

January 7, 2019

To,
**SINGAPORE EXCHANGE SECURITIES
TRADING LIMITED**
11 North Buona Vista Drive,
#06-07 The Metropolis Tower 2,
Singapore – 138589

Dear Sirs,

Sub: Update on Innovative R&D Capabilities and Pipeline Overview

Kindly find enclosed herewith the presentation on update on Innovative R&D Capabilities and Pipeline Overview. The same will also be made available on our website.

Request you to kindly take the same on record.

Thanking you,

Yours faithfully,

For Glenmark Pharmaceuticals Limited

HARISH
VINAYAK KUBER
Harish Kuber
Company Secretary & Compliance Officer

Digitally signed by HARISH
VINAYAK KUBER
Date: 2019.01.07 10:25:40
+05'30'

Encl: as above

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Glenmark Pharmaceuticals Ltd.

Update on Innovative R&D Capabilities and Pipeline Overview

January 2019

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The assets described herein are in different stages of development in clinical studies and the findings described herein are based on such studies. Accordingly, these findings are indicative only and can change from time to time as the studies are continued and the assets described herein advance through the applicable stages of development.

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Agenda

- **Glenmark Overview**
- **R&D Capabilities**
- **Information on select assets in clinical development**
 - **GBR 830**
 - **GBR 1302**
 - **GBR 1342**
 - **GRC 17536**
 - **GRC 27864**
- **Conclusions**

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Glenmark is a research oriented, integrated global pharmaceutical company



Annual revenue of ~\$1.5 bn in FY19E with CAGR of ~12% over last 5 years

Commercial infrastructure in the US, India, Europe, Russia and other emerging markets

EBITDA pre-R&D expenses at ~30% in FY19E

Global manufacturing footprint with 16 facilities and capability to manufacture small molecules and biologics

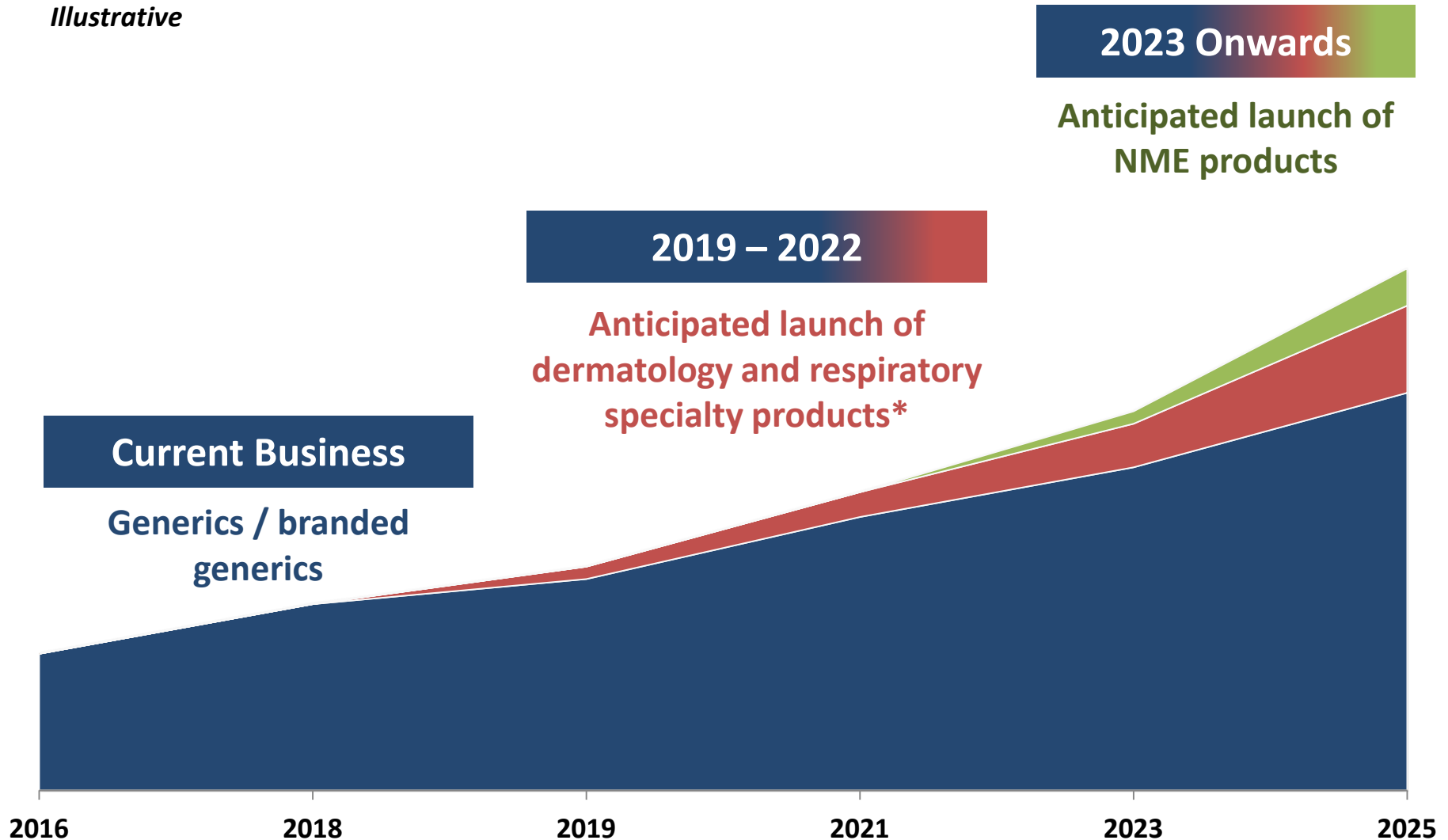
Consolidated R&D expense: 12-13% of revenue

Balanced portfolio of NCEs and NBEs with 9 assets in the pipeline

Projected revenue roadmap going forward – Moving up the value chain



Illustrative

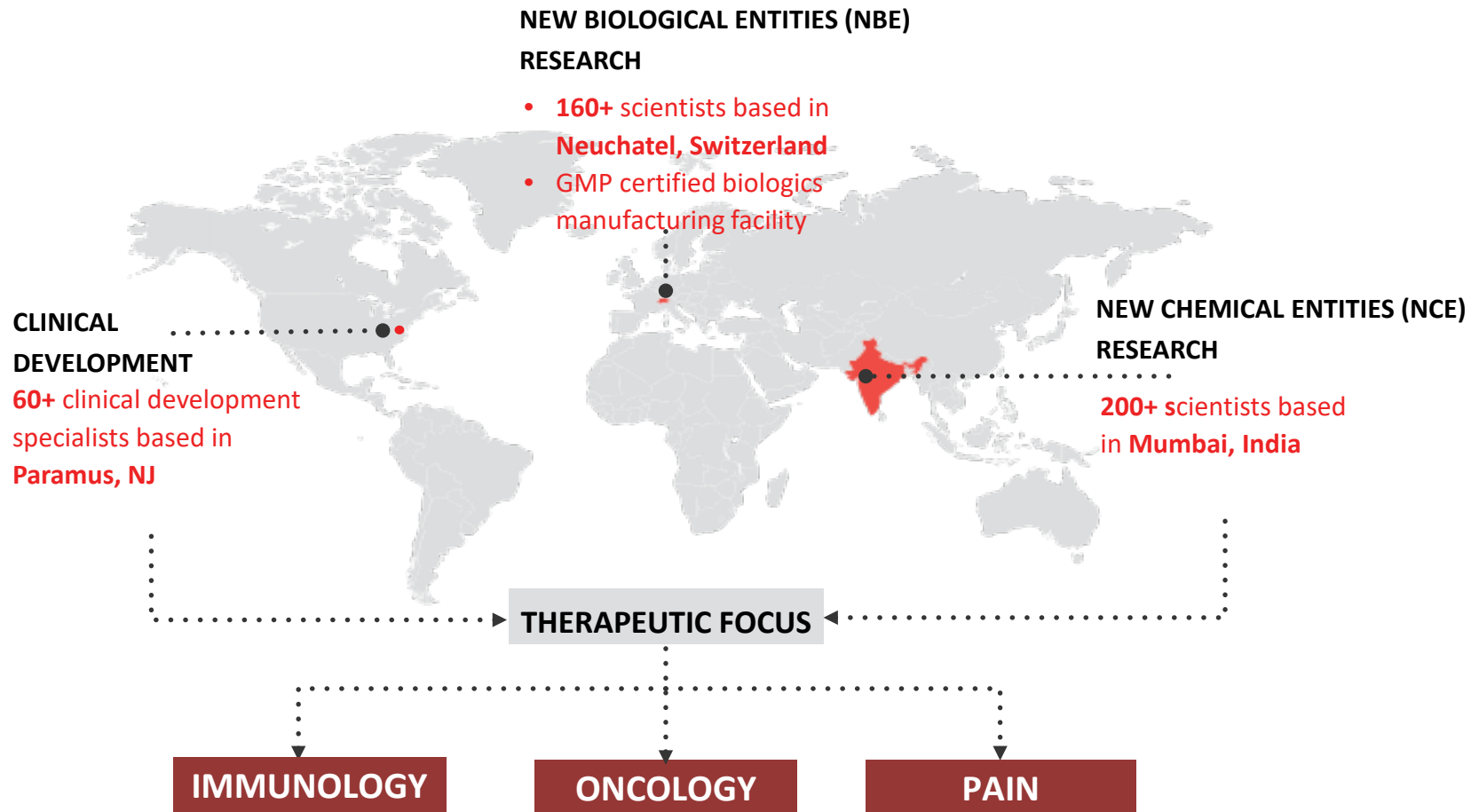


**Projections include additional assets not discussed in this document*

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Glenmark is one of the leading innovative R&D-focused companies from the emerging markets



Global innovative R&D infrastructure spread across the US, Switzerland and India

Leadership Team – Discovery Research

Dr. Lamine Mbow - PhD

Senior Vice President, Head NBE Discovery

- 25+ years in global life sciences Industry
- 18+ years experience in research programs in infectious diseases, human inflammatory and autoimmune diseases
- Prior Experience – Boehringer-Ingelheim
- PhD and Master's degree in Immunology, both from the University of Neuchâtel



Dr. Venkateshwar A Reddy - PhD

Senior Vice President, Global Head of Translational Sciences

- 20+ industry experience in translational drug discovery and development
- Prior Experience – Sanofi, Pfizer
- PhD in Human Biology from Ludwig Maximilian University (LMU) Munich, Germany and Master's from JNU, New Delhi, India



Dr. Kurt Stoeckli - PhD

President and Chief Scientific Officer

- 25+ years of industry experience focused on biopharmaceutical R&D
- Prior Experience – Sanofi, Novartis
- PhD in Molecular Cell Biology from Max-Planck-Institute for Neurobiology in Munich, graduated from University of Basel, ETH Zurich
- Postdoctoral Fellow & Assistant Professor - University of California Medical Center



Dr. Pravin S Iyer - PhD

Senior Vice President, Head NCE Research

- 20+ years of industry experience in Medicinal Chemistry and Drug Discovery
- Prior Experience – Roche, AstraZeneca, GVK Biosciences and Zydus Cadila
- PhD in Organic Chemistry from Duquesne University, Pittsburgh, Pennsylvania, USA
- Research Scientist in University of Pittsburgh



Dr. Roberto Giovannini - PhD

Vice President, Head of Process Sciences

- 18+ years of combined pharmaceutical and academia experience
- Prior Experience - Boehringer-Ingelheim
- PhD in Chemistry from the EPFL in Lausanne
- Masters and Bachelors in Chemistry from Univ of Geneva



Didier Moerenhout

Vice President, Manufacturing Science and Technology

- 25+ years in biopharmaceutical industry
- Prior Experience – Novartis, Pfizer, GSK, Monsanto
- Masters in Management and Biotechnology Engineer - Université catholique de Louvain, Switzerland



**Discovery
Research**

Leadership Team – Clinical Development

Dr. Ewa Matczak - MD

Vice President, Clinical Sciences – Oncology

- 27+ years of clinical and industry experience, with focus on oncology
- Prior Experience – Hengrui Therapeutics, Pfizer, Novartis, Eisai, Bayer Healthcare, NIH
- Hematology/Oncology Fellowship at Columbia Medical School, NY and HIV Oncology Fellowship at Harvard Medical School
- MD - SUNY Downstate, NY



Dr. Mahboob Rahman – MD, PhD

President and Chief Medical Officer

- 25+ years of clinical practice and industry experience
- Prior Experience – Novartis, Centocor/J&J, Pfizer
- PhD, Molecular Biology, from Medical College of Pennsylvania
- US Board Certified (Rheumatology) and licensed to practice medicine in multiple states (NJ, PA, MA)
- Teaching experience at 3 Medical Schools - Rutgers/RWJ, UPenn, and Harvard focus on Rheumatology

Dr. Gerhard Wolff – MD, PhD

Vice President, Clinical Sciences – Immunology

- 15+ years experience in translational medicine and early clinical development
- Prior Experience – Achillion, Mitsubishi Tanabe, Roche, Revotar Biopharma
- Board certified Internal Medicine physician and Immunologist
- Fellowship from Pulmonary Branch National Heart, NIH, MBA - from Rutgers University



Clinical Development

Cynthia Caracta – MD, PhD

Vice President, Clinical Sciences – Respiratory

- 20+ years combined experience in clinical and industry expertise
- Board Certified Physician and Fellow of the American College of Chest Physicians
- Prior Experience – Teva, Hurley Consulting, Forest Laboratories
- NYU (BA – Biochemistry), NY Medical College (PhD – Biochemistry & Molecular Biology), and SUNY Health Sciences Center at Brooklyn (MD)

