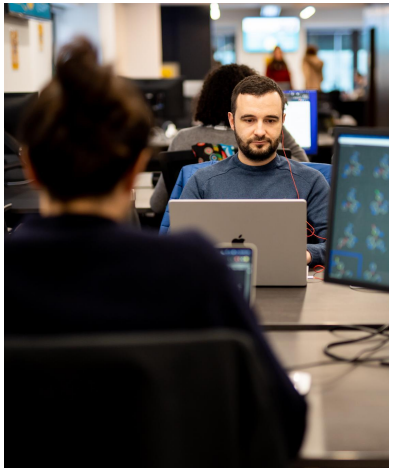


BenevolentAI
Preliminary results for the year ended 31 December 2022

16 March 2023

Benevolent AI

Disclaimer

Forward-Looking Statements

This document may contain forward-looking statements. Forward-looking statements are statements that are not historical facts and may be identified by words such as "plans", "targets", "aims", "believes", "expects", "anticipates", "intends", "estimates", "will", "may", "should" and similar expressions. Forward-looking statements include statements regarding objectives, goals, strategies, outlook and growth prospects; future plans, events or performance and potential for future growth; economic outlook and industry trends; developments in BenevolentAI's markets; the impact of regulatory initiatives; and/or the strength of BenevolentAI's competitors. These forward-looking statements reflect, at the time made, BenevolentAI's beliefs, intentions and current targets/aims. Forward-looking statements involve risks and uncertainties because they relate to events and depend on circumstances that may or may not occur in the future. The forward-looking statements in this release are based upon various assumptions based on, without limitation, management's examination of historical operating trends, data contained in BenevolentAI's records, and third-party data. Although BenevolentAI believes that these assumptions were reasonable when made, these assumptions are inherently subject to significant known and unknown risks, uncertainties, contingencies and other important factors which are difficult or impossible to predict and are beyond BenevolentAI's control.

Forward-looking statements are not guarantees of future performance and such risks, uncertainties, contingencies and other important factors could cause the actual outcomes and the results of operations, financial condition and liquidity of BenevolentAI or the industry to differ materially from those results expressed or implied by such forward-looking statements. The forward-looking statements speak only as of the date of this release. No representation or warranty is made that any of these forward-looking statements or forecasts will come to pass or that any forecast result will be achieved.

AGENDA

- The Benevolent Platform™ & Business Model
  Joanna Shields, CEO
- Preliminary Results for the year ended 31 December 2022
  Nick Keher, CFO
- Pipeline review
  Anne Phelan, CSO
- Outlook
  Joanna Shields, CEO

2022: A year of solid progress and growth for BenevolentAI

Enhanced the Benevolent Platform™, progressed our pipeline and delivered in commercial collaboration with AstraZeneca

BEN-2293: Completed Phase IIa study and expect top-line data in Q1 2023

BEN-8744: Submitted CTA in Dec 2022, expect to initiate a Phase I clinical trial in H1 2023

BEN-28010: Declared as a clinical candidate in July 2022, with preparation for IND-enabling studies ongoing

3 **year collaboration expansion** with **AstraZeneca's** into 2 new disease areas

3 **additional novel targets** selected for **AstraZeneca's** portfolio

3 in-house assets transitioned into **lead optimisation**

4 **new in-house drug programmes** generated using the Benevolent Platform™

FDA **approval** of **COVID-19 treatment** (baricitinib) first identified by BenevolentAI

BenevolentAI is a leading AI-enabled drug discovery & development company with a platform-generated pipeline

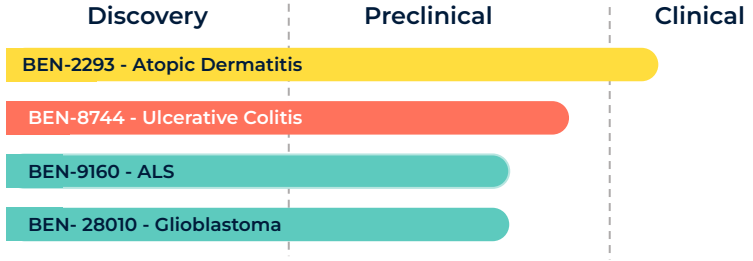
DISCOVERY

Industry-leading AI platform uncovers novel biology across diseases and drug modalities

- **Expansive knowledge graph** with multimodal data foundations
- **Disease** and **drug modality agnostic**
- Generate predictions with the aim to increase the **probability of success**

DEVELOPMENT

Portfolio of first-in-class and best-in-class programs, discovered using our proprietary AI platform



1 asset in **Phase II**

3 assets in **pre-IND/CTA**

15 **Platform-generated drug programmes**

Powerful data foundations generate a 360° view of disease biology

Experiments
Assay Data (Binding, Omics Comparison, CRISPR Screens)
Clinical Trial

Literature
Scientific Literature
Patent Literature
Regulatory Documents

OMICS
Genes
Proteins
Isoforms
Transcripts & Variants

Aetiology
Diseases
Symptoms

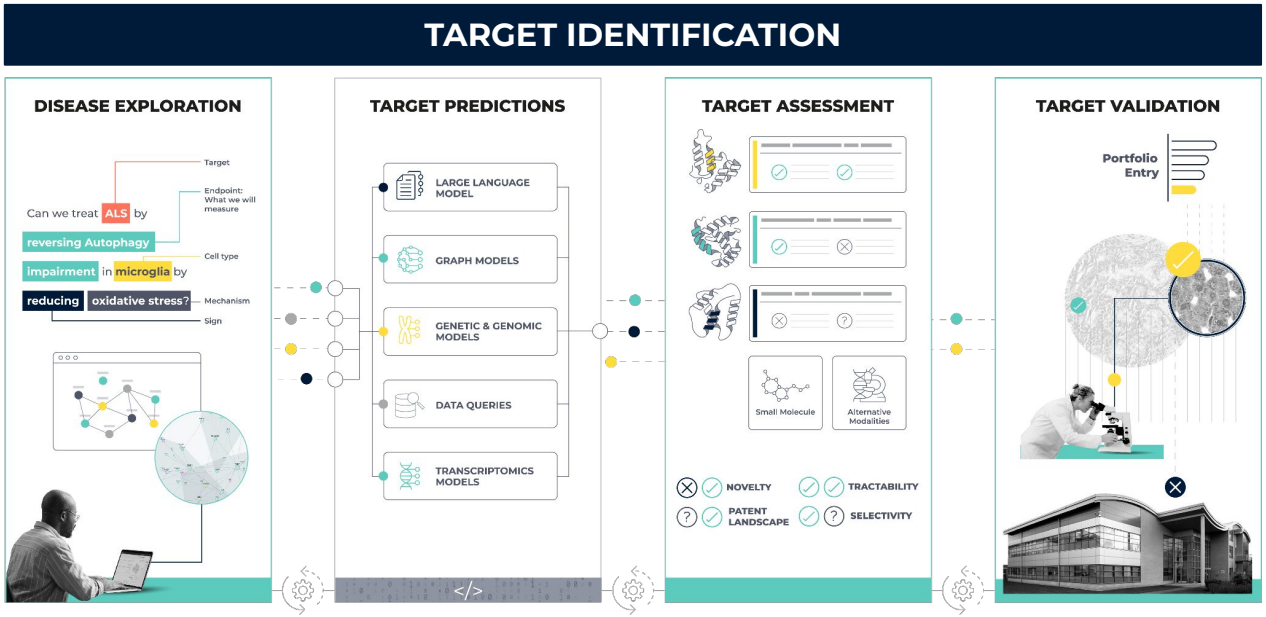
Biological Systems
Cellular Component
Molecular Function
Biological Process
Mechanism
Pathways

Molecules
Organic Compounds
Preclinical Candidates
Approved Drugs
Antibodies
Other Biologics
Pharmacology
Pharmacokinetics

