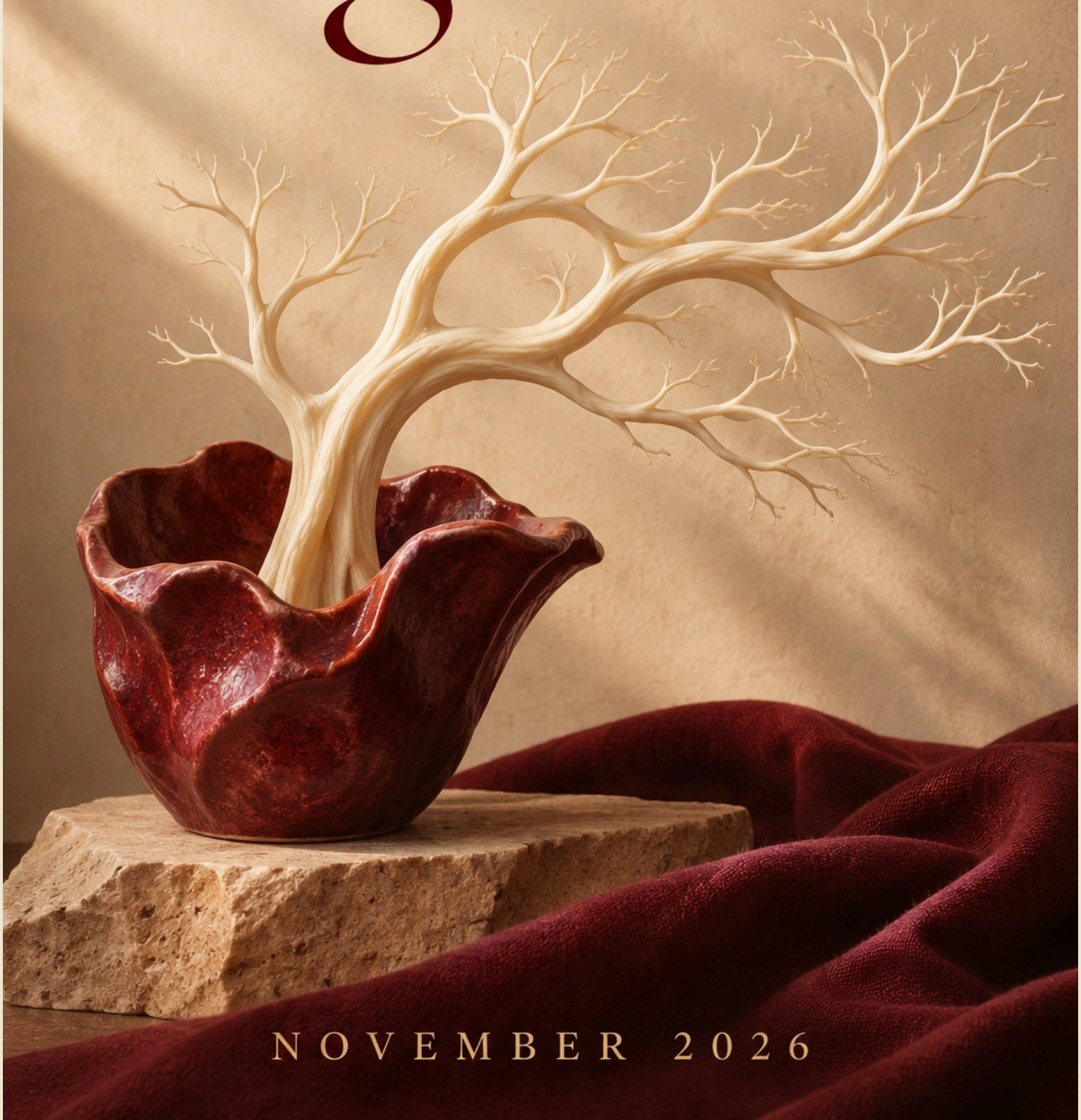
THE ARISING

Organ Regrowth



NOVEMBER 2026

An organ is an arrangement

Replacing an organ asks more of biology than making the right cell type. Cells must be arranged, supplied, connected, and able to perform coordinated work. The route by which fluid enters and leaves can be as consequential as the identity of the cells. This is why organ-regrowth stories should specify the scale of the achievement. A cell culture, a tissue fragment, and a transplantable organ solve different parts of the problem.

