

Correction for New perspectives of the cardiac cellular landscape: mapping cellular mediators of cardiac fibrosis using single-cell transcriptomics

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This is a correction to Krstevski, C., Cohen, C.D., Dona, M.S.I. and Pinto, A.R. (2020) New perspectives of the cardiac cellular landscape: mapping cellular mediators of cardiac fibrosis using single-cell transcriptomics. *Biochem. Soc. Trans.* **48**, 2483–2493. 10.1042/BST20191255

Following publication of the above article, the authors noticed a citation was missing from the revised article resulting in errors in [Table 1](#), where the reference numbers associated with studies summarised in the table are incorrect.

The authors apologise for the error and would like to correct the record by including a citation to the following paper as no. 101:

Gladka MM, Molenaar B, de Ruiter H, van der Elst S, Tsui H, Versteeg D, et al. Single-Cell Sequencing of the Healthy and Diseased Heart Reveals Cytoskeleton-Associated Protein 4 as a New Modulator of Fibroblasts Activation. *Circulation*. 2018 Jul 10;138(2):166–180.

And updating the reader to the corrected table below:

Table 1. Single-cell transcriptomic studies investigating cellular and molecular drivers of cardiac fibrosis in heart failure Part 1 of 2

Context	Reference	Cells	Mean Reads/ cell	Technology	Notes and key findings
Mouse myocardial infarction	(101)	426	16,874	Sort-Seq	<ul style="list-style-type: none"> • 3-days post-MI and sham control mice • $n = 3/\text{group}$. • <i>Ckap4</i> proposed as a novel driver of myofibroblast differentiation.
Mouse myocardial infarction	(62)	13,331	~35,000	10X Chromium	<ul style="list-style-type: none"> • 3- and 7-days post-MI and sham control. • $n = 1/\text{time point}/\text{group}$. • Identified heterogeneous fibroblast and myofibroblast subsets involved in fibrosis.
Mouse hypertension (Angiotensin II infusion)	(56)	29,615	~80,000	10X Chromium	<ul style="list-style-type: none"> • 2 weeks AngII-induced experimental hypertension and control mice. • $n = 4/\text{sex}/\text{group}$. • Novel fibroblasts <i>Fibro-Cilp</i> and <i>Fibro-Thbs4</i> implicated in AngII-induced fibrosis. • Almost all cardiac cell types contribute to ECM remodeling after AngII treatment. • Extensive cell and sex-specific gene expression patterns in the non-stressed and stressed hearts.

Continued

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Table 1. Single-cell transcriptomic studies investigating cellular and molecular drivers of cardiac fibrosis in heart failure

Part 2 of 2

Context	Reference	Cells	Mean Reads/ cell	Technology	Notes and key findings
Mouse myocardial infarction	(66)	36,874	~46,000	10X Chromium	<ul style="list-style-type: none"> • 1, 3, 5, 7, 14- and 28-days post-MI and sham control. • $n = \sim 3$/group. • Identified heterogeneous fibroblast and myofibroblast subsets involved in fibrosis. • Early differentiation of myofibroblasts are associated with cardiac rupture.
Healthy and heart failure human patients	(100)	21,422	~300,000 (Median reads/ cell)	iCell8	<ul style="list-style-type: none"> • Healthy organ donors and patients with HF. • $n = 14$ healthy controls, 8 HF patients. • Endothelial cells and fibroblasts are key players in cellular crosstalk in progression of HF.