UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, DC 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of report (Date of earliest event reported): March 30, 2022

Roivant Sciences Ltd.
(Exact Name of Registrant as Specified in Charter)

Bermuda	**001-40782**	**98-1173944**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(I.R.S. Employer Identification No.)**

Suite 1, 3rd Floor
11-12 St. James's Square
London SW1Y 4LB
United Kingdom
(Address of Principal Executive Offices, and Zip Code)

+44 207 400 3347
Registrant's Telephone Number, Including Area Code

Not Applicable
(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (*see* General Instruction A.2. below):

☐ Written communication pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Shares, $0.0000000341740141 per share	**ROIV**	**The Nasdaq Stock Market LLC**
Redeemable Warrants, each whole warrant exercisable for one Common Share at an exercise price of $11.50 per share	**ROIVW**	**The Nasdaq Stock Market LLC**

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (17 CFR §230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (17 CFR §240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☒

Item 7.01. Regulation FD Disclosure.

On March 30, 2022, the Company's subsidiary, Immunovant, Inc., will host a pre-announced R&D event via webcast. A copy of the presentation to be used during the webcast is attached hereto as Exhibit 99.1 and is incorporated herein by reference.

The information furnished under this Item 7.01, including Exhibit 99.1, shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended, or subject to the liabilities of that section or Sections 11 and 12(a)(2) of the Securities Act of 1933, as amended. The information shall not be deemed incorporated by reference into any other filing with the Securities and Exchange Commission made by the Company, regardless of any general incorporation language in such filing.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits.

Exhibit No.	Description of Exhibit
99.1	Presentation, dated March 30, 2022.
104	Cover Page Interactive Data File (embedded within the Inline XBRL document).

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

ROIVANT SCIENCES LTD.

By: /s/ Matt Maisak
Name: Matt Maisak
Title: Authorized Signatory

Dated: March 30, 2022

Exhibit 99.1

IMMUNOVANT

Immunovant R&D Day

Enabling normal lives for people with autoimmune disease

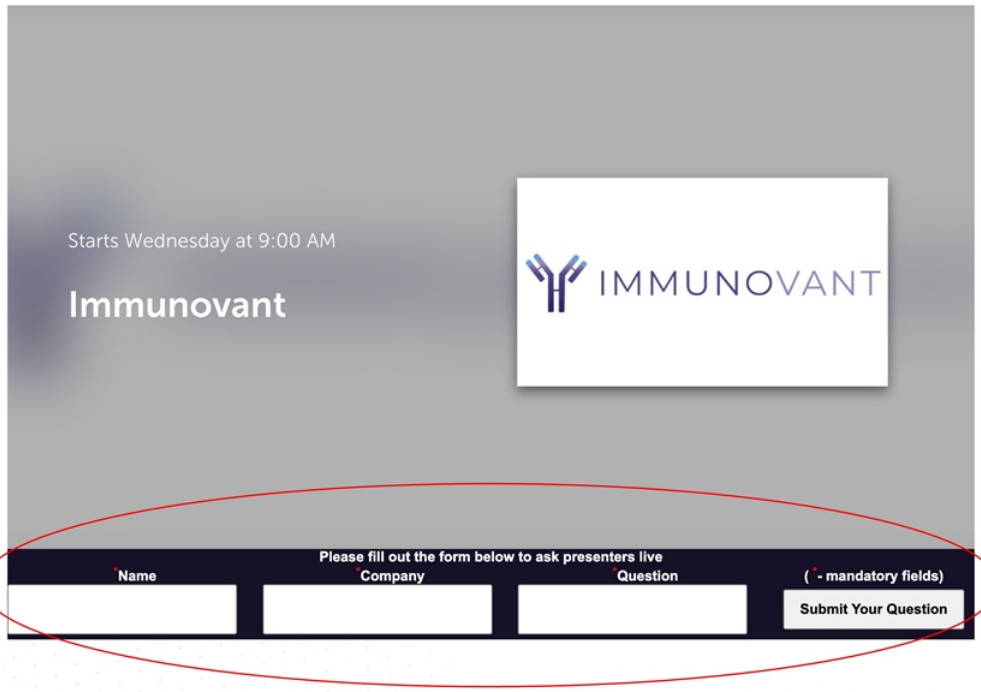
March 30, 2022

Forward-looking statements

This presentation contains forward-looking statements for the purposes of the safe harbor provisions under The Private Securities Litigation Reform Act of 1995 and other federal securities laws. The use of words such as “may,” “might,” “will,” “would,” “should,” “expect,” “believe,” “estimate,” “design,” “plan,” and other similar expressions are intended to identify forward-looking statements. Such forward looking statements include Immunovant’s plan to start a Phase 3 study for batoclimab in myasthenia gravis (MG) in the first half of calendar year 2022 with an expected data readout in 2024, and expectations with respect to the safety and monitoring plan and size of the safety database; Immunovant’s plan to explore in subsequent study periods follow-on treatment with alternative dosing regimens; Immunovant’s plan to develop batoclimab across a broad range of autoimmune indications; Immunovant’s expectations regarding timing, the design and results of clinical trials of its product candidates and indication selections; Immunovant's beliefs regarding its cash runway, and the potential benefits of batoclimab’s unique product attributes. All forward-looking statements are based on estimates and assumptions by Immunovant’s management that, although Immunovant believes to be reasonable, are inherently uncertain. All forward-looking statements are subject to risks and uncertainties that may cause actual results to differ materially from those that Immunovant expected. Such risks and uncertainties include, among others: initial results or other preliminary analyses or results of early clinical trials may not be predictive 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 candidate, including the timing of the commencement of additional clinical trials and resumption of current trials; Immunovant’s scientific approach, clinical trial design, indication selection and general development progress; future clinical trials may not confirm any safety, potency or other product characteristics described or assumed in this presentation; any product candidate that Immunovant develops may not progress through clinical development or receive required regulatory approvals within expected timelines or at all; Immunovant’s product candidate may not be beneficial to patients, or even if approved by regulatory authorities, successfully commercialized; the potential impact of the ongoing COVID-19 pandemic on Immunovant’s clinical development plans and timelines; Immunovant’s business is heavily dependent on the successful development, regulatory approval and commercialization of its sole product candidate, batoclimab; Immunovant is at an early stage in development of batoclimab; and Immunovant will require additional capital to fund its operations and advance batoclimab through clinical development. These and other risks and uncertainties are more fully described in Immunovant’s periodic and other reports filed with the Securities and Exchange Commission (SEC), including in the section titled “Risk Factors” in Immunovant’s most recent Annual Report on Form 10-K, its Form 10-Q filed with the SEC on February 4, 2022, and Immunovant’s subsequent filings with the SEC. Any forward-looking statement speaks only as of the date on which it was made. Immunovant undertakes no obligation to publicly update or revise any forward-looking statement, whether as a result of new information, future events or otherwise.

Q&A housekeeping

You may submit questions through the Q&A function at the bottom of the webcast player. Please see the image here for reference.



Today's agenda

Immunovant and batoclimab
vision and strategy | Pete Salzmann, MD, CEO Immunovant

Thyroid Eye Disease
an exciting opportunity | Bill Macias, MD, CMO Immunovant
- Andrea Kossler[1], MD, FACS, Stanford University School of Medicine
- George Kahaly[2], MD, PhD, Johannes Gutenberg University Medical Center
- Pete Salzmann, MD, CEO Immunovant

Myasthenia Gravis
a multifaceted disease | Pete Salzmann, MD, CEO Immunovant
- Katherine Ruzhansky[3], MD, MS, Medical University of South Carolina
- Nicholas Silvestri[4], MD, FAAN, University of Buffalo

Warm Autoimmune Hemolytic Anemia
opportunity for innovative treatment options | Pete Salzmann, MD, CEO Immunovant
- David Tucker[5] MB ChB, BSc, MD MRCP, FRCPath, Royal Cornwall NHS Hospitals Trust

Cholesterol management
what we know | Bill Macias, MD, CMO Immunovant
- Michael Davidson[6], MD, University of Chicago, Pritzker School of Medicine

Path forward
what comes next | Pete Salzmann, MD, CEO Immunovant

Q&A

Financial Disclosures: **1.** Consultant, Horizon & Immunovant; Research Funds Horizontal Pharmaceuticals, Viridian Pharmaceuticals, VasaraGen Inc. **2.** The Johannes Gutenberg University (JGU) Medical Center, Mainz, Germany (academic institution of George J Kahaly, MD, PhD) has received research-associated funding from the JGU Medical Faculty, AdvanceCor (Germany), Apitope (Belgium), Berlin-Chemie (Germany), Byondis (The Netherlands), GlycoEra (Switzerland), Horizon (USA), Immunovant (USA), ISAR (Germany), Mediomics (USA), Merck (Germany), Novartis (USA), Quidel (USA), River Vision (USA), and Roche (Switzerland). GJK consults for GlycoEra, Immunovant, ISAR, Mediomics, Merck, Novartis, Quidel, & VasaraGen (USA). **3.** Consultant/advisory board for: Alexion, Argenx, Ra/UCB, Immunovant; Current grant/research funding from: Alexion, Ra/UCB, Janssen, Myasthenia Gravis Foundation of America, MGNet **4.** medical advisory boards and speaker for argenx, UCB, advisory boards for Immunovant, Alexion, Biogen, Roche, speaker for Strongbridge/Xeris 5. Advisory board honoraria: Roche, Abbvie, Novartis, Consultant, Immunovant **6.** Consultant, Immunovant

Broad development plan enabled by an exciting mechanism of action, batoclimab's features and a strong balance sheet

01 Batoclimab's unique combination of attributes present a potentially significant commercial opportunity across multiple indications in rare autoimmune diseases

02 Immunovant is well-capitalized with $527M[1] in cash expected to fund its broad development plans into calendar year 2025[2]

03 Immunovant plans to initiate a Phase 3 study of batoclimab in Myasthenia Gravis in the first half of calendar year 2022 – topline results expected in 2024

04 Immunovant remains on track to start three pivotal studies in calendar year 2022[3], and to announce two new indications by August 2022

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1. As of December 31, 2021, per most recent Quarterly Report on Form 10-Q filed with the SEC on February 4, 2022
2. The assumptions upon which we have based our estimates are routinely evaluated and may be subject to change
3. Three pivotal trials include myasthenia gravis and two other indications

Vision and Strategy

Pete Salzmann, MD
Chief Executive Officer

IMMUNOVANT

Our vision:
Normal lives for people with autoimmune disease

Driven by our core values

Love Trailblazing

Bolder, Faster

All Voices

Our focus: unmet needs common to many IgG-mediated autoimmune diseases

Reliable treatment options

- Variable time to response for existing treatments (e.g., older non-steroidal immunosuppressants)
- Challenging safety profile or logistics (e.g., steroids, IVIg)

Tailored treatment options

- Symptoms may wax and wane
- Dosing that can be tailored based on the severity and stage of disease

Simpler route of administration

- Desire to feel like a person, not like a patient
- Considerations for chronic disease management

Significant improvement in Quality of Life

- Improve symptoms and reduce uncertainty
- More data to guide a proactive approach

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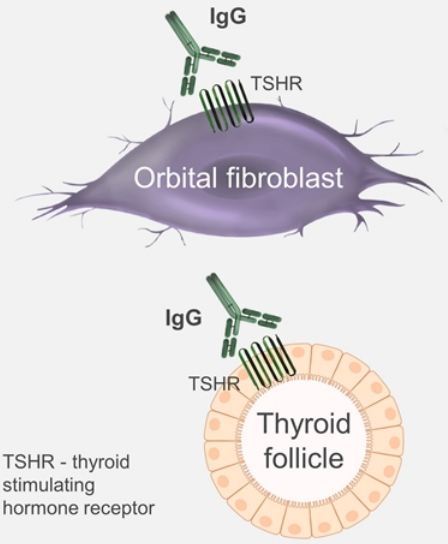
Source: Analysis – Social Listening on MG n=975 / Qualitative research – MG patient journey n=28 / MG Patient Advisory Council n=6 / MG Patient Quantitative Survey (n=50)

IgG antibodies mediate autoimmune disease pathogenesis

- In many autoimmune diseases, IgG antibodies develop that can recognize and bind to normal tissues[1]
- IgG targets may include cell-surface receptors or circulating proteins
- IgG autoantibodies trigger a harmful immune responses resulting in autoimmune symptoms and tissue damage
- **Disease severity may correlate with quantity of pathogenic IgG**

Normal tissues recognized by IgG autoantibodies in Thyroid Eye Disease[1]



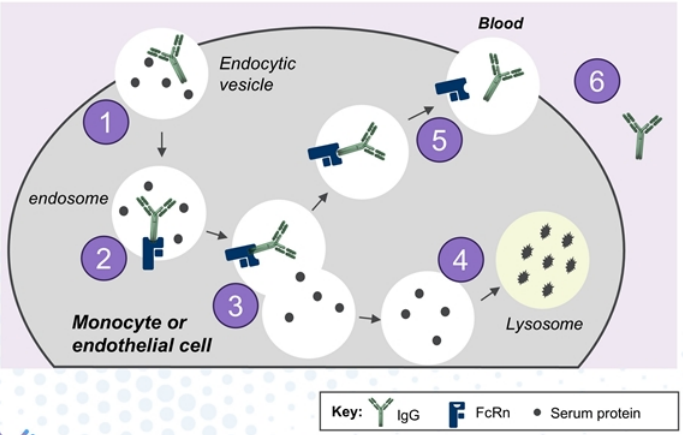
TSHR - thyroid stimulating hormone receptor

1. Kahaly GJ. J Clin Endocrinol Metab. 2020;105(12):3704–20

FcRn promotes recycling of IgG antibodies

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

FcRn maintains levels of IgG in circulation by preventing IgG degradation



FcRn Mechanism of Action

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

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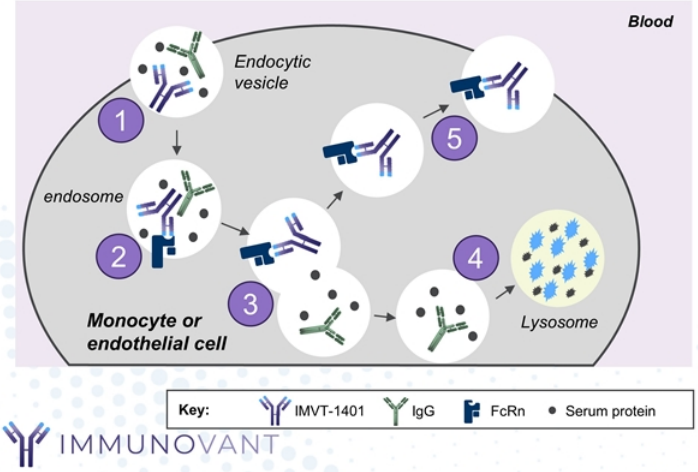
Batoclimab inhibits FcRn, promoting IgG degradation