- **Multimodal approach** combines over 85 data sources to provide a holistic view of disease biology
- **Maximise our probability of clinical success** by integrating disease traits, genetics and genomics data to generate endotype-specific target predictions
- **Breaks down silos** across therapeutic areas to connect shared mechanisms across disease
- **Provides a proprietary integrated view of biomedical data** that supports discovery and decision-making

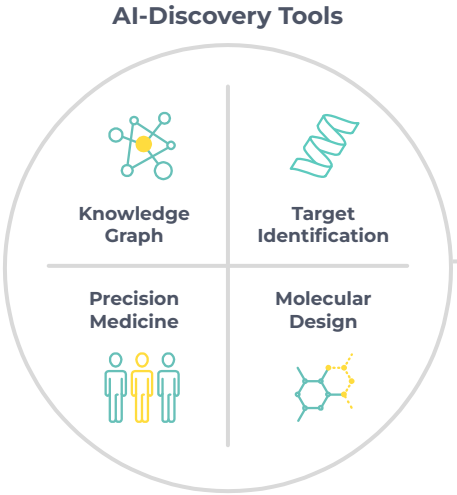
The Benevolent Platform™ empowers scientists with industry-leading drug discovery AI

Comprehensive data foundations **Biology first** **Hypothesis driven**



- Proprietary AI models reason across multi-modal data to **discover novel targets**
- Enables scientists to **assess** & **select** only the **most promising targets** to take into wet lab experiments
- Efficiently **surfaces scientific evidence** to support higher confidence decisions
- **Data fed back** into the Knowledge Graph to enhance future predictions

Our flexible business model unlocks multiple routes to value creation



OWNED PIPELINE
- Platform-generated assets
- In-house development

LICENSING
- Platform-generated assets
- Out-licensed at IND, end Phase I or end II

PLATFORM COLLABORATIONS
- Economic Benefits
- Platform Validation
- Data generated enriches the Benevolent Platform™

NON-COMMERCIAL COLLABORATIONS
- ESG
- Platform Validation
- Data generated enriches the Benevolent Platform™

Preliminary Results for the year ended 31 December 2022

Nick Keher, CFO

2022 Financial highlights

	31 December	
	2022	2021
	£'000	£'000
Revenue	10,560	4,625
R&D - Drug discovery ("DD")[1]	(43,179)	(27,129)
R&D - Product & technology ["P&T"][1]	(21,914)	(19,963)
G&A - Business operations ["Bus Ops"][1]	(16,500)	(13,944)
Underlying expenses related to share-based payments	(23,731)	(51,390)
Other income	166	90
Normalised operating loss	**(94,598)**	**(107,711)**
Normalised EPS (in pence)[2]	(72.6)	(104.6)
Weighted average ordinary shares outstanding (in millions)	109.1	89.9

Revenue increase across AstraZeneca collaboration, with 3 additional novel targets in chronic kidney disease (x1) and idiopathic pulmonary fibrosis (x2).

DD spend increase driven by advancing pipeline into later stages of development, particularly BEN-2293 adaptive Phase I/II and BEN-8744 CTA filing enablement in Dec-22. Net 4 named programmes added into pipeline during the year.

P&T spend increase reflecting increased headcount, expected to plateau.

Bus Ops spend +18%, driven predominantly by listing status and expected to maintain at this level.

1) Excludes exceptional costs related to the Business Combination
2) Normalised EPS also excludes taxation impact from exceptional items and finance income related to the Business Combination

Reported to Normalised[1]

	31 December	
	2022	2021
	£'000	£'000
Operating loss	**(197,034)**	**(121,322)**
Adjustments for:		
G&A - Direct Transaction costs	11,255	2,911
G&A - Transaction-related listing service SBP expense	83,067	-
G&A - Transaction-related employee-related SBP expense	3,883	-
G&A - Impairment of assets	-	10,700
G&A - Transaction-related stamp duty	3,740	-
Revaluation of investments	491	-
Normalised[1] group operating loss	**(94,958)**	**(107,711)**

Reported operating loss significantly driven by non-recurring costs of Business Combination.

Non-cash exceptional costs related to acceleration of share-based payments also incurred from Business Combination, and set to reduce beyond 2022.

Non-cash listing service expenses reflects cost of share issuance to Odyssey SPAC as part of Business Combination net of assets acquired (before PIPE and backstop)

1) Excludes exceptional costs related to the Business Combination

Cash flows focused upon drug and platform development

	31 December 2022 £'000
Normalised[1] operating loss	**(94,598)**
Depreciation & amortisation	3,058
Foreign exchange	(3,141)
Other employee-related SBP expense	29,935
Cash flows from changes in working capital	(13,094)
Cash expended from underlying operating activities	**(77,840)**
Opening cash, cash equivalents and short-term deposits	40,553
Closing cash, cash equivalents and short-term deposits	130,182

£1.7m property-related leases.

£3.2m gain from EUR holdings; £0.3m gain from USD holdings; £0.4m charge from operational.

Non-Transaction-related equity awards removed from the P&L (no cash impact)

Largely driven by increase in R&D tax credit receivable (£4.0m), as well as decrease in SBP employer-related tax provision (£6.2m).

Year end cash position of £130.2m at the top end of our stated guidance.

1) Excludes exceptional costs related to the Business Combination

Cash runway to Q4-2024 providing sufficient capital for key value inflection points

Cash Runway

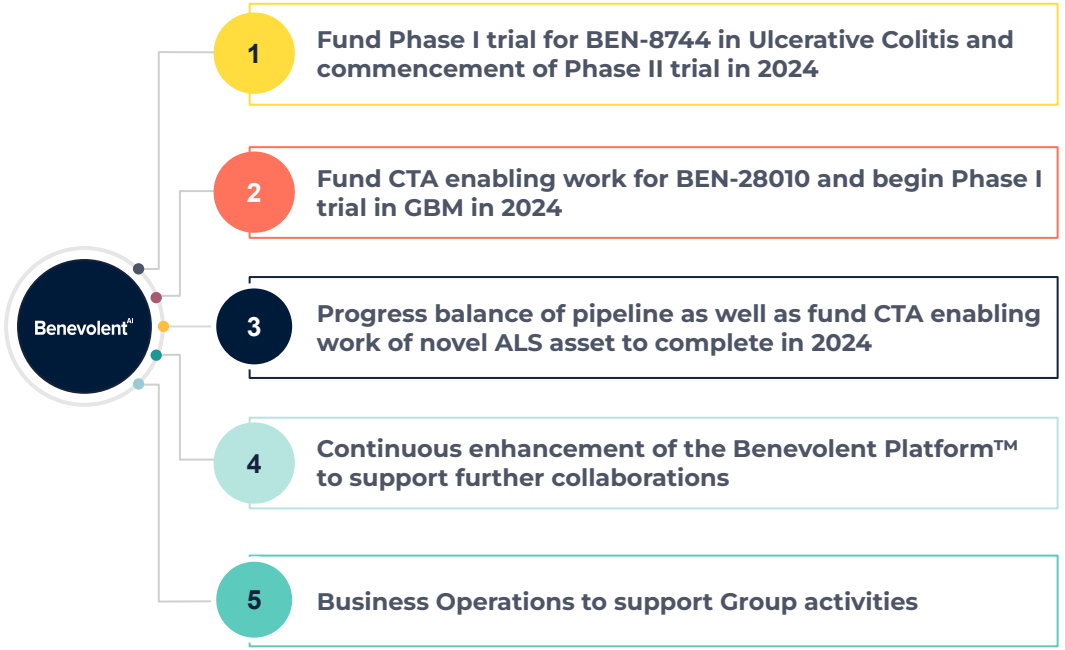
Cash[1] at 31 December 2022	£130.2m

2022 cash burn of £64.7m before working capital movements

Cash runway guidance assumes no future capital from licensing or collaboration agreements

