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Redx - Corporate Presentation - 21 June 2023
Our ambition: To create world leading medicines to transform patients’ lives

- **Lisa Anson**
  - CEO
  - High profile general manager, former President of AstraZeneca UK, with >25 years in biotech and pharma

- **Dr Richard Armer**
  - CSO
  - Accomplished drug discovery executive, with >25 years in biotech and pharma

- **Dr Jane Robertson**
  - CMO
  - Oncologist with >20 years in biotech and pharma drug development. Led the successful clinical development of Lynparza

- **Peter Collum**
  - CFO
  - Experienced finance and strategy executive with >25 years in biopharma
Clinical Stage Biotech Discovering Targeted Medicines for Fibrotic Disease and Cancer

Focus on progressing differentiated ROCK portfolio:

- **Zelasudil (RXC007)**
  - with potential in idiopathic pulmonary fibrosis, cancer-associated fibrosis and fibrostenotic Crohn’s disease

- **RXC008**

Multiple near-term value inflection points including clinical data readouts expected:

- **Zelasudil** Phase 2a IPF data Q1 2024
- **RXC004** Phase 2 combination data H2 2023
- **RXC008** CTA submission H2 2023

World-class Discovery Engine with experienced scientific team and track record of generating successful drug candidates:

- **5 clinical molecules**
  - Including FDA approved, Jaypirca™ (pirtobrutinib)*

Backed by blue chip specialist biotech investors. Funded into 2024 to deliver multiple value inflection points.

*the asset was subsequently sold outright to Loxo Oncology, now part of Eli Lilly, Redx has no remaining economic interest.
## Advancing a Robust Pipeline Built In-House

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<td>Potential best-in-class ROCK2 Selective Inhibitor zelasudil (RXC007)</td>
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<td>Potential best-in-class Porcupine Inhibitor (RXC004)</td>
<td>Genetically selected MSS mCRC Biliary tract cancer and pancreatic cancer</td>
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<td>Pan-RAF Inhibitor (JZP815)</td>
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GI: Gastrointestinal; IND: Investigational new drug application; MAPK: Mitogen-activated protein kinase; MSS mCRC: Microsatellite-stable metastatic colorectal cancer; RAF: Rapidly accelerated fibrosarcoma; ROCK: Rho associated protein kinase
Zelasudil (RXC007): A Selective ROCK2 Inhibitor for Fibrotic Diseases - Lead Indication is IPF with Phase 2a Data Expected Q1 2024

Highlights

- Zelasudil is a highly potent, selective and orally-active ROCK2 inhibitor
- ROCK2 is a validated, compelling target at a key junction in cell signalling pathways central to fibrosis
- Robust preclinical efficacy data across disease models supports clinical development plan in lung fibrosis - IPF and CF-ILD, as well as potential in cancer-associated fibrosis
- Phase 1 healthy volunteer data in single ascending dose and multi-dose cohorts confirms drug like profile for safety and PK
- Phase 2a in IPF recruiting - expected to report topline data Q1 2024
  - 12-week Phase 2a dose ranging study for early efficacy readouts, safety and tolerability in IPF patients +/- SoC, in addition to target and disease biomarker engagement
  - No safety signal in initial safety review enabling dose escalation to progress
- Phase 2b in IPF and CF-ILD planned for zelasudil with SoC over 12 months with lung function (FVC) as primary endpoint
- Potential to augment clinical development plan with Phase 1 study of zelasudil in combination with SoC chemotherapy in first line pancreatic cancer and other potential indications
ROCK is a Compelling, Nodal Target for Fibrotic Diseases

Why ROCK2 Selective?

- The role of ROCK2 in a diverse range of cellular process allows zelasudil to have pleiotropic effects
- Systemic inhibition of ROCK1&2 results in hypotension
  - Effect not seen with selective ROCK2 inhibition
- ROCK2 inhibition alone is sufficient to protect from pulmonary fibrosis in mouse models

Why Target ROCK?