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

Batoclimab removes pathogenic antibodies by binding to FcRn and promoting IgG degradation



Batoclimab Mechanism of Action

1. IgG and batoclimab are taken up into cells in endocytic vesicles
2. Batoclimab binds to FcRn in endosomes
3. FcRn-batoclimab complexes are sorted from unbound proteins
4. Non-receptor bound IgGs are degraded in lysosomes

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Our opportunity: FcRn inhibition has broad therapeutic potential

17 indications currently announced or in development across the anti-FcRn class

NEUROLOGY

Myasthenia Gravis
Chronic inflammatory demyelinating polyneuropathy
Myositis
Autoimmune encephalitis
Myelin oligodendrocyte glycoprotein antibody disorders (MOG-antibody disorder)

RENAL

Membranous nephropathy
Lupus Nephritis

ENDOCRINOLOGY

Thyroid eye disease

HEMATOLOGY

Warm autoimmune hemolytic anemia
Hemolytic disease of the fetus and newborn
Idiopathic thrombocytopenic purpura

RHEUMATOLOGY

Primary Sjogrens Syndrome
Systemic lupus erythematosus
Rheumatoid arthritis

DERMATOLOGY

Bullous pemphigoid
Pemphigus foliaceus
Pemphigus vulgaris
Cutaneous lupus erythematosus

Potential for anti-FcRn technology to help a broad range of people impacted by autoimmune disease

Estimated number of people with autoimmune diseases* driven by pathogenic IgG

EXPANDED OPPORTUNITY in US and EU

2.3M+*
PEOPLE

Additional, potentially IgG-mediated autoimmune diseases

*Note: Prevalence estimates for autoimmune diseases are illustrative, and are not necessarily indications Immunovant will pursue (supporting data on file at Immunovant). MG: Myasthenia Gravis; WAIHA: Warm Autoimmune Hemolytic Anemia; TED: Thyroid Eye Disease; Additional IgG-mediated autoimmune diseases include ITP: Idiopathic Thrombocytopenic Purpura; PV: Pemphigus Vulgaris; CIDP: Chronic Inflammatory Demyelinating Polyneuropathy; BP: Bullous Pemphigoid; PF: Pemphigus Foliaceus; AIE: Autoimmune Encephalitis LGI1+; MOG: Myelin oligodendrocyte glycoprotein antibody disorder; pSS: Primary Sjögren's Syndrome; SLE: Systemic Lupus Erythematosus; HDFN: Hemolytic Disease of the Fetus and Newborn; RA: Rheumatoid Arthritis; LN: Lupus Nephritis; CLE: Cutaneous Lupus Erythematosus
Europe includes all EU countries, the UK and Switzerland

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Batoclimab has a potentially unique combination of attributes within the anti-FcRn class to address unmet patient needs

Batoclimab

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

Demonstrated rapid and deep IgG reduction in studies to-date with subcutaneous injection

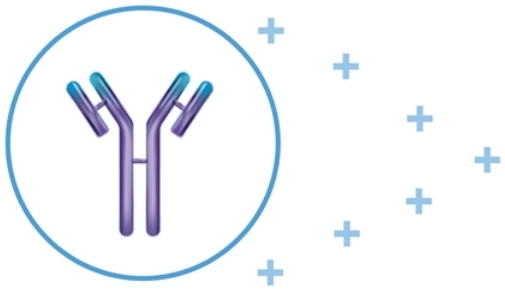
Tailored dosing to address varying symptom severity across indications and stage of disease

- Maximize IgG suppression initially
- Lower chronic doses when less IgG suppression needed
- Manage analyte changes

Simple, subcutaneous injection that will enable self-administration at home

Pioneering anti-FcRn technology to meaningfully advance the quality of care for people living with autoimmune diseases

Batoclimab



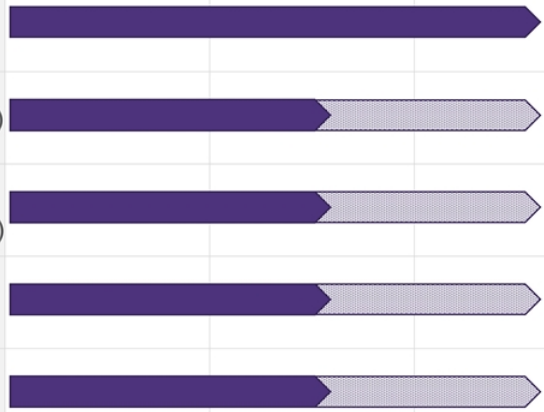
Novel, fully human, monoclonal antibody inhibiting FcRn-mediated recycling of IgG

Intentionally designed to deliver ***potent IgG reductions*** via ***a standard sub-cutaneous injection*** for ***tailored & sustained disease control***

Pursuing a broad development program with batoclimab

$527M[1] in cash expected to fund Immunovant's operating plans into calendar year 2025

Target Indication	Phase 1	Phase 2	Phase 3	Anticipated Milestones
Myasthenia Gravis (MG)				Phase 3 initiation planned in first half of 2022; top line results expected in 2024
Thyroid Eye Disease (TED)				Two of these four indications will be initiated as pivotal trials for a total of three pivotal trials to begin in 2022
Warm Autoimmune Hemolytic Anemia (WAIHA)				
Indication 4*				
Indication 5*				



***Two new indications to be announced by August 2022**

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1. As of December 31, 2021, per most recent Quarterly Report on Form 10-Q filed with the SEC on February 4, 2022

Thyroid Eye Disease (TED)

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Fireside chat on

Thyroid Eye Disease

Bill Macias, MD
Chief Medical Officer, Immunovant

Andrea Kossler, MD, FACS
Director, Oculoplastic Surgery & Orbital Oncology
Assistant Professor of Ophthalmology
Byers Eye Institute @ Stanford University

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Thyroid Eye Disease

Pete Salzmann, MD
Chief Executive Officer, Immunovant

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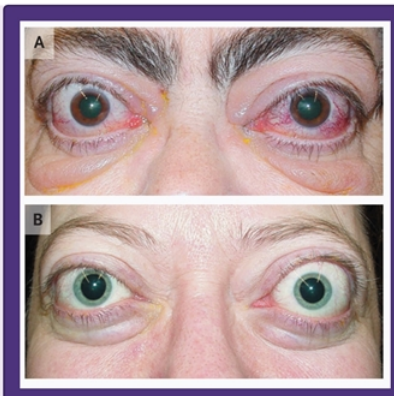
Thyroid eye disease presents with a variety of clinical symptoms

UNDERSTANDING TED:

- Also referred to as Graves' Ophthalmopathy or Graves' Orbitopathy (GO) due to close temporal relationship with Graves' Disease
- Progressive disease marked by inflammation that can lead to fibrosis
- Clinical features are variable, including but not limited to:[1]
  - Eye bulging ("proptosis")
  - Eye pain
  - Double vision ("diplopia")
  - Swollen/red eyes
  - Impaired visual ability
- May become sight-threatening if under-treated[2]
- Beyond IV teprotumumab, disease-modifying treatments are currently limited

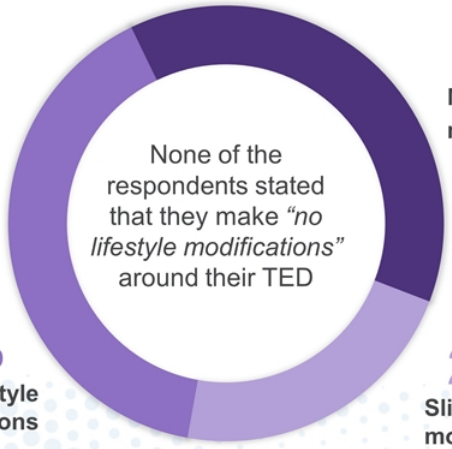


Bahn, 2010
Figure 1. Patients with Thyroid Eye Disease
Panel A shows a 59-year-old woman with excess proptosis, moderate eyelid edema, and erythema with moderate eyelid retraction affecting all four eyelids. Conjunctival chemosis (edema) and erythema with bilateral edema of the caruncles, with prolapse 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.

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

Patients with active TED report a substantial impact on their lifestyle and work-life

Extent of lifestyle modifications



34%
Major lifestyle modifications

46%
Moderate lifestyle modifications

20%
Slight lifestyle modifications

>75% report the impact of disease on their work-life as very challenging

"I cannot bear very bright light which makes it very difficult for me to work on screens"

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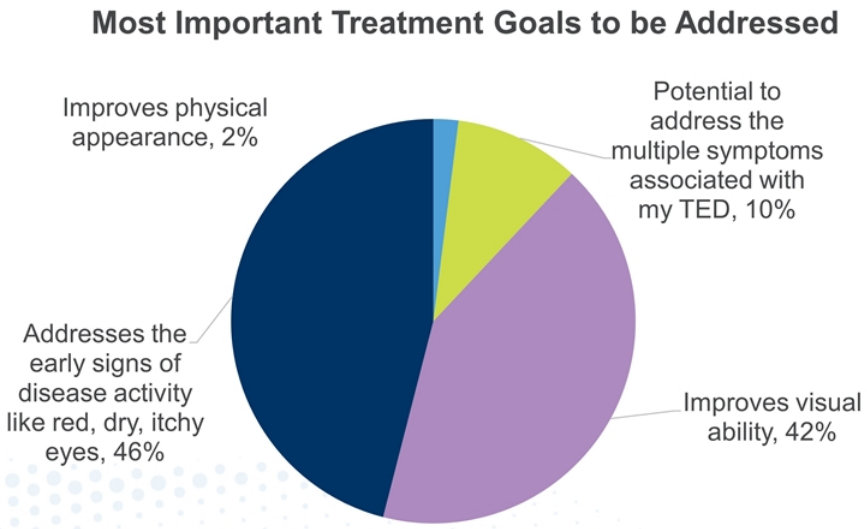
Source: TED Patient Quantitative Survey (n=50) by Immunovant, 2022

Not surprisingly for a heterogeneous disease, people with TED prioritize different treatment goals



Source: TED Patient Quantitative Survey (n=50) by Immunovant, 2022

Thyroid Eye Disease

Mechanism of Action

George J. Kahaly, MD, PhD
Professor of Medicine and Endocrinology/Metabolism
Johannes Gutenberg University (JGU) Medical Center
Department of Medicine I
ORPHAN Disease Center for Graves' Orbitopathy
and Autoimmune Polyendocrinopathy

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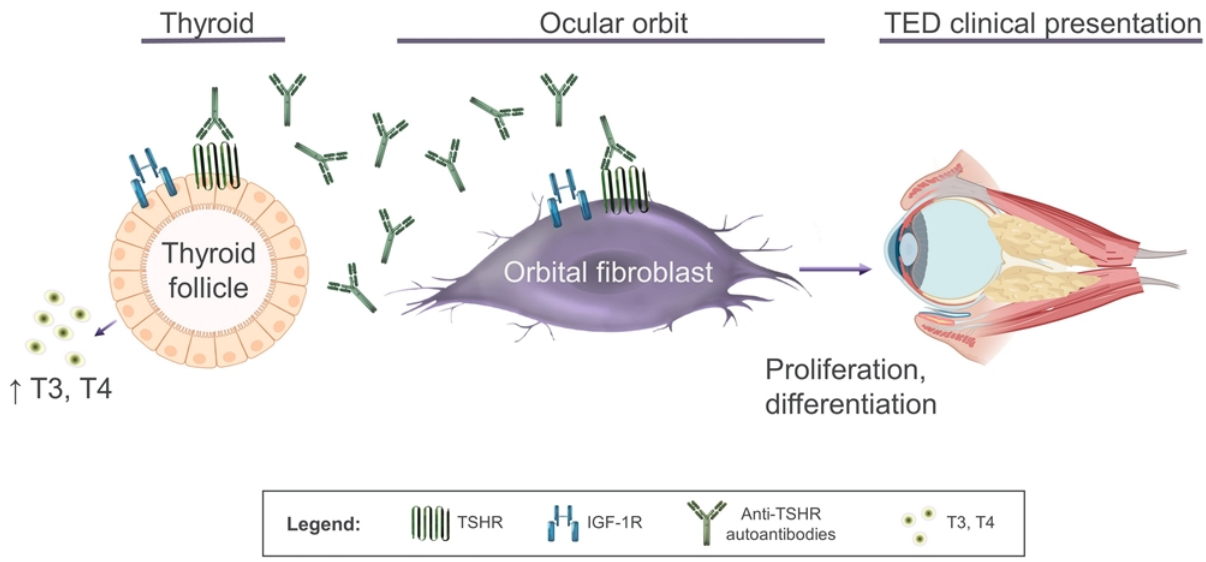
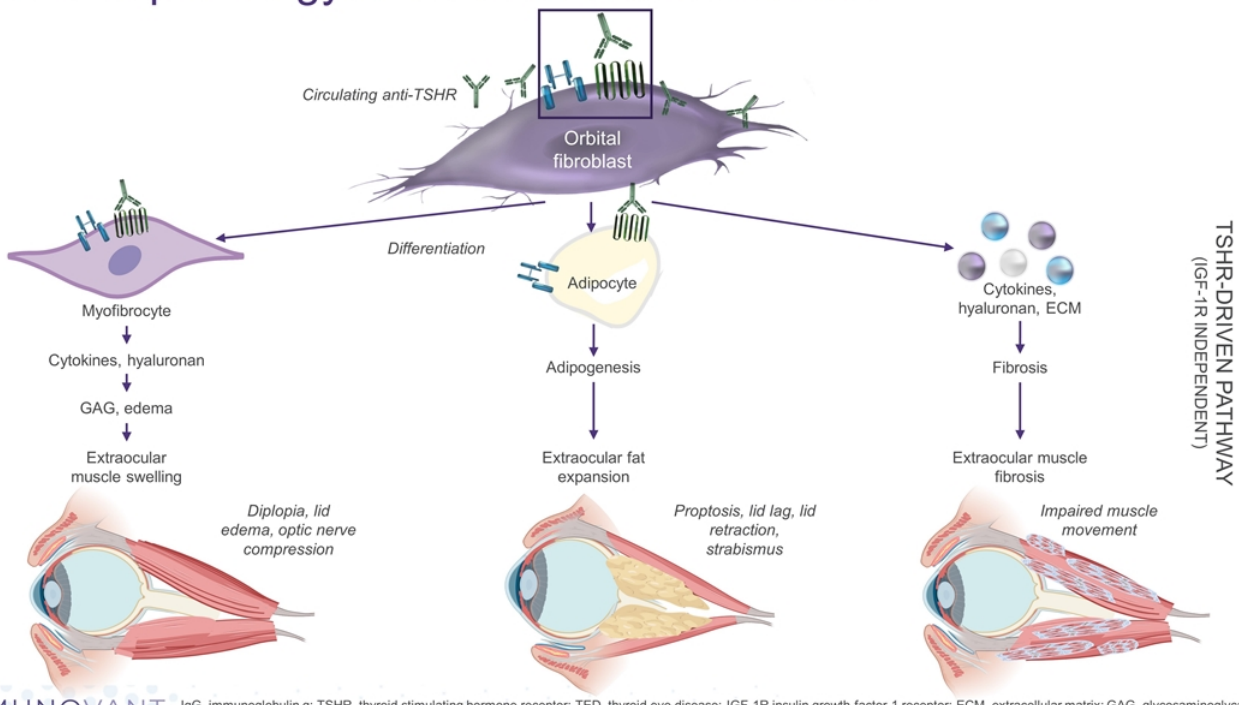
Understanding Thyroid Eye Disease

Anti-TSHR autoantibodies drive the pathogenesis of both Graves' Disease and Graves-Associated Orbitopathy (Thyroid Eye Disease)



IgG, immunoglobulin g; MOA, mechanism of action; TED, thyroid eye disease; TSHR, thyroid stimulating hormone receptor; IGF-1R insulin growth factor-1 receptor A.
Adapted from: Smith TJ, Hegedüs L. *N Engl J Med*. 2016;375(16):1552-1565. Kahaly GJ. *J Clin Endocrinol Metab*. 2020;105(12):3704–20.