The kidney makes that distinction unusually clear. Blood must reach a fine network of filters. Tubules must recover useful substances and adjust what remains. The resulting fluid needs a route out. Producing fluid alone does not establish that this system controls the body's chemistry effectively. A convincing replacement must combine structure with coordinated performance, rather than display one recognizable feature. [1]

Several research strategies address different pieces of this challenge. A scaffold preserves an existing organ's architecture and invites new cells to occupy it. Developing organ buds and organoids use cells' ability to organize into tissue. Cell replacement can supply a particular function without rebuilding the organ that originally provided it. These approaches share an ambition, but their experiments and measures of success are not interchangeable. [1] [2] [3] [4]

The word human also needs care. It may describe the origin of cells rather than the recipient of a transplant. Human-derived tissue working inside a mouse is a preclinical finding. It is not evidence that the same construct can replace an adult human organ, withstand that workload or remain safe for years. The species, implant location and duration of observation belong beside the result.

The studies in this issue illuminate that distance. Two foundational demonstrations appeared in 2013, another kidney experiment in 2018, and a human islet-cell study in 2025. Read together, they show why replacing useful function can advance before whole-organ reconstruction. They do not establish routine clinical regrowth of complete human kidneys, livers or pancreases.



Coordinated work, not four isolated signals

For a kidney, blood supply, filtration, selective recovery and drainage must work together. A functioning part is not yet a replacement organ. [1]

A kidney scaffold, with limits

A 2013 study by Jeremy Song and colleagues began with the organ's physical framework. Detergent perfusion removed cells from rat, pig and human kidneys while retaining a scaffold with vascular channels and a collecting system. The reseeding and transplant experiments used rat kidney scaffolds. Keeping those steps separate prevents an important misunderstanding: preparing a human scaffold was not the same as rebuilding and transplanting a working human kidney. [1]

The investigators introduced human umbilical-vein endothelial cells through the vascular route and neonatal rat kidney cells through the urinary route. A bioreactor supported the reseeded constructs. The cells occupied parts of the preserved architecture, and the grafts produced rudimentary urine during laboratory perfusion. After experimental transplantation into rats, blood flowed through the grafts and urine emerged through the ureteral conduit. [1]

That was a substantial proof of principle. The structure offered both a route for blood and a route for drainage, while the added cells supplied some selective functions. Yet the measurements also showed immaturity. Filtration was reduced compared with native kidneys, and recovery of glucose and electrolytes remained incomplete. The researchers described further work on cell delivery, maturation and scaling as necessary. [1]

The distinction between a demonstration and a dependable replacement is not semantic. An organ can make urine without doing enough useful work to free a recipient from dialysis. This experiment did not establish durable renal-replacement independence in people, and it should not be presented as a clinical transplant success. Its value lies in showing that preserved architecture can help organize living cells into a partly functioning graft.

A scaffold also does not solve the cell-supply problem by itself. An intact outline can still contain too few mature cells, incomplete coverage or poorly coordinated compartments. Readers should therefore look beyond the photograph of a translucent organ. The consequential evidence is whether its occupied channels, filters and tubules work together under the conditions a recipient would actually impose.



Song and colleagues preserved kidney architecture, added cells and tested the resulting grafts in rats. Human-sized scaffolds were not human replacement kidneys. [1]

Liver buds find a blood supply

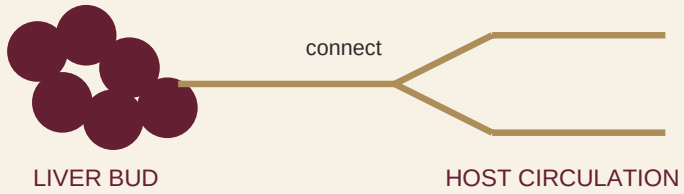
A different 2013 experiment, led by Takanori Takebe, began with developing tissue rather than an emptied organ. The team combined liver-destined cells derived from human induced pluripotent stem cells with endothelial and mesenchymal cells. Their interactions produced three-dimensional liver buds, small assemblies whose organization resembled an early developmental stage rather than a finished adult liver. [2]