1. RhoA/ROCK/ROCK2 downstream of many major profibrotic factors
2. ROCK is involved in diverse cellular processes
3. ROCK upregulates key profibrotic genes. Upregulation of these genes leads to actin cytoskeleton organisation, cell adhesion and motility, proliferation, and extra cellular matrix remodeling

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(1) Knipe et al., 2018

(1) Rho-associated protein kinase

Upregulated Genes:
- CXCL10
- COL1A1
- COL3A1
- CTGF
- FNPAT1
- WISP-1
- TIMP1
- ICAM-1
- α-SMA

Intermediate filament disruption

ROCK1/2

Signalling and transcription activation

Cytoplasm

Integrins

Stress fibre assembly and contraction

F-actin stabilisation

ROCK-GTP

GPCRs

RTKs

TGFβR
Zelasudil is a Next-Generation ROCK2 Selective Inhibitor With Potential to Improve Safety and Therapeutic Outcomes

Selectivity
- Highly selective with limited off target pharmacology

Drug-drug interaction
- Limited cytochrome P450 interaction supports combinability

PK / Bio-distribution
- Increased exposure at lower doses than previous ROCK2 inhibitors

zelasudil
- Selective ROCK2 inhibitor
- Best-in-class opportunity

Plot of kinases inhibited by RXC007 with IC50 < 1 µM
- Selectivity >100 fold vs ROCK 1 and vs 468 kinases

ROCK: Rho-associated protein kinase
Lung Fibrosis: A Growing Clinical Need with Limited Treatment Options

Lead indication is Idiopathic Pulmonary Fibrosis (IPF)

- IPF is a deadly fibrotic lung disease
- Median survival between 3 – 5 years\(^{(1)}\)
- High unmet need: over 170,000+ patients in the US, EU5 and Japan alone\(^{(2)}\)
- Global IPF market projected to reach $3.6 billion by 2029\(^{(3)}\)
- Nintedanib and pirfenidone are the only approved treatments but have significant side effects that limit use

Broader opportunity in Interstitial Lung Disease (ILD)

- IPF represents approx. one third of ILD patients\(^{(4)}\)
- Of the two thirds of ILD patients without IPF, up to 40% may develop a progressive fibrosing phenotype\(^{(5)}\) (PF-ILD) where new anti-fibrotic treatments are needed
- PF-ILD includes rheumatoid arthritis-ILD (RA-ILD), systemic sclerosis-ILD (SSc-ILD) and mixed connective tissue disease-ILD (mCTD-ILD)
- SSc-ILD market is particularly well-characterized, with this segment alone estimated to surpass $1.1 billion by 2030\(^{(6)}\)
Strong Literature Rationale on ROCK and ROCK2 Supports IPF as a Lead Indication

ROCK as a target in IPF\\(^{(1)}\\)

RhoA/ROCK/ROCK2 signalling pathway is upregulated in human IPF patients.

ROCK2 expression in IPF\\(^{(2)}\\)

Increased ROCK2 expression in IPF bronchial epithelial cells and fibroblasts.

Preclinical validation of selective ROCK2 inhibition\\(^{(3)}\\)

ROCK2+/- knockout mice are protected from bleomycin-induced pulmonary fibrosis.

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\(^{(1)}\) Zhou, 2013; \(^{(2)}\) Shimizu, 2014; \(^{(3)}\) Knipe, 2018

(Myo)fibroblasts isolated from lungs of patients with IPF (n = 8), non-IPF control subjects (n = 6). ROCK activity assays using a colorimetric approach. Data are mean ± SD. Intensity of immunostaining evaluated on a scale of 0 to 3. N=6 per group; NF: not found.