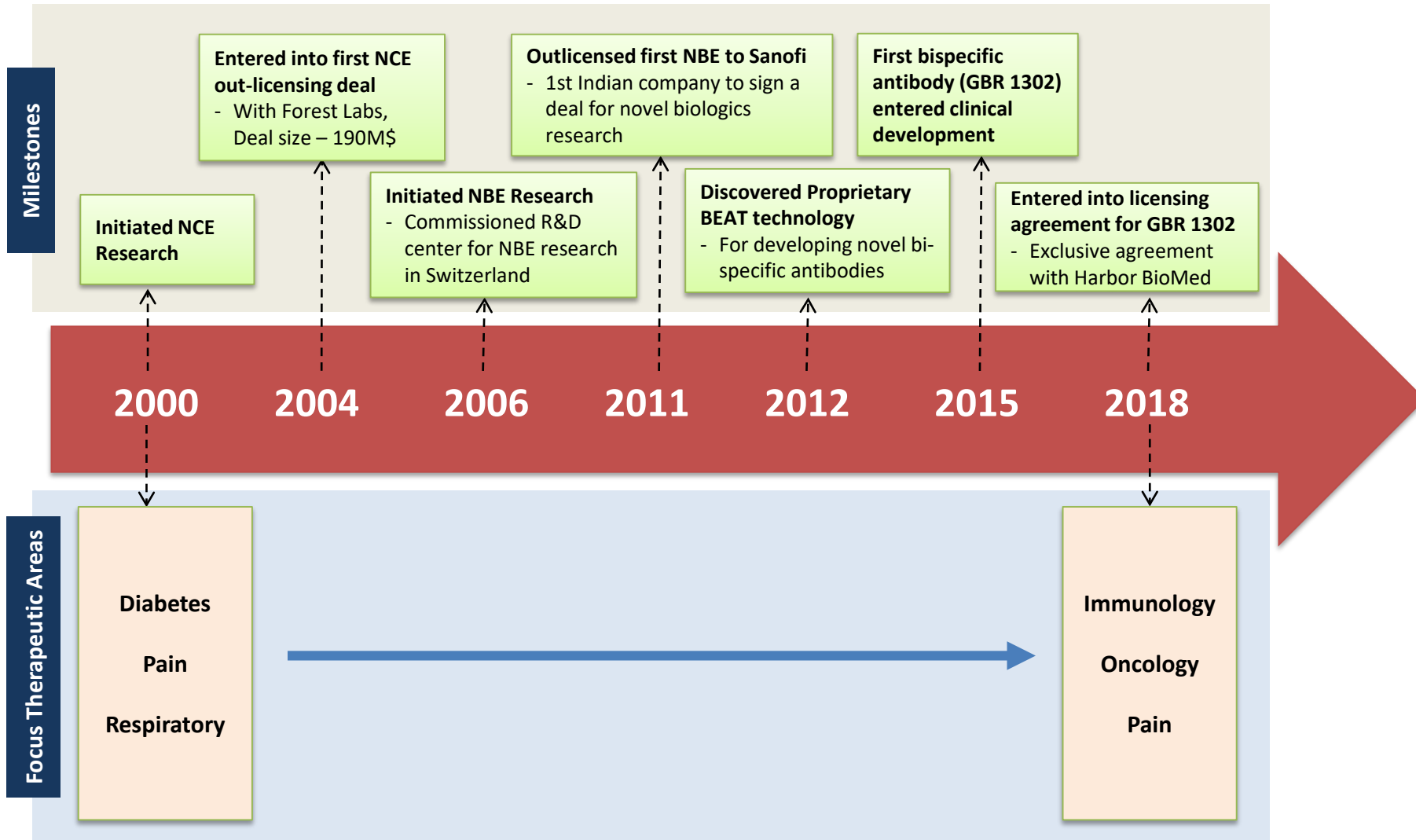


Dr. Monika Tandon – MD, DM

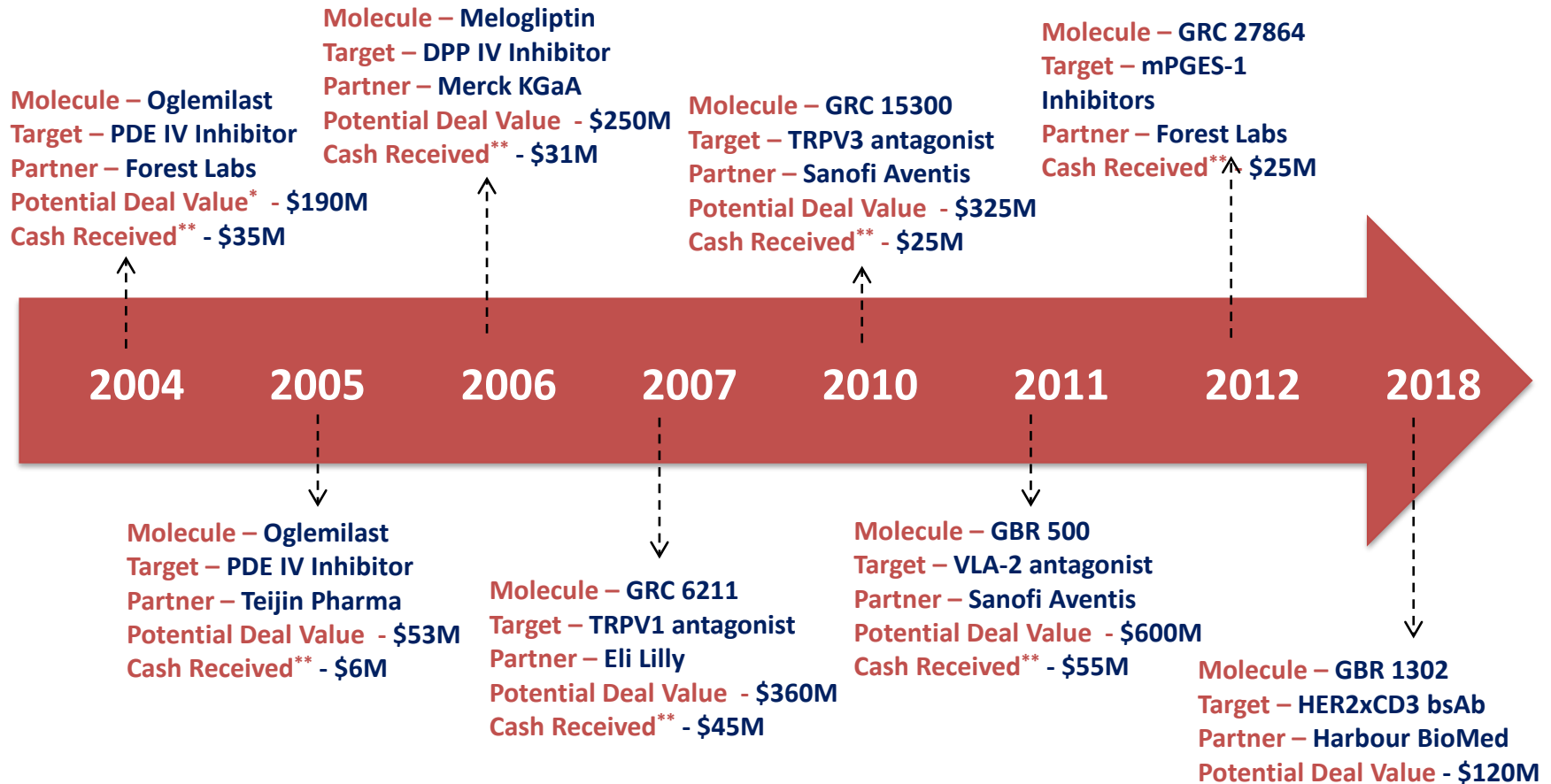
Vice President and Head, Clinical Development - ROW

- 19+ years of post MD experience in overseeing clinical programs and translational medicine
- Prior Experience – Ranbaxy
- MD in Pharmacology from Post Graduate Institute of Medical Sciences; DM Clinical Pharmacology – PGIMER Chandigarh, India

Glenmark's innovation journey



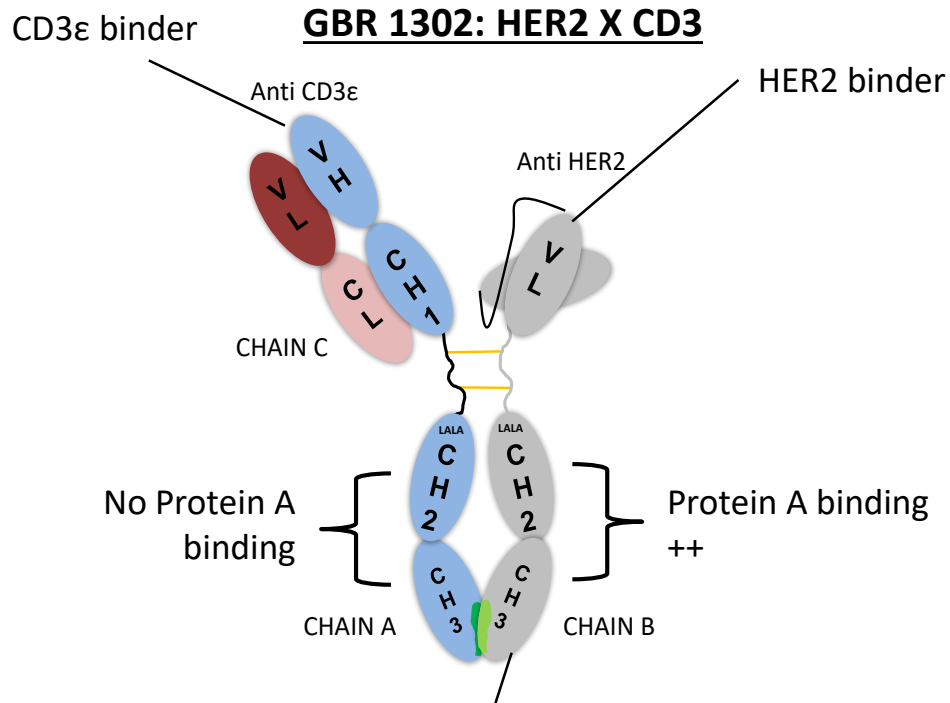
Eight out-licensing deals resulted in \$220+ mn of cash payment received



*Potential Deal Value refers to contingent payments over a period of years

**Cash payment received as upfront or milestone payment

Proprietary BEAT® Platform to develop bispecific antibody assets



Key features of BEAT

- **Efficiency** in transferring tumor antigen binding scFv's into BEAT format ("plug & play")
- **Flexibility** beyond CD3-mediated engagement immunocytes
- **Robustness** and **scalability** of platform similar to standard monoclonal antibody (mAb) production

- Tumor killing activity based on Redirected Lysis (RDL) of Tumor Cells by T cells
- Structure of bispecific antibodies
 - Primary arm to engage and activate T-cells through binding to the CD3 receptor on the T cell
 - Secondary arm to act solely as a binding motif to a target on the tumor cell surface
- Multiple bispecific antibodies under development targeting unmet needs in oncology

Robust relationships with academia and R&D service providers



Non-exhaustive



Innovative research pipeline

Therapy	Molecule	MoA/Class	Potential Indication	Pre Clinical	Phase 1	Phase 2a	Phase 2b	Phase 3	Approval
Immunology	GBR 830	OX40 Antagonist	- Atopic Dermatitis						
			- Systemic Lupus Erythematosus (SLE) - Ulcerative Colitis (UC)						
	GRC 39815	RORyt Inhibitor	- COPD						
Oncology	GBR 1302	HER2 X CD3	- Breast Cancer - Gastric Cancer						
	GBR 1342	CD38 X CD3	- Multiple Myeloma - Solid Tumors						
	GBR 1372	EGFR X CD3	- Colorectal Cancer						
	TBD	MAP4K1 Inhibitor	- TBD						
Pain	GRC 27864	mPGES-1 Inhibitor	- Osteoarthritic Pain						
	GRC 17536	TRPA1 Antagonist	- Diabetic Peripheral Neuropathic Pain						

Innovative research pipeline – Summary

5 clinical and 4 pre-clinical
assets currently in
development

2 clinical assets currently
in Phase 2b, and one
asset likely to enter Phase
2b in FY20

3 immuno-oncology
bispecific antibodies from
the BEAT[®] platform

All clinical assets
developed in-house, no
financial commitment to
any 3rd party

Balanced portfolio
consisting of NCEs and
NBEs

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GBR 830 Executive summary

- First-in-class, novel, investigational antagonistic mAb (monoclonal antibody) designed to selectively target OX40 receptor to treat autoimmune and chronic inflammatory diseases
- Inhibits pathologically activated T cells and effector memory T cells
- Moderate-to-severe AD
 - Phase 2a study provided proof-of-concept for in moderate-to-severe AD presented at the International Investigative Dermatology Meeting in May 2018
 - Phase 2b study in US, Canada and EU ongoing
- SLE
 - Phase 2a POC study anticipated
- UC
 - Phase 2a POC study anticipated
 - Initiated ex-vivo translational studies to evaluate GBR 830 in UC

