

Global Research Map of Potential Therapies and Pathways Towards a Cure for Type 1 Diabetes

SP Science Research Centre by Sugar Pulse

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Purpose of the document

This map is one of the first analytical outputs of SP Science. We have organised the global research landscape to establish the centre's priorities, prepare consultations with researchers and select testable questions for the two-year programme. It provides a common foundation for internal research planning and open dialogue with partners.

Key conclusion

For long-standing type 1 diabetes (T1D), islet cell replacement and graft protection are closest to restoring autonomous glucose regulation. Some people have achieved independence from external insulin, but the most mature approaches require immune therapy, complex infrastructure and strict patient selection. The next major milestone is reproducible, durable independence from both external insulin and chronic systemic immunosuppression. Broad access to this outcome, especially for children, has not been established. [1–12]

Early T1D calls for a different strategy: preserving remaining cells and slowing autoimmunity. Teplizumab is the most mature specific immunotherapy in this setting. These findings cannot automatically be applied to people with many years of disease and little remaining beta cell reserve. [17–18]

This document brings together a readiness scale, a tree of research directions, a catalogue of approaches and criteria for selecting projects. It distinguishes established findings, developer reports, early hypotheses and approaches without verifiable supporting evidence.

How to use this document

Readiness describes how far a particular technology has progressed. Promise is an analytical assessment of its potential to solve an important T1D problem. These are different properties: an early project may be strategically attractive, while a late-stage clinical programme may stop after a negative result.

Insulin independence means no need for external insulin during the stated observation period. It does not establish elimination of autoimmunity, lifelong benefit or freedom from other treatment. In this review, “functional cure” is used only in this limited sense.

Scope: major global research classes, leading programmes covered by the review and important experimental branches. This is an extended landscape map, not an exhaustive list of every registered trial or laboratory paper. A full systematic register requires a separate search strategy, registry deduplication and publication screening. Unresolved programme statuses are identified explicitly.

Purpose: research planning. The document is not a treatment protocol, an individual treatment recommendation or confirmation that an approach is authorised in Russia or the UAE. It contains no financial commitments by the centre.

1 Readiness for application

The order below reflects the readiness of specific approaches. Levels do not predict the number of years until treatment becomes available. Safety, manufacturing scale and the target population require separate assessment for each project.

Level	Evidence available	Examples and principal limitation
I Application	An authorised method or specialist clinical practice	Pancreas and donor-islet transplantation; Lantidra; teplizumab for specified indications. Patient and geographical restrictions.
II Advanced clinical development	A late-stage clinical programme with a strong human signal	Zimislecel. Insulin independence with immunosuppression; this review does not confirm approval.
III Early clinical development	Human trials, small series or randomised signals requiring further study	Donor islets with tegoprobart, Cell Pouch, UP421, Encellin, baricitinib, ustekinumab, verapamil.
IV Preclinical development	Human-cell and animal studies; preparation for first clinical studies	SC451 in the confirmed August update; iTOL-102; local immune protection; DYRK1A with GLP-1.
V Exploratory science	A testable mechanism and early proof of principle	Cell reprogramming, synthetic secretion systems, electrical and radiofrequency control.
VI Unsubstantiated ideas	Insufficient verifiable support for therapeutic use	Bioenergy treatment, wave rewriting and disease frequencies without a validated mechanism and reproducible data.

The separate marker X denotes discontinuation, a negative result or substantial uncertainty. It is not another step up the readiness scale. For example, reaching phase III does not make Diamyd ready for use after a negative interim analysis. [27]

The separate marker M denotes a research method. Bioinformatics, biophysics, omics, mathematical modelling and AI are tools used across all readiness levels, rather than approaches ranked below clinical medicines.

Priority definitions

P1: central to the centre's objective—cell mass restoration, immune protection and graft survival. P2: a promising component or a route to preserving residual function. P3: monitor until stronger evidence emerges. P0: exclude from a therapeutic programme without reproducible evidence. These priorities are the authors' analytical assessments, not official ratings assigned by developers.

2 Tree of research directions

The tree is organised by the task to be solved. Each branch extends from clinical methods to early approaches. A project may belong to several fields: a hypimmune islet combines cell therapy, genetic engineering and immunology.