Genetic reduction in either or both ROCK isoforms affords protection from bleomycin-induced pulmonary fibrosis. Significant reduction in hydroxyproline content (lung collagen).
Zelasudil Activity on Patient Tissue and in Preclinical Models Supports Core Development Plan in IPF and CF-ILDs

Reduction in Collagen Deposition with zelasudil in Therapeutic Murine Bleomycin-induced Lung Fibrosis Model

Significant Reduction of Collagen Content in Lungs in Murine Sclerodermatous chronic Graft versus Host Model

Gene Set Enrichment Analysis of PCLS Tissues Shows ROCK2 Relevance in Disease Modulation

Suppression of the expression of genes strongly associated with fibrosis in IPF

Source: Data generated by Redx
**Phase 1 Data in Healthy Volunteers Showed Good Safety and Pharmacokinetic Profile**

**Good safety profile**
- No SAEs reported in SAD or multidose cohorts
- Doses tested in SAD from 2mg-100mg QD and doses tested in MAD: 50mg BID and 70mg BID
- Safe and well tolerated with few treatment emergent adverse events reported
- All AEs transient, mild and reversible with no dose changes required
- No evidence of hypotension, validating rationale for selective ROCK2 inhibition

**Drug-like pharmacokinetic profile**
- Pharmacokinetics as predicted from preclinical data
- Mean half-life 9-11 hours, potentially suitable for once or twice daily dosing
- Essentially linear exposure in SAD from 2mg QD to 70mg BID
- No significant differences between 50mg fed and fasted cohorts
- 20mg BID selected as starting dose for Phase 2a, which achieved biologically relevant exposures based on preclinical models

**Multidose cohort 50mg and 70mg BID - total plasma concentration over time**

PK sampling up to 72 h; only 0-24 h plotted. On day 24 only 1 dose administered

Source: Data generated by Redx
Phase 2a Trial in IPF Expected to Report Q1 2024

Phase 2a dose ranging study to inform Phase 2b dose

Provides early efficacy readouts, safety and tolerability in IPF patients with or without standard IPF therapy

Key endpoints:
- Safety, PK Profile
- Changes from baseline in lung function - FVC and DLCO
- Changes from baseline in Quantitative Lung Fibrosis Score, airway volume and resistance on HRCT Scan

Dose Level - 70mg BID tbc
12:4 zelasudil:pbo
(n=16)

Dose Level - 50mg BID
12:4 zelasudil:pbo
(n=16)

Dose Level - 20mg BID
12:4 zelasudil:pbo
(n=16)

Dosing period 3 months
(Patients may elect to continue for longer if no signs of disease progression)

Translational Science Sub-Study to demonstrate effects on disease biomarkers e.g., α-SMA, TIMP-1, ICAM-1, and target engagement biomarkers e.g., pMYTP1 and pMLC2

Endpoints include:
- Changes from baseline in blood biomarkers
- Proteins and genes from broncho-alveolar lavage (BAL) fluid
- BAL-fluid cells and bronchial epithelial cells

Cohort 1
8 patients

Cohort 2
8 patients

Current Status: Six countries approved. US IND open for 28-day dosing with further information required for three-month dosing cohorts. More than 30 sites expected to be active by end Q2 2023.

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Zelasudil Unlocks ROCK as a Key Fibrosis Target Across Indications

- Murine & Rat Bleomycin-Induced IPF Models
- PCLS from Patients with IPF
- Murine Sclerodermatous chronic Graft versus Host Model

Metabolic diseases
- NASH
- Kidney fibrosis

Cardiovascular disease

Oncology
- Pancreatic
- Pancreatic metastatic patient-derived PDAC (PDX-05) murine xenograft
- FOLFIRINOX resistant PDX-08 model
- Murine KPC syngeneic model

Fibrosis
- IPF
- ILD

Neuromuscular

CNS

Auto-immune
- cGvHD
- Systemic sclerosis

Supportive preclinical ROCK2 inhibitor data available

Murine Sclerodermatous chronic Graft versus Host Model

IPF: Idiopathic pulmonary fibrosis; NASH: Nonalcoholic steatohepatitis
ROCK2 expression is elevated in patients with advancing tumour stage*

