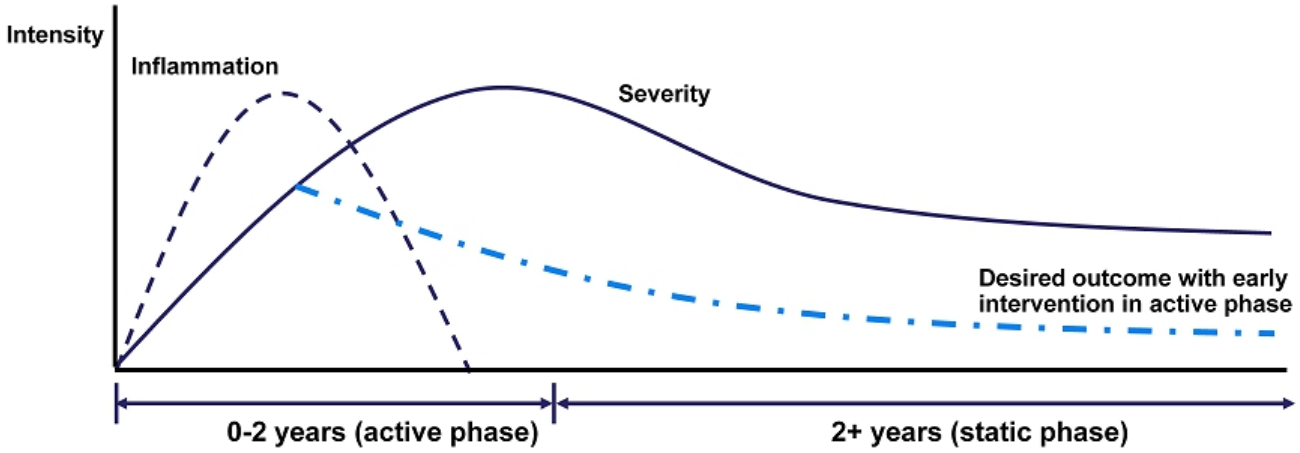
1. Smith T.J., et al. Role of IGF-1 pathway in the pathogenesis of Graves' orbitopathy. Best Pract Res Clin Endocrinol Metab, 2013
2. Liaboe C.A., et al. An Introductory Tutorial and Overview of Disease - Thyroid Eye Disease (TED), 2016
3. Varewijck A.J., et al. Circulating IgGs may modulate IGF-I receptor stimulating activity in a subset of patients with Graves' ophthalmopathy. J Clin Endocrinol Metab., 2013

GO characterized by an active phase, followed by a static phase

Rundle's Curve describes natural history of disease[1]



- Orbital tissue actively inflamed
- Steroids and other immunosuppressive treatments can be effective
- Inflammatory tissue replaced by fibrotic tissue
- Steroids and immunosuppression no longer effective
- Patients to be evaluated for surgery

IMMUNOVANT 1. Adapted from Hiromatsu Y., et al. Graves' ophthalmopathy: epidemiology and natural history. Intern Med., 2014 31

Limited treatment options for GO

Current treatment paradigm[1]

1st Line	2nd Line	3rd Line	Inactive disease
• Corticosteroids	• Orbital radiotherapy • Immunosuppressive agents	• Rituximab (off-label)	• Orbital surgery

Unmet need

- Currently no FDA-approved therapies for GO
- Corticosteroids are not effective in all patients, and approximately one-third of patients will relapse
- Sight-threatening disease may occur in 3-5% of patients with Graves' disease[2]
  - Medical emergency requiring immediate hospitalization and evaluation for surgery[2]
- Up to 20% of GO patients require surgical invervention[2]

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1. Bothun E.D., et al. Update on thyroid eye disease and management. Clin Ophthalmol., 2009
2. Bartalena L., et al. Management of Graves' Ophthalmopathy: Reality and Perspectives. Endocrine Reviews, 2000