Main branch	Subfields	Intended patient outcome
A Restore cells	Donor transplantation; ESC/iPSC-derived cells; autologous cells; xenogeneic islets; regeneration in vivo	Adequate glucose-responsive insulin secretion
B Protect cells	Systemic immunotherapy; encapsulation; hypoimmune editing; local protection; antigen tolerance	New or remaining cells escape destruction
C Sustain function	Vascular support; oxygen and nutrients; biomaterials; fibrosis control; implant monitoring and retrieval	Viable and controllable cells
D Preserve reserve	Teplizumab; other immune targets; cell-stress protection; antiviral strategies	Slower loss of endogenous secretion
E Control without cell restoration	A1D and CGM; new insulins; adjunctive medicines; synthetic delivery systems	Better control without necessarily removing the cause of T1D
F Understand and predict	Genetics and omics; immune endotypes; biophysics; AI and models; biomarkers	More precise selection and validation of treatments
G Test unconventional ideas	Reproducible physical hypothesis; controlled experiment; independent replication	Evidence of an effect first; treatment claims only after clinical validation

Why directions cannot be ranked on a single scale

For long-standing T1D, restoring adequate cell mass provides the most direct route to insulin independence. Before clinical onset, immune prevention is more relevant. For a child with a recent diagnosis, preserving residual secretion matters. Readiness is assessed against a defined objective and population, rather than the apparent sophistication of a technology.

Long-term planning should compare combinations: cell source, protection strategy, delivery site and controllability. A membrane or immune drug alone rarely addresses all four tasks.

3 Approaches closest to a practical outcome

01 Donor pancreas transplantation

I • transplantation • P1 as a clinical benchmark. Organ transplantation can restore insulin secretion. It requires major surgery and immune therapy; combined kidney and pancreas transplantation is a particularly important context. Donor availability and the balance of risk and benefit limit broad use in T1D. For the centre, it establishes the possibility of durable functional restoration, but is not a simple scalable platform. [1]

02 Donor islets and Lantidra

I • cell therapy • P1. Lantidra consists of donor islets, not islets grown from stem cells. The FDA label covers selected adults with recurrent severe hypoglycaemia despite intensive diabetes management and education, together with immunosuppression. Donor scarcity, variability of cell material and prolonged immune therapy remain barriers. US approval does not establish universal availability. [1–2]

03 Zimislecel formerly VX 880

II • Vertex • stem cell derived islets with immunosuppression • P1. In the June 2025 data report, 10 of 12 full-dose participants with one year of follow-up were insulin independent at month 12. All 12 achieved HbA1c below 7% and time in range above 70%. This was a specific evaluable group, not everyone ever enrolled. The company reported two deaths in the programme judged unrelated to zimislecel itself; this does not remove the need to assess risks of the entire treatment regimen. [3]

The Vertex pipeline page lists the programme as phase 1/2/3. Potentially reproducible islet manufacturing is an advantage; the need for anti-rejection therapy is the principal limitation. Previously announced filing plans do not establish regulatory approval or a launch date. [4, 41]

04 Donor islets with tegoprubart

III • UChicago and Eledon • cell therapy with anti-CD40L • P1. In its 3 September 2026 announcement, Eledon reported insulin independence in all 12 treated participants, HbA1c below 7% and stable graft function with follow-up reaching 22 months. This is a small research series, not evidence of equivalent outcomes in every population. Twenty-two months is the maximum follow-up, not the duration for every participant. [5]

The company submitted an IND for its own registrational islet transplantation study. IND submission is not drug approval, and phase III plans in kidney transplantation do not constitute phase III T1D evidence. [5, 42]

4 Bioengineering and cell delivery

05 Sernova Cell Pouch

III • bioengineering with donor islets • P1. Cell Pouch creates a site for cells and their vascular support. It is not automatically an immunoprotective capsule: the clinical donor-islet programme uses immunosuppression. The company reports long-term cell survival and insulin independence in cohort A. A rigorous comparison must distinguish the contribution of the implant from any additional islet infusions. [9, 43]

On 8 September 2026, Sernova and Seraxis announced a merger agreement to form BetaNova. Closing was expected later and should not be described as completed. The announcement states that treatment and follow-up in the donor-islet Cell Pouch trial had been completed; older website pages may still describe the study as ongoing. [8]

06 Encellin ENCRT and ENC 201 CED

III • encapsulation • P1. A nanoporous membrane is designed to pass required molecules and support enclosed cells. A July 2026 announcement described seven adults, signs of non-fibrotic engraftment and surrounding vascularisation. The phase I study primarily assessed safety and was not designed to demonstrate clinical efficacy. These data do not establish broad insulin independence without immunosuppression. [11–12, 45]