Multiple assets at, or close to, key value inflection points

Capital allocation

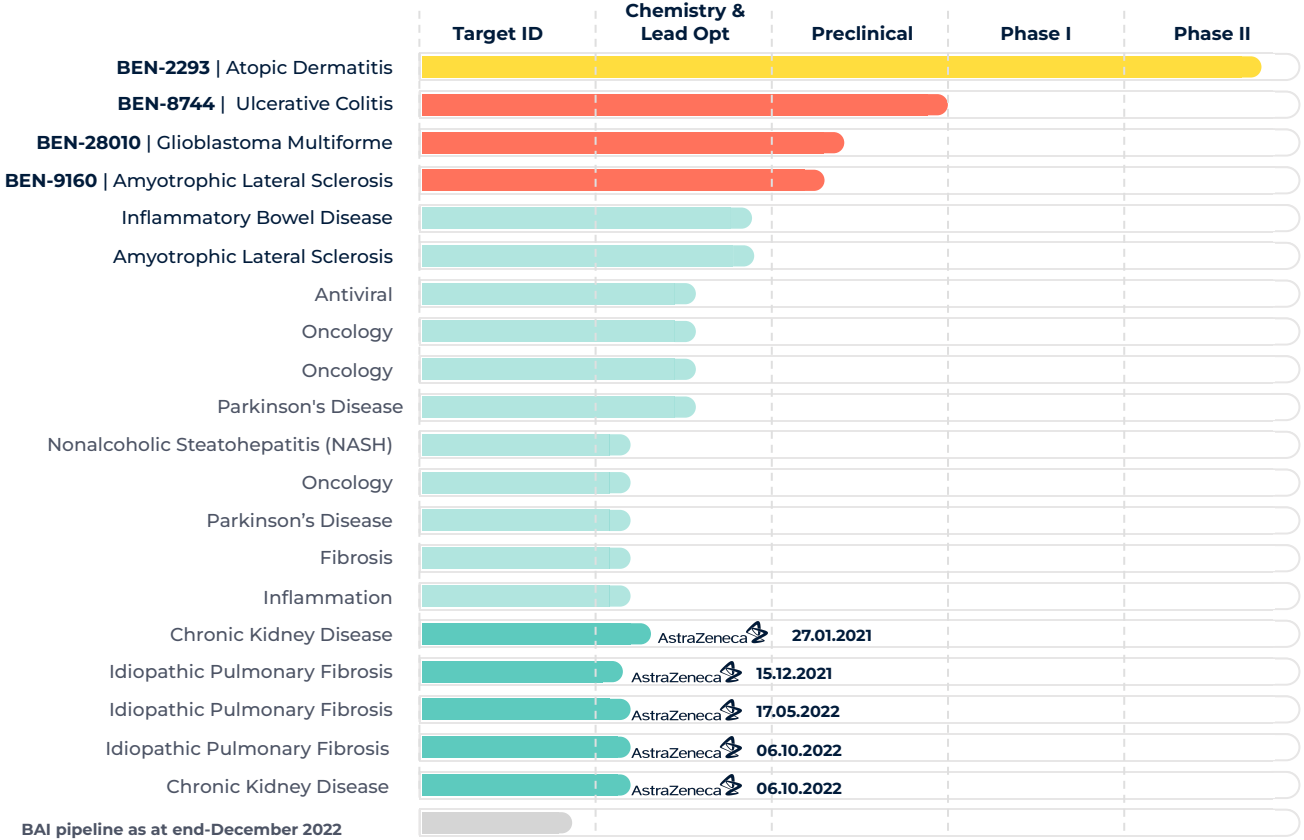


1) Includes cash, cash equivalents and short-term deposits (maturity between 3 and 12 months).

Pipeline review

Anne Phelan, CSO

Robust pipeline entirely generated by the Benevolent Platform™



BAI pipeline as at end-December 2022

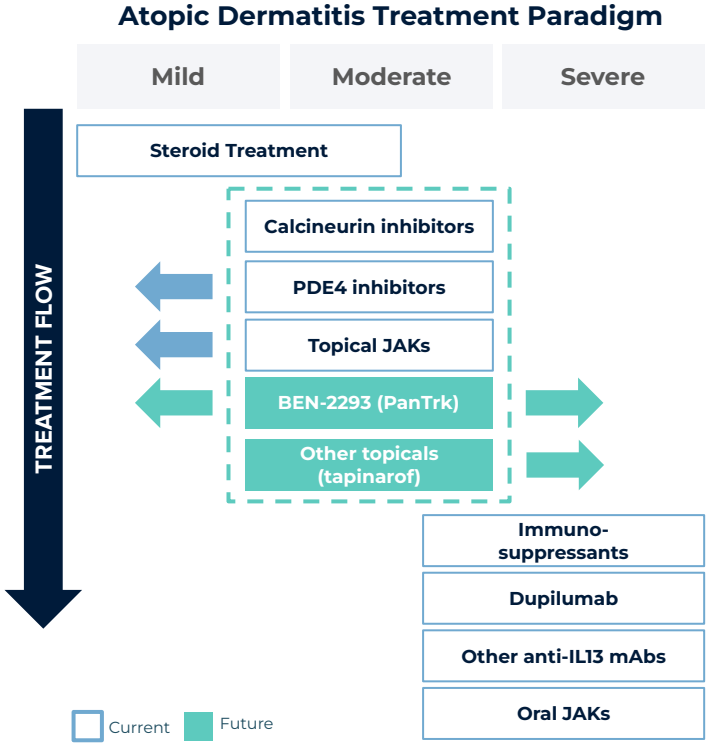
+10 Exploratory stage programmes

Highlights

- **All pipeline programmes** generated using the **Benevolent Platform™**
- **BEN-2293**: Phase IIa read out Q1-2023
- **BEN-8744**: **Novel target** with zero prior linkage to UC. Delivered **drug candidate within 2 years** from programme initiation, **starting Phase 1 H1-2023**
- **AstraZeneca:** multi-year Target-ID collaboration **expanded in Jan 2022** to include systemic lupus erythematosus & heart failure.

BEN-2293 for Atopic Dermatitis

- Atopic dermatitis (AD) is the **most common chronic inflammatory skin disease**, characterized by intensely itchy, red, and swollen skin[1]
  - Affects **10-20% of children** and up to **3% of adults**[2]
  - Approximately **60-70% of all cases** present with mild-moderate disease severity[3]
  - Prevalence is rising[3], with market value in 7MM **forecast to exceed $16.7 billion** by 2030[4]
- BEN-2293 is expected to **treat atopic dermatitis** by: inhibiting **itch signaling** and blocking nerve sensitization (TrkA) in addition to inhibiting Th1 /Th2-mediated **dermal inflammation** (TrkB, TrkC).
- Using our Molecular Design expertise we were able to design a PanTrk inhibitor, equipotent against the 3 receptors



BEN-2293 will target **Mild, Moderate and Severe AD patients**, addressing unmet need as a steroid sparing alternative and in more severe patients undergoing treatment with systemics (e.g. dupilumab) that require add-on treatment

Sources: (1) Weidinger et al. Nat Rev Dis Primers 2018; (2) GlobalData Report 2018: Atopic Dermatitis: Global Drug Forecast and Market Analysis to 2027; (3) GlobalData Report 2018: Atopic Dermatitis: Epidemiology Forecast to 2027; (4) GlobalData Report 2022: Atopic Dermatitis: Global Drug Forecast and Market Analysis to 2030

BEN-2293 Phase IIa clinical study designed to demonstrate efficacy in mild-moderate Atopic Dermatitis

STUDY DESIGN: SAFETY & EFFICACY

Recruitment completed end of 2022
91 patients randomised 1:1 for BEN-2293:Vehicle