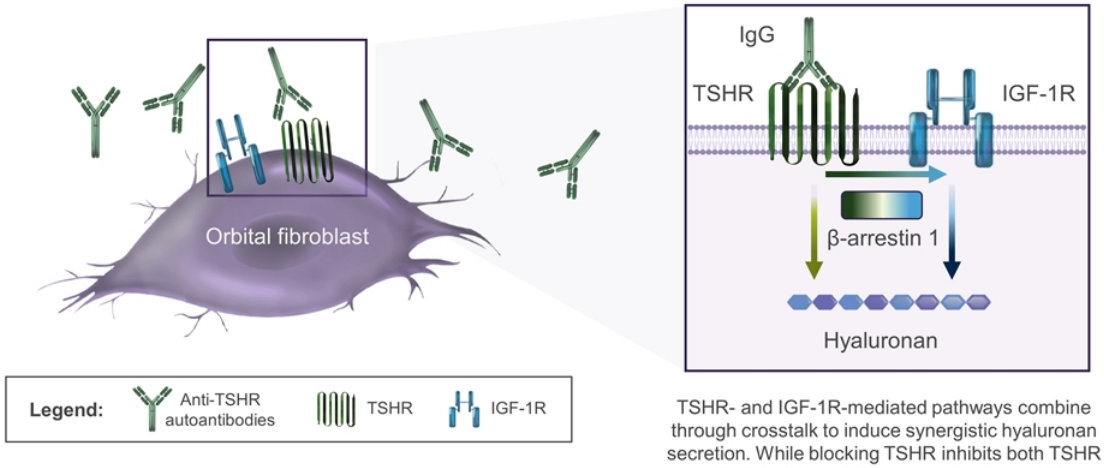
Anti-TSHR autoantibody-mediated activation of orbital fibroblasts drives TED pathology and clinical manifestations



IgG, immunoglobulin g; TSHR, thyroid stimulating hormone receptor; TED, thyroid eye disease; IGF-1R insulin growth factor-1 receptor; ECM, extracellular matrix; GAG, glycosaminoglycans.

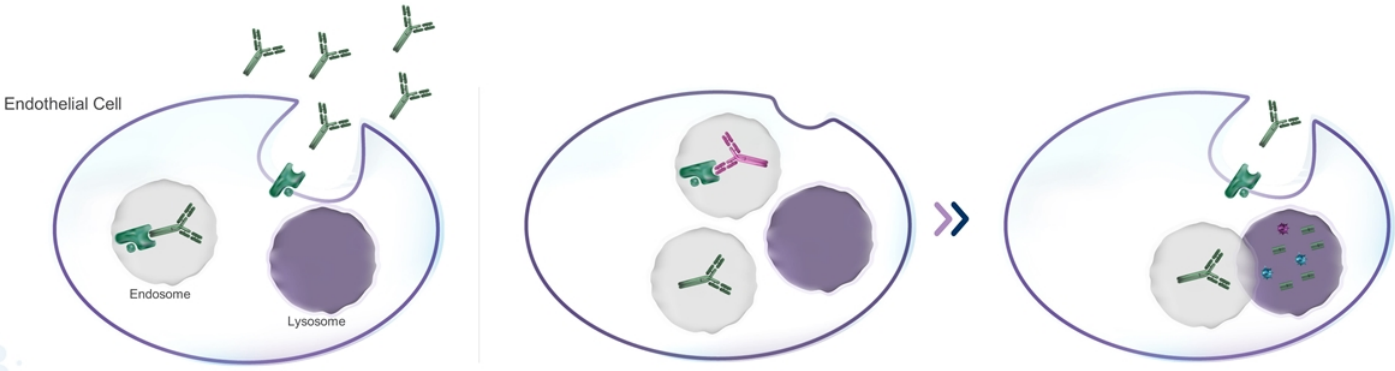
Adapted from: Bahn RS. *Horm Metab Res.* 2015;47(10):773-8. Wiersinga WM. *Lancet Diabetes Endocrinol.* 2017;5(2):134-142. Diana T, et al. *J Endocrinol Invest.* 2021;44(4):703-712. Neumann S, et al. *Eur Thyroid J.* 2020;9(Suppl 1):59-65.

Within the orbit, anti-TSHR autoantibodies bind and activate fibroblasts via crosstalk between TSHR and IGF-1R



TSHR- and IGF-1R-mediated pathways combine through crosstalk to induce synergistic hyaluronan secretion. While blocking TSHR inhibits both TSHR and IGF-1R signaling, partial activation of TSHR may still be possible when IGF-1R is inhibited.

Batoclimab's mechanism of action is designed to inhibit FcRn, potentially fostering the degradation of circulating pathogenic autoantibodies



In the absence of batoclimab, FcRn binds to the anti-TSHR autoantibodies, inhibiting their degradation and returning them into the circulation

With batoclimab, FcRn is blocked from binding to anti-TSHR autoantibodies

Anti-TSHR autoantibodies are transported to the lysosome for degradation decreasing their levels in the circulation

Legend: FcRN | Anti-TSHR autoantibodies | Batoclimab

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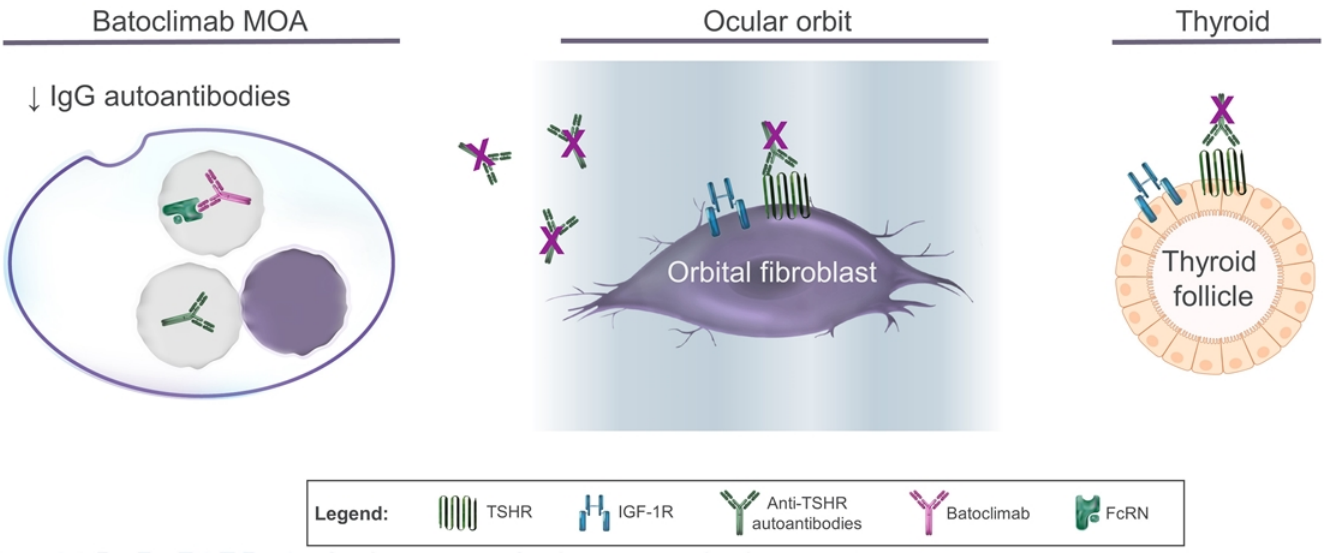
Batoclimab has been observed to reduce pathogenic anti-TSHR autoantibodies that drive Thyroid Eye Disease



IgG, immunoglobulin g; MOA, mechanism of action; TED, thyroid eye disease; TSHR, thyroid stimulating hormone receptor; IGF-1R insulin growth factor-1 receptor.
Adapted from: Smith TJ, Hegedüs L. *N Engl J Med*. 2016;375(16):1552-1565. Kahaly GJ. *J Clin Endocrinol Metab*. 2020;105(12):3704–20.

Thyroid Eye Disease

Pete Salzmann, MD
Chief Executive Officer, Immunovant

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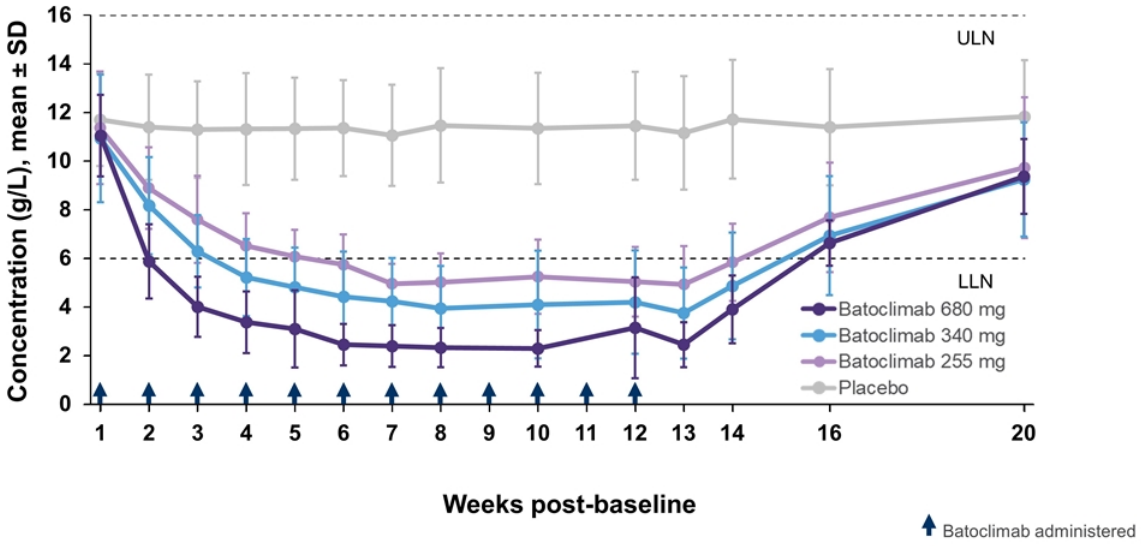
Batoclimab in Thyroid Eye Disease

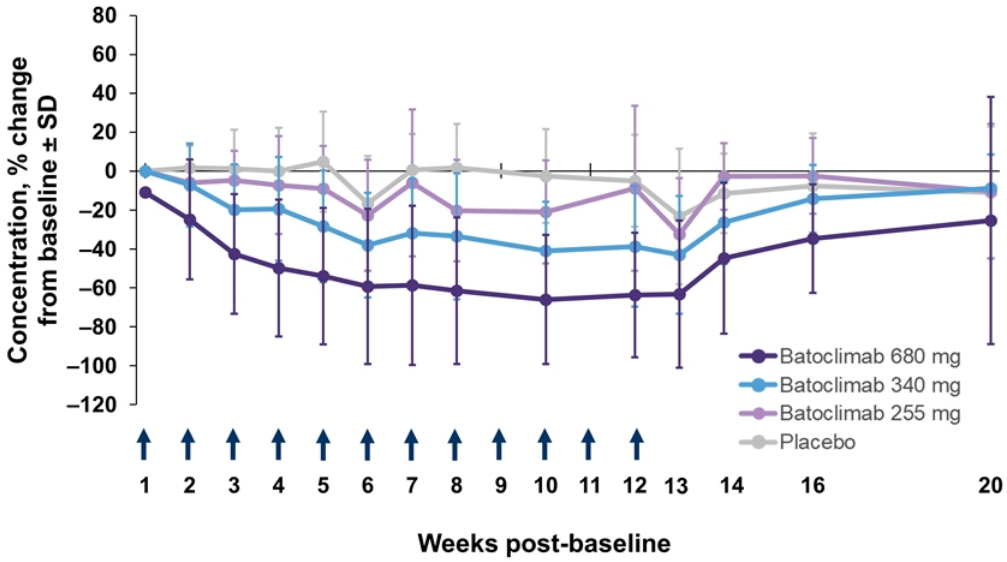
Exploratory analyses used to inform further development because trial voluntarily halted with inconclusive primary endpoint

Observed reductions in total IgG with 12 weeks of batoclimab treatment



LLN: lower limit of normal. **ULN:** upper limit of normal. % changes are not a formal statistical output.
Source: Data on File, Immunovant, Inc.
The efficacy of batoclimab and clinical outcomes were deemed inconclusive, in part, because the study was terminated early.

