

Astinova AI Platform — Drug Repurposing Report

Indication context: idiopathic pulmonary fibrosis (IPF) · target/mechanism-anchored hypotheses

Illustrative sample — synthetic data for format demonstration; not a specific client deliverable.

Objective

Identify approved or late-stage agents with a mechanistic rationale for repurposing in IPF, ranked by strength of target linkage and translational evidence. Candidates and rationales below are illustrative and drawn from publicly discussed repurposing biology.

Repurposing candidates

#	Candidate (class)	Primary target / mechanism	Repurposing rationale	Evidence level
1	AMPK activator (biguanide class)	AMPK activation -> inhibits TGF-beta/SMAD fibrotic signalling and myofibroblast differentiation.	Anti-fibrotic in preclinical lung-fibrosis models; large safety database.	Preclinical + epidemiological
2	PDE inhibitor (xanthine class)	Phosphodiesterase inhibition raises cAMP, dampening fibroblast proliferation.	Rationale overlaps with approved anti-fibrotics; oral, well-characterised.	Mechanistic + early clinical
3	ROCK inhibitor	Rho-kinase blockade reduces myofibroblast contractility and ECM deposition.	Strong mechanistic tie to fibrogenesis; inhaled delivery feasible.	Preclinical
4	Ang-(1-7)/MAS agonist	Counter-regulatory RAS arm; opposes pro-fibrotic angiotensin-II signalling.	Attenuates bleomycin-induced fibrosis in models.	Preclinical

Interpretation

Candidate 1 leads on the combination of mechanistic linkage and an established safety/PK profile, which lowers repurposing risk. Candidates 3–4 carry stronger target rationale but thinner clinical evidence and would need dedicated translational studies. Target-linkage claims should be confirmed against current pathway and genetic-association evidence before pursuit.

Repurposing hypotheses are computational/knowledge-graph proposals and require target validation and disease-model confirmation before clinical translation.