## IMAGE



## Insights Image for "Human ucMSCs seeded in a decellularized kidney scaffold attenuate renal fibrosis by reducing epithelialmesenchymal transition via the TGF- $\beta$ /Smad signaling pathway"

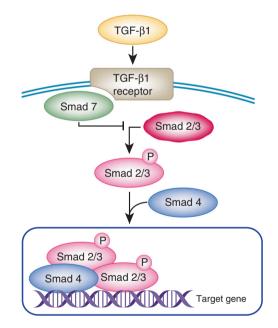
Dong Hu<sup>1,2,3</sup>, Deying Zhang<sup>1,2</sup>, Bo Liu<sup>1,2</sup>, Yang Liu<sup>4</sup>, Yu Zhou<sup>1,2</sup>, Yihang Yu<sup>1,2</sup>, Lianju Shen<sup>2</sup>, Chunlan Long<sup>2</sup>, Dan Zhang<sup>2</sup>, Xing Liu<sup>1,2</sup>, Tao Lin<sup>1,2</sup>, Dawei He<sup>1,2</sup>, Tao Xu<sup>5</sup>, Peter Timashev<sup>6</sup>, Denis Butnaru<sup>7</sup>, Yuanyuan Zhang<sup>8</sup> and Guanghui Wei<sup>1,2</sup>

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Transforming growth factor- $\beta$ 1 (TGF- $\beta$ 1)/Smad signaling pathway. TGF- $\beta$ 1 can activate Smad 2/3 by binding to the membranebound TGF- $\beta$ 1 receptor (T $\beta$ R). Thereafter, Smad 2/3 bound to Smad 4 enters the nucleus and regulates target gene transcription. Smad 7 can bind to the T $\beta$ R and inhibit the activation of Smad signaling [1]. **Publisher's note** Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

## REFERENCE

 Hu, D. et al. Human ucMSCs seeded in a decellularized kidney scaffold attenuate renal fibrosis by reducing epithelial-mesenchymal transition via the TGF-β/Smad signaling pathway. *Pediatr. Res.* https://doi.org/10.1038/s41390-019-0736-6 (2020).



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