ROCK2i combined with SoC chemotherapy increases survival in metastatic PDX pancreatic cancer mouse models

ROCK2i combined with gemcitabine/Abraxane in pancreatic cancer PDX model

ROCK2i combined with FOLFIRINOX (FXN) in FNX-resistant pancreatic cancer PDX model

Results based on quantification of ROCK2-immunohistochemistry stained sections in normal and stage I, II and III/IV cases of human pancreas adenocarcinoma

- Hypothesis is that ROCK2 antifibrotic action allows entry of tumour fighting T cells and reprogrammes the TAMs and CAFs to reduce their protumourogenic role
- Potential to initiate Phase 1 study of zelasudil (RXC007) in combination with standard of care chemotherapy in first line pancreatic cancer, early 2024

Source: Data generated by the Garvan Institute of Medical Research
**Highlights**

- RXC008 is a potent, oral, small molecule non-systemic ROCK 1/2 inhibitor
- Fibrostenotic Crohn’s disease is a significant unmet need - only treatment option for patients is successive surgical intervention
- RXC008 is a potential first-in class treatment - no approved therapies for underlying fibrosis and no curative treatments available
- ROCK is a key target involved in fibroblast activation, and is upregulated in fibrostenotic Crohn’s disease
- RXC008 is GI-targeted, selectively active in gut without risking systemic exposure
- RXC008 has demonstrated robust preclinical efficacy *in vivo*
- Phase 1 enabling work underway - **CTA submission planned for end 2023**
  - CMC API manufacture complete
  - Toxicology studies ongoing
**Potential First-in-Class Treatment for Fibrostenotic Crohn’s Disease**

**Clinical progression in Crohn’s**
- Intervention with anti-inflammatories, suppresses inflammation but not fibrosis

**Fibrotic stricture formation**
- Fibrotic lesion
- Smooth muscle cells
- Intestinal tract

**Surgical interventions**
- Stricture-plasty
- Balloon dilation
- Resection

---

1.7 million\(^1\) patients globally affected by Crohn’s disease

>50% of patients\(^2\) develop fibrostenosis and strictures within 10 years of first diagnosis

**No approved** therapies for underlying fibrosis only treatment options are debilitating surgical intervention

---

\(^1\) Clarivate, Crohn’s disease disease landscape & forecast pg 39, Published Sep 2022; \(^2\) Chan et al. 2018
RXC008: GI-targeted pan-ROCK Inhibitor Targets a Fibrotic Pathway Nodal Point without Systemic Breakthrough

- ROCK is a nodal point in the fibrotic signalling pathway
- Inhibiting ROCK 1&2 systemically is known to result in hypotension
- GITR inhibitors are specifically designed to avoid hypotensive effects associated with systemic ROCK inhibition
- RXC008 designed to be retained in the GI tract via high efflux and low permeability, rapidly metabolised by paraoxonase enzymes in the plasma
- Result of this is virtually no systemic breakthrough

GI: Gastrointestinal; GITR: Gastrointestinal targeted ROCK; MRTF: myocardin-related transcription factor; MLC: myosin light chain
Preclinical Package Has Shown Promising Anti-fibrotic Effects in Multiple Translatable Models

In adoptive T-cell transfer models RXC008 reduces fibrosis and smooth muscle hyperplasia

In a therapeutic 12-week DSS model GI-targeted ROCK inhibition was able to reverse fibrosis

Significant reduction in fibrosis, measured by histology score. Entropy changes correlate with progression of IHC confirmed fibrosis in the DSS model

Endpoint imaging translatable to the clinic using non-invasive MRI scans

Source: Data generated by University of Ghent on behalf of Redx. Data generated by Redx. REDX8087 is similar to RXC008 1-way Anova with Dunnet's multiple comparison. # T-cells/vehicle vs untreated controls, * RXC008 10mg/kg QD or anti-p40 v T-cells/vehicle.