ASCEND-GO Phase 2 clinical program

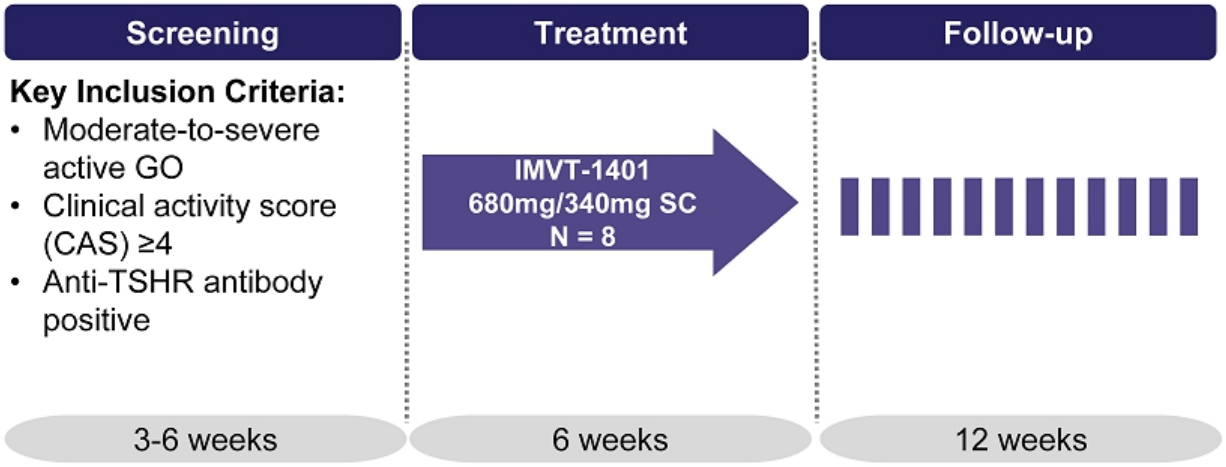
ASCEND-GO 1

- **Phase 2a**
  - Trial ongoing in Canada
  - Single arm, open label
  - N=8
  - 6 weeks of dosing
    - 680mg weekly x 2 doses
    - 340mg weekly x 4 doses

ASCEND-GO 2

- **Phase 2b**
  - Trial ongoing in USA and Europe
