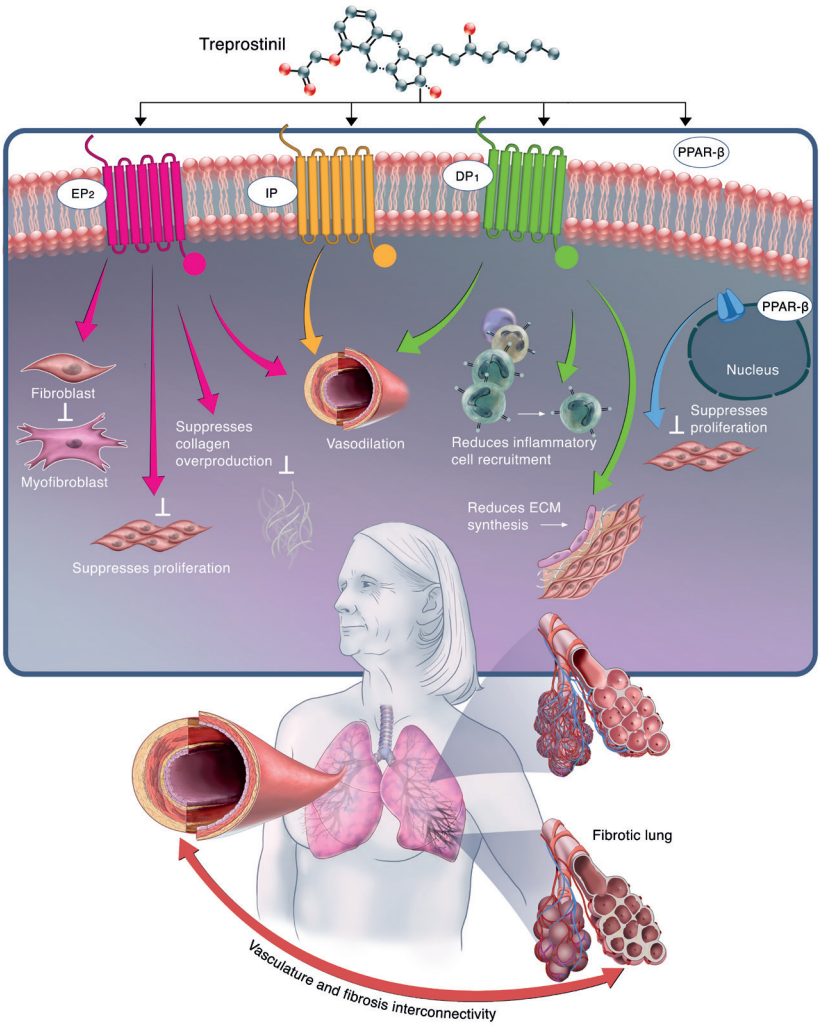
The Antifibrotic Effects of Treprostinil: An Emerging Option for ILD

Martin Kolb, Stylianos Orfanos, Chris Lambers, Kevin Flaherty, Alison Masters, Lisa Lancaster, Adam Silverstein, Steven D. Nathan



The antifibrotic effects of treprostinil are mediated through the activation of the prostaglandin E receptor 2 (EP_2), prostacyclin receptor (IP), prostaglandin D receptor 1 (DP_1), and peroxisome proliferator-activated receptors (PPAR-β)

- Activation of the EP_2, IP, and DP_1 receptors leads to vasodilation
- Activation of EP_2 inhibits fibroblast to myofibroblast differentiation, suppresses fibroblast proliferation, and suppresses collagen overproduction
- Activation of DP_1 reduces inflammatory cell recruitment and reduces extracellular matrix synthesis
- Activation of the nuclear receptor, PPAR-β, leads to suppressed fibroblast proliferation

Summary: Through the activation of EP_2, IP, DP_1 and PPAR-β, treprostinil leads to vasodilation, reduced vascular remodeling, reduced fibroblast activity, reduced proliferation and collagen deposition, and reduced inflammation, thereby promoting antifibrotic activity.

The graphical abstract represents the opinions of the authors.
For a full list of declarations, including funding and author disclosure statements, please see the full text online.

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