Barcelona 2026 ueg week

Redx

View the ROCKETEER Phase 2 Trial

Fira Gran Via
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RXC008: First-in-class gastrointestinal-targeted potent pan-ROCK inhibitor for treatment of Fibrostenotic Crohn's Disease

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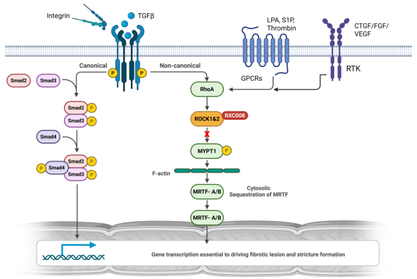
Introduction

Redx Pharma is developing RXC008, a gastrointestinal (GI)-restricted highly potent and selective 'pan-ROCK' inhibitor (inhibits rho-associated coiled-coil kinase ROCK1 and ROCK2) to treat fibrostenosis due to Crohn's disease (FSCD).

ROCK1/2 are key signalling kinases in fibrosis and suppression of ROCK activity is highly efficacious in multiple rodent models of fibrotic disease.

ROCK sits at a nodal point in fibrotic signaling pathways[1,2]



GI-Restricted Mechanism of RXC008 Removes the Limitations of Systemic pan-ROCK Inhibition

Systemic ROCK1/2 inhibition is associated with hypotension, limiting therapeutic utility. RXC008 was therefore designed to be GI-restricted and minimise systemic exposure through three complementary mechanisms: low permeability with high efflux, rapid plasma metabolism by paraoxonase enzymes, and rapid hepatic clearance.

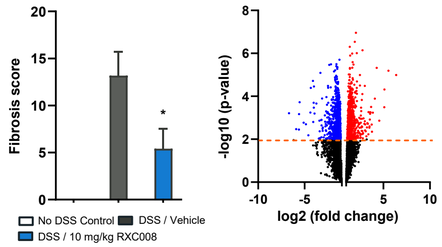
Preclinical Methods

Anti-fibrotic activity was assessed in the chronic Dextran Sulfate Sodium (DSS) and 45-day adoptive T-cell transfer murine models of colitis. Fibrosis was evaluated by Masson's Trichrome staining and University of Ghent fibrosis scoring. RNA-seq was used to assess gene expression changes, while digital pathology quantified smooth muscle thickness and collagen area. Colon and plasma drug concentrations were measured to confirm GI restriction of RXC008.

(1) Julian and Olson, 2014. (2) Knipe et al., 2015.

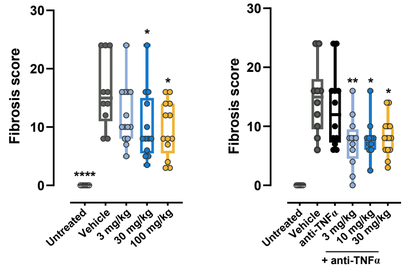
Preclinical Results

RXC008 Demonstrated Monotherapy Activity in the DSS Model

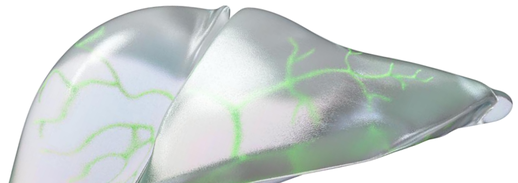


- In the DSS model, oral dosing (po) of RXC008 10 mg/kg once daily (QD) significantly suppressed colon fibrosis.
- Using RNA-seq analysis RXC008 modulated the expression of >2000 genes (p≤0.01, log2 fold change).

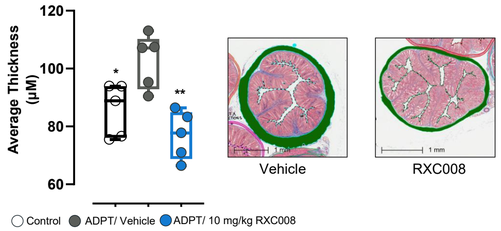
RXC008 Demonstrated Both Monotherapy and Combination Activity in the Adoptive T-cell Transfer Model (ADPT)



- Monotherapy RXC008 (30 or 100 mg/kg po QD) reduced the fibrosis score in the ADPT model, anti-TNFα alone had no effect on fibrosis.
- RXC008 (3, 10 and 30 mg/kg po QD) significantly reduced fibrosis in combination with anti-TNFα.



RXC008 Reduced the Muscularis Propria Thickness in the Adoptive T-cell Transfer Model (ADPT)



- Digital pathology demonstrated monotherapy RXC008 (10mg/kg po QD) reduced muscularis propria thickness observed in Mouse ADPT model.

Clinical Methods

Randomised, placebo-controlled Phase 1 study comprising single ascending dose (SAD) and multiple ascending dose (MAD) cohorts. Healthy participants were randomised 2:1 to receive single doses of RXC008 (10-1000 mg) or placebo in the SAD phase, and 3:1 to receive once-daily RXC008 (30, 100, or 300 mg) or placebo in the MAD phase. The primary objective was to evaluate the safety and tolerability of single and multiple oral doses of RXC008, while the secondary objective was to characterise the pharmacokinetic profile of RXC008 in plasma and colonic tissue.

Clinical Results

RXC008 was generally well tolerated with a favourable safety profile

- Favourable safety profile with no SAEs reported
- No clinically relevant breakthrough observed
- No hypotension observed
- Tissue exposure confirmed
- Minimal treatment emergent adverse events reported (TEAEs)

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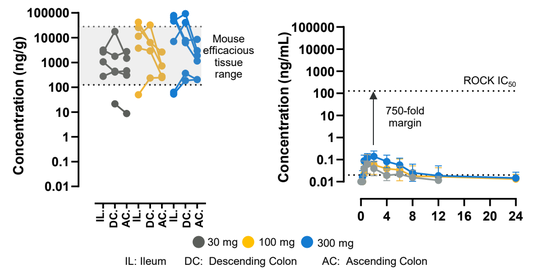
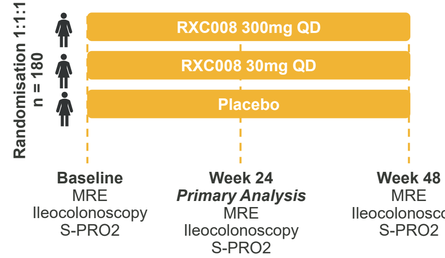
Tissue Exposure Achieved Clinically at Predicted Efficacious Concentrations with Virtually No Systemic Exposure



- RXC008 achieved Ileum & colonic tissue concentrations consistent with preclinical anti-fibrotic efficacy, while maintaining systemic exposure ~750-fold below pharmacologically active levels.

ROCKETEER: A Global Phase 2 Trial to assess the efficacy of RXC008 in FSCD Patients



Patient population

- Symptomatic Fibrostenotic Crohn's patients
- Stable on background SOC anti-inflammatory

Primary endpoint

- Change in stricture length at week 24 (MRE)

Secondary endpoints include

- Safety, PK, Radiographic response, endoscopic passibility , Symptomatic response