Patients (Inclusion Criteria)
- Mild-Moderate AD
  - Baseline vIGA 2-3
  - Baseline Itch NRS ≥4
  - Atopic dermatitis affecting 1-30% BSA of treatable skin
- 18-65 years

Dosing
- 7 day medication washout followed by 3 day placebo run-in prior to dosing
- 1% w/w BEN-2293, or placebo/vehicle applied twice daily to treatable lesioned skin up to 30% BSA
- 28 days on treatment

STUDY EFFICACY OUTCOMES

Itch
- Change from baseline in the **Pruritus NRS** (Worst itch over 24 hours *and* Current itch)
- **Time to itch reduction**
- Fraction of patients achieving **itch reduction**

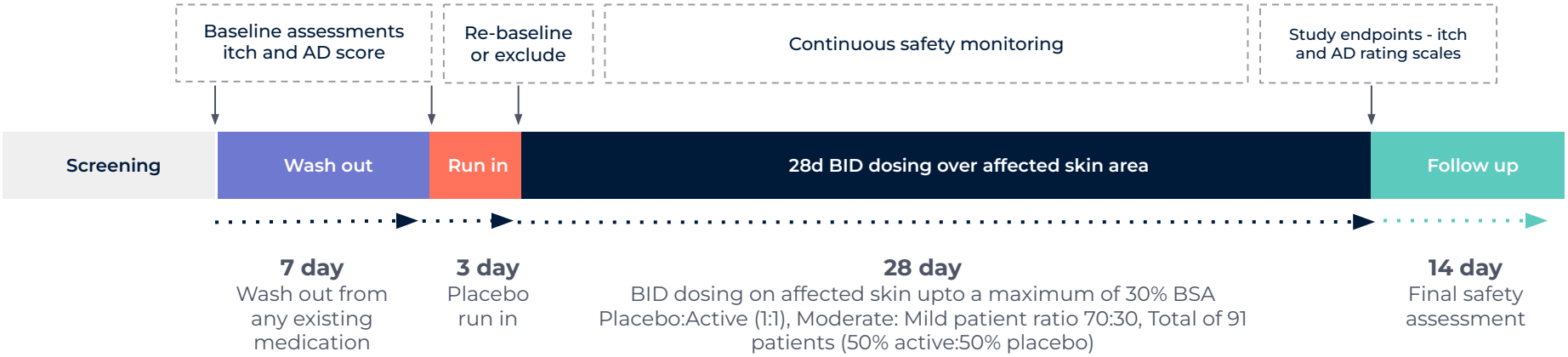
Inflammation
- Change from baseline in **EASI** score
- Fraction of patients achieving improvement in **EASI** score
- Change from baseline in **BSA** affected by AD in treated area(s)
- Change from baseline in **vIGA**

Quality of life
- Change from baseline in **POEM**
- Change from baseline in **DLQI**
- Change from baseline in **EQ-5D**
- Change from baseline in **PROMIS** (sleep subscale)

= Powered endpoints

BEN-2293 - Phase IIa study recruitment completed

Phase IIa clinical study design



Current Status

- LPLV achieved end January 2023
- 91 patients randomised (65 moderate and 26 mild)
- DBL completed 1st March
- TLR anticipated end March

Our intention is to to partner this asset with a pharmaceutical company that has a focus in dermatology for continued clinical development and, if approved, commercialisation

LPLV - last patient last visit | DBL - database lock | TLR - Top line results

BEN-8744
Ulcerative Colitis (UC)

BEN-8744: Oral, peripherally-restricted, PDE10 inhibitor under development as a first-in-class treatment for refractory UC

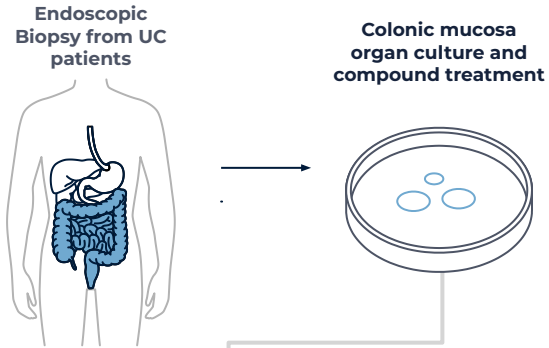
Affects 0.4% US population[1], 1.7 million patients in 7MM[1], forecast $7.8bn market by 2026[2]

- **A chronic, lifelong disease** that causes inflammation and ulceration of the inner lining of the colon and rectum
- 64% of patients are mild-moderate, 31% of patients are moderate-severe and 5% of patients are severe-fulminant
- **Efficacy** - 20-40% of Moderate-severe patients do not respond to anti-TNF (main treatment paradigm)[3]
- **Safety** - Treatments have many side effects — from steroids to anti-TNF and JAK inhibitors (black box warnings)[4]
- High unmet need for an alternative **oral** small molecule treatment option with **improved safety profile** and efficacy in treatment of **refractory patients**

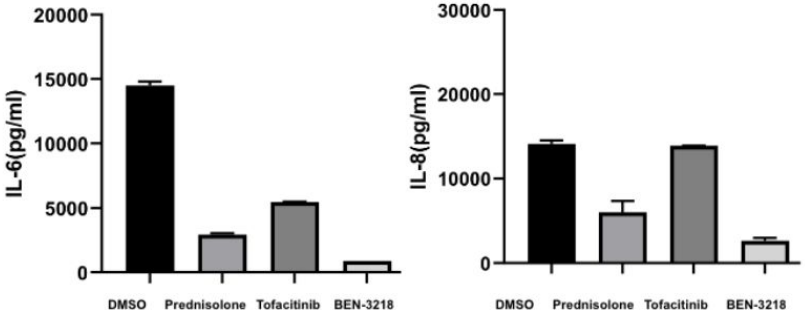
- **Phosphodiesterase 10 (PDE10)** was identified by our TargetID platform as **an entirely novel target for the treatment of UC/IBD**
- Using our Molecular Design expertise we optimally designed a best in class peripherally restricted PDE10 inhibitor: **BEN-8744**
- **BEN-8744** is expected to provide an efficacious disease modifying oral treatment for UC/IBD
- **BEN-8744** will target **Moderate and Severe UC/IBD patients**, meeting the unmet need left by existing therapies including:
  - Patients refractory to anti-TNFs or other biologics
  - Improved safety and tolerability profile compared to competitors
  - Aiming to use a Precision Medicine approach to target key responder patient cohorts and avoid the safety risks associated with ineffective therapies

Sources: (1) GlobalData: Ulcerative Colitis, Global Drug Forecast and Market Analysis to 2026; (2) Evaluate Pharma: Gastro-intestinal, Inflammatory bowel disease (IBD), Ulcerative colitis, Worldwide Overview (report 17th Sep 2021); (3) Roda et al. Clin Transl Gastroenterol 2016; (4) Kobayashi et al Nat Rev Dis Primers 2020 and US FDA Drug Safety Communication 2021

BEN-8744: potent activity in UC colon biopsies

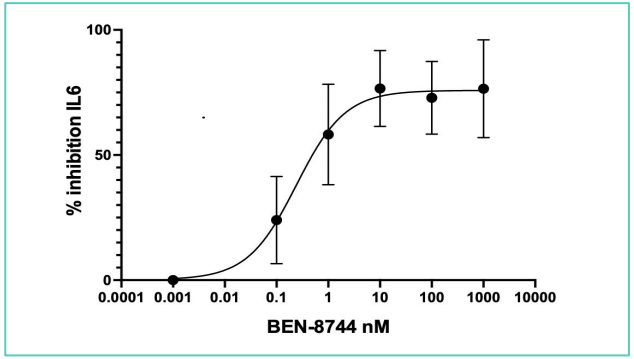


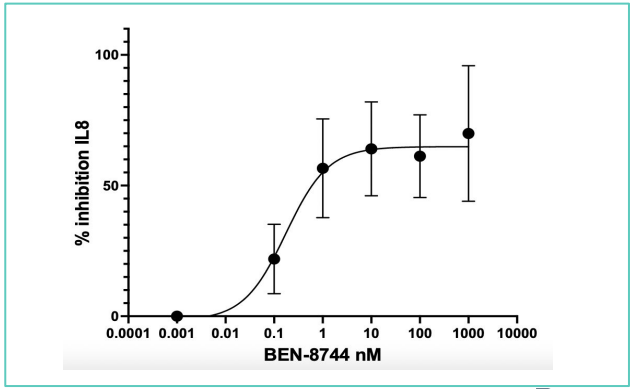
Inflammatory cytokine measurement



Selective PDE10 inhibition showed comparable reduction of IL-6 and IL-8 to the positive controls in ex vivo UC biopsies