GBR 830 Differentiation

Differentiation versus targeting OX40L

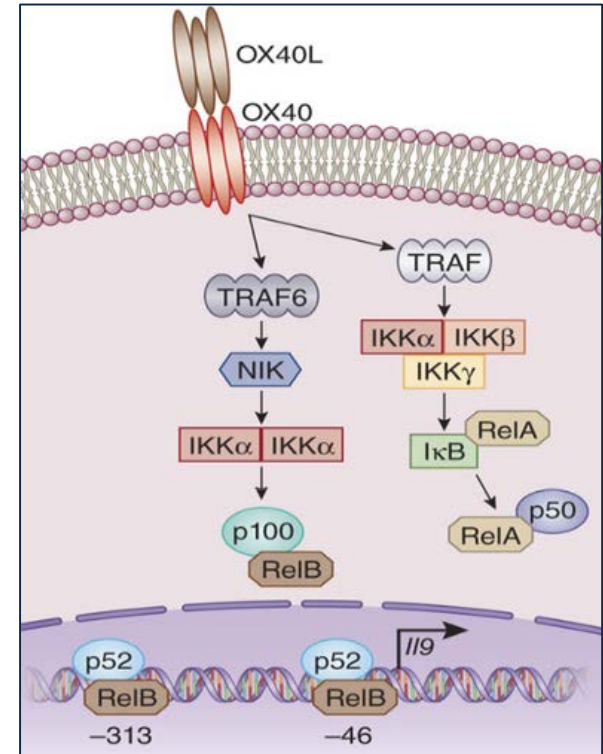
- OX40 expression on T-cells is more defined than OX40L expression on antigen-presenting cells suggesting OX40 as a better therapeutic target

Differentiation versus other strategies

- OX40 pathway controls key drivers of autoimmunity and chronic inflammation including Atopic Dermatitis pathology
- Directly targeting dysfunctional T cells addresses the plasticity of disease pathology

Other inflammatory diseases

- Genetic association studies and specific expression of OX40 and OX40L indicates involvement in many autoimmune pathologies
- Potential application across multiple indications beyond AD, such as UC and SLE
- Expression of OX40 and OX40L is specific to sites of autoimmunity and correlates with disease severity



Webb et al. *Clin Rev Allergy Immunol.* 2016 Jun;50(3):312-32

Coustet et al., 2012. PMID: 22422496;
Bossini-Castillo et al.,

GBR 830 is Glenmark's leading OX40 antagonist molecule currently in clinical development

GBR 830 Pre-clinical review – Translational pharmacology



Translation relevant pharmacology¹

- GBR830 has been shown to block OX40 mediated pathological T cell responses
 - Naive and memory allogeneic T cells
 - T Helper cell-mediated response
- GBR 830 has been shown to block memory T Helper cell responses
 - Generation of memory T Helper cell pool
 - Reactivation of autoimmune memory T cells

Translation of preclinical results into human Atopic Dermatitis²

- An exploratory phase 2a study provided proof-of-concept in moderate-to-severe AD patients by investigating the safety, efficacy, and tissue effects of GBR830.
 - Two doses of GBR830, 4 weeks apart, were well-tolerated and induced significant, progressive tissue and clinical improvement

1. <https://acrabstracts.org/abstract/gbr830-a-true-ox40-antagonist-antibody-with-potent-suppressive-effects-on-t-cell-mediated-pathological-responses/>
2. Guttman-Yassky et al. 2018 Oral presentation at the International Investigative Dermatology. [https://www.jaad.org/article/S0190-9622\(18\)31773-0/fulltext](https://www.jaad.org/article/S0190-9622(18)31773-0/fulltext)

GBR 830 Clinical studies – Summary of healthy volunteers studies after IV or SC administration*



Safety*

- 98 healthy volunteers exposed to GBR 830 in 3 phase 1 studies
- Safe and well tolerated up to 40 mg/kg IV and 600 mg SC fixed dose in the studies conducted to date
- Similar rates of TEAEs in GBR 830 and placebo groups without dose relationships observed

Pharmacokinetics*

- Similarity of GBR 830 half-life in IV or SC forms: ~11-15 days
- Good bioavailability after IV and SC injection
- Dose proportional increase in C_{max} and AUC after IV and SC dosing

IV: Intravenous; SC: subcutaneous

* [https://www.jidonline.org/article/S0022-202X\(18\)31333-2/fulltext](https://www.jidonline.org/article/S0022-202X(18)31333-2/fulltext)

GBR 830 Clinical studies – Results from Phase 2a POC study in moderate-to-severe AD (NCT02683928)^{1,2}



- GBR 830 was observed to be safe and well tolerated in this first-in-patient study
 - The most common treatment emergent adverse event was headache, with no meaningful differences observed between GBR 830 (13%) and placebo (25%) treated patients.
- Although not powered for statistical differences between GBR 830 versus placebo, improvement in clinical scores were observed as compared to placebo
- Clinical improvement was associated with decline in related biomarker mRNA for disease activity in all 4 pathways analyzed, including Th1, Th2, Th17 and Th22
- Based on positive Phase 2a POC study results, a Phase 2b study was initiated in June 2018

1. [https://www.jaad.org/article/S0190-9622\(18\)31773-0/abstract](https://www.jaad.org/article/S0190-9622(18)31773-0/abstract)

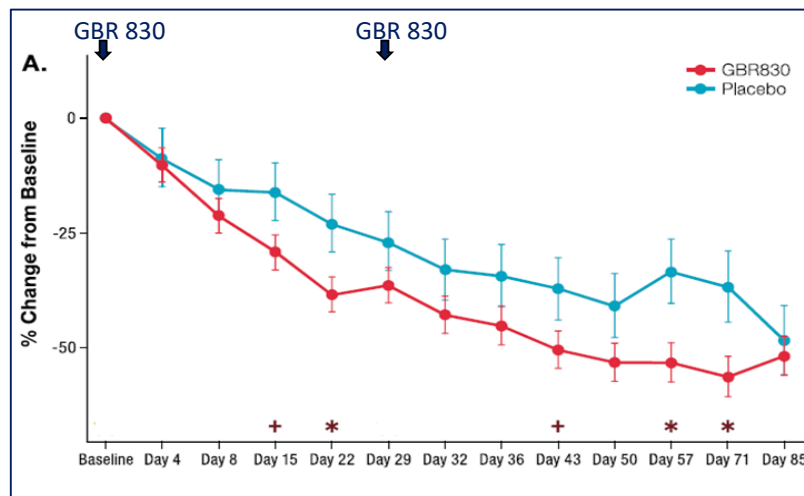
2. Guttman-Yassky et al. 2018 Oral presentation at the International Investigative Dermatology. [https://www.jidonline.org/article/S0022-202X\(18\)30686-9/fulltext](https://www.jidonline.org/article/S0022-202X(18)30686-9/fulltext)

GBR 830 Clinical studies – Phase 2a POC study in moderate-to-severe AD (NCT02683928)*

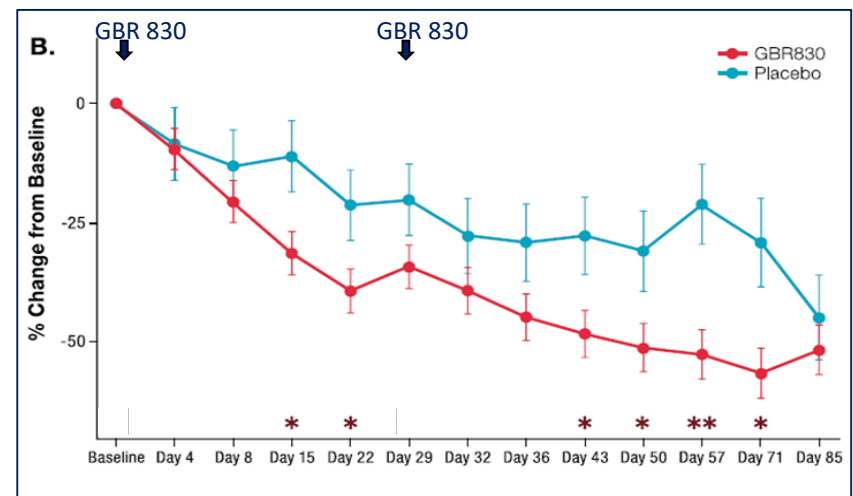


Two IV doses of GBR 830, 4 weeks apart, induced clinically meaningful and sustained improvements in Eczema Area and Severity Index (EASI) clinical severity

ITT population



Subjects with severe AD at baseline (SCORAD >50)



+P<0.1 (trend), *P<0.05, **P<0.01 (GBR 830 vs placebo)

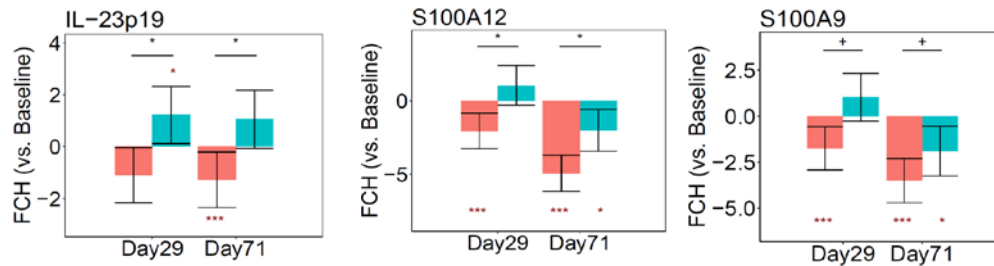
AD, atopic dermatitis; EASI, Eczema Area and Severity Index; ITT, intent-to-treat; SCORAD, Scoring of Atopic Dermatitis

GBR 830 Clinical studies – Phase 2a POC study in moderate-to-severe AD (NCT02683928)*

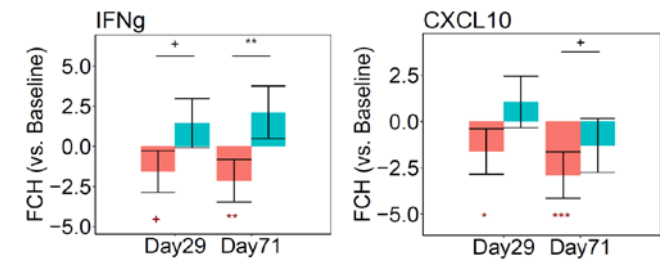


GBR 830 reduced key mRNA biomarkers of disease activity in lesional skin

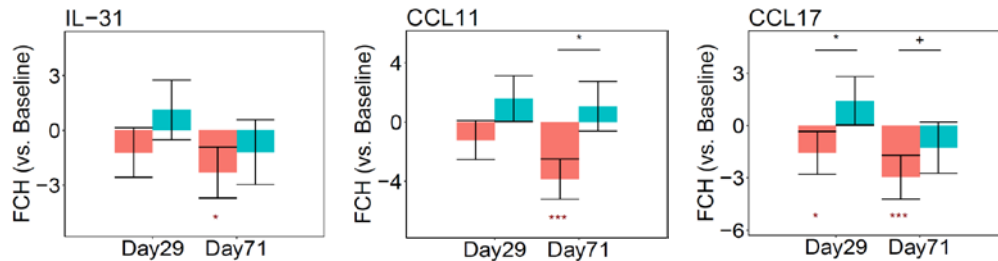
Th17/Th22



Th1



Th2



Treatment
■ Drug
■ Placebo

Quantitative RT-PCR mRNA expressions following treatment

- GBR 830 vs placebo: +P<0.1 (trend), *P<0.05, **P<0.01, ***P<0.001.
- FCH, fold change; mRNA, messenger ribonucleic acid.

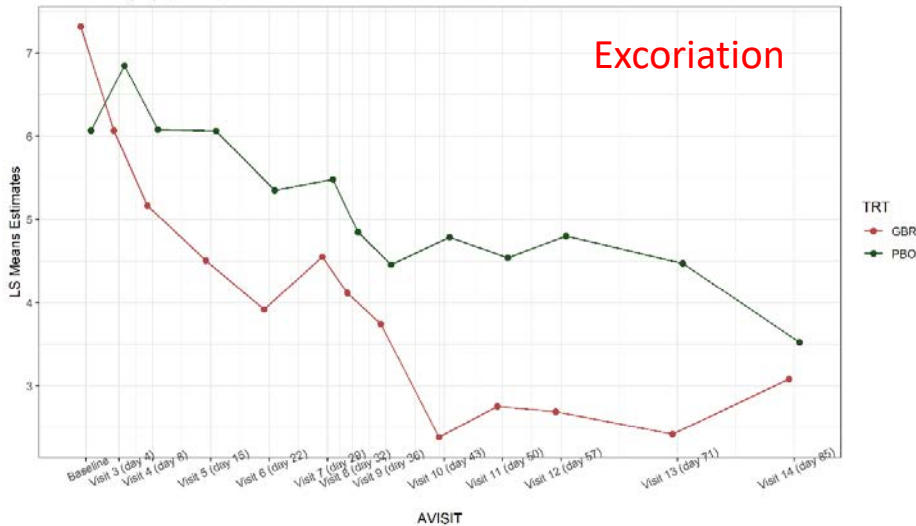
* Guttman-Yassky et al. 2018 Oral presentation at the International Investigative Dermatology [https://www.jidonline.org/article/S0022-202X\(18\)30686-9/fulltext](https://www.jidonline.org/article/S0022-202X(18)30686-9/fulltext)

GBR 830 Clinical studies – Phase 2a POC study in moderate-to-severe AD (NCT02683928)*