After transplantation into mice, vessels in the buds connected with the host circulation within 48 hours. The resulting tissue matured and performed selected liver functions, including protein production and human-specific drug metabolism. Transplantation also rescued a drug-induced lethal liver-failure model. These findings were important evidence that a developing tissue could gain circulation and contribute useful biological work inside an animal. [2]

The result did not mean that the investigators had grown a complete adult liver ready for a patient. The recipients' livers were not replaced. Human described the source of key cells and the character of certain measured functions; the recipients in these experiments were mice. A small tissue graft helping an experimental injury model is different from an organ reliably carrying a person's full metabolic workload.

That difference explains why vascularization receives so much attention. Growing tissue needs access to oxygen and nutrients, and its products need a route into circulation. A vessel-like structure in a dish, a perfused vessel in a recipient and a durable vascular system suitable for an organ transplant are distinct accomplishments. Establishing a connection is a milestone, not a complete answer to scale, stability or safety.

The broader lesson is that tissue assembly and function can develop together. Cells respond to neighboring cells and to their environment, not just to a laboratory instruction naming their desired identity. The experiment supports that developmental strategy. It does not supply a timetable for whole-liver replacement. A useful next claim would need to specify the amount of tissue, the work it performs and the recipient burden that remains.



Human-derived liver buds connected to mouse host vessels and performed selected liver functions. The recipients' own livers were not replaced. [2]

A nephron is not a renal tract

In 2018, Ioannis Bantounas and colleagues reported another route toward kidney tissue. They differentiated human pluripotent stem cells into kidney precursors and implanted them beneath the skin of immunodeficient mice. By approximately 12 weeks, the implants contained kidney structures with blood-perfused glomeruli, including human capillaries, alongside tubules. The study therefore joined human cell identity to a distinctly animal experimental setting. [3]

The team did more than inspect shape. After injecting a small fluorescent dextran into the host circulation, they detected fluorescence in a subset of tubules. Together with the vascular and structural observations, this supported filtration followed by tubular uptake. It was evidence of functioning nephron components, the kidney's filtering-and-processing units, rather than just expression of kidney-related molecular markers. [3]

The full report also identified missing infrastructure. The tissue lacked large arteries, branched collecting ducts were sparse, and no structures resembling a renal pelvis or ureter formed. Those last two structures matter because a useful kidney must deliver urine into the lower urinary tract. A local filtration signal does not show that waste leaves the recipient through a complete, controlled drainage system. [3]

This is a practical way to interpret organoid headlines. An organoid is a tissue model that reproduces aspects of organ organization; the name does not certify a miniature organ with every adult function. Its value may be considerable for studying development or disease even when transplantation remains far away. The same experiment can be a strong research tool and an incomplete replacement strategy.

Long-term fate also remained open. The authors called for longer observation, and their tissue included immature areas and occasional cartilage. The absence of typical teratomas in the reported kidney-progenitor implants was encouraging, but it did not settle long-term human safety. The relevant question is not simply whether the intended cells appeared. It is what else developed, how consistently the tissue formed and whether its behavior remained controlled. [3]



In the 2018 mouse implants, filtration-related evidence did not establish a complete urinary route. No renal pelvis or ureter formed. [3]

Function can arrive in smaller units

Cell replacement may sometimes address a narrower function without rebuilding an entire organ. The 2025 study of zimislecel, an allogeneic stem-cell-derived islet-cell therapy, makes that distinction concrete. Islets provide hormone-producing cells involved in glucose control. Delivering those cells is a different undertaking from constructing a complete pancreas with all of its endocrine and digestive functions. [4]

The phase 1-2 report analyzed 14 participants with type 1 diabetes who had completed at least 12 months of follow-up. Two received a half dose and 12 received a full dose. The cells were infused into the portal vein, and every participant received immunosuppressive treatment. After infusion, detectable C-peptide supported engraftment and islet function in all 14 participants. [4]