Cross donor response summary: % inhibition cytokine release (mean % inhibition from N=15 IBD donors)





BEN-8744 results and progress to date

TARGET IDENTIFICATION

Novel target for UC

✔ Discovered using Benevolent **TargetID tools**

✔ PDE10 has **zero linkage to UC** in all available biomedical literature

✔ Experimentally **validated in ex-vivo** UC colon samples from patients refractory to SoC treatment

CHEMISTRY

Rapid and efficient lead optimisation

✔ **Molecular Design tools** enabled rapid and efficient lead optimisation

✔ **Candidate nominated in Sep '21** Novel, potent, selective,peripherally restricted PDE10. Inhibitor, with low dose prediction

✔ Delivered drug candidate within **2 years** from programme initiation

CLINICAL DEVELOPMENT

Developing responder and progression endotypes

✔ We will develop responder and progression endotypes**, adding molecular descriptors**

✔ These will inform our trial design, **patient selection** and further target identification in UC

✔ Augmenting a further loop of iteration on an enriched graph

Outlook

Joanna Shields, CEO

“With the world’s attention on AI applications that deliver real- world impact, we are strongly positioned to capitalise on this moment. With our substantial portfolio of platform-generated drugs, our work with big pharma and research collaborators, and our continued investment in state-of-the-art technology, we are showing every day how AI can be used to unlock the next wave of biopharma innovation.”

Joanna Shields, CEO

Poised for growth: multiple value inflection points expected

BEN-2293

- Top-line results of the Phase IIa clinical trial expected in Q1 2023
- Out-license in H2 2023 subject to results

BEN-8744

- Initiate the Phase I study in H1 2023

BEN-28010

- Submit the CTA in H2 2023

2023

- Continued investment in state- of-the-art technology
- Aim to sign additional commercial collaboration
- Commence IND-enabling studies for at least one additional asset
- Continue to progress pipeline assets to value inflection points

Thank you

 benevolent.com

 @benevolent_ai

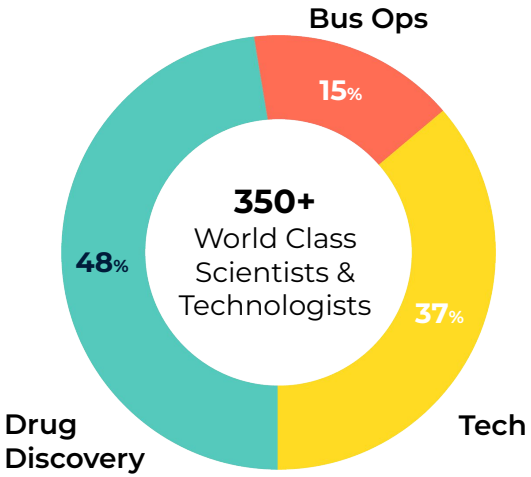
 benevolentai

 hello@benevolent.ai

Appendix

World-class team

We “build tech in the service of science”



Board of Industry Luminaries

Combines deep expertise across AI, pharma, & drug discovery & development

Baroness Joanna Shields
CEO & Executive Director

François Nader
Chairman

Susan Liautaud
Non-Executive Director

Olivier Brandicourt
Non-Executive Director

Jean Raby
Non-Executive Director

Jackie Hunter
Non-Executive Director

Nigel Shadbolt
Non-Executive Director

John Orloff
Non-Executive Director

As of December 2022

Proven to enhance drug discovery

DISEASE-AGNOSTIC

We can work on any therapeutic area due to the breadth and diversity of our data foundations.

MODALITY-AGNOSTIC

The Benevolent Platform™ can be applied to antibody and biologic targets, in addition to small molecule targets.

BUILT FOR SCALE

Our scalable and versatile Platform can support multiple in-house drug programmes and commercial collaborations.

ACCELERATES DISCOVERY

By combining our AI Platform, scientific expertise and wet lab facilities, we accelerate discovery and reduce discovery and development timelines.

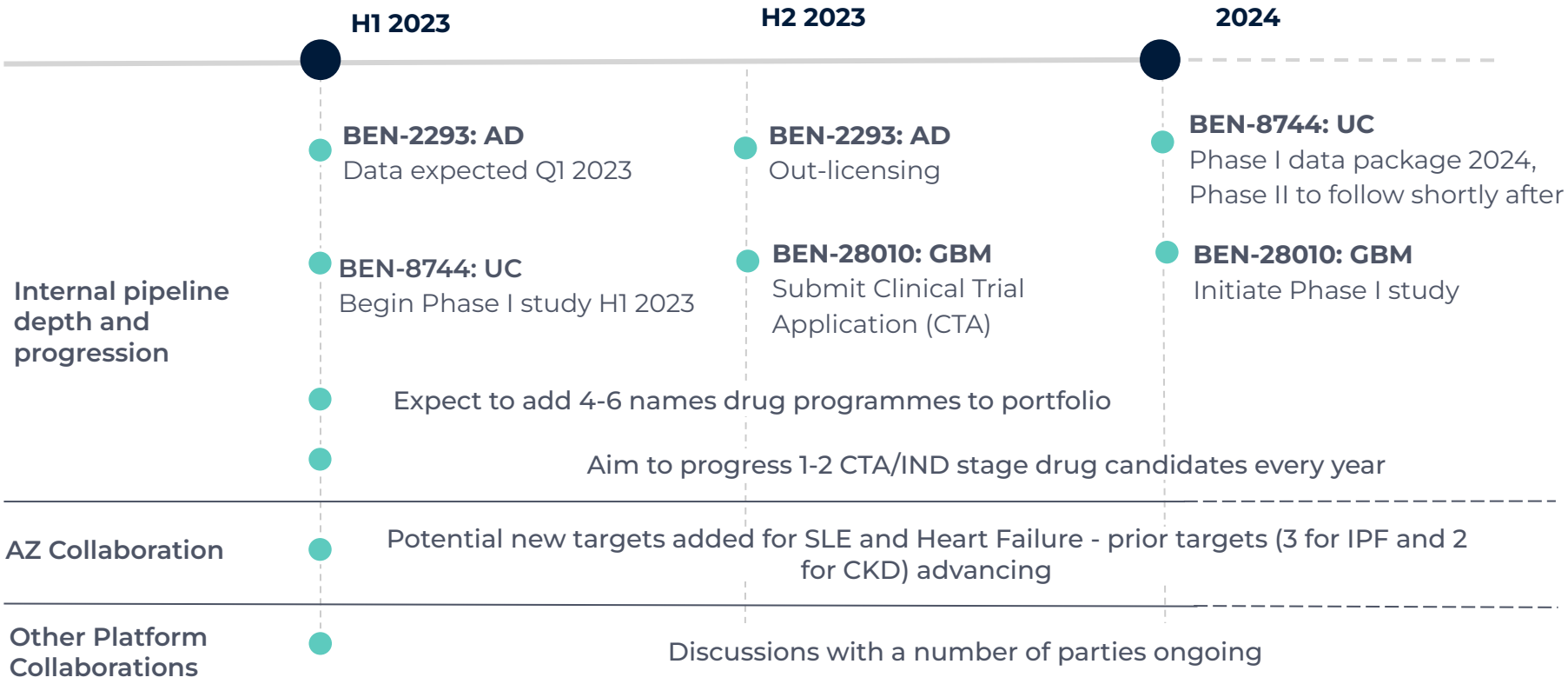
IDENTIFIES NOVEL TARGETS

Our predictive tools can surface targets that have never been considered for a disease before.