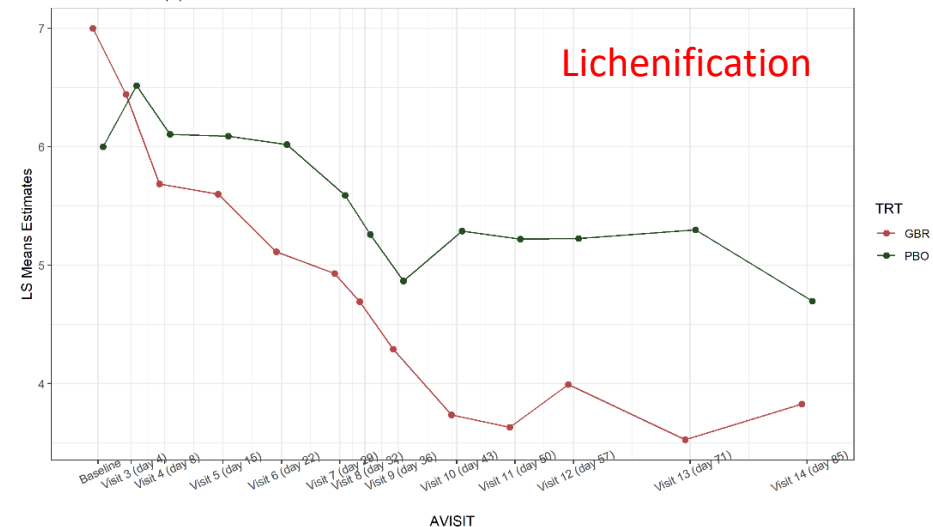


Post-Hoc Analysis of EASI Subscores* - Subjects treated with GBR 830 had sustained and significant improvement in hallmarks of chronicity of atopic dermatitis (i.e., Excoriation and Lichenification)

Excoriation (Ex): Score Overtime



Lichenification (L): Score Overtime



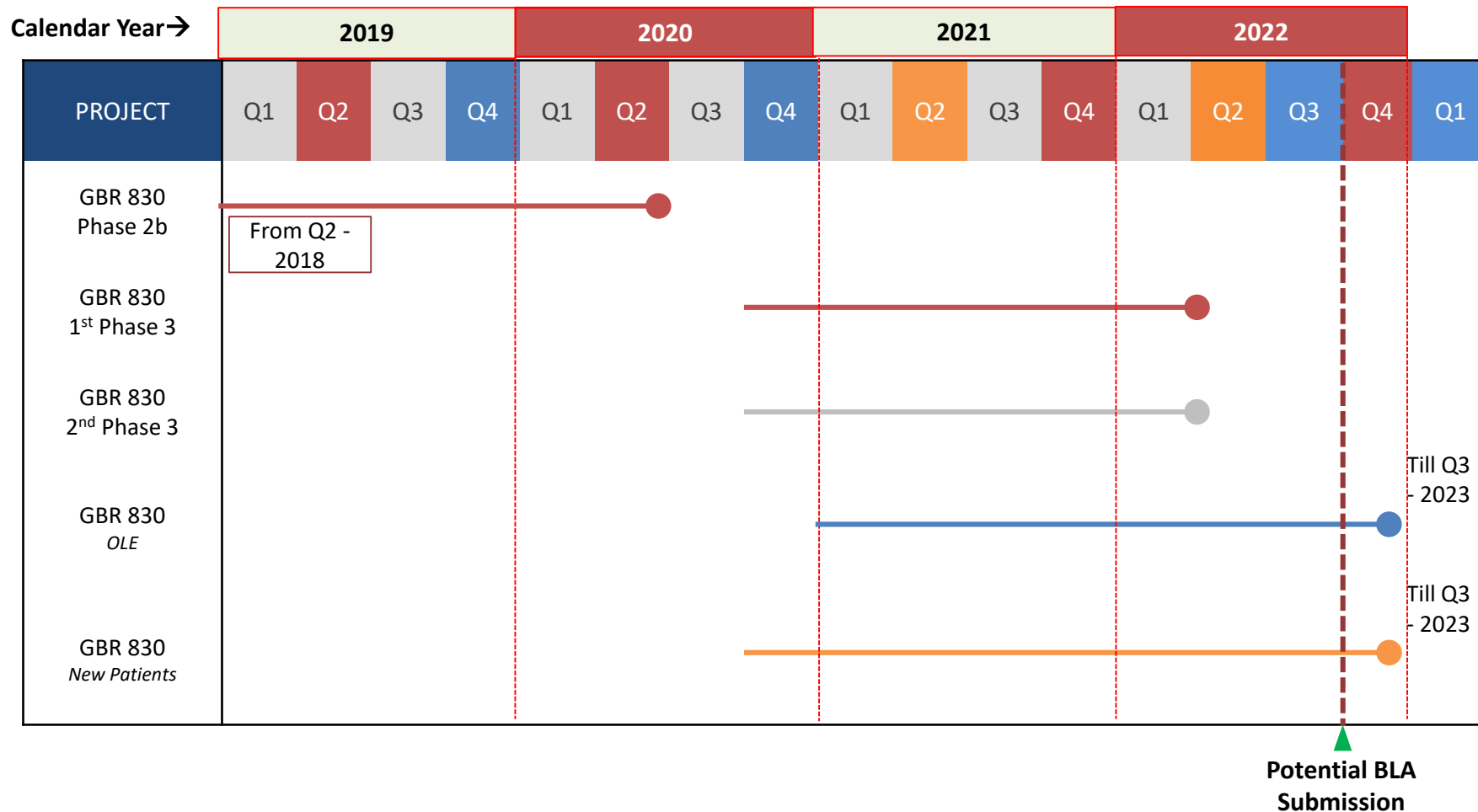
*Post-hoc analysis used a Mixed-Effect Model Repeated Measure (MMRM) model to handle missing data. Data on File. Glenmark Pharmaceuticals Ltd.

GBR 830 Clinical studies – Ongoing Phase 2b study in moderate to severe AD (NCT03568162)



- Multicenter, randomized, double-blind, placebo-controlled, parallel-group study of GBR 830 in adult subjects with moderate to severe AD in US, Canada, and EU
- Primary endpoint: To characterize the efficacy of GBR 830 monotherapy in adults with moderate-to-severe AD compared to placebo as measured by Investigator's Global Assessment (IGA) at week 16
- Key secondary endpoint: To evaluate proportion of subjects with EASI 75 ($\geq 75\%$ improvement from baseline) at Week 16
- Subcutaneous administration of GBR830 or corresponding placebo to 312 patients
- Treatment Groups:
 - 600 mg 1X SC loading dose followed by 300 mg SC every 2 weeks for 16 weeks
 - 600 mg 1X SC loading dose followed by 300 mg SC monthly for 16 weeks
 - 150 mg 1X SC loading dose followed by 75 mg SC monthly for 16 weeks
 - Placebo every 2 weeks for 16 weeks
- Top-line Results: Target Q4 CY19

GBR 830 Clinical studies – Timeline*



*As of December 2018

GBR 830 Publications*

Publication Type	Phase of Study	Citation
Poster	Phase 2 (GBR 830-201)	Guttman-Yassky E, et al. Results from a randomized, double-blind, placebo-controlled, exploratory, multicenter study of GBR 830 in adult patients with moderate-to-severe atopic dermatitis. American Academy of Dermatology Annual Meeting; 2018 Feb 16-20; San Diego, CA.
Poster	Phase 2 (GBR 830-201)	Guttman-Yassky E, et al. Results from a phase 2a randomized, double-blind, placebo-controlled, exploratory, multicenter study of GBR 830 in adult patients with moderate-to-severe atopic dermatitis. The 10 th Georg Rajka International Symposium on Atopic Dermatitis. 2018 Apr 11-13; Utrecht, Netherlands.
Poster	N/A	Macoin J, et al. Targeting OX40 with GBR 830, an OX40 antagonist, inhibits t cell-mediated pathological responses. International Investigative Dermatology Meeting; 2018 May 16-19; Orlando, FL.
Poster	Phase 2 (GBR 830-201)	Gudi G, et al. Clinical pharmacokinetics and immunogenicity of GBR 830, a first-in-class humanized monoclonal antibody inhibiting OX40 to treat atopic dermatitis. International Investigative Dermatology Meeting; 2018 May 16-19; Orlando, FL.
Poster	N/A	Macoin J, et al. GBR 830: An OX40 antagonist antibody with a favorable toxicity profile in non-human primates. International Investigative Dermatology Meeting; 2018 May 16-19; Orlando, FL.
Poster and Oral Presentation	Phase 2 (GBR 830-201)	Guttman-Yassky E, et al. GBR 830 induces progressive and sustained changes in atopic dermatitis biomarkers in patient skin lesions. International Investigative Dermatology Meeting; 2018 May 16-19; Orlando, FL.
Poster (encore)	Phase 2 (GBR 830-201)	Guttman-Yassky E, et al. GBR 830 induces progressive and sustained changes in atopic dermatitis biomarkers in patient skin lesions. Fall Clinical Dermatology Conference; 2018 Oct 18-21; Las Vegas, NV.
Poster (encore)	N/A	Macoin J, et al. Targeting OX40 with GBR 830, an OX40 antagonist, inhibits t cell-mediated pathological responses. Fall Clinical Dermatology Conference; 2018 Oct 18-21; Las Vegas, NV.
Poster	N/A	Macoin J, et al. GBR 830, A true OX40 antagonist antibody with potent suppressive effects on t cell-mediated pathological responses. 2018 ACR/ARHP Annual Meeting; 2018 Oct 19-20; Chicago, IL.
Manuscript	Phase 2 (GBR 830-201)	<i>Manuscript has been accepted for publication in the Journal of Allergy and Clinical Immunology.</i>

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GBR 1302 Executive summary

- Novel, first-in-class, investigational bispecific antibody designed to function as T cell redirector for treatment of HER2 expressing tumors
- In preclinical studies, GBR 1302, an HER2xCD3bsAb, demonstrated faster and more complete killing of Her 2+ tumor cells compared to current 1st and 2nd line HER2 targeted monoclonal antibodies
- Ongoing Phase 1 study (first-in-human study) to determine maximum tolerated dose in patients
 - Dose escalation continues at 9 participating clinical trial sites across Germany and the US
- Translational data in trastuzumab-resistant cancers presented at the 2018 Annual Meeting of the American Society of Clinical Oncology
- Results on early biomarker data accepted for presentation at European Society of Medical Oncology conference in October 2018
- Out-licensed rights for Greater China territory to Harbour Biomed; potential deal value >\$120M, in addition to sales-linked royalties for Glenmark

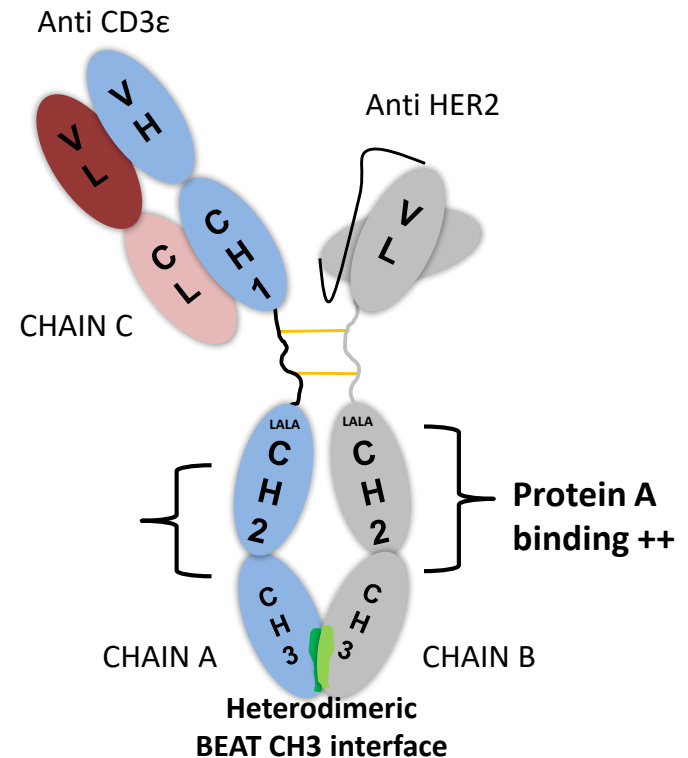
GBR 1302 Pharmacology overview

In vitro pharmacology

- Activity of GBR 1302 requires bridging of T cells to HER2+ cells to create an immune synapse
 - Limited CD3 bystander effect in the absence of HER2+ cells
- Leads to dose dependent T cell activation, cytokine production and cytotoxicity against HER2+ target cells
- Similar binding efficiency on HER2 cancer cells and T lymphocytes
- Consistent high killing by GBR 1302 on IHC 3+ or 2+ cell lines

In vivo pharmacology

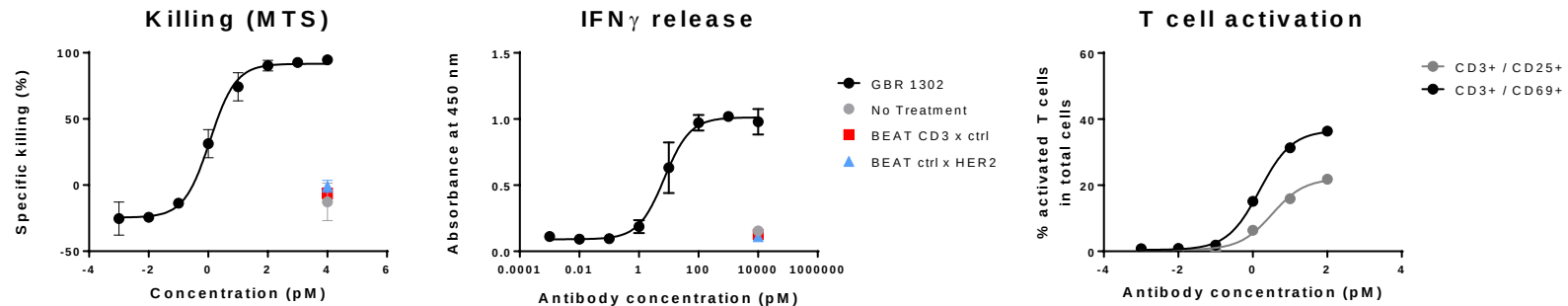
- GBR 1302 has shown activity in tumors derived from IHC3+ Gastric cancer cells compared to TDM1 in Subcutaneous as well as metastatic models
- GBR 1302 has shown activity in TDM1 resistant JIMT-1 breast cancer cell line



