SCE 0808 Public Evidence Appendix

Kidney disease

Literature review date: September 13, 2026

Controlling principle. Renal evidence is syndrome-, etiology-, product-, route-, stage-, and endpoint-specific. Biomarker, histologic, or mechanistic signals must not be converted into claims of durable kidney recovery or improved patient-important outcomes.

Point-by-point evidence framework

Controlling Point	Evidence focus	Interpretive boundary
Acute kidney injury	Tubular epithelial injury, microvascular dysfunction, inflammation, cytoprotection, and maladaptive repair.	Creatinine or early biomarker changes do not by themselves establish reduced dialysis, durable recovery, prevention of CKD, or improved survival.
Chronic kidney disease	Progressive nephron loss, hyperfiltration, inflammation, vascular injury, interstitial fibrosis, and declining filtration.	Mechanistic or histologic change does not establish class-wide restoration of kidney function or reversal of advanced CKD.
Diabetic kidney disease	Metabolic, hemodynamic, vascular, inflammatory, and fibrotic injury, including glomerular hypertension and albuminuria.	Experimental albuminuria or histology changes do not establish preserved human kidney function or superiority to established kidney-protective therapy.
Transplant-related injury	Ischemia-reperfusion, delayed graft function, rejection, calcineurin-inhibitor toxicity, infection, and chronic allograft dysfunction.	MSC research does not replace standard transplant immunosuppression, rejection treatment, infection control, or graft surveillance.
Renal fibrosis	Extracellular-matrix accumulation, myofibroblast activation, tubular atrophy, capillary rarefaction, TGF-beta signaling, and maladaptive repair.	Reduced fibrosis in a model does not establish restored nephron number, freedom from dialysis, durable filtration recovery, or improved survival.

Evidence-handling rules

Positive, negative, null, mixed, and inconclusive findings are weighted by design quality and directness rather than by whether they favor the intervention.

Mechanistic evidence, development stage, clinical efficacy, safety, regulatory status, guideline position, and manufacturing quality remain separate analytical dimensions.

Hemodynamic and cause-directed AKI care, CKD risk reduction, diabetes and blood-pressure treatment, kidney-protective pharmacotherapy, dialysis when required, and evidence-based transplant care remain the appropriate comparator.

Principal references

1. Galipeau J, Sensebé L. Mesenchymal stromal cells: clinical challenges and therapeutic opportunities. Cell Stem Cell. 2018;22:824-833.

2. Pittenger MF, Discher DE, Péault BM, Phinney DG, Hare JM, Caplan AI. Mesenchymal stem cell perspective: cell biology to clinical progress. NPJ Regen Med. 2019;4:22.

3. Relevant contemporary clinical and translational literature concerning MSC/EV interventions in acute kidney injury, chronic kidney disease, diabetic kidney disease, kidney transplantation, and renal fibrosis.

4. Disease-specific nephrology, diabetes, critical-care, and transplantation guidelines should control statements concerning established standard care.

5. Product identity, renal diagnosis and stage, route, dose, dialysis or transplantation context, background therapy, comparator, endpoint, and follow-up must be matched before evidence is generalized.