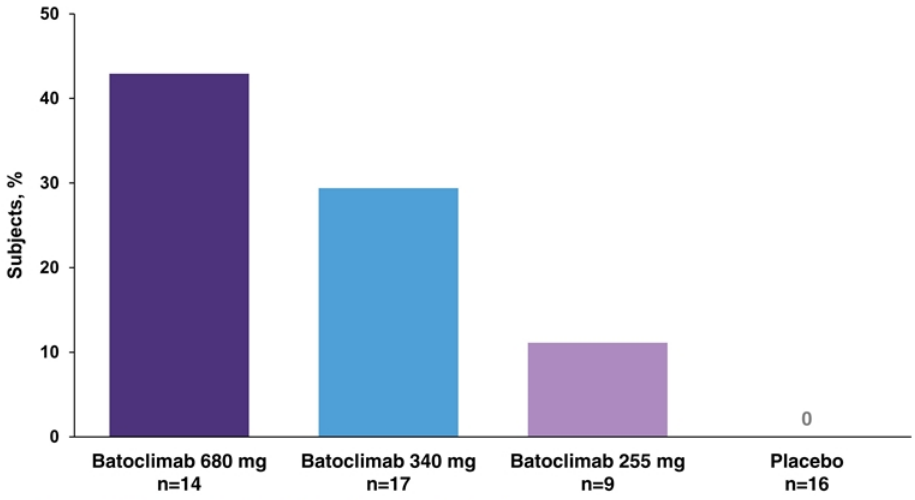
Observed reductions in stimulatory anti-TSHR antibodies with 12 weeks of batoclimab treatment



Percentage of subjects with Stimulatory Anti-TSHR antibody below 140 at week 12[1]	
680 mg	50%
340 mg	15%
255 mg	0%
Placebo	0%

Source: Data on File, Immunovant, Inc.
[1]SRR is the "Sample to Reference Ratio". This cell-based assay readout is the ratio of the sample signal to that of a reference control; avalue less th 140 is considered negative for stimulatory antibody; a value greater than or equal, positive for stimulatory antibody.
The efficacy of batoclimab and clinical outcomes were deemed inconclusive, in part, because the study was terminated early.

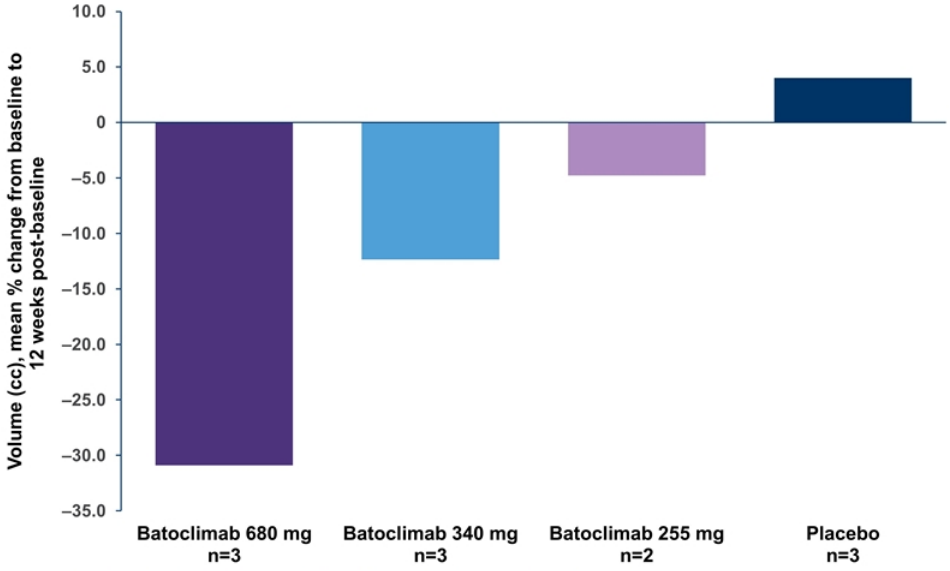
Post-hoc analysis of proptosis response at week 6[1]



Effect size similar at week 12 though confidence intervals wide

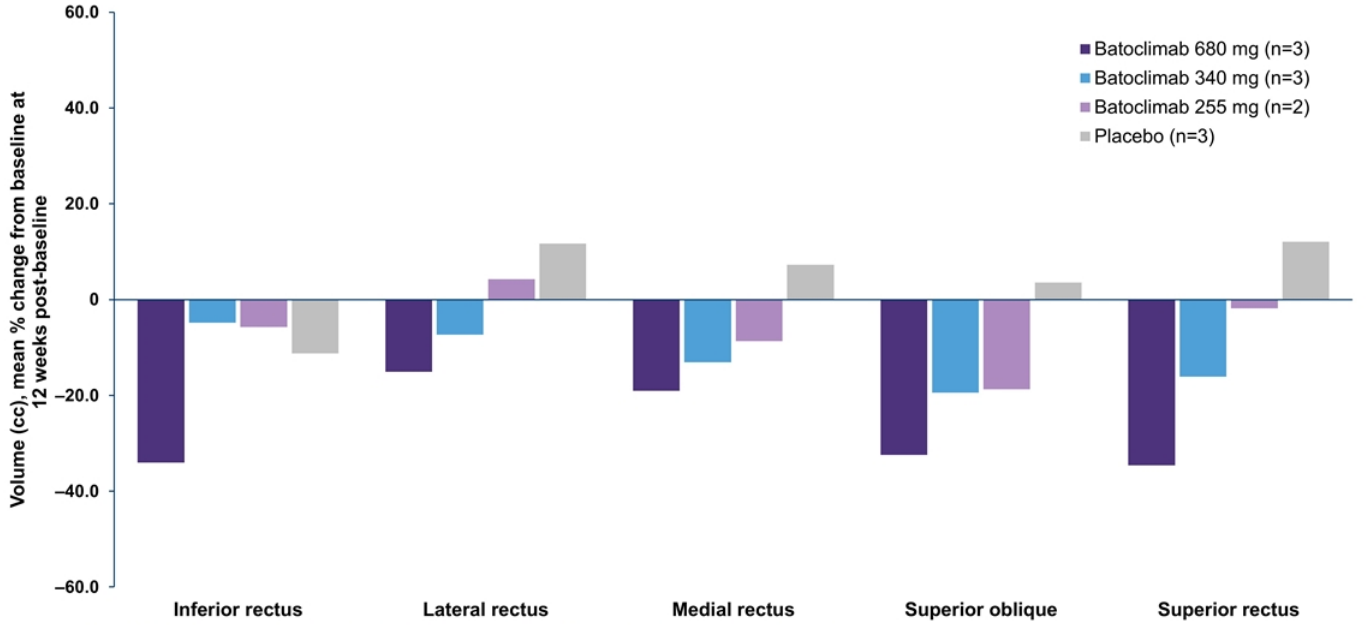
1 Proptosis response defined as proptosis reduction ≥2 mm in study eye, without ≥2 mm increase in non-study eye at same visit. Week 6 data selected as it represents the latest time point at which the largest amount of patient data is available prior to the voluntary pause
Source: Data on File, Immunovant, Inc.
The efficacy of batoclimab and clinical outcomes were deemed inconclusive, in part, because the study was terminated early.

Total muscle volume at 12 weeks post-baseline in all subjects with baseline and end of treatment CT scans



CT: computed tomography.
Represents all patients who had a baseline and week 12 CT scan, a subset of all study participants
Source: Data on File, Immunovant, Inc.
The efficacy of batoclimab and clinical outcomes were deemed inconclusive, in part, because the study was terminated early.

Individual muscle volume at 12 weeks post-baseline in all subjects with baseline and end of treatment CT scans



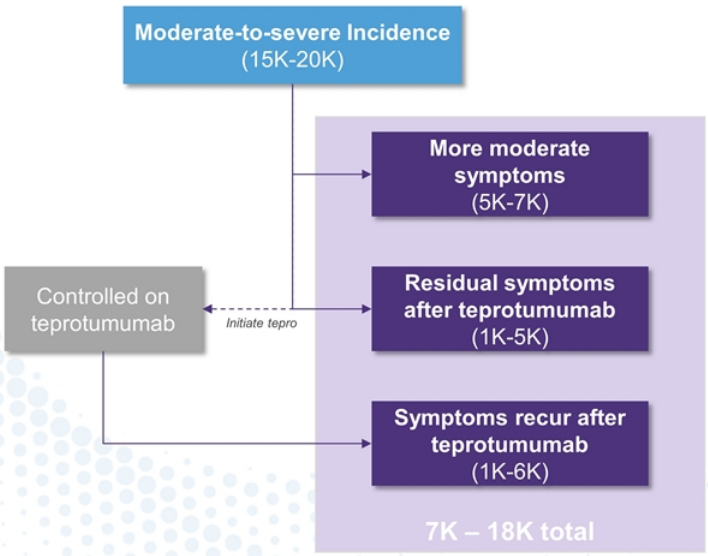
CT: computed tomography. Analysis conducted in the intent to treat population
Represents all patients who had a baseline and week 12 CT scan, a subset of all study participants
Source: Data on File, Immunovant, Inc.
The efficacy of batoclimab and clinical outcomes were deemed inconclusive, in part, because the study was terminated early.

Thyroid Eye Disease

An exciting opportunity

Many patients with Thyroid Eye Disease may benefit from a new therapy

Addressable Population (US)



- 15K-20K newly diagnosed moderate-to-severe TED patients annually[1]
- Of these cases, 1/3 have less severe disease or may benefit from an alternative therapeutic approach[2]
- HCP survey[3] indicates that physicians expect to prescribe teprotumumab to 75% of their TED patients by 2025, up from roughly 30% in March 2022
  - 20%-35% of patients treated with teprotumumab may have residual symptoms warranting treatment[4,5]
  - 25%-40% of patients treated with teprotumumab may experience a disease flare warranting additional TED treatment[6]
- 47-90% of total incidence potentially addressable

Sources: 1. Lazarus JH et al. *Best Practice & Research Clinical Endocrinology & Metabolism*. v26 (2012) 273-279 . 2. HCP Qualitative Research, Immunovant, 2020 3. 2021 Cowen Equity Research, March 2022 - surveyed 25 clinicians who treat 3,000+ patients with TED annually 4. Horizon Therapeutics Investor Presentations. 5. Teprotumumab's US Prescribing Information. 6. Douglas R et al. *American Academy of Ophthalmology*, v129, No. 4,

Thyroid Eye Disease key take-aways

1

TED is a multi-faceted condition – presenting with a range of clinical manifestations and substantial impact on patient functioning and quality of life

2

Data from batoclimab's clinical program are encouraging for continued development in TED

3

Batoclimab represents a potentially new and differentiated approach to treating TED

Myasthenia Gravis (MG)

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Fireside chat on

Myasthenia Gravis

Pete Salzmann, MD
Chief Executive Officer,
Immunovant

Katherine Ruzhansky, MD, MS
Clinical Neurologist, Associate Professor of Neurology and Director of the EMG lab at the Medical University of South Carolina

Nicholas Silvestri, MD, FAAN
Clinical Neurologist, Associate Professor of Neurology, and Assistant Dean for Student and Academic Affairs at the University of Buffalo

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A Beautiful Life

An Unpredictable Journey with Myasthenia Gravis (MG)

Jonni's story

IMMUNOVANT

Myasthenia Gravis – a multifaceted disease

Pete Salzmann, MD
Chief Executive Officer, Immunovant

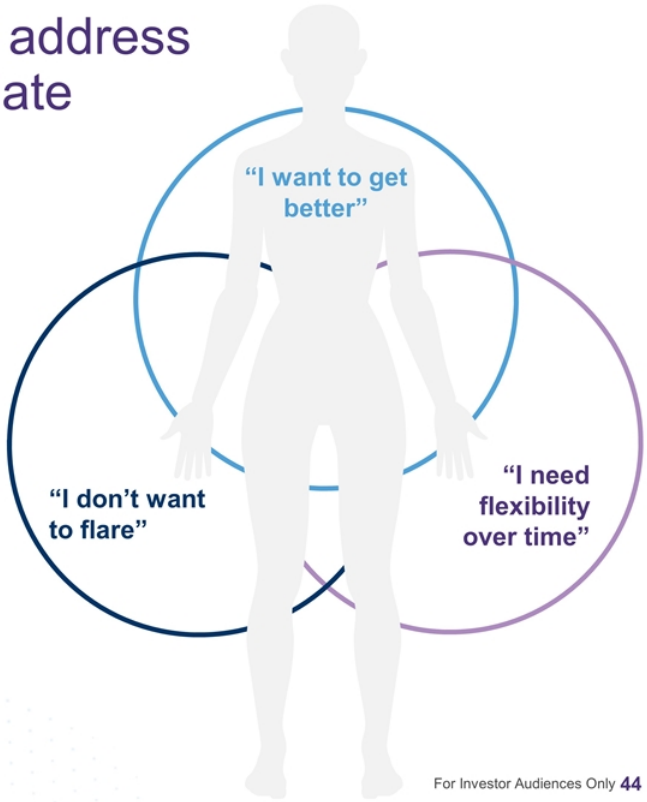
IMMUNOVANT

Phase 3 trial in MG is designed to address unmet patient needs and differentiate batoclimab

Need for significant improvement initially:
High doses included in the induction period to achieve maximum efficacy at the beginning of treatment

Peace of mind over time:
Chronic treatment to provide consistent symptom relief while lowering the dose to maintain efficacy with potentially fewer side effects

Flexible dosing to match disease fluctuations:
Myasthenia gravis waxes and wanes over time; clinicians and patients desire a data-driven approach to optimize care over time



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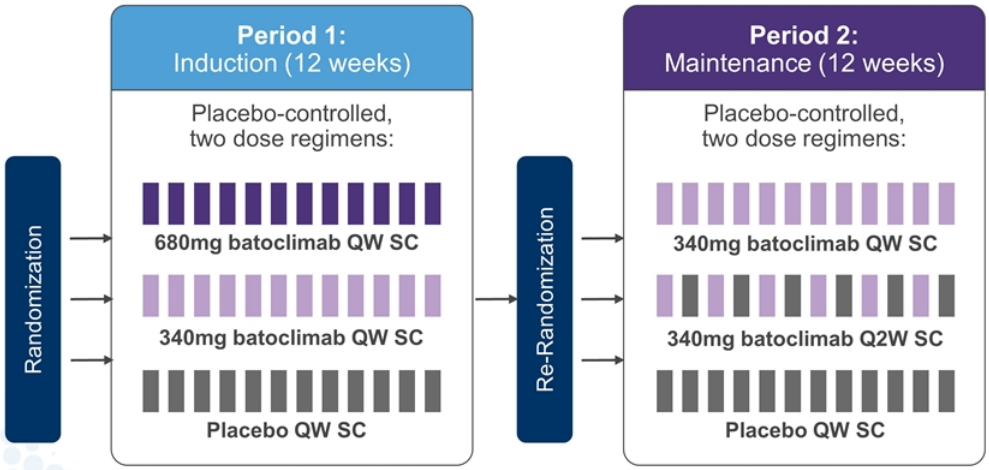
Flexible Phase 3 design that is common in immunology trials but a first for an MG trial

MG Phase 3 trial design (N ~ 200)



*Maximize efficacy through primary endpoint**

Maintain efficacy with anchor dose and lower dose

Primary analysis population:
AChR Ab+

***Primary endpoint:**
change in MG-ADL through 12 weeks

Period 2 followed by **Long-Term Extension** (LTE) study. Rescue therapy available during LTE per protocol.