07 Seraxis SR 02 and SR 03

SR-02: transition to clinical development; SR-03: IV • P1. The September merger plan describes SR-02 as stem cell derived islets, with first dosing in the new programme planned for Q1 2027, and cites NCT07581197. An IND for genetically modified SR-03 is planned for the second half of 2027. These are developer plans. They must not be replaced by older forecasts, and donor-islet successes cannot be attributed to these new products. [8]

08 Oxygen delivery devices and Beta O2

IV for new designs • biophysics and biomaterials • P2. The objective is to prevent islet loss from oxygen shortage inside a device. Experimental publications report improved cell viability with enhanced oxygen supply. Key questions include oxygen replenishment, system size, durability and safety. A historical paper does not establish the current availability of a particular device. [38]

09 Vascular support and fibrosis control

III–V depending on platform • P1. Prevascularisation, hydrogels, pore architecture, antifibrotic coatings and supporting cells address practical engraftment problems. Good secretion in vitro does not guarantee adequate oxygen supply in vivo. VEGF and endothelial-cell studies demonstrate the importance of the microenvironment; excessive or poorly organised vessel growth also requires control. [12, 47]

5 Genetic engineering and cell sources

10 Sana UP421

III • hypoimmune donor islets • P1. Sana's August 2026 update describes one patient with cell survival and function through 14 months without immunosuppression, supported by C-peptide and meal-response testing. This is important proof of concept for immune evasion. It does not demonstrate complete insulin independence: the programme had a different primary objective and cell dose. [10, 44]

11 Sana SC451

IV based on the confirmed 10 August 2026 announcement • P1. SC451 combines hypoimmune technology with iPSC-derived islets. The company reported preparation of toxicology studies, manufacturing and an IND, with a possible phase 1/2 start in 2026. The materials covered by this review do not establish that first dosing has occurred. UP421 findings cannot automatically be treated as SC451 findings. [10]

12 CRISPR Therapeutics CTX211

III based on a registered early clinical programme • P1. The approach combines genetically modified stem cell derived islets with a delivery device, addressing immune evasion and cell survival. Trial identifier: NCT05565248. The full current recruitment status and final outcomes were not confirmed from the accessible registry display. It is included as an important clinical branch, not an established treatment. [13]

13 Autologous iPSC and chemical reprogramming

III • personalised cell therapy • P1/P2. Wang and colleagues reported insulin independence in 2024 after transplantation of islets derived from a patient's chemically induced pluripotent cells. Concomitant immunosuppression in that clinical context matters: the case does not establish that a person's own reprogrammed cells resist autoimmunity. Barriers include individual production costs, genetic stability and tumour-risk control. [15]

14 Islet xenotransplantation

III for selected programmes; IV–V for new designs • P2. Pigs may provide a more available islet source. OPF-310 has a clinical programme, NCT06575426; other work combines genetically modified donor animals with immune protection. Questions include cell maturation and dose response, cross-species immunity, infectious safety and durability. An individual patient account cannot replace study results. [14]

15 Safety control for edited cells

IV–V • enabling technology • P1. Removing immune-recognition signals must be coupled with control of unwanted growth, genomic changes and the ability to destroy or retrieve the graft. Immune invisibility is both an advantage and a potential risk. Product purity, residual pluripotent cells and controllability must be assessed. This is a project-selection criterion, not a separately established treatment.

6 Immunology and pharmacology

16 Teplizumab

I for specified indications • anti-CD3 • P1 for early T1D. In the EU, Teplizumab is indicated to delay progression from stage 2 to clinical stage 3. Sanofi's June 2026 announcement also reports accelerated US approval of Teplizumab for children aged 8–17 with recently diagnosed stage 3 disease to slow loss of endogenous insulin production. This preserves function rather than replacing cell mass already lost in long-standing T1D. Indications in different jurisdictions must be distinguished. [17–18]

17 Tegoprunar and CD40L blockade

III in islet transplantation • P1. The drug reduces immune activation and protects a graft; it does not replace islets. The research centre describes maintenance infusions every three weeks following the initial regimen. The appropriate description is insulin independence during anti-rejection therapy. [6–7]

18 Baricitinib and the JAK pathway

III • pharmacological immunomodulation • P2. The randomised BANDIT trial demonstrated preservation of beta cell function in recently diagnosed T1D. This supports further research, not proven regeneration in long-standing disease. Durability after treatment withdrawal and the balance of immune risks are critical. [19]

19 Ustekinumab and IL 12 and IL 23

III • immunology • P2. A randomised phase II trial in adolescents showed a signal of preserved secretion. The target differs from anti-CD3 and JAK, making it relevant to endotype comparisons and combinations. Preserving C-peptide is not equivalent to insulin independence. [20]

