SCE 0803 Public Evidence Appendix

Neurology

Literature review date: September 13, 2026

Controlling principle. Neurologic indications are disease-specific. Investigational MSC/EV mechanisms must remain distinct from established neurologic care and from true neural-cell replacement strategies.

Evidence map

Domain	Evidence posture	Interpretive boundary
Ischemic stroke	Clinical research has focused mainly on post-acute repair, neuroinflammation, vascular support, and plasticity.	Investigational MSC/EV approaches are not substitutes for proven reperfusion therapies such as thrombolysis or mechanical thrombectomy.
Hemorrhagic stroke	Evidence is primarily preclinical or early clinical, with interest in inflammation, edema, vascular injury, and repair.	Findings from ischemic stroke should not be transferred automatically to intracerebral or subarachnoid hemorrhage.
Traumatic brain injury	Preclinical and early human work explores neuroinflammation, trophic signaling, secondary injury, and recovery.	Spontaneous recovery, injury heterogeneity, timing, and rehabilitation complicate causal interpretation.
Spinal-cord injury	Research investigates neuroprotection, inflammation, axonal injury, glial scar biology, remyelination, and functional recovery.	Neurologic improvement must be separated from procedure effects, rehabilitation, and spontaneous recovery.
Multiple sclerosis	Immune modulation and repair-related mechanisms are under study; disease-modifying therapies remain established standard care.	Potential anti-inflammatory or remyelinating mechanisms do not establish superiority to approved MS therapies.
Parkinson’s disease	MSC/EV research focuses mainly on trophic, inflammatory, and supportive signaling.	This is distinct from true dopaminergic-neuron replacement, which represents a different therapeutic strategy.
Alzheimer’s disease	Preclinical work examines neuroinflammation, amyloid/tau-related pathways, synaptic support, and trophic effects.	Biomarker or mechanistic changes do not establish durable cognitive benefit.
ALS	Research explores motor-neuron support, inflammation, trophic signaling, and disease progression.	Small, heterogeneous studies cannot establish class-wide efficacy or meaningful disease modification.
Peripheral neuropathy	Research includes inflammatory, vascular, Schwann-cell, axonal, and trophic mechanisms in diverse neuropathies.	Evidence should be matched to the exact neuropathy, product, route, and clinically meaningful functional outcome.

Key evidence conclusions

Neurologic diseases are not interchangeable. Stroke, traumatic injury, demyelinating disease, neurodegeneration, motor-neuron disease, and peripheral neuropathy involve materially different biology and endpoints.

Mechanistic plausibility is not neural replacement. Anti-inflammatory, trophic, vascular, or repair-related signaling should not be described as replacement of lost neurons unless direct evidence supports that claim.

Controlled functional outcomes are essential. Imaging, biomarkers, or laboratory signals must not substitute for durable patient-important neurologic outcomes.

Established care remains separate. Emergency stroke treatment, approved disease-modifying therapies, symptomatic treatment, and rehabilitation should not be displaced by unproven extrapolation.

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 stroke, traumatic CNS injury, multiple sclerosis, neurodegenerative disease, ALS, and peripheral neuropathy.

4. Disease-specific neurologic guidelines and regulatory sources should control statements concerning established care, emergency treatment, and jurisdiction-specific authorization.

5. Product identity, route, dose, disease stage, comparator, endpoint, and follow-up must be matched before evidence is generalized across neurologic indications.