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QW = weekly; Q2W = bi-weekly; SC = subcutaneous injection; AChR Ab+ = acetylcholine receptor antibody-positive; MG-ADL = Myasthenia Gravis Activities of Daily Living scale

Warm Autoimmune Hemolytic Anemia (wAIHA)

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Disease state of Warm Autoimmune Hemolytic Anemia

David Tucker, MB ChB, BSc, MD MRCP, FRCPath
Consultant Haematologist
Blood Transfusion and Patient Blood Management Lead
NIHR CRN Regional Subspecialty Lead for Malignant and Non-Malignant Haematology
Royal Cornwall NHS Trust

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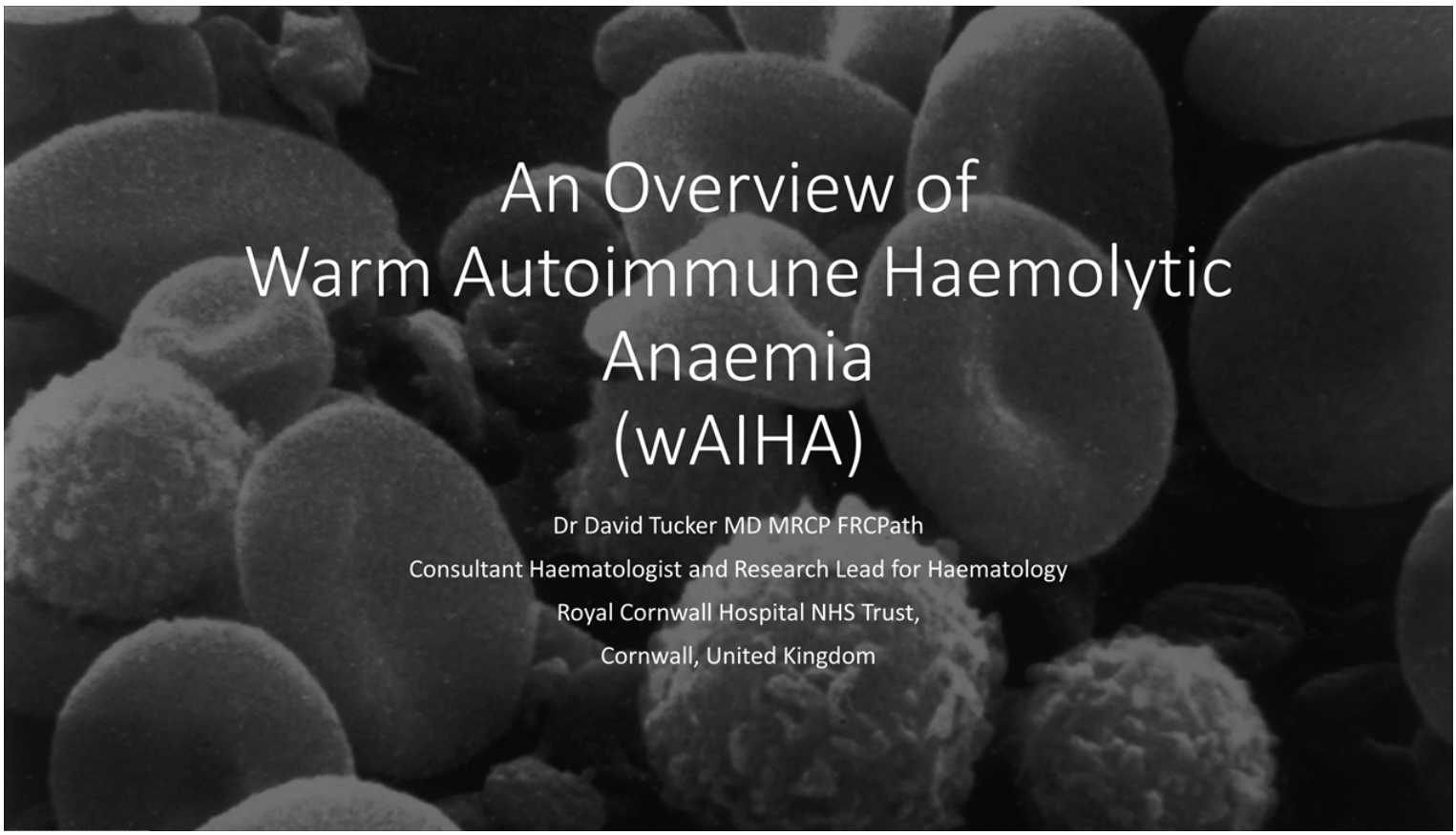


An Overview of Warm Autoimmune Haemolytic Anaemia (wAIHA)

Dr David Tucker MD MRCP FRCPath

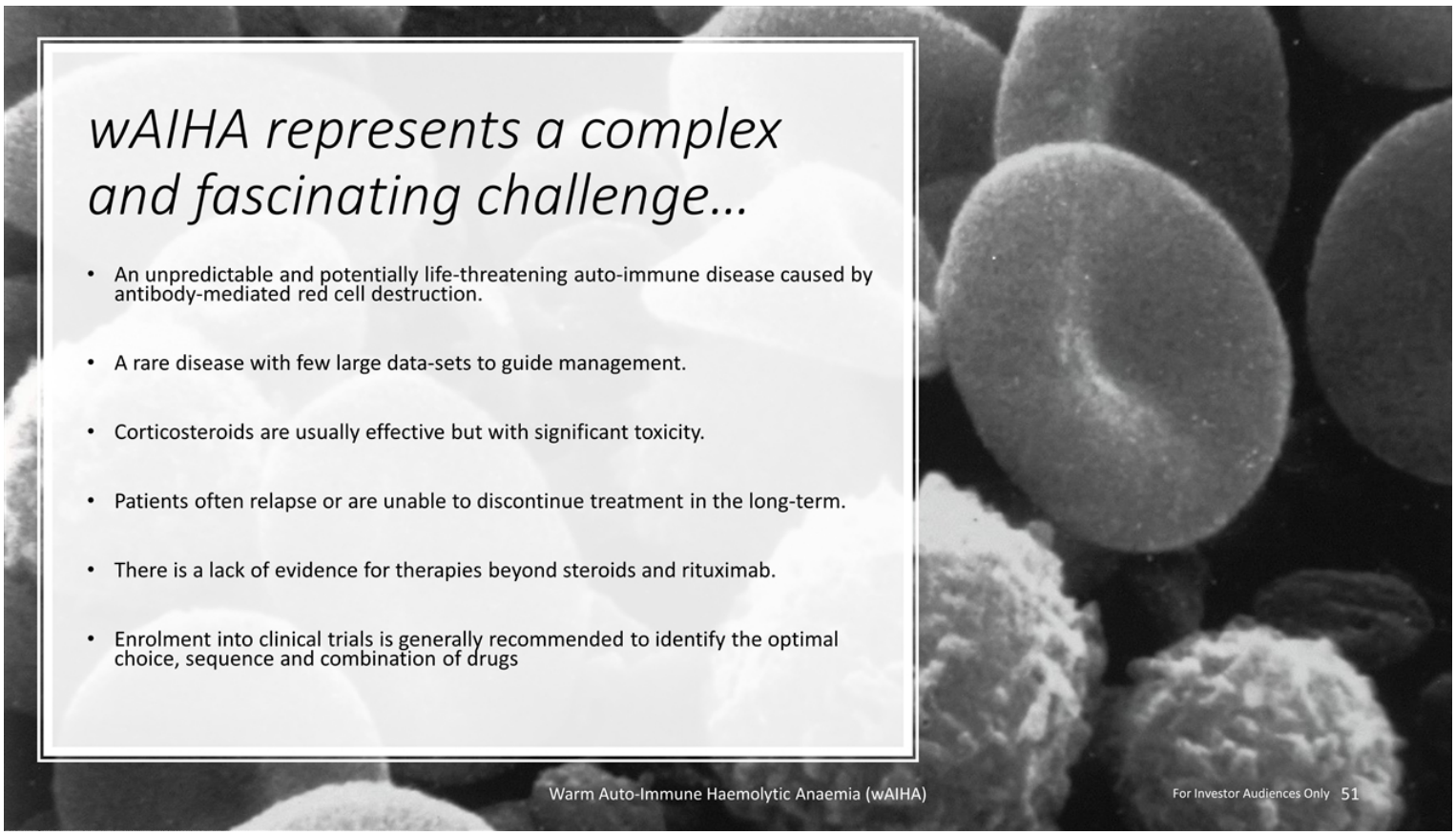
Consultant Haematologist and Research Lead for Haematology

Royal Cornwall Hospital NHS Trust,

Cornwall, United Kingdom

Conflicts of Interest

- Advisory Board – Roche, Abbvie, Novartis
- Conference attendance: Amgen, Takeda



wAIHA represents a complex and fascinating challenge…

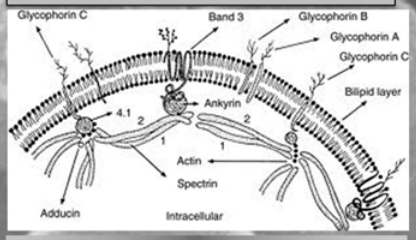
- An unpredictable and potentially life-threatening auto-immune disease caused by antibody-mediated red cell destruction.
- A rare disease with few large data-sets to guide management.
- Corticosteroids are usually effective but with significant toxicity.
- Patients often relapse or are unable to discontinue treatment in the long-term.
- There is a lack of evidence for therapies beyond steroids and rituximab.
- Enrolment into clinical trials is generally recommended to identify the optimal choice, sequence and combination of drugs

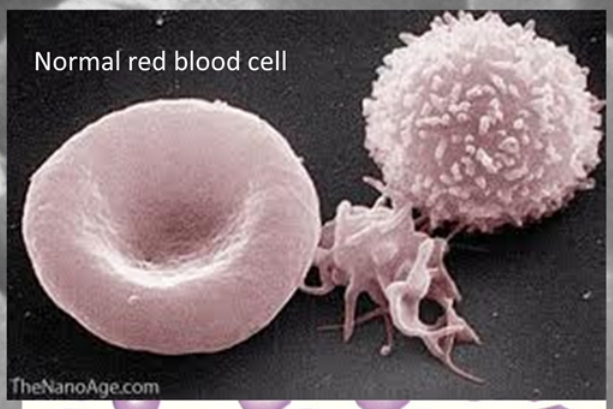
Normal Red Cell Physiology

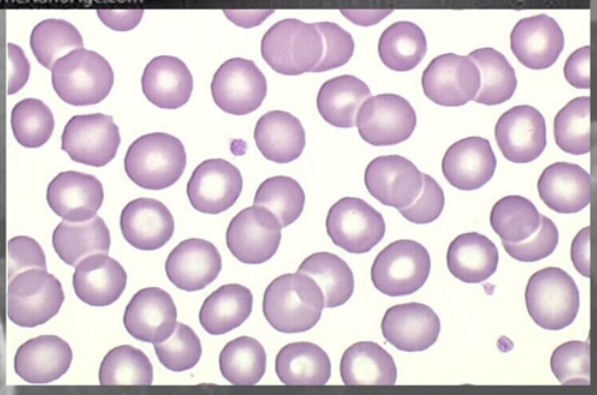
100 – 120 day lifespan

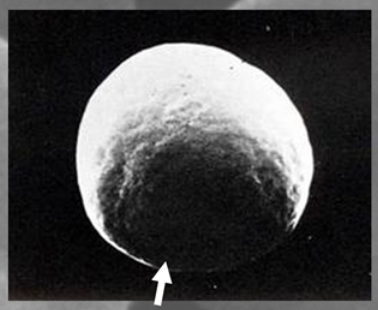
Travels 250km

3 layered elastic membrane allows erythron to “tank-tread”

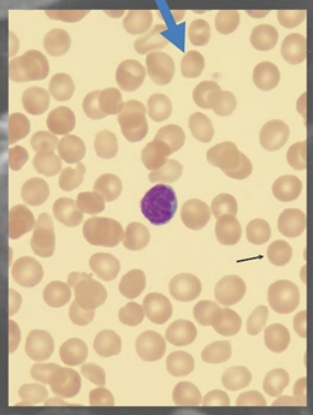




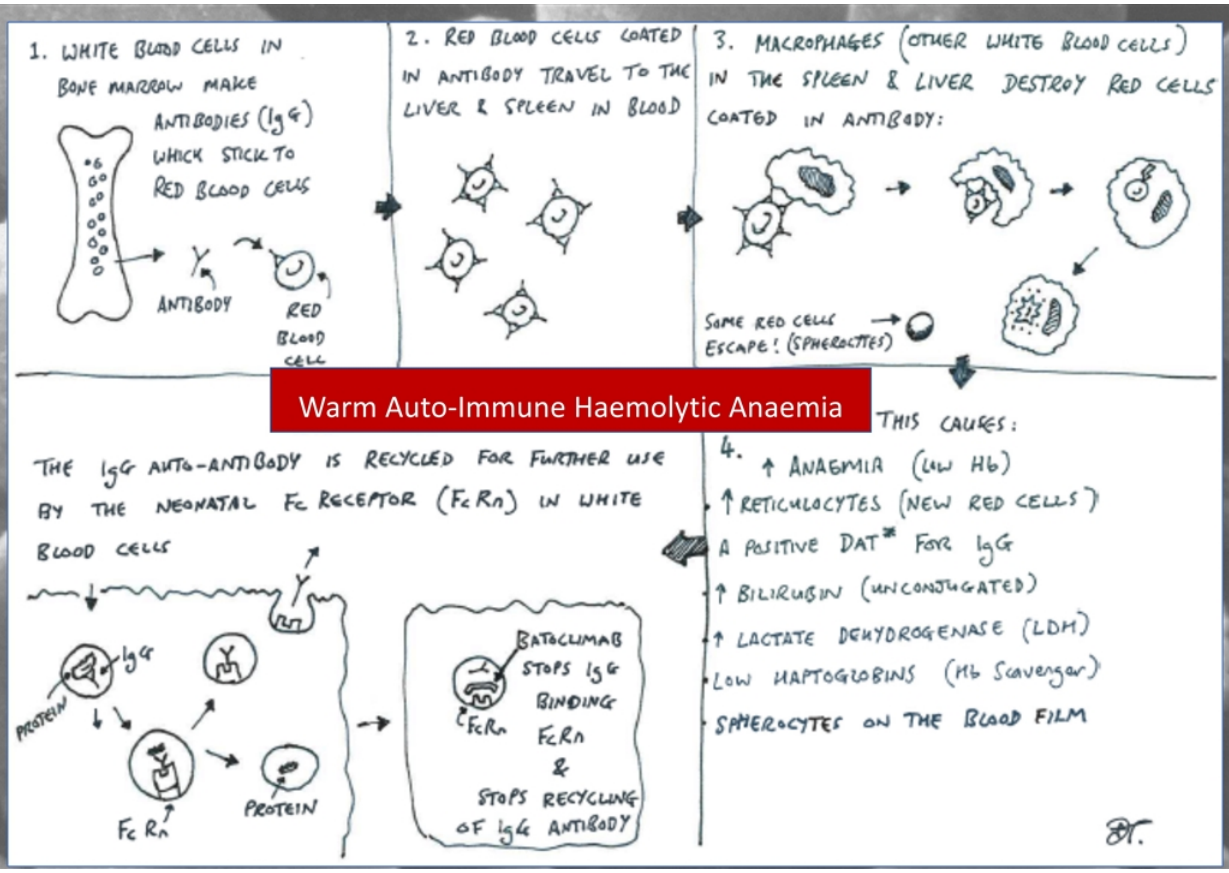




Red cell spherocyte



Warm Auto-Immune Haemolytic Anaemia (wAIHA)



wAIHA – Who is Affected?