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RXC004 is a highly potent, orally active, once daily Porcupine inhibitor.
Porcupine inhibition blocks secretion of all Wnt ligands, preventing both tumour growth and immune evasion.
RXC004 demonstrated clinical target engagement at all doses and has optimal PK profile with once daily, oral dosing.
RXC004 was well tolerated in Phase 1, as both monotherapy and in combination with nivolumab.
RXC004 shown to be active as a monotherapy in Phase 1, having differential clinical efficacy in Wnt-ligand dependent tumours (ESMO 2021).
Primary efficacy hypothesis is that combination with anti-PD-1 treatment can overcome anti-PD-1 resistance, which could open new patient segments (SITC 2022).
Recruitment ongoing in Phase 2 combination programme for RXC004 with anti-PD-1 in Wnt-ligand dependent tumours - topline data H2 2023.
Aim to seek a partner to continue development post Phase 2 data.
Porcupine Inhibition Targets Wnt-Ligand Dependent Tumours

Wnt signalling pathway is well established as a critical driver of cancer

1. Porcupine is a key enzyme upstream in the Wnt pathway, which adds a lipid chain to all 19 Wnt ligands (palmitoylation) enabling secretion.

2. Wnt ligands activate both canonical and non-canonical signalling pathways.

3. Canonical and non-canonical target genes drive tumour growth and tumour immune evasion.

4. By inhibiting Porcupine, RXC004 blocks the release of all Wnt ligands from cells inhibiting tumour growth and reversing immune evasion.

5. Targeting Wnt-ligand dependent tumours provides a clear patient selection strategy. Tumours with RSPO fusion or RNF43 mutations see upregulation of the Wnt pathway (increased number of frizzled receptors, through impaired degradation). Beyond genetic mutations, some other tumours display elevated Wnt-ligand expression.

RNF43: Ring finger protein 43; RSPO: R-spondin; Wnt: Wingless/integrated

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Mechanism of Action Demonstrated Preclinically Supports Combination Efficacy Hypothesis

**Inhibits tumour growth**

RXC004 inhibits tumour growth in an RNF43 mutant human pancreatic cancer model.

**Decreases tumour metabolism**

RXC004 treated tumour cells are less metabolically active. Cells also change morphology and increase mucous secretion.

**As a monotherapy**

Increased survival rate post-RXC004 monotherapy treatment in an anti-PD-1 resistant 'cold' mouse melanoma syngeneic model (B16F10).

**In combination with anti-PD-1**

Combination of RXC004 + anti-PD-1 showed a statistically relevant change in ratio of Cytotoxic T cells: Regulatory T cells, compared to either monotherapy alone in PD(L)-1 axis dominated CT26 CRC model.

**Survival: B16F10 tumours**

Time to tumour volume of 2500mm$^3$ for RXC004 treated tumours is significantly shorter than control.

Data as of 7th June 2021
Source: Redx posters, [https://aacrjournals.org/cancerrescommun/article/2/9/914/708958/The-Wnt-Pathway-Inhibitor-RXC004-Blocks-Tumor](https://aacrjournals.org/cancerrescommun/article/2/9/914/708958/The-Wnt-Pathway-Inhibitor-RXC004-Blocks-Tumor)

CRC: Colorectal cancer; RSPO: R-Spondin
18/25 monotherapy patients had RECIST-evaluable disease

Disease stabilisation observed in Wnt-ligand dependent tumours (5/7 patients)

Median treatment duration higher in patients with Wnt-ligand dependent tumours (13.1 weeks vs 6.6 weeks)

4/6 patients in the 1.5mg cohort had RECIST stable disease as best response

Changes in peripheral immune cell compartments are consistent with pre-clinical data and suggest an anti-tumour immune response

RNF43 LoF mt: loss of function mutation in Ring finger protein 43; M-MDSCs: monocytic myeloid derived suppressor cells

