

Why an Organ May Remain Diseased After Its Biochemistry Improves

Paper 20 · Editorial concept paper · Created 15 October 2026

Abstract. Biochemical normalization and structural recovery are different endpoints. Loss of specialized cells, fibrosis, microvascular remodeling and altered innervation can sustain impaired organ function despite correction of an upstream metabolic or inflammatory measure.

A marker can improve faster than a tissue

Blood measurements often change in hours or days; structural repair may require weeks to years, and some changes cannot reverse. A patient may achieve better glycemic control while established neuropathy persists, or inflammatory markers may fall while lung fibrosis continues limiting gas exchange. These examples do not mean the treatments failed. They reveal distinct timelines for controlling an initiating process and restoring damaged functional architecture.

The structural inventory

Tissue performance depends on the number and identity of living parenchymal cells, the integrity of capillaries, the arrangement of extracellular matrix and the continuity of neural and mechanical connections. A biomarker may report one molecular domain accurately while saying little about these structures. Loss of cardiomyocytes after infarction, fibrotic lung changes or irreversible nephron loss need not disappear because systemic biochemistry becomes favorable.

A distinction among three kinds of improvement

Biochemical response means a measurable pathway changed. **Clinical improvement** means symptoms, function or risk changed. **Structural regeneration** means damaged tissue architecture or specialized cells were restored. They can coincide, but one must not be inferred from another. Serious claims of reversal should specify which outcome was documented and by what method.

Residual injury versus active maintenance

Sometimes dysfunction persists because permanent damage remains; at other times feedback from fibrosis, altered perfusion or chronic immune activity continues producing new damage. The distinction matters. A stable scar might require supportive care and adaptation; an actively fibrosing organ might require disease-modifying therapy. Calling both conditions 'unfinished detoxification' would obscure rather than illuminate their clinical meaning.

Evidence that would strengthen the model

Longitudinal studies integrating functional outcomes, disease activity and structural measures could show whether persistent tissue abnormalities account for residual disability after biomarker correction. Such evidence could identify realistic expectations and additional treatment targets. It should not be interpreted as an argument against evidence-based biomarker-directed therapy, which can prevent future damage even where it cannot reverse old injury.

Conclusion

A corrected laboratory value is meaningful but not automatically a regenerated organ. The Institute's proposal is simply to make that difference explicit whenever we discuss cure, remission, compensation and recovery.

The problem of irreversible units

An organ is not defined solely by its momentary biochemical outputs. It is built from specialized units that may be lost or scarred. A kidney with reduced nephron number may maintain an acceptable filtration measure through compensation while possessing less reserve. A heart with substantial postinfarction scar may produce clinically adequate resting output while its response to demand remains limited. These examples emphasize why function, structure and reserve are distinguishable.

Recovery may be real without complete restitution

Clinical success can mean stabilization or adaptation rather than recreating the pre-injury organ. Treatment of heart failure can improve symptoms and survival without erasing every scar; controlling metabolic risk may prevent progression without fully reversing neuropathy. A rigorous Institute paper should celebrate such outcomes rather than framing them as failures because the organism has not returned to an idealized untouched state.

A practical reporting taxonomy

For every intervention, record whether the evidence concerns a biochemical change, a symptom change, improved function, demonstrated structural remodeling or reduced future events. These dimensions should be independently stated. The taxonomy can prevent exaggerated claims about 'full reversal' while allowing genuinely meaningful partial recovery to be described with precision and dignity.

Selected scientific reading

1. [Liu F et al. Mechanosignaling through YAP and TAZ drives fibroblast activation and fibrosis. Am J Physiol Lung Cell Mol Physiol. 2015.](#)
2. [Campisi J et al. Cellular senescence: a link between cancer and age-related degenerative disease? Semin Cancer Biol. 2011;21:354–359.](#)

3. [Østergaard L et al. Microcirculatory dysfunction and tissue oxygenation in critical illness. Acta Anaesthesiol Scand. 2015;59:1246–1259.](#)

Editorial status: Conceptual scientific essay, not peer reviewed. Not original clinical data, a treatment protocol or medical advice. Selected readings are not a systematic review. Evidence and interpretation should be assessed independently.