POTENTIAL TO INCREASE PROBABILITY OF SUCCESS

By building higher confidence hypotheses in the earliest stages of drug discovery, we aim to reduce costly failures down the line.

Poised for growth: multiple value inflection points



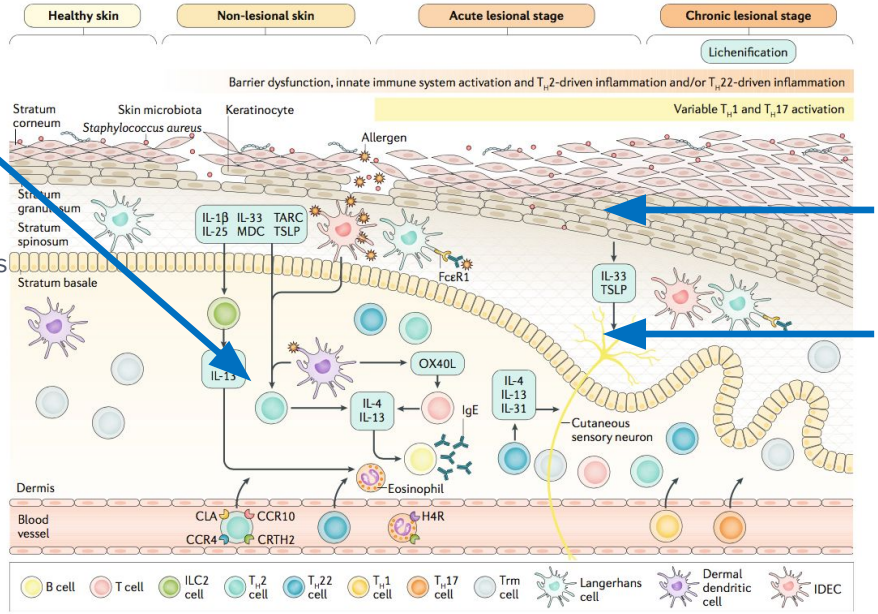
Atopic Dermatitis – BEN-2293, pan-Trk inhibition rationale

TrkC

- NT3/TrkC potentiates stimulated ***Th2 T-cell*** inflammatory responses and synergistically enhances T-cell receptor induced IL-4 production by Th2 cells
- ***Mast cells*** within AD skin lesions express high levels of NT3 compared to normal controls

TrkB

- AD Skin-resident ***eosinophils*** express elevated levels of TrkB (together with TrkA and C) and functionally respond to BDNF
- BDNF/TrkB inhibit eosinophil
- apoptosis and increase chemotactic index



TrkA

- TrkA levels in skin dramatically increase in response to inflammatory stimuli
- NGF produced by AD ***keratinocytes***, is a major mediator of cutaneous hyperinnervation
- Increased NGF in the skin sensitizes ***primary afferents*** contributing to peripheral itch sensitization and chronic pruritus
- Involved in the inflammatory activation of ***mast cells*** and ***basophils***

BEN-2293: Excellent skin penetration

- Experimental evidence supports high exposure in human skin at >IC90 free, and low exposure in blood with proposed clinical 1% ointment strength[1].
- 1% BEN-2293 ointment BID exceeds the exposure needed for PanTrk inhibition in both epidermis/upper dermis and lower dermis even at IC_{90} [1]

Human *in vitro* >>IC90 √

Minipig *in vitro* >> IC90 √

Minipig *in vivo* >IC90 √

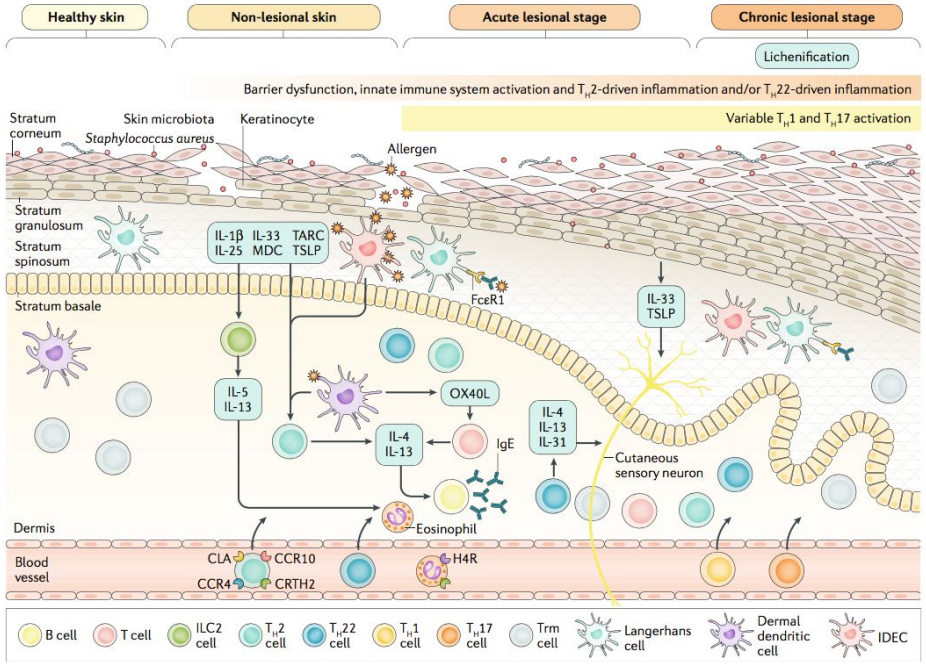
Epidermis and upper dermis

Lower Dermis

Minipig *in vivo* √

Blood

Free plasma levels <<400 below IC50



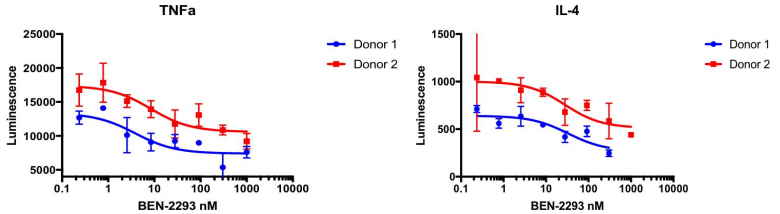
CTA-enabling 28d Tox Package: Rat (IV) and Mini-pig (topical) = Safety margins > 20 fold for AUC and > 269 fold for Cmax to dose limited NOAELs[1]

[1] (internal Company drug programme data)

We expect BEN-2293 to provide both symptomatic relief and to reverse disease progression in atopic dermatitis

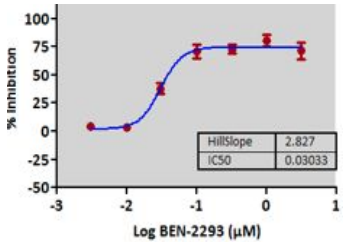
- **BEN-2293 is highly selective for Trk receptors,** with IC50 potencies in the low nM range for TrkA, B, and C
- **BEN-2293 dose dependently inhibits release of inflammatory Th1 and Th2 cytokines** TNFα, IFNɣ, IL-13, and IL-4 in human peripheral blood mononuclear cells (PBMCs) stimulated with an inflammatory T-cell stimulus (anti-CD3/CD28)
- **BEN-2293 inhibits the release of Calcitonin Gene-Related Peptide (CGRP)**, a mediator of itch, sensory nerve hypersensitisation and neurogenic inflammation, in dorsal root ganglion (DRG) isolated from adult rats and stimulated with NGF
- **BEN-2293 series significantly (p<0.05) reduced mouse ear inflammation** following administration of PMA, significantly reducing expression of cytokines IL-1β, IL-4, IL-6, CXCL1, MCP-1, and Tarc
- **BEN-2293 demonstrates excellent tolerability and safety margins** in IND/CTA-enabling toxicology studies