  - Double masked, placebo controlled, randomized
  - N=77
  - 3 drug arms vs placebo
  - 12 weeks of dosing

ASCEND-GO 1: Phase 2a study design



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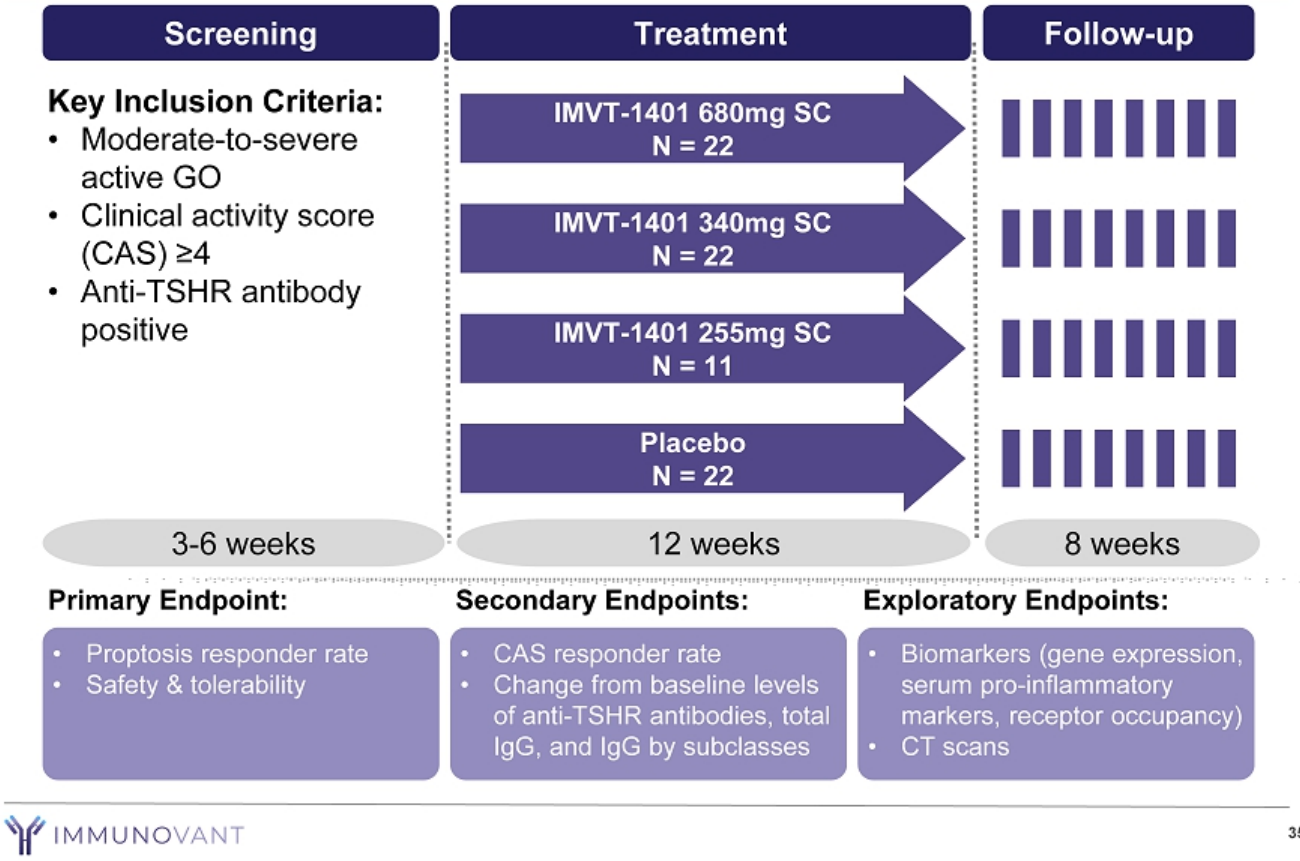
ASCEND-GO 2: Phase 2b study design