Causes

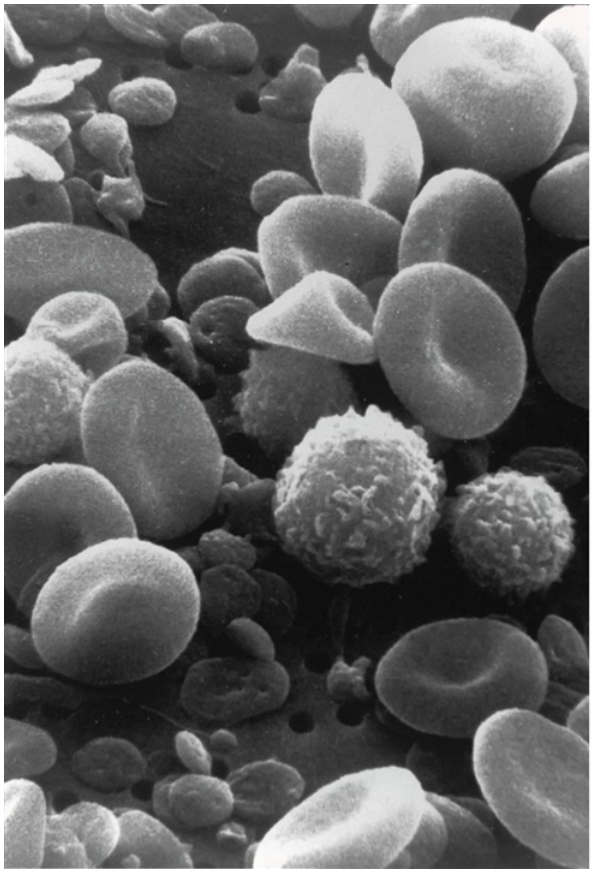
- Primary (Idiopathic) wAIHA
  - (40-60% of cases) (Hill et al. Roumier et al.)
- Secondary wAIHA
  - (50% of cases) (Hill et al. Roumier et al.):
    - Auto-immune diseases e.g. SLE, ITP, Rheumatoid arthritis
    - Lymphoproliferative diseases e.g. CLL, Lymphoma (NHL and HL)
    - Infections (e.g. mycoplasma, EBV)

Epidemiology

- Annual incidence:
  - 1-3/100,000 (Eaton *et al.* 2007)
- Prevalence:
  - ~ 0.17/1000 (Eaton *et al.* 2007)
- Addressable patients (US):
  - approximately 40,000 (*McCrae et al. 2021*)

wAIHA – The Patient Experience

- **Mild / Onset** (Hb >10g/dL): often gradual with mild fatigue, breathlessness and mild icterus (jaundice)
- **Moderate** (Hb 8.0 – 10g/dL): breathlessness and fatigue on moderate exertion (climbing stairs), ankle swelling, palpatations, more obvious jaundice, dark urine.
- **Severe** (Hb 6 - 8.0g/dL): fatigue at rest, breathless on mild exertion (walking room to room), light headed, dizzy on standing.
- **Life-threatening** (Hb < 6.0g/L): unable to mobilise, can precipitate cardiac dysrhythmia, chest pain / cardiac ischaemia.



How do we manage wAIHA?

- Because wAIHA is a rare disease there are few large data sets to guide management which is mainly empirical and based on expert opinion.
- The cornerstone of management is immunosuppression with corticosteroids.
- Historically, the treatment-related mortality rate is 8 to 15%
- The major issue with treatment is that 60% of patients become steroid-dependent. (Roumier et al.)

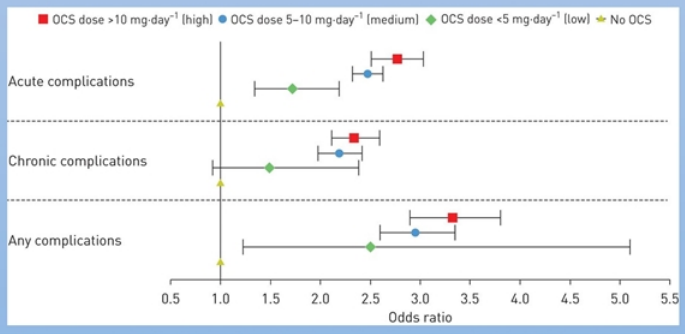
wAIHA Management – 1st line treatment

Corticosteroids

- 80% of patients respond (Allgood *et al.* ; Zupanska *et al.*)
- Only 20% remain in remission when steroids are discontinued (Allgood *et al.* Roumier et al.)
- 40% can maintain control on long-term steroids but side effects are significant (Roumier et al. Hill et al.; Zupanska et al.)

Side Effects of Corticosteroids (Yasir et al. Volmer et al.)

- New diabetes / worsening diabetes (30%)
- Osteoporotic fractures (10%)
- Osteonecrosis of femoral head (~4%)
- 2-4% risk of peptic ulcer disease with steroids (vs 0.1% in general population)(Hill et al.)
- Insomnia, weight gain, mood disturbance
- Reduced quality of life (Sweeney et al. 2016)



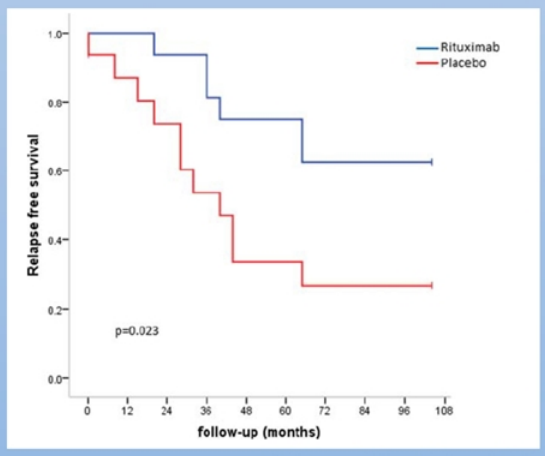
wAIHA Management – beyond 1st line

Rituximab (Monoclonal Antibody) (Maung *et al.* 2013; Michel et al. 2017; Birgens et al.)

- 70% of patients respond (about half of these respond completely);
- Median time to response 3 – 6 weeks;
- Relapses occur in 40-50% of cases after 30 months
- More than half of patients need further therapy
- The long-term remission rates are not well known.

Side Effects

- Infusion-related reactions (>1/10 emc data)
- Neutropenia (15%)
- Infections (Pneumonia, viral reactivation) (12%)
- Hypogammaglobulinaemia (12% emc data)
- JC-virus leukoencephalopathy (<1/10,000 emc data)
- Drug not available world-wide



Michel *et al.* Am. J. Hem. 2016

wAIHA Management – beyond 2nd line

Splenectomy

- 70% response rates (but response unpredictable) (Barcellini et al.)
- Irreversible and not definitively curative
- Risk of severe infection after splenectomy (3 – 5%) which is life-threatening in 50% of cases (Roumier et al.)
- Venous thrombosis risk: ≥2%
- Portal / splenic vein thrombosis risk 8% (Roumier et al. Hill et al.)
- Mortality rate ~ 10% (Balague et al.)

Steroid-sparing Agents

- Azathioprine (60% response rates, but number achieving steroid independence is unclear) (Zupanska et al.)
- Ciclosporin (evidence of efficacy is unclear and limited to case reports) (Hershko et al.)
- Mycophenolate mofetil (MMF) – responses take 3 – 4 months; evidence is from case series / report (Howard et al.)

Chemotherapy

- Few data on dosing / response rates (Moyo et al.)
- Has mutagenic potential.

Haematopoeitic Stem Cell Transplant

- very few data, high risk procedure. (Passweg et al.)

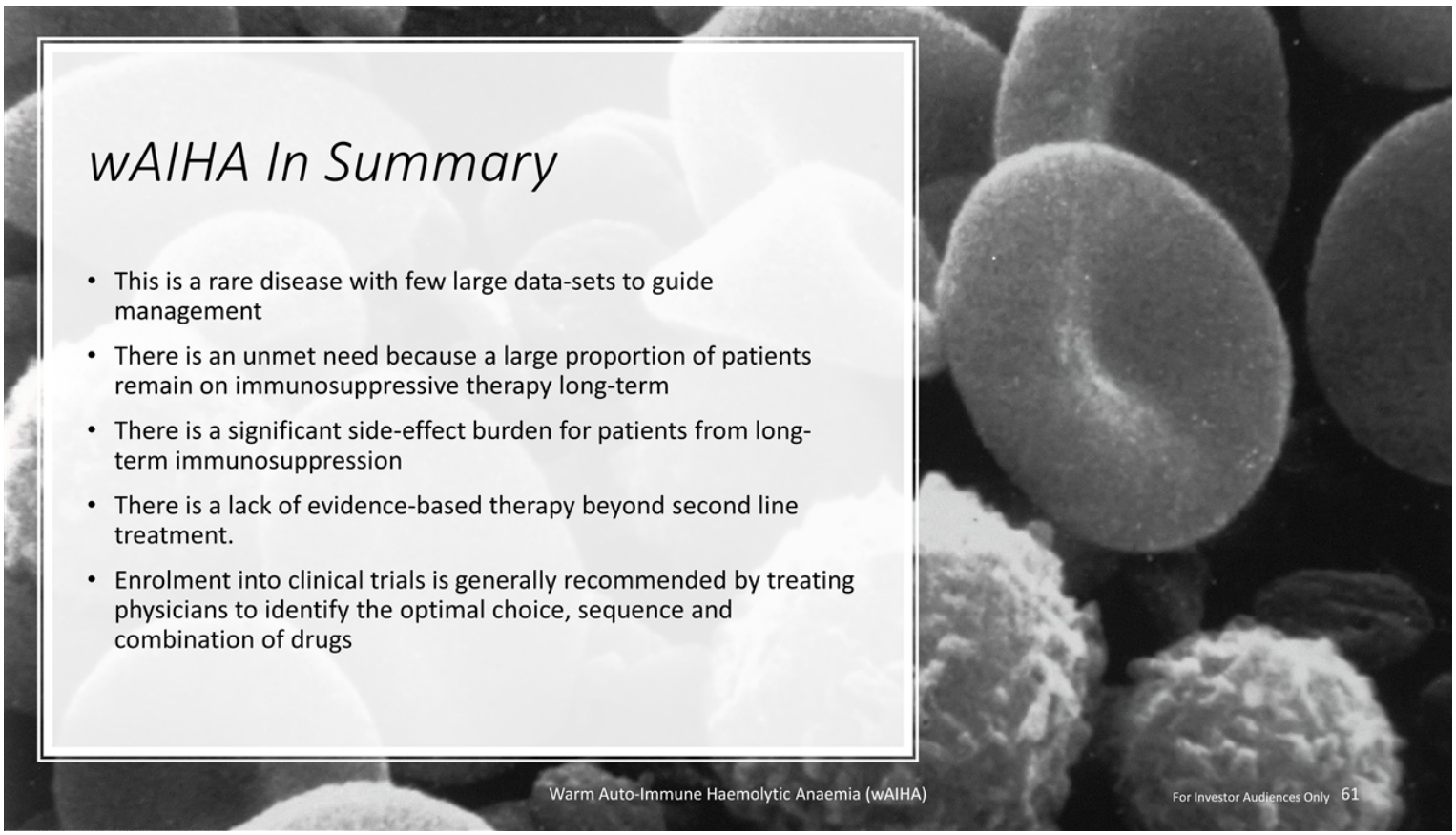
wAIHA – Evidence for a need for new therapeutic options

In studies of patients with wAIHA over 3 – 4 years:

- Less than half (47%) of patients remain in remission off treatment
- 25% of patients remain on low dose steroids
- 28% have ongoing disease and require higher dose steroids or other immunosuppressive drugs(Roumier et al. 2014)

There are serious ongoing complications of uncontrolled wAIHA:

- Risk of deep vein thrombosis (20%) (Hendrick 2003, Roumier et al.)
- Risk of pulmonary embolus (8%) (Roumier et al.)
- Excess mortality (8%) (Roumier et al.)



wAIHA In Summary

- This is a rare disease with few large data-sets to guide management
- There is an unmet need because a large proportion of patients remain on immunosuppressive therapy long-term
- There is a significant side-effect burden for patients from long-term immunosuppression
- There is a lack of evidence-based therapy beyond second line treatment.
- Enrolment into clinical trials is generally recommended by treating physicians to identify the optimal choice, sequence and combination of drugs

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Thank you for listening
Dr David Tucker
Warm Auto-Immune Haemolytic Anaemia (wAIHA)

Latest thinking on cholesterol management

Bill Macias, MD
Chief Medical Officer, Immunovant

Michael Davidson, MD
Professor, Director of the Lipid Clinic
The University of Chicago Pritzker School of Medicine

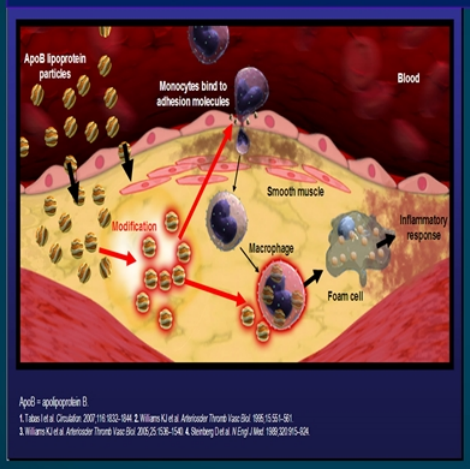
IMMUNOVANT

Relationship between CVD and Hypercholesterolemia

- The causes of cardiovascular disease (CVD) are multifactorial
  - Modifiable risk factors: lifestyle (especially an unhealthy diet) tobacco use and sedentary habits, high blood pressure, diabetes and dyslipidemias
  - Nonmodifiable risk factors: age, gender
- Control of lipid levels is one of the most effective strategies for CVD prevention
- Epidemiologic data have demonstrated the crucial role of dyslipidemia, especially hypercholesterolemia, in the development of CVD
- It is well understood that accumulation of cholesterol-rich low-density lipoprotein (LDL-C) over time leads to formation of lipid-laden foam cells and proliferation of atherosclerotic lesions, increasing the risk of CVD

Agabiti Rosei E, Salvetti M. *High Blood Press Cardiovasc Prev*. 2016;23(3):217-230.