BEN-2293 Inhibition of human primary T-cell activation

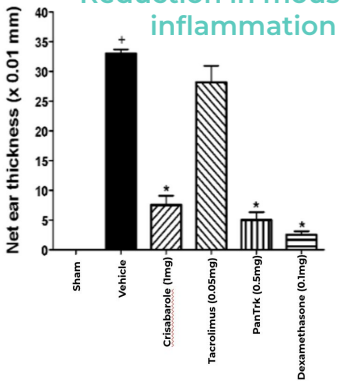


Human PBMCs from 2 donors. Anti-CD3/CD28 stimulus +/- BEN-2293

Inhibition of sensory neuron activation



Reduction in mouse ear inflammation



Key: Tropomyosin-related kinase (Trk) receptor tyrosine kinase family, namely TrkA, TrkB, and TrkC; Nerve Growth Factor (NGF); Brain Derived Neurotrophic Factor (BDNF); Neurotrophin-3 (NTF-3)/NT3

BEN-2293 - Phase Ib safety and tolerability study successfully completed

Phase Ib/IIa double-blind, placebo-controlled, first-in-patient, two-part clinical study

Phase Ib

1 → 2 → 3 → 4

First-in-human dose escalation

Phase Ib completed Dec 2021

8 Mild-Moderate AD patients (18-65 years) per cohort, randomised 3:1 BEN-2293:Placebo

Safety, Tolerability, PK

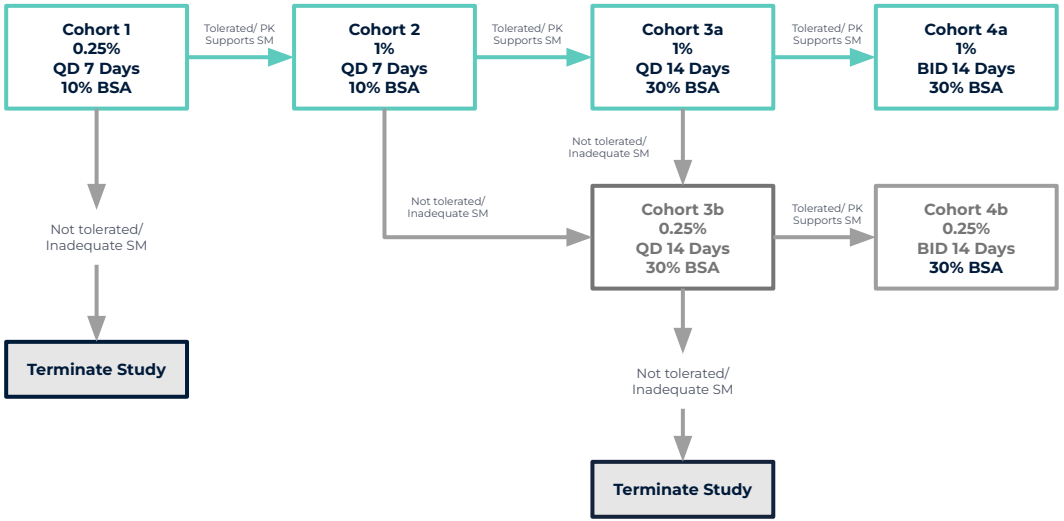
- Adaptive multiple ascending dose cohort design
- Includes efficacy endpoints
- MALDI imaging
  - To evaluate human skin PK

Phase Ib:

✔ **Successfully completed**

Safety and Tolerability arm

Adaptive design for Phase Ib of the clinical study



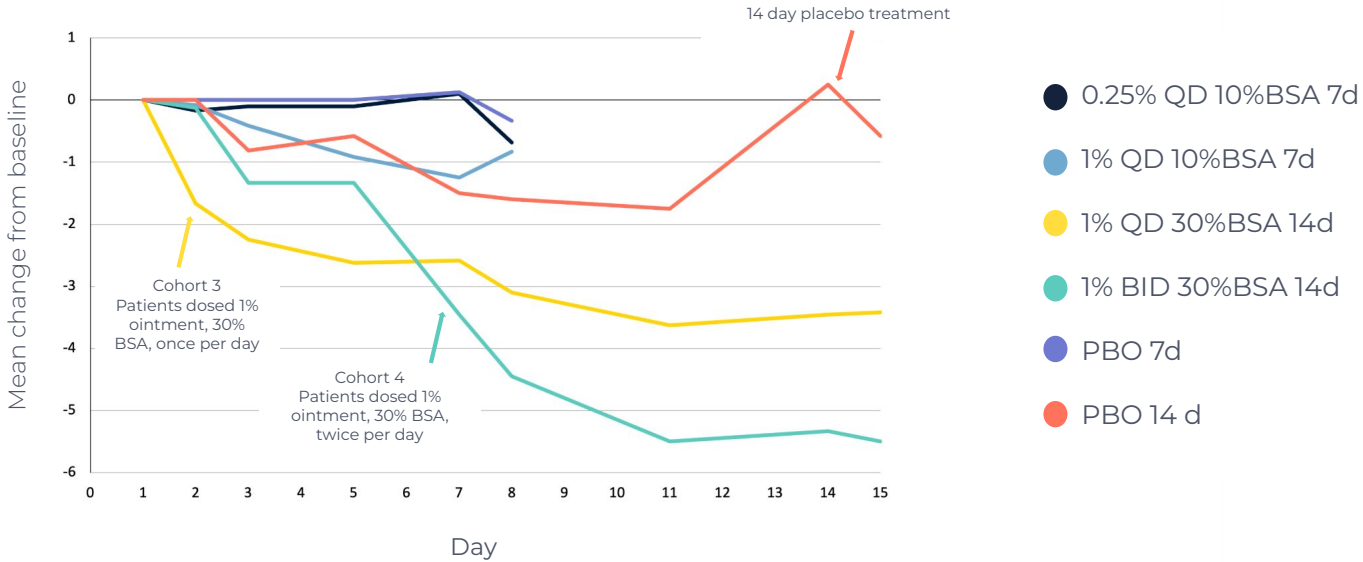
BSA= Body surface area. QD = once per day dosing BID= twice per day dosing

BEN-2293 - indicative data from Phase Ib

Caveats:

- Phase Ib was **NOT** powered to meaningfully assess efficacy - only 6 patients dosed with active per group
- Maximum duration of dosing 14 days (EASI score changes typically measured at 28 days)

Mean Change from Baseline %BSA affected in treated areas



EASI: Eczema Area and Severity Index

BEN-8744 - Phosphodiesterase 10 (PDE10) - a novel target for UC

Transcriptomics data support the rationale for PDE10 as a novel target for UC

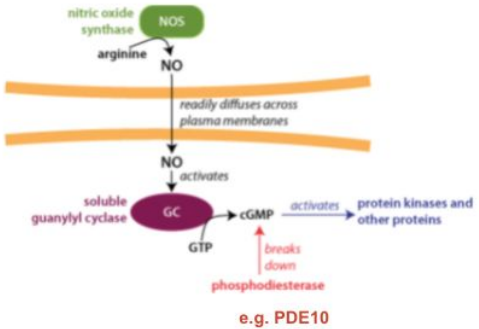
- PDE10 regulates signal transduction by hydrolysing cGMP
- PDE10 is significantly upregulated in UC-derived colon and colonic mucosa samples, whilst guanylyl cyclase, which makes cGMP, is down-regulated

