SCE 0807 Public Evidence Appendix

Liver and gastrointestinal disease

Literature review date: September 13, 2026

Controlling principle. Liver and gastrointestinal evidence is disease-, product-, route-, stage-, and endpoint-specific. Biological repair signals must not be converted into claims of restored organ function or durable clinical benefit.

Point-by-point evidence framework

Controlling Point	Evidence focus	Interpretive boundary
Cirrhosis	Fibrosis, hepatic stellate cells, portal hypertension, hepatic reserve, and endogenous repair.	Laboratory or short-term score changes do not establish reversal of cirrhotic architecture, fewer decompensations, or improved transplant-free survival.
Acute liver failure	Rapid hepatocellular injury, coagulopathy, encephalopathy, immune signaling, and recovery.	Investigational cell/EV therapy does not replace cause-specific treatment, intensive supportive care, or urgent transplant assessment.
Metabolic liver disease	Steatosis, insulin resistance, lipotoxicity, inflammation, hepatocyte injury, and fibrosis.	Improvements in enzymes, liver fat, or experimental biomarkers do not establish fewer liver-related events, cardiovascular events, or deaths.
Crohn’s fistulas	Local immune modulation and healing of complex perianal fistulas with defined local products.	Product-specific fistula evidence cannot be generalized to intravenous MSCs, luminal Crohn disease, unrelated fistulas, or ulcerative colitis.
Ulcerative colitis	Colon-limited inflammation, epithelial-barrier support, mucosal healing, and validated disease activity.	Mechanistic signals must remain separate from corticosteroid-free remission, hospitalization, colectomy, durability, and safety.
Intestinal injury	Barrier disruption, epithelial restitution, ischemia-reperfusion, radiation, infection, treatment toxicity, and short-bowel contexts.	Improved histology or permeability in one model does not establish restored human intestinal function, nutritional independence, sepsis reduction, or 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, efficacy, safety, regulatory status, guideline position, and manufacturing quality remain separate analytical dimensions.

Established hepatology, gastroenterology, surgical, transplant, metabolic, nutritional, and infection-management care remains 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 cirrhosis, acute liver failure, metabolic liver disease, Crohn fistulas, ulcerative colitis, and intestinal injury.

4. Disease-specific hepatology, gastroenterology, transplantation, surgical, and nutrition guidelines should control statements concerning established standard care.

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