GBR 1302 activates T cells to kill and release pro-inflammatory cytokines (in-vitro study)

Redirected Lysis (RDL) assay against NCI-N87 (gastric carcinoma, IHC 3+)

Effector cells: unstimulated PBMCs from healthy donor. E:T ratio 10:1. Duration 48 hours



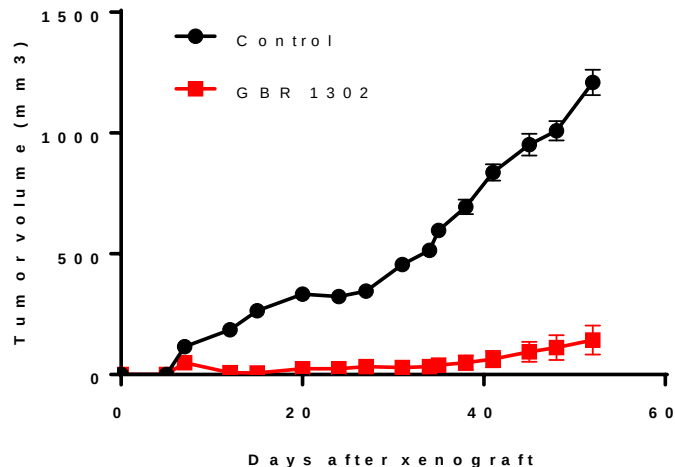
- GBR 1302 leads to complete killing of a gastric carcinoma IHC 3+
- GBR 1302 leads to T cell activation and proliferation
- GBR 1302 leads to pro-inflammatory cytokine production (e.g. IFN γ)
- CD3 only or HER2 only control BEAT do not activate T cells to kill and release cytokines

GBR 1302 demonstrates potent inhibition of tumor growth in Trastuzumab resistant & metastatic xenograft models



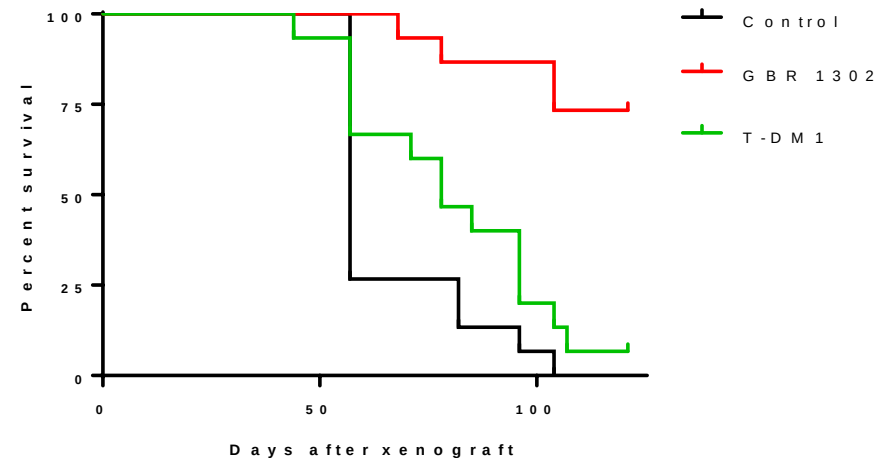
Mouse models: Mix of human HER2 expressing tumor cells and human PBMCs xenografted subcutaneously (JIMT-1) or intravenously (NCI-N87) in immunodeficient mice (NOD SCID).

JIMT-1 Trastuzumab resistant model



- GBR 1302 prophylactic treatment administered at 50 µg/kg in i.v. 3 times/week for 2 weeks (6 doses total).
- 4 different PBMCs donors for each treatment group (20 mice/group total).
- *Report GBR1302_IMM_1514*

Metastatic NCI-87 model



- Prophylactic treatments administered at 50 µg/kg for GBR 1302 and 15 mg/kg for T-DM1 both i.v. 1 time/week for 3 weeks (3 doses total).
- 3 different PBMCs donors for each treatment group (15 mice/group total).
- *Report GBR1302_IMM_1513*

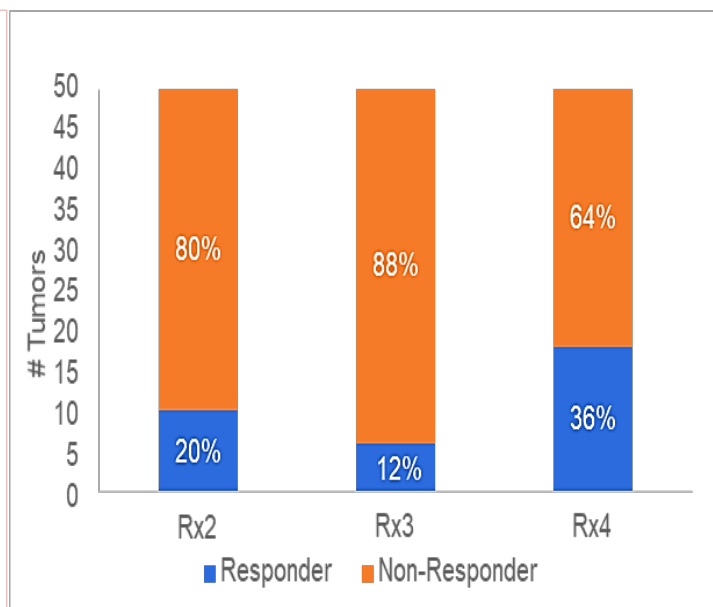
GBR 1302 Ex-vivo Translational Study

Rationale

- Identify Responder and Non-Responders to GBR 1302 in metastatic Breast cancer and Gastric cancer
- Increase confidence in rationale of using checkpoint inhibitor as a combination agent with GBR 1302
- Identify differentiation features of CD3 engagers
 - Induction of T cell memory responses
 - Induction of effector T cells
 - Mechanisms that tilt the balance between regulatory T cells vs Effector T cells

Outcome

- Overall ~20% response rates to single agent
- ~36% response rates with combination agent anti-PD1/PD-L1 (published clinical data, as well as non-related Canscript study data anti-PD1/PD-L1 response rates are ~10-15%)
- Out of 50 gastric and metastatic breast tumors
 - 19/50 tumors responded to at least one treatment arm.
 - Maximum response (18/50) tumors (36%) was observed in arm Rx4
 - 10 out of 50 tumors (20%) responded to Rx2 arm
 - 6 out 50 tumors (12%) responded to Rx3 arm (trastuzumab)



Rx2 – GBR 1302, Rx3 – Trastuzumab, Rx4 - Combination agent anti-PD1/PD-L1

CANscript® data sets reveal predicted responders with M-score >25

GBR 1302 Phase 1 study

Development Phase

- First-in-human, multicenter, open-label, dose-escalation study of single-agent GBR 1302 in subjects with HER2 positive cancers
- Study sites – Germany and US
- Primary objective
 - Safety and Maximum Tolerated Dose
- Secondary objectives
 - Antitumor activity of GBR 1302
 - Pharmacokinetics (PK) of GBR 1302
 - Immunogenicity of GBR 1302
- Exploratory objectives
 - Pharmacodynamic (PD) biomarkers, MOA biomarkers
 - Clinical activity of GBR 1302 in terms of duration of treatment and time to disease progression compared to previous line of cancer treatment

Biomarker Data

Assessment of Preliminary Cytokine Data:

- Overall cytokine profiles indicates T-Cell activation
- Interferon-gamma and IL-2 are (and other relevant biomarkers) consistently elevated with GBR 1302 beginning with Cohort 6, with an exponential increase in Cohort 8
 - Suggestive of activation of cytolytic T-Cells

Dose escalation (part 1a) is being conducted on all HER2 patients where as safety expansion (part 1b) will be conducted on only breast cancer HER2 patients

GBR 1302 Clinical development plan in Breast Cancer

4 Phase 1 clinical studies are currently planned, of which safety expansion is imminent

- Safety expansion study – Monotherapy
 - Biweekly dosing
 - Breast cancer: Her2 2+ and 3+ population
- Dose escalation study – Monotherapy
 - Weekly dosing supported by PK data in biweekly dosing
 - Breast cancer: Her2 2+ and 3+ population
- Combination therapy with PD-1
 - Weekly and biweekly dosing
 - Breast cancer: Her2 2+ and 3+ Population
- Evaluation in patients exposed to lesser lines of therapy
 - Weekly and biweekly dosing
 - Breast cancer: Her2 2+ and 3+ Population

GBR 1302 Publications*

Publication Type	Phase of Study	Citation
Poster	Phase 1 (GBR 1302-101)	Wermke M, et al. A phase 1 study of GBR 1302 in subjects with HER2-positive cancers. 4th Annual ESMO Symposium on Immuno-oncology; 2016 Nov 4-6; Lausanne, Switzerland.
Poster (encore)	Phase 1 (GBR 1302-101)	Wermke M, et al. A phase 1 study of the bispecific antibody t-cell engager GBR 1302 in subjects with HER2-positive cancers. The Annual Meeting of the American Society Of Clinical Oncology; 2017 Jun 2-6; Chicago, IL.
Poster	Phase 1 (GBR 1302-101)	Wermke M, et al. Preliminary biomarker and pharmacodynamic data from a phase 1 study of single-agent bispecific antibody t-cell engager GBR 1302 in subjects with HER2 positive cancers. ASCO-SITC Clinical Immuno-oncology Symposium; 2018 Jan 25-27; San Francisco, CA.
Poster	Phase 1 (GBR 1302-101)	Back J, et al. GBR 1302: Effect of CD3-HER2, a bispecific t cell engager antibody, in trastuzumab- resistant cancers. The Annual Meeting of the American Society Of Clinical Oncology; 2018 Jun 1-5; Chicago, IL.
Poster	Phase 1 (GBR 1302-101)	Wermke M, et al. Preliminary Results from a phase 1 study of single-agent bispecific antibody t-cell engager GBR 1302 in subjects with HER2 positive cancers. The European Society for Medical Oncology Congress; 2018 Oct 19-23; Munich, Germany.

*Current through Nov 2018

Agenda

- Glenmark Overview
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 - GBR 830
 - GBR 1302
 - **GBR 1342**
 - GRC 17536
 - GRC 27864
- Conclusions

GBR 1342 Executive summary

- GBR 1342 is a novel bispecific antibody (CD38xCD3) that is designed to function as a T-cell engager for treatment of CD38 over-expressing tumors
- GBR 1342 is currently being developed in Multiple Myeloma which express CD38
- GBR 1342-101 is an ongoing First-in-Human, Phase 1, dose-escalation study, in patients with MM
 - Total Dosed: 19, Active Patients: 4 (1 patient - Cohort 7, 3 patients - Cohort 8)
 - The longest duration on therapy to date is over 5 cycles
 - Safety: 28 TEAEs in 14 pts., 2 were drug related and reversible: CPK elevation (DLT) and IRR, no neurotoxicity observed to date
- MITRA - an Ex-vivo translational assay system was used to predict responses in patients of selected solid tumors – Head and Neck SCC, prostate, colorectal cancers
 - Results predicted a higher response in Prostate, Head & Neck SCC and Colorectal cancers for GBR 1342 vs. daratumumab.
- GBR 1342 is also being considered in solid tumors and in combination with other therapies.