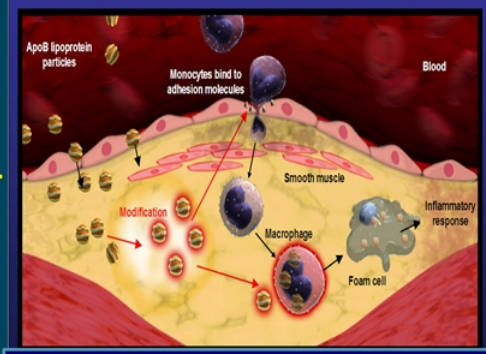
The Theory of Circulating Low Density Lipoproteins (LDL) and Causation of Atherosclerosis



ApoB = apolipoprotein B

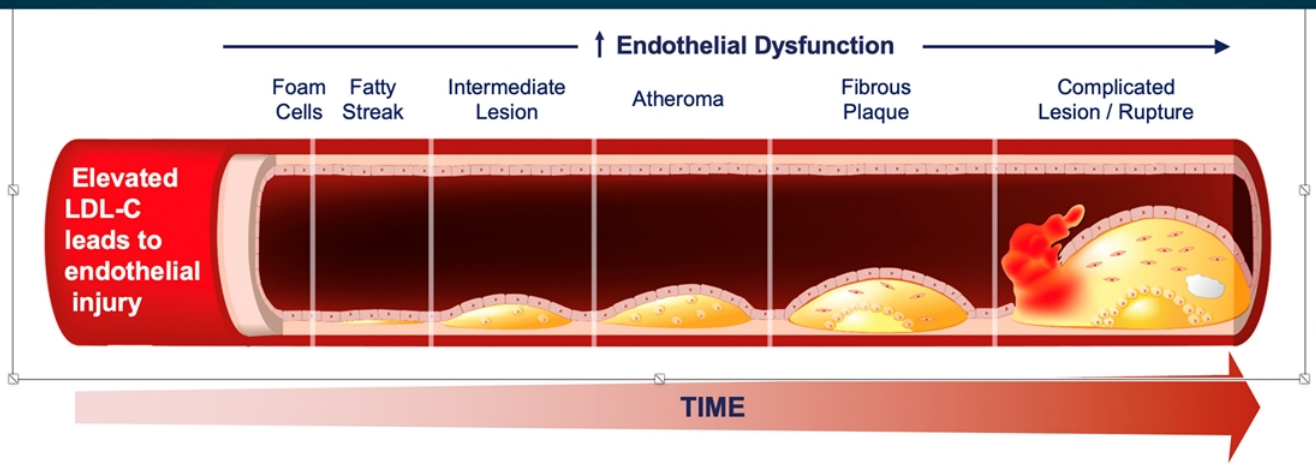
1. Tabas I et al. Circulation. 2007;116:1832–1844. 2. Williams KJ et al. Arterioscler Thromb Vasc Biol. 1995;15:551–561.
3. Williams KJ et al. Arterioscler Thromb Vasc Biol. 2005;25:1536–1540. 4. Steinberg D et al. N Engl J Med. 1989;320:915–924.

Reduce LDL



Healing lesion less likely to rupture or cause thrombosis - (fewer endpoints).

Elevated LDL-c over time associated with atherosclerosis development

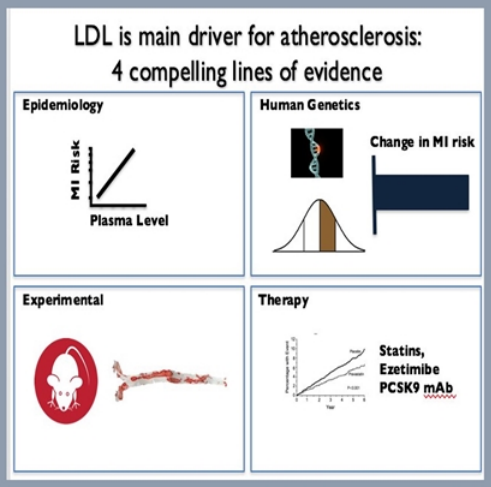


- Plaque rupture can occur at any time in advanced lesions[2,3]
- Patients with FH are at risk for events at a younger age[4]

1. Pepine CJ. *Am J Cardiol*. 1998;82:23S-27S. 2. Ross R. *N Engl J Med*. 1999;340:115-126. 3. Stary HC, et al. *Circulation*. 1995;92:1355-1374. 4 Jellinger P, et al. *Endocr Pract*. 2017;23(suppl 2):1-87

Support for LDL Causality in ASCVD

- **Observational data**
- **Interventional data**
- **Genetic studies**
- **Experimental**



Evidence: Statins Reduce CVD Events—Trials in the 1990s

No data that lipid-lowering drugs reduced mortality
No large-scale trials with statins completed

4S (n=4444)

WOSCOPS (n=6595)

CARE (n=4159)

LIPID (n=9014)

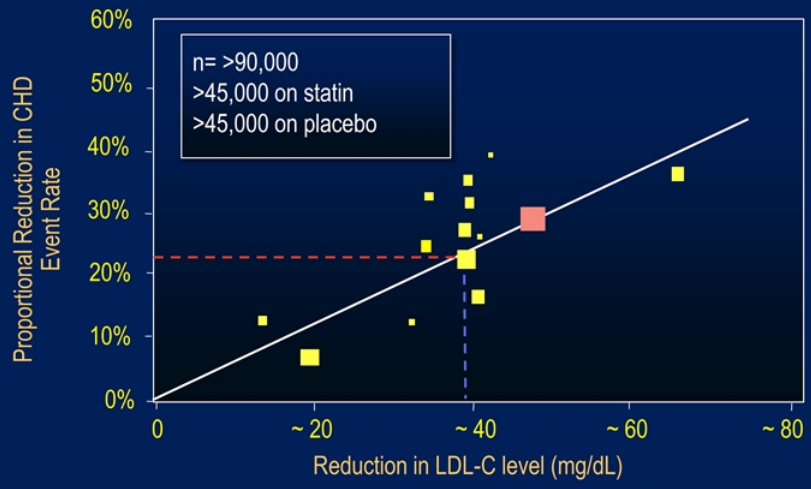
AFCAPS/TexCAPS (n=6605)

5 major trials with statins completed, enrolling 30,000 patients

WOSCOPS = The West of Scotland Coronary Prevention Study; CARE = Cholesterol and Recurring Events (trial); LIPID = Long-term Intervention with Pravastatin in Ischemic Disease; AFCAPS/TexCAPS = Air Force/Texas Coronary Atherosclerosis Prevention Study.

Relation Between Reduction in Incidence of Major CVD Events and Mean Absolute LDL-C

*Reduction at Year 1**



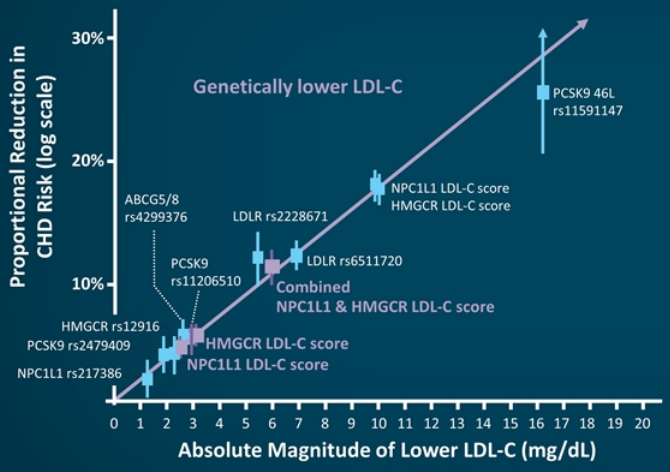
1 mmol/L reduction in LDL-C results in 20% reduction in CVD risk at 1 year

***Meta-analysis of 14 statin trials (CTT collaborators)**

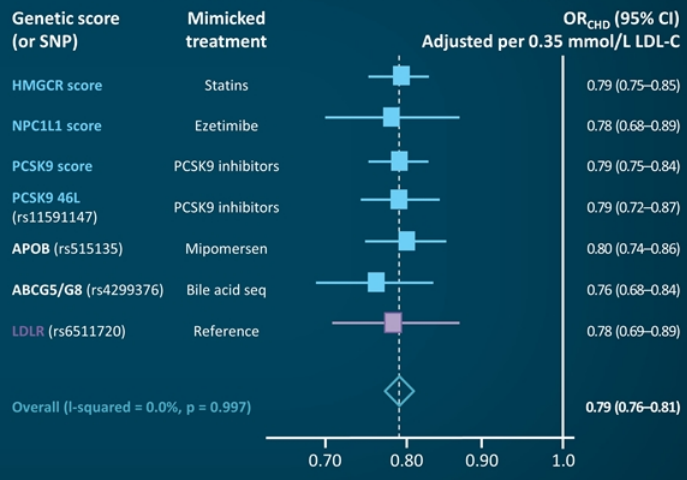
Boekholdt SM et al. *J Am Coll Cardiol.* 2014;64:485-494.

We Have Observed That it Does Not Matter How You Lower LDL-C (Evidence from Mendelian Randomization Studies)

Log-linear Association Between Genetically and Pharmacologically Mediated Lower LDL-C and Risk of CHD



Effect of Exposure to Lower LDL-C by Mechanism of LDL-C Lowering: Effects of Genetic Variants or Genetic Score

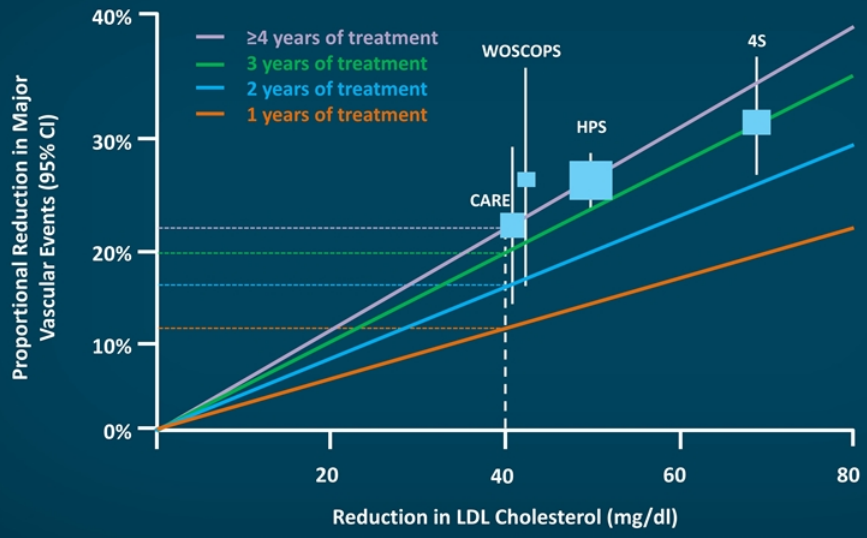


Ference BA, et al. *J Am Coll Cardiol.* 2012;60:2631–2639.
Ference BA, et al. *J Am Coll Cardiol.* 2015;65:1552–1561.

Ference BA, et al. EAS Consensus Statement on LDL Causality. *Eur Heart J.* 2017; doi:10.1093/eurheartj/ehx144.

ABCG5/G8 = ATP binding cassette subfamily G member 5/8; APOB = apolipoprotein B; CHD = coronary heart disease; CI = confidence interval; HMGCR = 3-hydroxy-3-methylglutaryl-CoA reductase;
LDL-C = low-density lipoprotein cholesterol; LDLR = low-density lipoprotein receptor; NPC1L1 = Niemann-pick C1-like 1; OR = odds ratio; PCSK9 = proprotein convertase subtilisin/kexin type 9.

We Have Observed That Benefit is Related to Absolute Reductions in LDL-C and the Duration of That Absolute Reduction

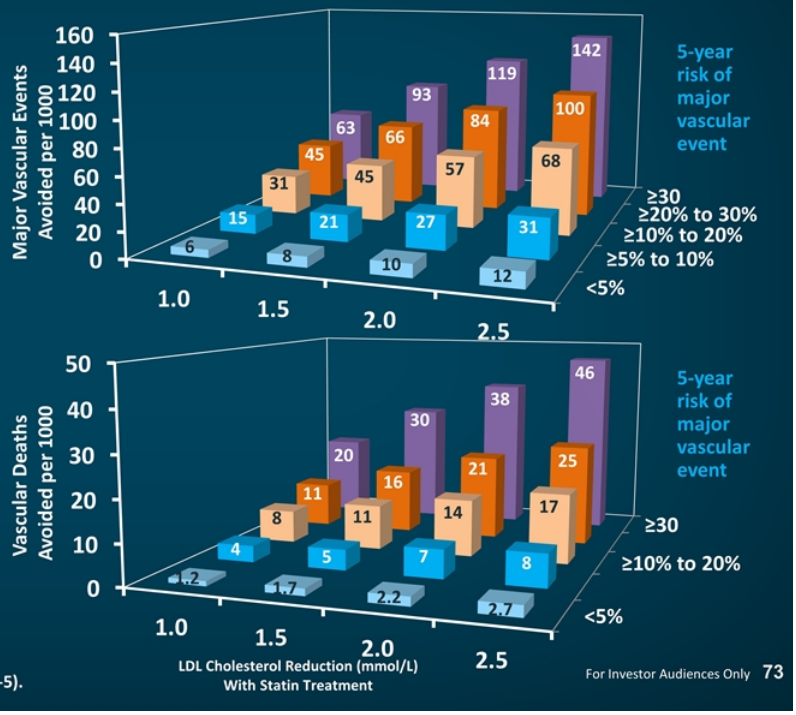


CI = confidence interval; LDL = low-density lipoprotein.

Ference BA, et al. *Eur Heart J*. 2017; doi: 10.1093/eurheartj/ehx450.

GUIDELINES Match the Intensity of the LDL-C Reduction to the Level of Risk in the Individual The Basis for Each Individual Consult in Every Clinic!