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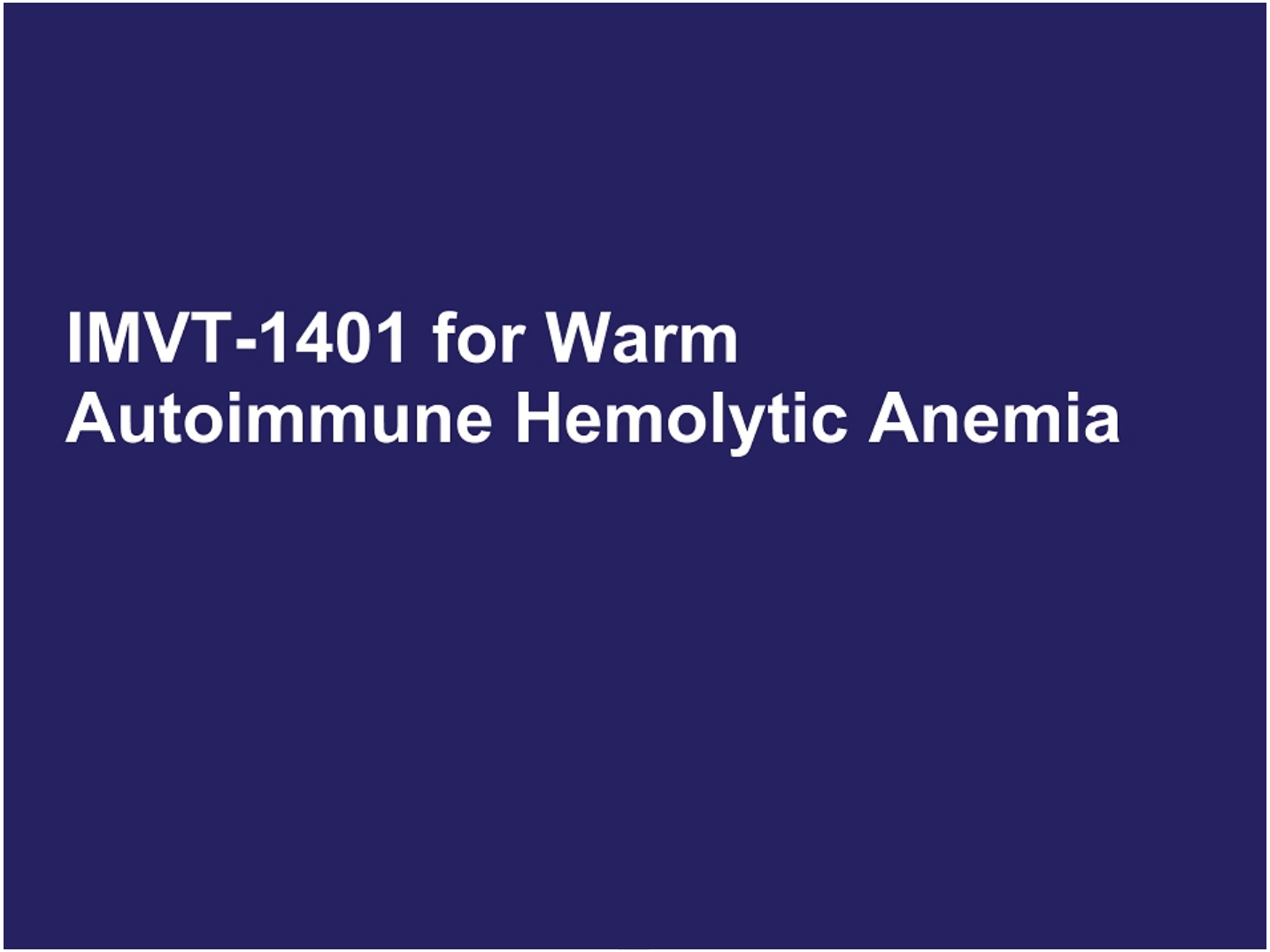
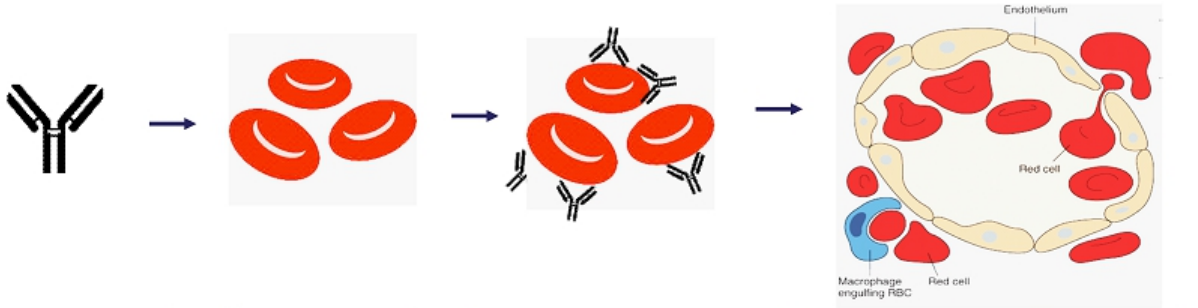