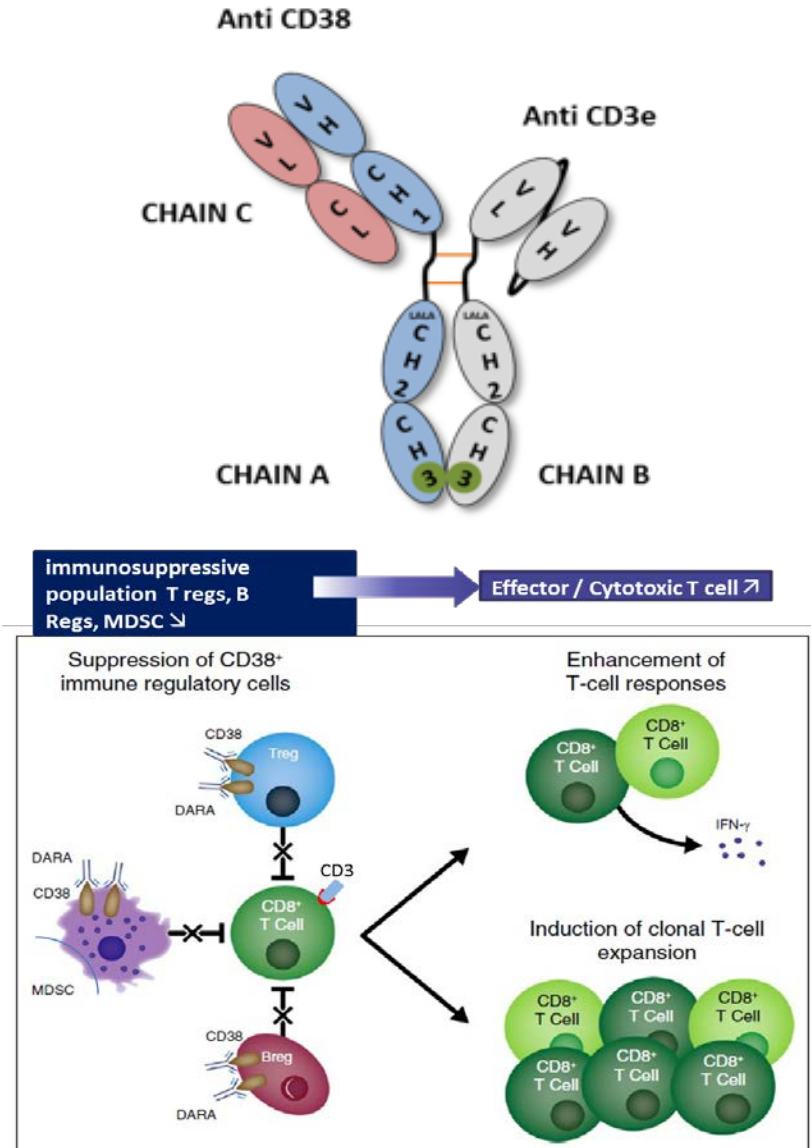
GBR 1342 Key MOA characteristics

CD38 an emerging new therapeutic target

- CD38 as a therapeutic target is validated in multiple myeloma (MM)
- CD38 is highly expressed on plasma cells, and is present on NK, B and T cells
- Targeting CD38 can release the suppression of CD38+ immune regulatory cells, leading to enhancement of T cell responses
- Emerging target beyond MM in other hematological malignancies and solid tumors targeting tumor micro environment (TME)

Characteristics

- Humanized proprietary CD38 binder (CD38 binding epitope has been mapped; parental antibody = 9G7)
- IgG-like PK due to unaltered FcRn binding
- Cross-reactivity:
 - Binding of scFv arm to human/cyno CD3ε (SPR): 100/50 nM
 - Binding of FAB arm to human/cyno CD38 (SPR): 0.6/1.2 nM



GBR 1342 Translational study in solid tumors

Translation Medicine Approach for Clinical Development

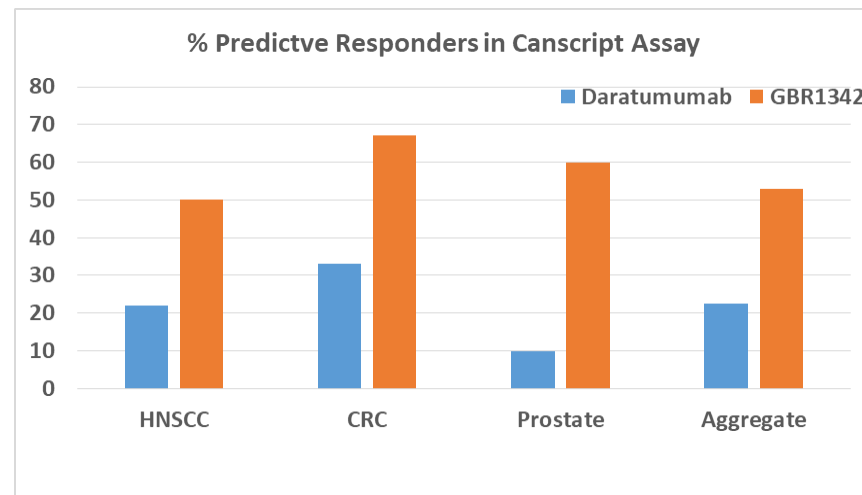
Tumor Identification

- CANscript assay for a monotherapy approach will be utilized to identify potential CD38+ solid tumors (4 different types)
- Full dataset available in September 2018:
 - Part 1 pertains to indication scouting in solid tumors based on CD38 expression
 - Part 2 will be performed in a chosen solid tumor indication based on Part 1 and identify potential combination partner

Dose Regimen

- DMPK analysis from Phase 1 study:
 - Core validation of GBR 1342 PK assay by Immunocapture LC/MS/MS in human serum

Results



Tumor Type	N (20 biopsies per group)	GBR 1342	Daratumumab
HNSCC	N=20	50%	20%
CRC	N=12	67%	42%
Prostate	N=17	47%	12%
Pancreatic	N=2	NA	NA
Aggregate	N=51	53%	22%

In addition to M score, Expression of CD38 on tumor cells as well immune cells is taken into account in predicting responders

GBR 1342 Phase 1 study

Two part dose escalation and cohort expansion study in subjects with previously treated Multiple myeloma (MM)

Part 1 (Dose Finding)

Primary Objective

- Safety and tolerability of GBR 1342

Secondary Objectives

- Pharmacokinetics of GBR 1342
- Immunogenicity of GBR 1342

Exploratory Objectives

- Pharmacodynamic biomarkers
- Explore efficacy of GBR 1342

Part 2 (Efficacy Exploration)

Primary Objective

- Explore efficacy of GBR 1342

Secondary Objectives

- Safety and tolerability of GBR 1342
- Pharmacokinetics of GBR 1342
- Immunogenicity of GBR 1342

Exploratory/Other Objective

- Pharmacodynamic (PD) biomarkers

GBR 1342 Phase 1 study

Study Design

- Study sites in US Only
- Modified 3+3 design
 - First 4 cohorts single patient only
- Administration - every 2 weeks in four week cycles
- First administration same dose as safe dose from previous cohort, second and subsequent at designated higher dose
- Started at 1 ng/kg (MABEL), to planned maximum of 1000 ng/kg
- 1 patient in cohort 7, currently dosed 400 ng/kg, has entered 6th cycle of dosing
- Enrollment in cohort 8 completed

Biomarker Plan

- Peripheral Blood based biomarkers, Cytokines, FACS based Analytes,
- Bone Marrow Biopsies: MRD, IO 360

GBR 1342 Publications*

Publication Type	Phase of Study	Citation
Poster	Phase 1 (GBR 1342-101)	Richter J, et al. Phase 1, multicenter, open-label study of single-agent bispecific antibody t-cell engager GBR 1342 in relapsed/refractory multiple myeloma. The ASCO-SITC Clinical Immuno-oncology Symposium; 2018 Jan 25-27; San Francisco, CA.
Poster (encore)	Phase 1 (GBR 1342-101)	Wermke M, et al. Phase 1, multicenter, open-label study of single-agent bispecific antibody t-cell engager GBR 1342 in relapsed/refractory multiple myeloma. The Annual Meeting of the American Society Of Clinical Oncology; 2018 Jun 1-5; Chicago, IL.

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GRC 17536 Executive summary

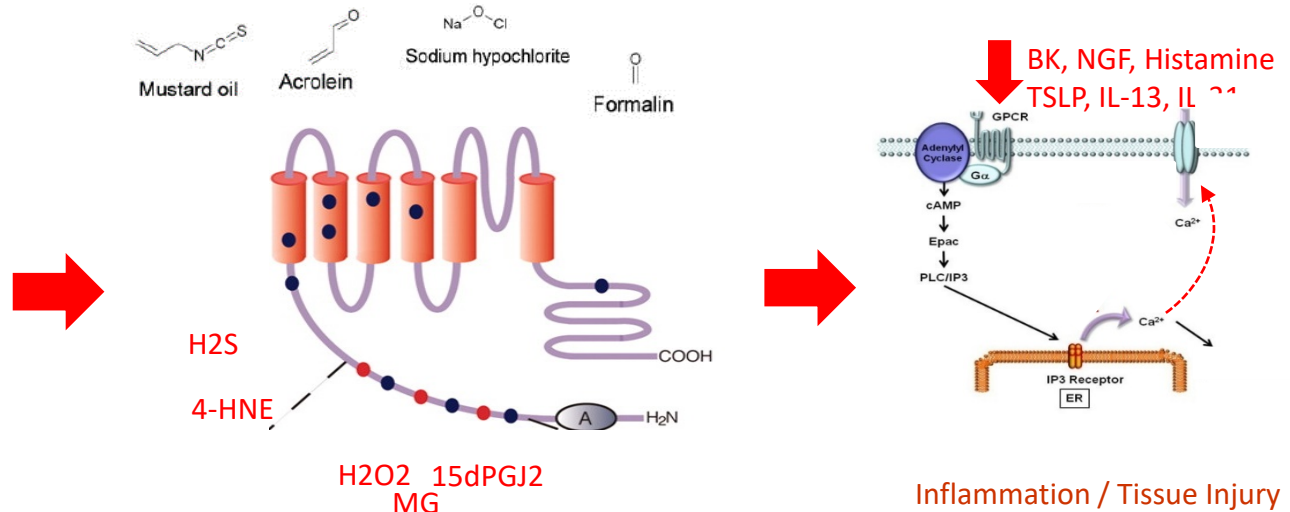
- Novel, non-cannabinoid, non-opioid, non-steroidal TRPA1 antagonist analgesic mechanism with a potential identifiable responder population based on MOA
- Demonstrated dose-dependent modulation of inflammatory and neuropathic pain conditions in rats
- Observed no maternal toxicity or fetal abnormalities in rats and mouse
- Safe and well tolerated based on human Phase 1 studies, after both single and multiple dose administration
- Phase 2a provided proof of concept in moderate to severe pain in patients with diabetic peripheral neuropathy (DPN) having preserved small fiber function
- GCR 17536 is currently on clinical hold from FDA. The hold is to resolve preclinical (nonclinical) issues
 - Working to get the hold lifted and are presently anticipating resumption of clinical activity in the second half of 2019

TRPA1 receptor

Neuronal Receptor



TRPA1



- TRPA1: A poly-modal receptor activated directly by exogenous / endogenous chemical and biochemical stimuli and indirectly by GPCR and cytokine receptor mediated cross talk
- Expressed on peripheral and spinal nerves and implicated in sensory nerve activation due to increased signalling by pathologically relevant ligands during inflammation / pain / nerve injury
- A broad target implicated in several chronic diseases involving sensory nerve activation
 - Chronic pain conditions (Diabetic Peripheral Neuropathy, Chemotherapy Induced Neuropathy , Post Herpetic Neuralgia and Post Traumatic Neuropathy)
 - Dermatological diseases (Pruritus)
 - Respiratory diseases (COPD, Chronic cough, Asthma)

Phase 2a POC study in Painful Diabetic Peripheral Neuropathy (DPN)



Methodology

- Phase 2a randomized, double-blind, parallel group, placebo controlled study to evaluate efficacy, safety and tolerability of oral GRC 17536
- 138 patients with painful DPN - pain for at least 6 months and no greater than 5 years
- Patient willing to withdraw their medication for neuropathic pain for the entire study period
- Treatment arms : Oral 250 mg BID GRC 17536, or Placebo
- Dosing Duration: 4 weeks

Conclusions:

- Clinically meaningful and statistically significant-improvement in pain in patients treated with GRC 17536 compared with placebo in a predefined subgroup of patients with a phenotype indicating functional peripheral small fiber function
- In the ITT population, a small but consistent trend towards reduction in pain scores by GRC 17536, more than placebo across multiple end-points suggests efficacy via TRPA1 blockade

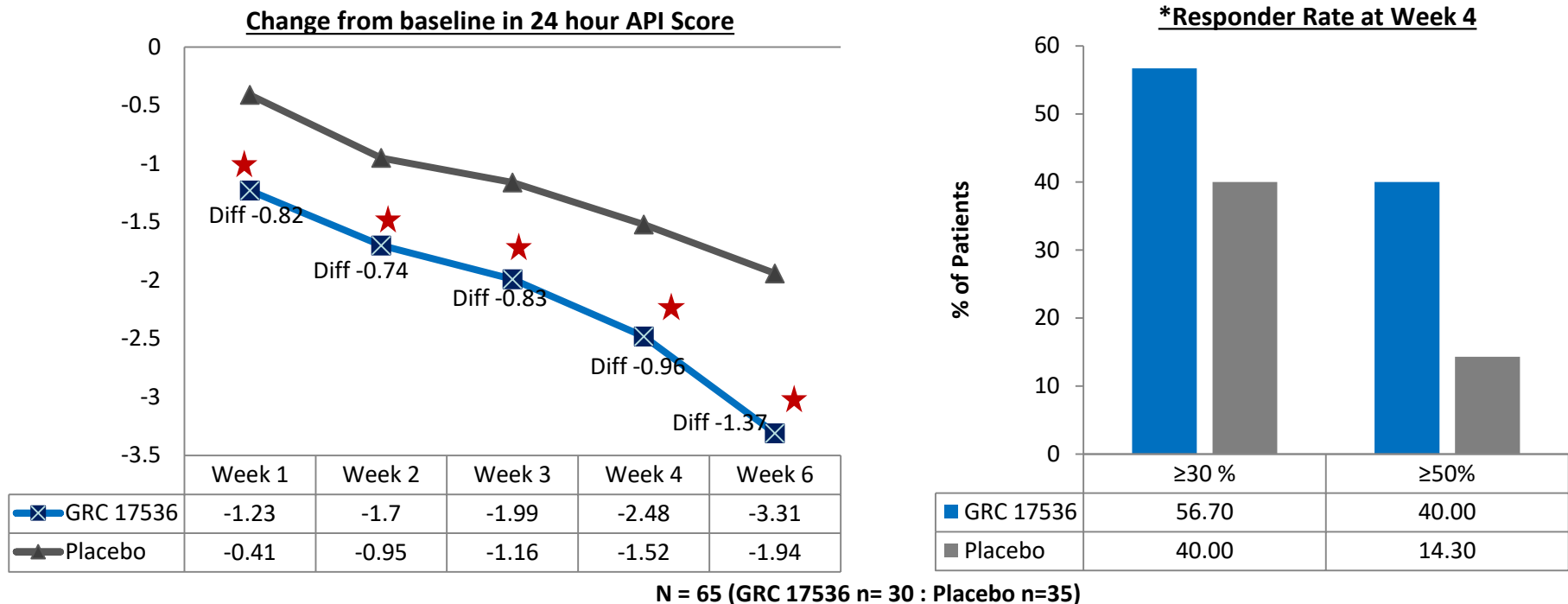
Available data suggests GRC 17536 is peripherally acting for painful diabetic peripheral neuropathy

Phase 2a POC study in Painful Diabetic Peripheral Neuropathy (DPN)



Efficacy: Clinically meaningful and statistically significant reduction in mean 24- hour API pain score by GRC 17536 over placebo in subjects with intact small fiber function (API ≥ 5 and CDT $>18^{\circ}\text{C}$ and/or WDT $<49^{\circ}\text{C}$ at Baseline)

Safety: Safe and well-tolerated; No CNS related adverse events



★ Statistically significant reduction in mean 24 hour API pain score (based on 11-point NRS scale) over placebo

*Responder Rate: Number of patients achieving 30% and 50% of reduction from baseline in the mean 24-hour API score.

