

Redx Pharma announces new drug development candidate for fibrosis

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RXC006, a novel, oral porcupine inhibitor, to be developed as a first-in-class treatment for the orphan disease, idiopathic pulmonary fibrosis

First-in-man studies earmarked for 2020

Preclinical data to be presented at the Anti-Fibrotic Drug Development summit, Cambridge, USA on 29 November 2018

Alderley Park, 14 November 2018 Redx (AIM: REDX), the drug discovery and development company focused on cancer and fibrosis, is pleased to announce the nomination of its first development compound to treat fibrosis. The new development candidate, RXC006, is an oral porcupine inhibitor that will be developed as a first-inclass treatment for the orphan disease, idiopathic pulmonary fibrosis (IPF), a severe and life-threatening chronic lung condition with very poor prognosis and limited treatment options.

RXC006 represents a novel approach to treat this debilitating and progressive disease through targeting porcupine, a component enzyme of the Wnt pathway. There is strong scientific evidence that this pathway is critically involved in the scarring process (fibrosis) in the lung that is a hallmark of IPF.¹ This leads, over time, to the lungs being unable to function effectively, ultimately resulting in suffocation and death. Porcupine inhibition suppresses the release of all Wnt ligands and therefore should eliminate one of the major drivers of fibrosis in IPF. The median survival from IPF diagnosis is 3 years and the annual incidence is between 6.8-16.3/100,000 population in the U.S.²

Extensive pre-clinical testing has revealed that RXC006 is very potent and highly effective at suppressing the Wnt pathway, and hence fibrosis, *in vivo* in the lung as well as in the liver and the kidney. Evidence shows that involvement of the Wnt pathway increases with disease severity³ and Redx believe that RXC006 may also prove effective in more severe IPF patients where there is currently no effective therapy beyond palliative care.

Lisa Anson, Chief Executive Officer at Redx Pharma plc commented: "IPF is a devastating disease with little effective treatment and there is, therefore, a clear unmet need for new therapies. Redx is excited to bring its precision medicinal



chemistry expertise to bear with the discovery of this novel drug candidate. We look forward to taking RXC006 into clinical development; we plan to enter first in man clinical trials during 2020, in line with our strategy."

Dr. Peter Bunyard, Redx's Head of Fibrosis will be presenting preclinical data at the 2nd Anti-Fibrotic Drug Development summit in Cambridge USA on Thursday the 29th of November 2018.

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About Redx Pharma Plc

Redx is a UK based biotechnology company whose shares are traded on AIM (<u>AIM:REDX</u>). Redx's vision is to become a leading biotech focused on the



development of novel precision medicines that have the potential to transform treatment in oncology and fibrotic diseases.

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About IPF

IPF is a life threatening fibrotic lung condition with diagnosed prevalence projected to increase from 119,000 (2015) to 138,000 (2025). Current treatment options are OFEV®(nintedanib) and Esbriet® (pirfenidone); both slow progression of disease by approximately 50%. Product sales in IPF are projected to increase from US\$ 0.9b (2015) to US\$3.2b (2025).²

About RXC006

Redx has invested into research to target the Wnt /ß-Catenin signalling pathway by inhibition of the upstream porcupine enzyme and has built considerable knowledge and expertise in this scientific area. Our most advanced porcupine inhibitor, RXC004, is currently being investigated clinically for the treatment of a range of cancers. RXC006 is a potent porcupine inhibitor protected by discrete Intellectual Property and has a predicted human PK profile which will allow flexibility in dosing regimens to balance efficacy with potential side effects. RXC006 is the first porcupine inhibitor aimed at treating IPF.

References

1. Newman DR, Sills WS, Hanrahan K, Ziegler A, Tidd KM, Cook E, Sannes PL.

Expression of WNT5A in Idiopathic Pulmonary Fibrosis and Its Control by TGF-β and WNT7B in Human Lung Fibroblasts. *J Histochem Cytochem. 2016 Feb;64(2):99-111.*

2. Global Data Opportunity Analyser 2015, based on 7 major markets



3. Meuten T, Hickey A, Franklin K, Grossi B, Tobias J, Newman DR, Jennings SH, Correa M, Sannes PL. WNT7B in fibroblastic foci of idiopathic pulmonary fibrosis. *Respir Res. 2012 Jul 28;13:62*.