IMVT-1401 for Warm
Autoimmune Hemolytic Anemia

Warm Autoimmune Hemolytic Anemia overview



Anti-red blood cell autoantibody → **Red blood cells (RBCs)** → **Anti-RBC autoantibodies bind to RBCs** → **Antibody-coated RBCs undergo extravascular hemolysis**

- Blood disorder marked by red blood cell destruction
- Estimated prevalence of 42,000 patients in US and 66,000 patients in EU[1]
- Presentation typically non-specific and occurs over several weeks to months
  - Fatigue, weakness, skin pallor, shortness of breath
- Severe cases can be fatal[2]

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1. Park S.H. Diagnosis and treatment of autoimmune hemolytic anemia: classic approach and recent advances. Blood Res., 2016
2. Roumier M., et al. Characteristics and outcome of warm autoimmune hemolytic anemia in adults: new insights based on a single-center experience with 60 patients. Am J Hematol., 2014

Limited options for treating WAIHA

Current treatment paradigm[1,2]

1st Line	2nd Line	3rd Line	4th Line
• Corticosteroids • RBC transfusion	• Immunosuppressive agents	• Rituximab (off-label)	• Splenectomy

Unmet need

- Currently no FDA-approved therapies for WAIHA
- Only one-third of all patients maintain sustained disease control once steroids are discontinued
  - Majority of patients will require either long-term steroid treatment or additional therapies[1]
- No clear guidelines on choice of treatment in patients failing treatment with corticosteroids
- RBC transfusions are indicated in patients who require immediate stabilization, despite the fact that autoantibodies present in WAIHA patients may react against RBCs in the transfusion product[1,2]

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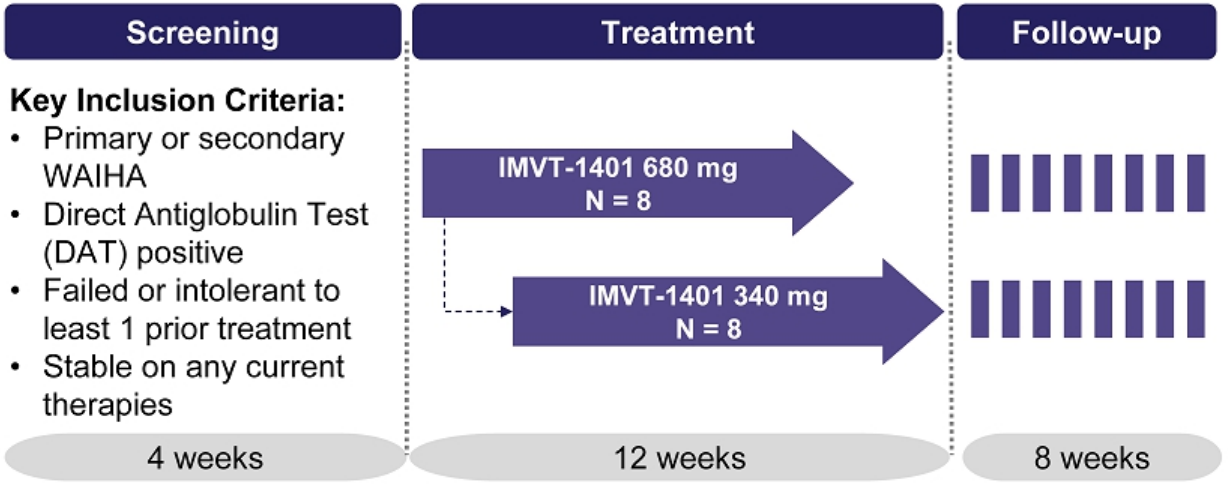
1. Salama A. Treatment Options for Primary Autoimmune Hemolytic Anemia: A Short Comprehensive Review. Transfus Med Hemother., 2015
2. Park S.H. Diagnosis and treatment of autoimmune hemolytic anemia: classic approach and recent advances. Blood Res., 2016

ASCEND-WAIHA: Phase 2 study design



Primary Endpoint:

- Hemoglobin response rate*
- Safety & Tolerability

Secondary Endpoints:

- Change in hemoglobin, LDH, bilirubin, & haptoglobin
- Time to response
- QOL measures
- PK/PD
- Anti-drug antibody levels

Exploratory Endpoints:

- Biomarkers (gene expression, serum pro-inflammatory markers, receptor occupancy)

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* Defined as hemoglobin level ≥10 g/dL with at least a ≥2 g/dL increase from baseline

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Immunovant Recap

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Our vision: Normal lives for patients with autoimmune diseases

Our asset: IMVT-1401, a novel, fully human monoclonal antibody inhibiting FcRn-mediated recycling of IgG

Our strategy for IMVT-1401:

- **Be best-in-class** in target indications where anti-FcRn mechanism has already established clinical proof-of-concept
- **Be first-in-class** in target indications with clear biologic rationale and no known in-class competition

Our near-term value drivers: Four anticipated data readouts over the next 20 months

IMVT-1401: Multiple anticipated near-term value inflection points

MG

- ☑ Phase 2a open for enrollment
  - ☐ Top-line results of Phase 2a study expected in 1H 2020
- ☐ Pivotal Phase 3 study initiation expected in 2020

GO

- ☑ Phase 2a open for enrollment
  - ☐ Initial results of Phase 2a study in Q1 2020
- ☑ Phase 2b proof-of-concept study open for enrollment
  - ☐ Top-line results of Phase 2b study expected in early 2021

WAIHA

- ☐ IND submission expected in 2H 2019


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