API: Average pain intensity; CDT: Cold detection threshold; WDT: Warm detection threshold; NRS: Numeric Rating scale

GRC 17536 Publication

Treatment of Pain Associated with Diabetic Peripheral Neuropathy with the Novel TRPA1 Antagonist GRC 17536 in Patients with Intact Peripheral Nerve Function

M. Tandon¹, S.M. Jain², R. Balamurugan³, M. Koslowski¹, P. Keohane¹

¹Glenmark Pharmaceuticals, Watford, UK, ²TOTAL Diabetes Hormone Institute, Indore, India,

³Koval Diabetes Specialty Centre and Hospital, Coimbatore, India



Introduction

10-20% of patients with Diabetes mellitus develop painful Diabetic Peripheral Neuropathy (DPN). Adverse effects of currently available centrally acting DPN therapies and a low responder rate contribute to suboptimal clinical outcomes in a large proportion of patients resulting in a high unmet need for specific treatments with better safety profile and novel approaches to identify patients likely to respond to treatment. We have developed a potent and selective antagonist (GRC 17536) targeting transient receptor potential ankyrin 1 (TRPA1), a non-selective cation channel that is primarily expressed on pain-mediating primary afferent peripheral nerve fibers. Activation of TRPA1 by reactive metabolites generated in diabetic patients (e.g. advanced glycation products) has been identified as a mediator of DPN pain and is discussed to have an important role in the pathophysiology of DPN. GRC 17536 shows dose-dependent modulation of neuropathic pain in various preclinical models with efficacy comparable to current standard of care drugs. GRC 17536 showed good safety and tolerability without any signs of supra-spinal mechanism of action in Phase 1a/b healthy volunteer studies up to 500mg daily dose.

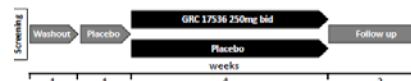
Objectives

We performed a Phase 2a proof-of-concept study to assess efficacy, safety and tolerability of oral GRC 17536 in patients with painful DPN. Based on TRPA1 expression on small nerve fibers GRC 17536 was expected to be more effective in DPN patients with preserved small peripheral nerve fiber function compared to those patients with progressive loss of small nerve fibers. Quantitative sensory testing (QST) was applied in the total DPN population to prospectively identify responder populations and assess GRC 17536 efficacy in distinct pain phenotypes based on differences in small nerve fiber function.

Methods

Study Design

Randomized, double-blind, parallel group, placebo controlled study. Patients with painful DPN for at least 6 months and no greater than 5 years. Patients had to withdraw their neuropathic pain medication for the entire study period. Treatment arms: oral GRC 17536 250mg bid, or placebo 4 weeks treatment administration followed by 2 week follow up



Primary Endpoint

Change from baseline to end of treatment (EOT) in the mean 24-hour API score based NRS

Secondary Endpoints

Number of patients achieving 30% and 50% of reduction from baseline in the mean 24-hour API score.

The change from baseline to EOT in

- Mean night-time API Score
- Mean night-time worst pain intensity Score
- Mean sleep interference Score.
- Patient Global Impression of Change (PGIC)
- Clinician Global Impression of Change (CGIC)
- Mean daily dose of rescue medication
- Neuropathy Pain Symptom Inventory (NPSI)
- Quantitative Sensory Testing (QST)

Time to onset of sustained improvement

Effect of GRC 17536 in patients with painful DPN who have mechanical hyperalgesia and/or cold allodynia

Results

- ITT population: 136 patients with painful DPN from Germany, India and Czech Republic
- 70 patients were randomized to GRC 17536, 66 patients to placebo

Table 1: Demographics of ITT population

Demographics	GRC 17536	Placebo	Total
Male	51	49	99
Female	19	23	42
Age (years)	55.0	57.3	56.1
Weight (kg)	75.9	73.1	75.5
Baseline pain score (NRS)	6.0	6.1	6.0
Time since diagnosis of DPN (months)	27.2	25.8	26.8
Time since diagnosis of DM (years)	16.2	9.6	9.9

Efficacy

Figure 1: Primary endpoint in ITT population

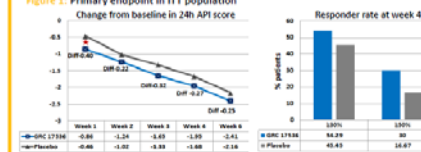
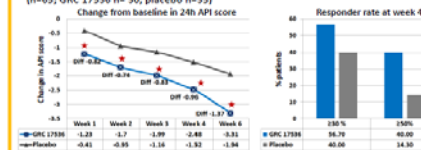


Figure 2: Primary endpoint in patients with API ≥5 and cold detection threshold >18°C and/or warm detection threshold <49°C at baseline as determined by QST (n=65; GRC 17536 n=30, placebo n=35)



Safety

Table 2: Adverse events with incidence >2% in total study population

Predefined Term	GRC 17536 n (%)	Placebo n (%)	Total n (%)
Abdominal distension	1 (1.4)	2 (3.0)	3 (2.2)
Constipation	2 (2.8)	0	2 (1.4)
Dyspepsia	2 (2.8)	3 (4.5)	5 (3.6)
Pain	2 (2.8)	0	2 (1.4)
Pyrexia	1 (1.4)	3 (4.5)	4 (2.9)
Increase in serum CRP	1 (1.4)	3 (4.5)	4 (2.9)
Increase in serum potassium	2 (2.8)	2 (3.0)	4 (2.9)
Hyperglycemia	2 (2.8)	2 (3.0)	4 (2.9)
Hypoglycemia	0	3 (4.5)	3 (2.2)
Dyspnoea	2 (2.8)	1 (1.5)	3 (2.2)
Pruritis	0	2 (3.0)	2 (1.4)
Throat irritation	1 (1.4)	3 (4.5)	4 (2.9)

Conclusions

- GRC 17536 shows a small but consistent trend towards reduction in pain scores more than placebo across multiple end-points in the ITT population.
- GRC 17536 shows clinically meaningful and statistically significant efficacy over placebo in a prospectively defined subgroup of patients (48% of ITT population) with a pain phenotype indicating functional peripheral small fiber function based on thermal threshold detection.
- GRC 17536 shows no CNS adverse events and has a safety profile similar to placebo.
- TRPA1 blockage using GRC17536 offers a novel therapeutic approach and peripherally acting mechanism for pain control, with a low potential for CNS adverse effects.

Treatment of pain associated with diabetic peripheral neuropathy with the novel TRPA1 antagonist GRC 17536 in patients with intact peripheral nerve function.

In Proceedings of the Fifth International Meeting of The Special Interest Group on Neuropathic Pain (NeuPSIG), Nice, France, 14–17 May 2015.

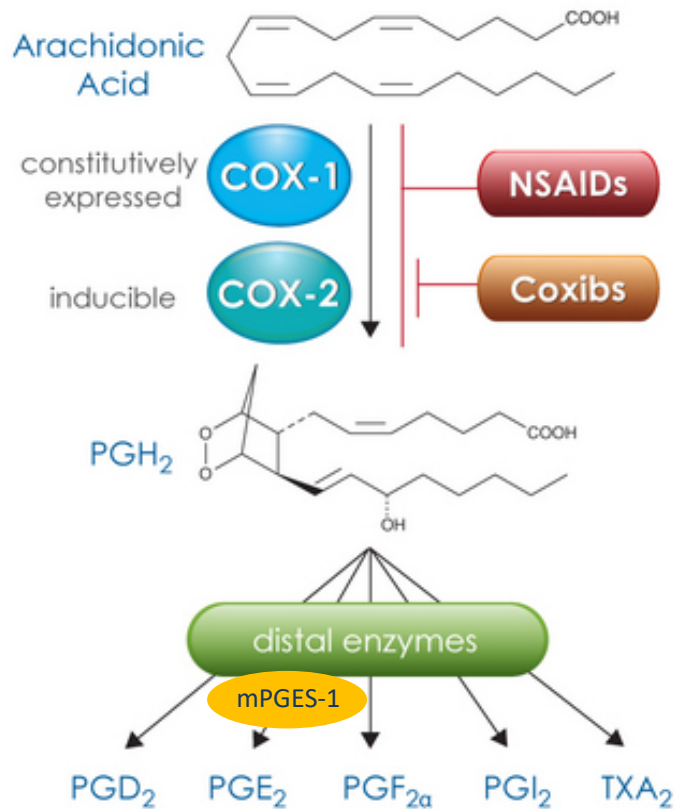
Agenda

- Glenmark Overview
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 - GBR 830
 - GBR 1302
 - GBR 1342
 - GRC 17536
 - **GRC 27864**
- Conclusion

GRC 27864 Executive summary

- A potent, selective, and orally bioavailable inhibitor of mPGES-1
- First-in-class mPGES-1 inhibitor currently undergoing clinical development for osteoarthritis (OA) pain of knee and hip.
- Safety, tolerability, and PK/PD evaluated in three Phase 1 studies in healthy volunteers in UK and France
 - Single dose tolerability studied up to 1000 mg
 - Multiple dose (28 days) tolerability studied up to 130 mg. Most common AEs observed were nausea, diarrhea and abdominal pain.
 - Formulation bridging study with tablet formulation successfully completed
 - No dose-limiting toxicities observed in Phase 1 studies
 - Pharmacodynamic and urinary biomarkers (prostanoid metabolites) evaluated in Phase 1 studies confirming PGE₂ inhibition without suppression of other prostanoids (prostacyclin and thromboxane)
- Phase 2b study currently in progress

mPGES-1 Inhibition: Blocking PGE₂ in a “new way” that is efficacious & safe



Novel biochemical MOA of GRC 27864

- Selectively inhibit inflammatory PGE₂ without effecting cardio-protective PGI₂ & TXA₂
- Do not inhibit COX-1 or COX-2 enzyme activity
- Do not inhibit multiple, constitutively produced prostanoids
- Novel biochemical MOA distinct from tNSAIDs/COXIBs
- Downstream blockade at mPGES-1 is also expected to translate into analgesic benefit with improved GI, renal and CV safety in man

Selective mPGES-1 inhibitors are expected to relieve pain while mitigating the dose limiting GI, renal and CV side effects of tNSAIDs and COXIBs

GRC 27864 Pre-clinical studies

In vitro profile

- Exhibits potent inhibition of human, guinea pig and dog mPGES-1 enzyme
- Shows no significant inhibitory effect on mouse, rat, rabbit and monkey enzyme up to a conc. of 10 μ M

Ex vivo profile

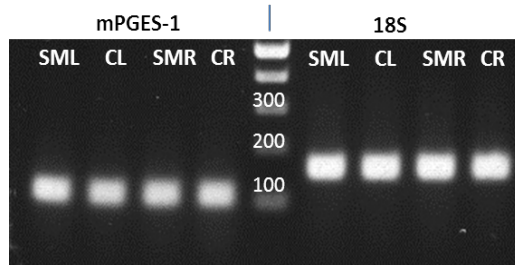
- Target Engagement in OA, RA and Periodontitis Patient Biopsy Tissues
- Observed inhibition of PGE₂ release from OA patient tissue explants
- Inhibits LPS induced PGE₂ release in blood of OA and RA patients with IC50 comparable to Celecoxib
- Inhibits PGE₂ release from gingival tissues of periodontitis patients

In vivo profile

- Shows dose dependent modulation of analgesia in animal models of pain, osteoarthritis & inflammatory arthritis comparable to standard of care (Celecoxib, Naproxen)
- Selectively attenuates PGE₂ while sparing other prostanoids in guinea pig and dog
- Selectively inhibits LPS induced PGE₂ release in whole blood of healthy subjects as well as OA patients at therapeutically relevant plasma concentrations