Among the 12 full-dose participants, 10 were insulin independent and not using injected insulin at day 365. All 12 were free of severe hypoglycemic events in the assessed period and had glycated hemoglobin below 7%. These are clinically meaningful observations, but the denominator and follow-up belong with the headline. Ten of 12 is not an expected success rate for every person with type 1 diabetes. [4]

The burdens were consequential. Neutropenia was the most common serious adverse event, affecting three participants. Two deaths occurred: one from cryptococcal meningitis and one from severe dementia with agitation associated with progression of preexisting neurocognitive impairment. Listing those events does not by itself establish that the cell product caused them. It does show why efficacy and the risks of the whole treatment strategy must be considered together. [4]

The analyses were interim and not prespecified, and the authors described the study as small and short term. Longer-term durability, safety and applicability require further investigation. The result supports restoration of a specific function in selected study participants; it does not establish pancreatic regrowth or a universal cure. It also cautions against treating immunosuppression as a minor footnote to an otherwise complete solution. [4]



10 / 12

Insulin independent at day 365

Full-dose group | Immunosuppression for every participant

At day 365, 10 of 12 full-dose participants were insulin independent. All participants received immunosuppression; this was islet-cell therapy, not pancreatic regrowth. [4]

Judge what the recipient can rely on

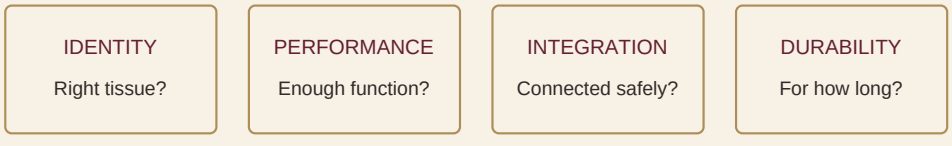
A dramatic image is informative only when paired with the right measurements. Start with identity: which cells and structures are present, and which are missing? Then ask about performance: what does the tissue actually do, how much work can it sustain and how does that compare with the function being replaced? These questions prevent appearance from standing in for capability.

Next examine integration. Where was the tissue placed? How did blood reach it? Where did its waste or secretions go? Was the recipient an animal, a healthy volunteer or a patient with severe disease? A result beneath a mouse's skin and a result in a human clinical study belong at different points in the evidence, even when both use human-derived cells. [2] [3] [4]

Durability changes the meaning of success. A useful signal during an experiment, a functioning graft after weeks and stable benefit over years are not equivalent. Neither is a favorable measurement the same as freedom from an existing treatment. Look for the duration actually observed, the support still required and the adverse events reported alongside benefit. Leave unanswered questions open rather than filling them with optimism.

Manufacturing deserves the same attention as biology. A future replacement must be made reproducibly, with an understood cell composition and dependable function. The foundational kidney studies point to incomplete maturation, cell distribution and drainage as real engineering problems. The islet study shows that even a narrower functional replacement can bring substantial treatment burdens. Progress in one approach does not remove the requirements facing another. [1] [3] [4]

A future success would be judged by what the recipient can rely on, not simply by whether the graft resembles the organ it is intended to replace. The possibility of rebuilding tissue is extraordinary. Its practical meaning will be earned through the patient, persistent work of making the new structure function as part of a living person. For now, the most useful reading habit is precise curiosity: appreciate each achieved function, name the remaining gap and resist turning a promising component into an entire organ.



Identity, useful performance, integration and durability answer different questions. A strong result in one does not establish the others.

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Reading across different kinds of evidence

The kidney-scaffold, liver-bud and kidney-progenitor studies are preclinical demonstrations. Their human-derived material does not turn them into human transplant trials. The islet-cell report concerns a specific cellular function in a small phase 1-2 study, with immunosuppression and important safety events.

These selected studies illustrate different regenerative strategies. They do not constitute an exhaustive survey, a treatment recommendation or proof of whole-organ regrowth in people. Organ preservation, donor-organ transplantation and cell replacement should each be assessed on their own evidence.