- Global guidelines identify four groups in whom LLT should be considered
- Established ASCVD
- Diabetes mellitus
- Primary LDL elevations >190mg/dL
- Primary prevention but high global risk (risk calculator)



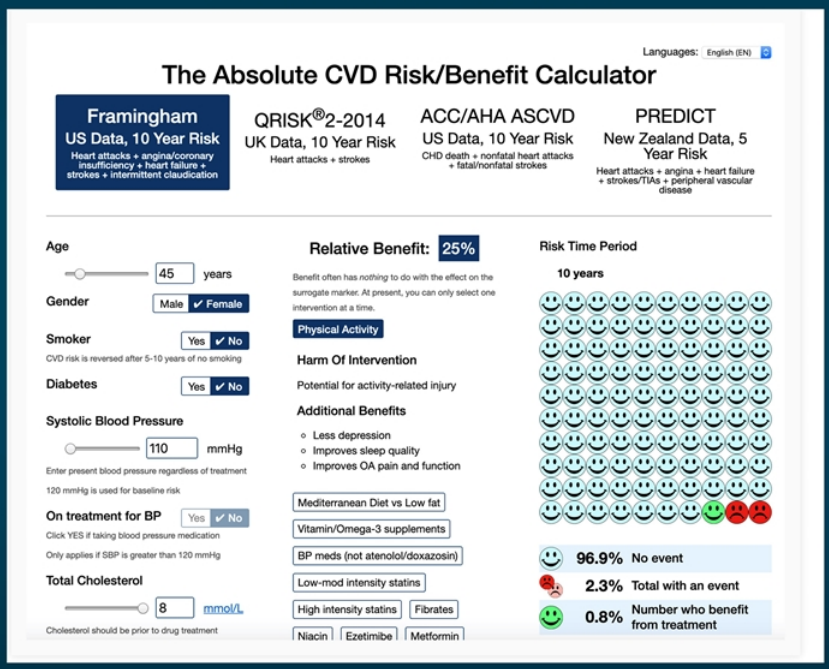
CTT Colaborators. *The Lancet* 2012 380, 581-590. DOI: (10.1016/S0140-6736(12)60367-5).

Risk Stratification: ACC/AHA Guidelines Define Four Statin Benefit Groups

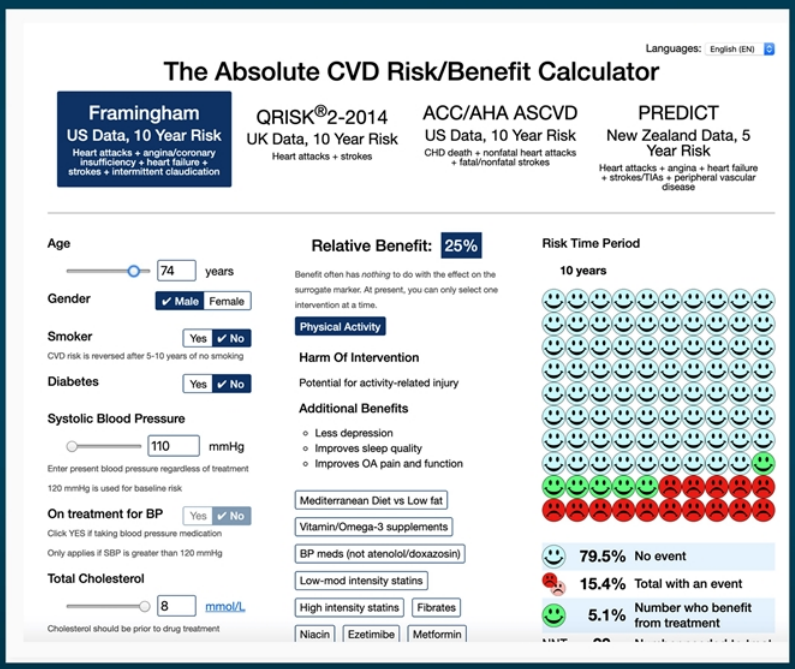
Category	Recommendation
Clinical ASCVD	**Secondary prevention** •High-intensity statin if age ≤ 75 years •Moderate intensity statin if age > 75 years or not a candidate for high-intensity statin •Combination therapy if 50% LDL-C lowering not reached
Primary elevations of LDL-C ≥190 mg/	**Primary prevention** •High-intensity statin
Diabetes (type 1 or 2), without clinical ASCVD, 40-75 years of age, LDL-C 70 to 189 mg/dL	**Primary prevention** •Low risk – moderate-intensity statin •High risk – high intensity statin
No diabetes, estimated 10-year ASCVD risk ≥7.5%, 40-75 years of age, LDL-C 70 to 189 mg/dL	**Primary prevention** •Moderate- to high-intensity statin

In Patients with Low Absolute Risk the Number Who Benefit with LDL-C Reduction is Modest

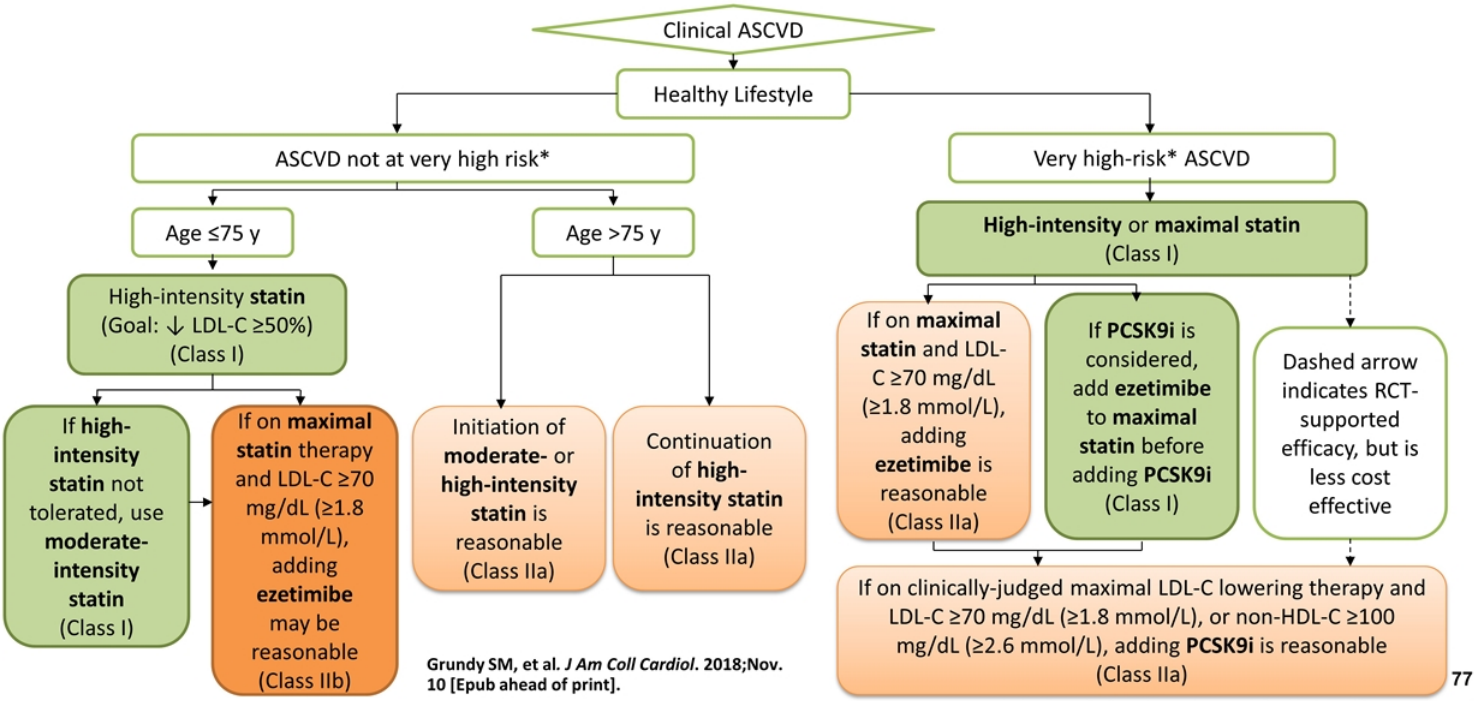
8 mmol/L total cholesterol is about 320 mg/dl in these units



In Patients with High Absolute Risk the Number Who Benefit with LDL-C Reduction is More Pronounced



Secondary Prevention: Patients with Clinical ASCVD



Grundy SM, et al. *J Am Coll Cardiol*. 2018;Nov. 10 [Epub ahead of print].

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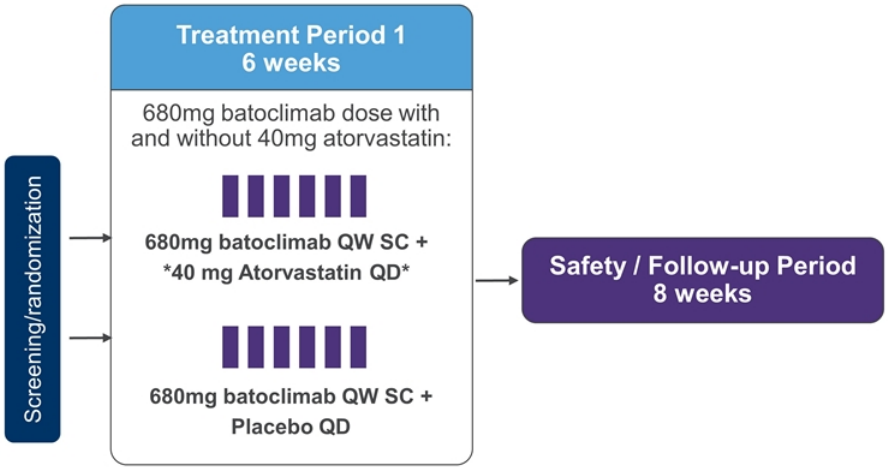
Conclusions

- LDL-C is causal for atherosclerotic cardiovascular disease
- The risk is determined by the absolute levels of LDL-C and the duration of the elevation measured in years (i.e cholesterol-years)
- National guidelines have been developed to match the intensity of therapy to the absolute risk of the patient
- Statins due to LDL-C lowering efficacy, safety, proven CV benefits and cost are the primary therapy for the treatment of elevated LDL-C
- In medical practice, there are a number of therapies that increase LDL-C such as SGLT2 inhibitors and beta blockers that have proven CV benefits or anti-cytokine therapy such as IL-6 inhibitors in which potential elevations are managed with statin therapy
- In general, LDL-C elevation with a therapy (or a lifestyle intervention, for example the keto diet) should be judged based on the absolute risk vs benefits

Cholesterol management

Healthy Volunteer study preliminary data

Healthy volunteer study initiated to measure LDL impact of concomitant statin therapy with batoclimab

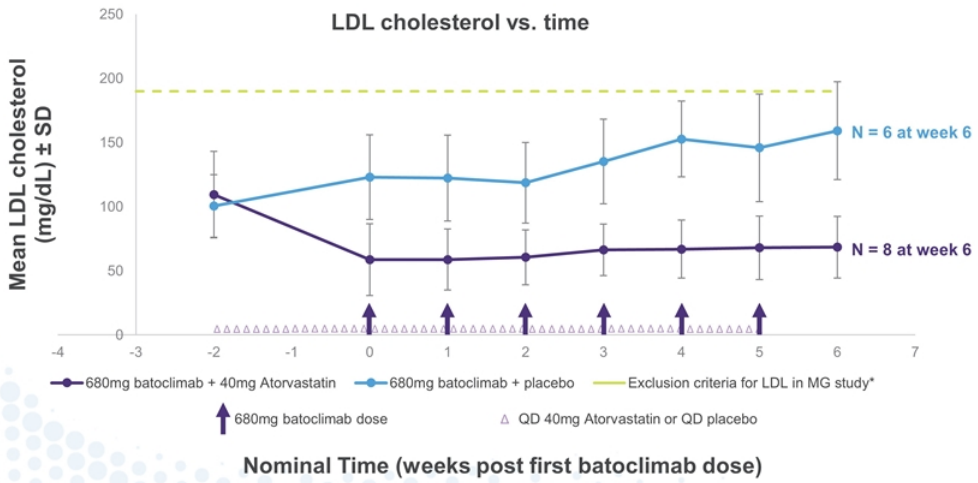


40mg atorvastatin dosing initiated 14 days prior to initiation of 680mg batoclimab dosing

QW = weekly; QD = daily, SC = subcutaneous injection

Healthy volunteer study preliminary data

Mean LDL cholesterol for first two cohorts of patients reflects impact of statin treatment on LDL cholesterol when co-administered with 680mg batoclimab



Distribution of Atorvastatin in US (2019)**

Strength	% of dispensed products
80 mg	13.8
40 mg	36.0
20 mg	29.1
10 mg	20.6
Other, unspecified, or misc.	0.5

*Note – 190mg/mL exclusion criteria in MG Ph3 study for batoclimab applies to subjects without a history of cardiovascular disease.
**All doses in tablet/capsule form: Data source Medical Expenditure Panel Survey (MEPS) 2013-2019. Agency for Healthcare Research and Quality (AHRQ), Rockville, MD. ClinCalc DrugStats Database version 2021.10

Key take-aways on the impact of batoclimab on LDL cholesterol

1

Mechanism is not unique to batoclimab

LDL changes correlated with on target changes in albumin

2

Cholesterol changes are reversible

Dose dependent changes in LDL returned to normal with cessation of dosing

3

Cholesterol changes expected to be manageable

Batoclimab dose titration and use of statins or other cholesterol-lowering therapies provide levers for maximizing benefit-risk

Closing and Q&A

Normal lives for people with autoimmune disease

IMMUNOVANT

What comes next

Path forward for Immunovant

Pete Salzmann, MD
Chief Executive Officer

IMMUNOVANT

Broad development plan enabled by an exciting mechanism of action, batoclimab's features and a strong balance sheet

01 Batoclimab's unique combination of attributes present a potentially significant commercial opportunity across multiple indications in rare autoimmune diseases

02 Immunovant is well-capitalized with $527M[1] in cash expected to fund its broad development plans into calendar year 2025[2]

03 Immunovant plans to initiate a Phase 3 study of batoclimab in Myasthenia Gravis in the first half of calendar year 2022 – topline results expected in 2024

04 Immunovant remains on track to start three pivotal studies in calendar year 2022[3], and to announce two new indications by August 2022

IMMUNOVANT

1. As of December 31, 2021, per most recent Quarterly Report on Form 10-Q filed with the SEC on February 4, 2022
2. The assumptions upon which we have based our estimates are routinely evaluated and may be subject to change
3. Three pivotal trials include myasthenia gravis and two other indications

Investor Q&A

Pete Salzmann, MD
Chief Executive Officer
Renee Barnett
Chief Financial Officer
Bill Macias
Chief Medical Officer

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