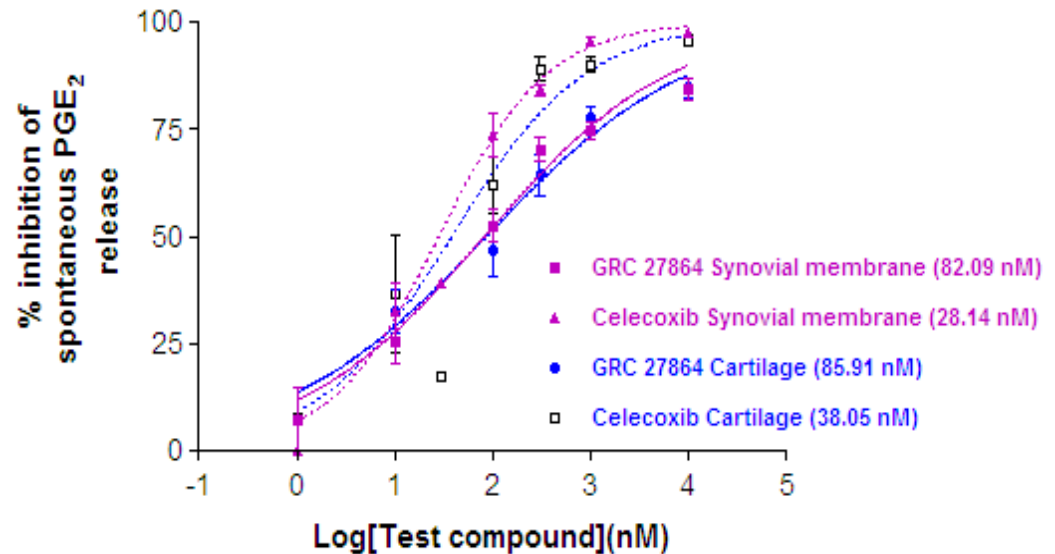
GRC 27864 – Inhibition of PGE₂ release from OA patient tissue explants

PCR Gel Electrophoresis



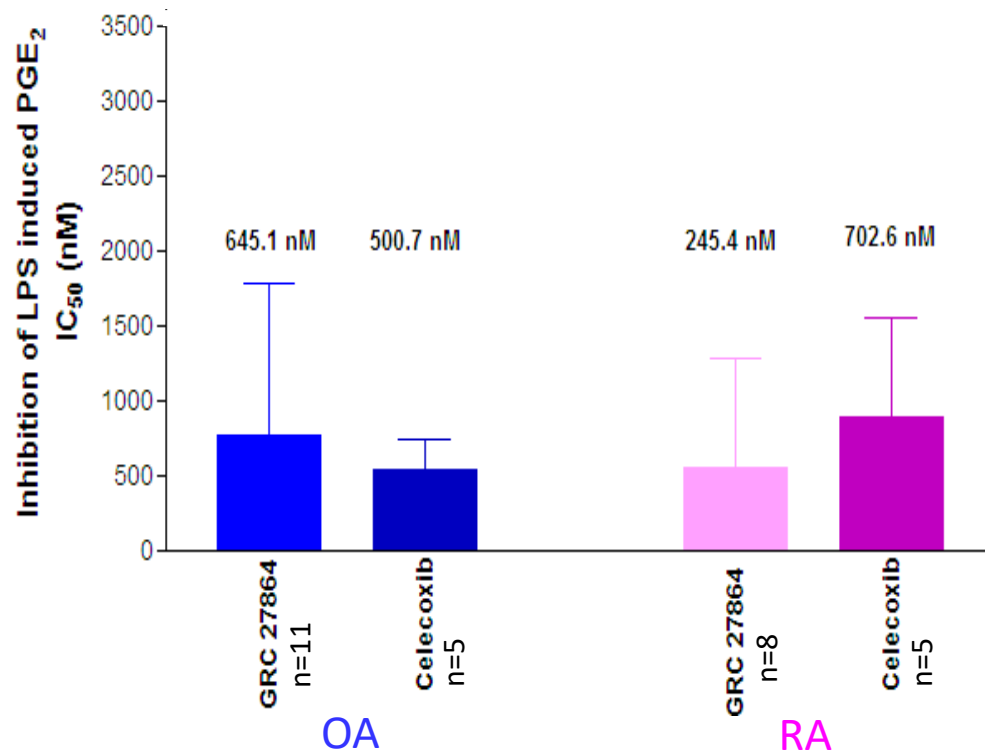
SML – left knee synovial membrane
SMR – right knee synovial membrane
CL – left knee cartilage
CR – right knee cartilage

Robust mPGES-1 expression was observed in osteoarthritis patient synovial membrane and cartilage



In synovial and cartilage tissues of OA patients, GRC 27864 exerts robust *ex vivo* PGE₂ inhibition of >70% at clinically relevant concentrations

GRC 27864 – PGE₂ release Inhibition: OA and RA patient blood



GRC 27864 inhibits LPS induced PGE₂ release in blood of OA and RA patients with IC₅₀ comparable to Celecoxib

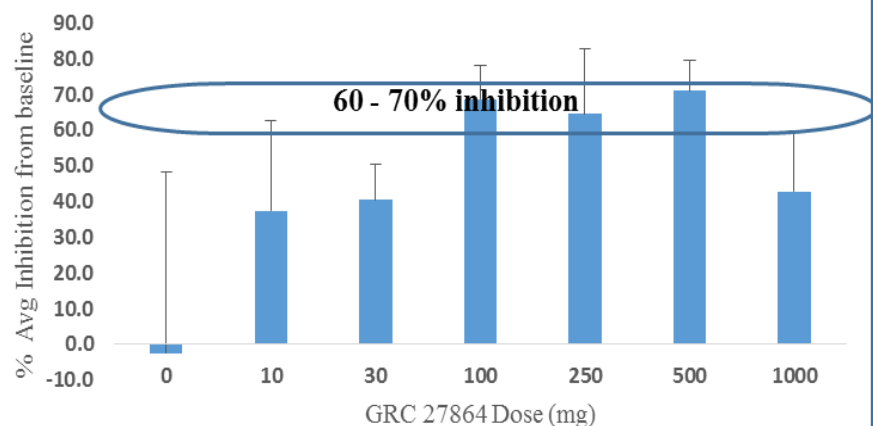
GRC 27864 Phase 1 – Study summary

- Safety, tolerability and PK and PD evaluated in three Phase 1 studies in healthy volunteers in UK and France.
- Single dose tolerability studied up to 1000 mg in healthy volunteers
 - Multiple dose (28 days) tolerability studied up to 130 mg
 - Formulation bridging study with tablet formulation successfully completed.
 - No dose limiting toxicities observed in Phase 1 studies
- Pharmacodynamic and urinary biomarkers (prostanoid metabolites) evaluated in Phase 1 studies confirming PGE₂ inhibition without suppression of other prostanoids (prostacyclin and thromboxane).
- Showed robust inhibition of ex-vivo LPS induced release of PGE₂ release reflecting its clinically relevant anti-inflammatory potential, during SAD and MAD studies, comparable to Celecoxib

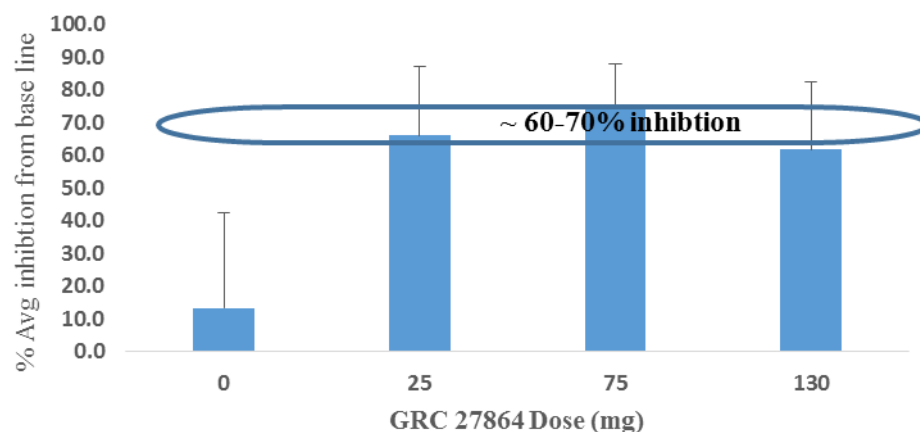
GRC 27864 Phase 1 – Study summary

Ex vivo LPS-induced PGE₂ release whole blood from SAD and MAD Studies

SAD - Dose vs Avg (0-24 hr) LPS induced PGE₂ inhibition



MAD - Dose vs Avg (0-24 hr) LPS induced PGE₂ inhibition (Day 28)



- After a single dose administration, GRC 27864 inhibited ex vivo LPS-induced PGE₂ release (Eavg_{0-24h}) in the range of 40 to 71% compared to placebo. ~60-70% inhibition was maintained in the 100-500 mg dose range.
- Further, the inhibition (~ 60%) was comparable between GRC 27864 (1000 mg) and celecoxib (400 mg)

Ref: GRC 27864, Novel, Microsomal Prostaglandin E Synthase-1 Enzyme Inhibitor: Phase 1 Study To Evaluate Safety, PK and Biomarkers In Healthy, Adult Subjects. Osteoarthritis Research Society International, OARSI 2018 World Congress, Liverpool, UK, April 2018 Volume 26, Supplement 1, Pages S351–S352

Following multiple oral doses of GRC 27864 from 25 to 130 mg QD,

- There was a marked drug-related inhibition of ex vivo LPS-induced PGE₂ release on Days 1, 10 and 28.
- Onset and extent of inhibition was generally similar across the dose range and dosing days, with mean Epeak values ranging from 78% to 90%.
- Near maximum inhibition was retained over each 24-hour dosing period, with Eavg_{0-24h} inhibition ranging from 60% to 71%

Ref: Phase 1 study to assess the Safety, Tolerability, Pharmacokinetics and Pharmacodynamics of Multiple Ascending Doses (MAD) of GRC 27864 oral administration in Healthy or Elderly volunteers IASP 17TH WORLD CONGRESS ON PAIN, September 12-16, 2018 Boston, USA

GRC 27864 – Differential mechanism of action validated through biomarker data



- In Phase 1 studies GRC 27864 showed marked inhibition of ex-vivo LPS-induced PGE2 release and decrease in urinary excretion of tetranor PGEM* without any decrease in urinary excretion of other prostanoid metabolites (prostacyclin and thromboxane).
- Pharmacodynamic and urinary biomarkers (prostanoid metabolites) evaluated in Phase 1 studies confirm PGE2 inhibition without suppression of other prostanoids that supports differential mechanism of action.
- Pharmacodynamic and prostanoid biomarkers are being evaluated in the ongoing Phase II study of GRC 27864 in patients of hip and knee osteoarthritis to confirm the novel and differentiated mechanism of action.

*PGEM: PGE metabolites

GRC 27864 Phase 2b study design

- Patient Population – OA (Hip or Knee)
- Randomized, double-blind, parallel group, placebo-controlled study
- Treatment Duration: 12 weeks
- Treatment arms: N=624 (156 per arm)
 - Arm 1: GRC 27864, 10 mg QD for 12 weeks
 - Arm 2: GRC 27864, 25 mg QD for 12 weeks
 - Arm 3: GRC 27864, 75 mg QD for 12 weeks
 - Arm 4: Placebo, QD for 12 weeks
- Primary endpoint: Mean change from baseline in pain intensity end of 12 weeks of treatment in the most severely affected joint (target joint) over the previous 48 hours, using a 100mm Visual Analogue scale
- Pharmacodynamic and urinary biomarkers being evaluated to support novel and differentiated mechanism of action.
- Current status:
 - ~200 patients randomized across 30+ sites
 - Top line results expected in Q3 CY19

GRC 27864 Publications

- GRC 27864, novel, microsomal prostaglandin E synthase-1 enzyme inhibitor: phase 1 study to evaluate safety, PK and biomarkers in healthy, adult subjects
 - Osteoarthritis Research Society International, OARSI 2018 World Congress, Liverpool, UK, April 2018 Volume 26, Supplement 1, Pages S351–S352 [https://www.oarsijournal.com/article/S1063-4584\(18\)30798-2/pdf](https://www.oarsijournal.com/article/S1063-4584(18)30798-2/pdf)
- Phase 1 study to assess the Safety, Tolerability, Pharmacokinetics and Pharmacodynamics of Multiple Ascending Doses (MAD) of GRC 27864 oral administration in Healthy or Elderly volunteers
 - IASP 17th World Congress On Pain, September 12-16, 2018 Boston, USA
<https://www.iaspworldcongressonpain.org/wp-content/uploads/2018/11/Program-Book-Final-only-posters.pdf>
- A three-cohort phase I study to evaluate the pharmacokinetics of GRC 27864 after single dose oral administration of a tablet formulation (25 mg, 50 mg and 75 mg) and relative bioavailability of the tablet formulation in comparison to the granule formulation of 75 mg
 - Annual Conference Of Indian Rheumatology Association, 6-9th December, 18. Guwahati, Assam
<http://www.indianjrheumatol.com/article.asp?issn=0973-3698;year=2018;volume=13;issue=6;spage=93;epage=241;aulast=>

Agenda

- Glenmark Overview
- R&D Capabilities
- Information on assets in clinical development
 - GBR 830
 - GBR 1302
 - GBR 1342
 - GRC 17536
 - GRC 27864
- **Conclusions**

Glenmark's Innovative R&D pipeline is based on robust scientific rationale and driven by experienced team



Rich pipeline addressing unmet needs across multiple therapy areas and potential indications

Leadership team derived from Big Pharma and Biotech with experience of developing and launching blockbuster molecules

Strong business cash flows to support ongoing clinical development

Strong network of KOLs and academia for continuous feedback on programs

Selective licensing partnerships to share risk and leverage additional capabilities



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A new way for a new world