20 Golimumab and TNF

III • immunology • P2. A study in young people with recent onset showed preservation of beta cell function. Remaining questions concern durability, patient selection and the justification for prolonged systemic treatment. Evidence from other autoimmune indications does not replace T1D-specific evidence. [21]

21 Rituximab and B cells

III • immunology • P2. B-cell depletion has been studied to slow loss of secretion at onset. A research signal has been demonstrated, but immune intervention does not automatically create new islets. Rational sequences with other immune targets are a possible future direction. [22]

22 Abatacept and costimulation

III with negative findings for certain objectives • P2. In a prevention trial involving relatives with stage 1 T1D, the primary endpoint of delaying progression was not statistically significant. The approach therefore cannot be described as generally proven to prevent T1D. The drug, disease stage and endpoint must be specified. [23]

23 Low dose ATG

III • immune remodelling • P2. Clinical studies report preserved C-peptide after recent onset. The intervention affects the immune system and is not a universal ready-to-use solution. Dose, age, safety and residual reserve matter. [24]

7 Tolerance and local protection

24 Polyclonal regulatory T cells

III • cellular immunology • P1/P2. Treg infusion seeks to strengthen natural suppression of autoimmunity. Early studies established feasibility; a later randomised programme illustrates why cell safety and persistence cannot be equated with efficacy. Each product requires evidence of durable control of the relevant immune response and preserved secretion. [25–26]

25 Antigen specific Treg and CAR Treg

IV–V as a class for T1D • P1. The aim is to direct regulatory cells towards an islet or graft antigen and avoid unnecessary systemic suppression. This is a promising engineering field, but CAR-T success in cancer or other autoimmune diseases does not establish a cure for T1D. This review does not identify a mature, effective CAR-Treg treatment for human T1D.

26 GAD65 and Diamyd retogatein

X following phase III • antigen-specific immunotherapy • P3. DIAGNODE-3 did not meet prespecified continuation criteria. A July 2026 analysis discusses administration technique, treatment timing and product characteristics, but these explanations remain exploratory. They are not established causes of failure or proof of benefit in a selected subgroup. [27]

27 Proinsulin peptides and IMCY 0098

III for IMCY-0098; IV–V for new peptides • P2. Modified antigenic peptides seek to redirect the pathological immune response. IMPACT investigates biomarkers and beta cell function. A peptide vaccine is not necessarily an infectious-disease vaccine and does not replace insulin. A locally developed proinsulin peptide must be assessed on its own molecule and data; a shared theme does not establish product identity. [28, 39]

28 Tolerogenic nanoparticles and CNP 103

III based on a registered trial • P2. Antigen delivery in a context intended to promote tolerance rather than immune attack. CNP-103 is registered for recently diagnosed adolescents and adults aged 12–35. A protocol establishes a research programme, not a positive outcome. Long-standing T1D still requires restoration of cell mass. [29]

29 iTolerance and Kadimastem iTOL 102

IV based on confirmed available evidence • P1. The approach combines IsletRx islets with iTOL-100 for local immune tolerance. The pre-IND meeting announcement reports animal data. A meeting with the FDA is not permission to treat people or clinical evidence that immunosuppression is unnecessary. [16]

30 Other routes to immune tolerance

III–V, a heterogeneous class • P2. Low-dose IL-2, tolerogenic dendritic cells, antigenic DNA/RNA constructs, oral or nasal insulin, engineered microorganisms and local delivery of immune signals form

distinct research branches. They cannot be assigned one phase or class-wide efficacy. They are included for scientific screening; each product requires a separate current dossier.

8 Regeneration and protection of endogenous cells

31 Verapamil and cell stress

III for function preservation in T1D • P2. A randomised study in children and adolescents with recent diagnosis found partial preservation of secretion. This investigates protection of residual cells, not restoration of a destroyed pancreas in long-standing T1D. Cardiovascular effects require separate assessment. [30]

32 DYRK1A inhibitors and GLP 1 agonists

IV • pharmacological regeneration • P1. Harmine and exendin-4 increased the mass of human beta cells transplanted into mice. This is a strong preclinical finding, but human cells in a mouse are not a treated human. Clinical translation requires selectivity, safe proliferation control and protection from autoimmunity. [31]

33 Menin inhibition and BMF 219

III based on registered phase II • P2 with elevated uncertainty. BMF-219, also called icovamenib, is being investigated for effects on beta cell mass and function; the T1D identifier is NCT06152042. This review does not establish its current operational status or convincing clinical benefit in T1D. Type 2 diabetes findings cannot be transferred to autoimmune T1D. [32]

34 Converting other cells into beta cells

IV–V • regeneration and genetic engineering • P1/P2. Alpha, ductal, acinar and other cells are being considered as sources of new insulin-secreting cells. Principal barriers are fully glucose-responsive secretion, stable cell identity, factor delivery and immune protection. This branch is a research platform; the reviewed sources do not establish a ready clinical solution.