Reduced levels of guanylyl cyclase correlate with increased TNF-α in UC colonic mucosa[1]

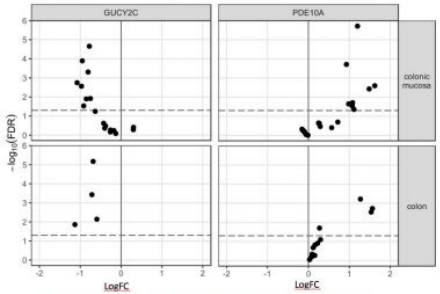
- cGMP is downregulated in UC and its expression inversely correlates to disease severity
- PDE10 is well-studied in CNS disorders but not in inflammation with zero linkage to UC

PDE10 demonstrates restricted expression in peripheral tissue

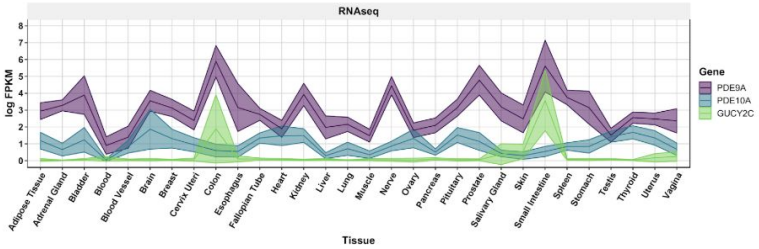
- Reduces the safety liability of targeted inhibition



PDE10 degrades cGMP



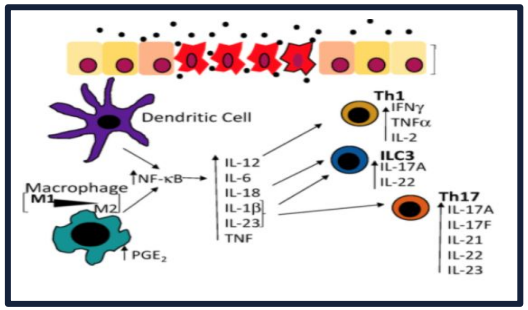
Differential RNA expression of PDE10A and GUCY2C: normal vs UC



Low basal PDE10 expression, highest levels in brain. Low basal soluble guanylate cyclase (GUCY2C) levels except in colon and sm intestine

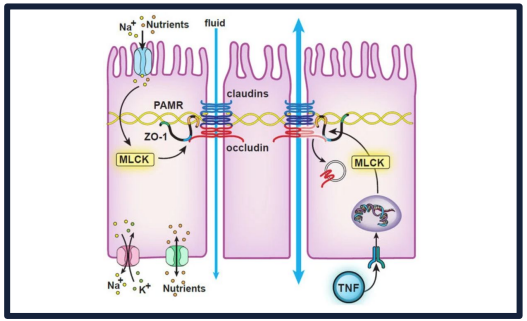
Source: (1) Brenna et al. Scand J Gastroenterol 2015
Image (left): Rashed, Second Messenger System 2018

PDE10 inhibition: potential to normalise dysregulated mechanisms in IBD



PDE10 inhibition

↑cGMP ↑cAMP



- Reduced inflammatory cytokine release from intestinal epithelia via ↓NFκB[1]
- Reduced tissue-resident macrophage activation[1]

Reduced intestinal inflammation

- Improved TJ assembly via PKG/PKA-mediated ↓pMLC[2]
- Improved fluid/mucus homeostasis via PKG phosphorylation of intestinal CFTR[3]

Improved barrier integrity

Sources: (1) Harmel-Laws et al PLoS One 2013: (2) Han et al PLoS One 2011; (3) Brenna et al Scand J Gastroenterol 2015
Images: Nettleford and Prabhu, Antioxidants 2018 (left); He et al. Int J Mol Sci 2020 (right)

Strategic validation: successful collaboration with AstraZeneca

Multi-year Target-ID collaboration is delivering multiple, novel targets for complex diseases with high unmet need

Separate data environment established to integrate AstraZeneca's data into a bespoke Knowledge Graph

BenevolentAI and AstraZeneca teams working in close collaboration to explore, identify and validate targets

Deal structure of upfront license fee, milestone payments and downstream royalties

Data generated via the collaboration enriches the Benevolent Platform™

AstraZeneca

Five novel targets selected for AstraZeneca's portfolio to date

2019

Initial deal focussed on Chronic Kidney Disease & Idiopathic Pulmonary Fibrosis

2022

3-year collaboration expansion to include Heart Failure & Systemic lupus erythematosus

Using our platform for wider societal benefit

Identified a COVID-19 treatment now approved for use by the FDA

RAPID
Identified baricitinib as a treatment in just **48 hours**, published research in The Lancet in Feb 2020

NOVEL
Our technology and AI workflows identified a **previously unknown antiviral mechanism**[1]

EFFECTIVE
COV-BARRIER trial showed baricitinib reduces mortality by 38% in hospitalised patients[2], and by **46% in ventilated or ECMO patients**[3]

FDA approved the baricitinib to treat COVID-19 in **May 2022**[4] after first granting EUA in **Nov 2020**[5]

Led to equity investment from Eli Lilly

DNDi
Drugs for Neglected Diseases *initiative*

Non-commercial collaboration

→ Focused on Dengue fever - a major healthcare burden

→ Aims to deliver biological targets and drug repurposing candidates

→ Experimental validation in progress - 6 assays

Categorisation of AI/ML companies in biotech, hit & target-ID

Companies can be characterized across two key dimensions: **Original technology focus** and **drug discovery approach**

Target-ID

WHAT TARGET DO WE NEED TO HIT TO BE EFFECTIVE AND SAFE IN A SPECIFIC DISEASE (pathways, cellular processes)?

HIGH COMPLEXITY THROUGH BIOLOGY

Many layers of knowledge needed, and many areas where research is not yet complete

Hit-ID

HOW DO WE NEED TO HIT THE TARGET WE HAVE IDENTIFIED (specific drug characteristics)?

HIGH COMPUTATIONAL COMPLEXITY

Atom-to-atom interaction is relatively well known, but requires many calculations and simulations

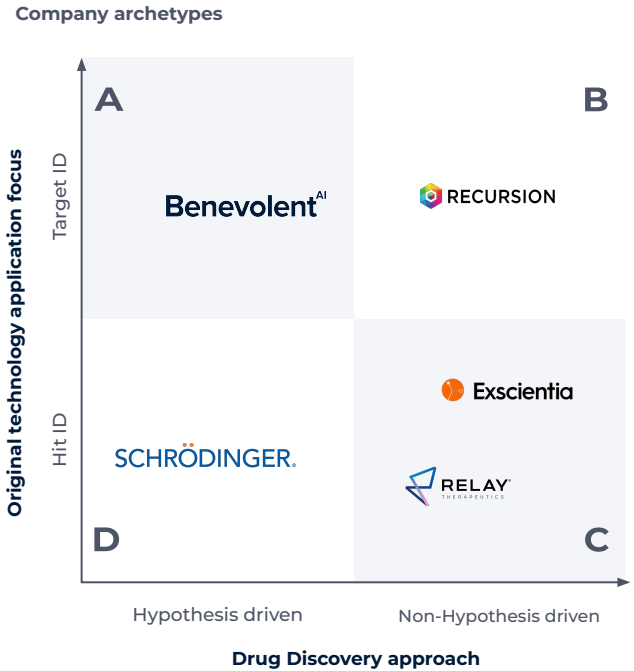


Figure: Oliver Wyman Analysis (listed companies only)

Hypothesis driven

Involves a **data-driven hypothesis-led** approach to therapeutic target identification

Non hypothesis driven

Leverages technology to identify solutions **without specific conditions to target specified at the outset**

Pharma companies also active in the space, through **internal development** and/or **collaborations**

Source: Company Websites, Oliver Wyman Analysis