PD: Progressive disease; RECIST: Response evaluation criteria in solid tumours; SD: Stable disease
Phase 2 Combination Programme in Wnt-Ligand Dependent Tumours Expected to Deliver Topline Data During 2023

**PORCUPINE**
MSS Metastatic Colorectal Cancer

- **Combination** Genetically Selected RXC004 + Nivolumab
  (n=20)

**Endpoints**
- ORR
- Secondary DCR,
  Tumour size change, DoR, PFS

**Status**
Recruiting - US, UK, Spain, South Korea

**Topline data expected**
H2 2023

**Analysis**
Key translational endpoints will characterise mechanism of action
- FDG-PET scans to assess metabolic effect of RXC004 on tumours
- Tracking circulating tumour DNA (ctDNA)
- Baseline and on-treatment biopsies to assess changes to tumour and immune microenvironment

**PORCUPINE2**
Biliary Tract Cancer

- **Combination** Biliary Tract Cancer
  RXC004 + Pembrolizumab
  (n=15)

**Endpoints**
- ORR
- Secondary DCR,
  Tumour size change, DoR, PFS

**Status**
Recruiting - UK, Australia

**Topline data expected**
H2 2023

DCR: Disease control rate; DoR: Duration of response; ORR: Overall response rate; OS: Overall survival; PFS: Progression free survival
Discoidin Domain Receptor (DDR) Inhibition as a Potential Novel Therapeutic Class for Fibrosis

**DDR is a collagen target**
- Non-integrin tyrosine kinase collagen receptors
- Collagen binding initiates downstream fibrotic signalling pathways

**DDR inhibition is a novel approach**
- Novel, druggable therapeutic target for fibrosis
- Recent publication of pre-clinical PoC studies for small molecule inhibitors in models of lung and kidney fibrosis
- Potential for disease modifying efficacy

**Redx has a discovery programme**
- Potent and selective DDR inhibitors identified
- In lead optimisation phase
- Pre-clinical PoC in lung and kidney fibrosis models

Redx - Corporate Presentation - 21 June 2023
Preclinical Efficacy of DDR1 Inhibition Demonstrated in a Kidney Fibrosis Model

Murine Unilateral Ureteral Obstruction Model

Reduction of tubulointerstitial damage

<table>
<thead>
<tr>
<th>% Positive Area</th>
<th>UUO Vehicle</th>
<th>UUO REDX12271 15 mg/kg BID</th>
<th>UUO REDX12271 50 mg/kg BID</th>
</tr>
</thead>
<tbody>
<tr>
<td>Interstitial Damage</td>
<td></td>
<td></td>
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</tbody>
</table>

10-Day therapeutic murine UUO kidney fibrosis model (Oral dosing from day 5 post surgery)

Reduction of Fibrosis

Representative images

Kidney histology Picrosirius Red

- Vehicle
- 15mg/kg BID REDX12271
- 50mg/kg BID REDX12271

Target Engagement in Kidney Tissue

- UUO Vehicle
- UUO REDX12271 15 mg/kg BID
- UUO REDX12271 50 mg/kg BID

- 8-Day prophylactic murine UUO kidney fibrosis model (Oral dosing from day -1 with surgery performed on day 1)

Source: Data Generated by Redx, as presented at ASN Kidney Week 2022
Financed into Q1 2024 and Through Significant Catalysts to Continue Portfolio Momentum

Cash runway to support near-term milestones

- RXC004 Phase 2 combination data
- RXC008 CTA submission

Medium/long-term value expansion

- zelasudil Phase 2a topline data
- RXC008 Development in fibrostenotic Crohn’s
- RXC004 Explore partnership
- zelasudil Potential in ILD and cancer-associated fibrosis
- Discovery Engine 2 further INDs by 2025

Supported by top-tier specialist investors

AIM (UK) listed Ticker: REDX
Total shares in issue: 334,911,458*
Fully diluted: 487,776,686**

* As of 31 March 2023
** Assuming full conversion of loan notes and exercise of employee share options. Updated 31 March 2023