35 Epigenetics and microRNA

IV–V • molecular biology • P2. Gene-expression regulation may affect beta cell maturation, stress and secretion. For example, miR-375 has an experimentally demonstrated role in insulin secretion. Translation requires delivery to the right cells, avoidance of unwanted effects and evidence of durable benefit. [46]

36 Mesenchymal cells and extracellular vesicles

Early research approaches • P3. The main areas of study are immunomodulation and tissue support. These products must be distinguished from functional ESC/iPSC-derived islets: the term stem cells does not imply a shared mechanism. Each product needs standardised composition, a control group and demonstrated function. A universal therapeutic effect of the class is not established here.

37 Organoids and tissue engineering

IV–V • P1 as a research platform. Organoids, vascular and immune-cell co-culture, matrices and bioprinting help study the islet as a tissue system. Implantation requires scale, nutrient supply, reproducibility and safety. An organoid can be a useful screening model before it is suitable as a transplant product.

9 Other biological hypotheses

38 Antiviral therapy

III • P2. The DiViD programme investigated antiviral treatment near onset, with a published three-year follow-up. It tests a viral contribution in a subset of patients. It does not mean all T1D is persistent infection or that an antiviral drug can restore long-lost islets. Durability and identification of the appropriate subgroup matter for further development. [33]

39 Vaccines against possible viral triggers

Investigational prevention • P2/P3. Enterovirus vaccines, including those targeting coxsackie B viruses, aim to prevent a possible infectious trigger. This differs from GAD or proinsulin immunotherapy. Antiviral immunogenicity does not establish a reduction in T1D incidence. The objective is long-term prevention, not cell replacement in established disease.

40 BCG and immunometabolic remodelling

III, insufficient for an established T1D treatment • P3. Studies describe long-term glycaemic changes after BCG. These do not justify promises of insulin independence or automatic regeneration. Prioritisation requires independent replication, adequate sample sizes, transparent endpoints and assessment of background treatment. [34]

41 Microbiome and metabolites

From mechanistic research to small clinical trials • P3. Microbiota transplantation, dietary substrates and bacterial metabolites are studied in immune regulation. Association between microbiome composition and T1D does not prove causality. The centre should prioritise prespecified metabolic endpoints: a change in microbial composition alone is not therapeutic success.

42 Immune reset through autologous HSCT

III • a clinically investigated high-risk approach • P3. Publications report insulin independence after autologous haematopoietic stem cell transplantation with immune conditioning in recently diagnosed patients. This does not transplant new beta cells. Conditioning, infection and late complication risks are fundamental; onset findings do not transfer to long-standing T1D. [35]

43 Mixed chimerism and graft tolerance

IV–V for a broadly applicable T1D solution • P2. The aim is durable acceptance of donor tissue through changes to the immune system. Potential value must be weighed against conditioning and immune complications. Successful transplantation of another organ does not establish evidence for an islet programme.

44 Combination therapy

Readiness depends on the specific combination • P1. Cell preservation or replacement is combined with immune protection and engraftment support. Two promising components do not automatically create an effective regimen: interactions, toxicity and clinical value must be tested together. For the centre, this is more useful than seeking a single decisive molecule.

10 Biophysics and unconventional testable approaches

45 Electrical control of engineered cells

V • synthetic biology • P2/P3. Huang and colleagues demonstrated an interface using direct current to control gene expression, with engineered model cells supporting regulated insulin production. This is a defined physical signal, cellular switch and product—not an intervention on an abstract disease field. Durability, safety and human applicability remain to be established. [36]

46 Radiofrequency and magnetic control

V • nanobiotechnology • P3. Stanley and colleagues used radio-wave heating of magnetic nanoparticles to control insulin production and glucose in mice. The physical carrier, exposure parameters and biological target are defined. This does not show that external irradiation without an engineered cellular system eliminates T1D. [37]

47 Optogenetics and light switches

V • synthetic biology • P3. Light can control specifically engineered cells. Therapy requires delivery of light to its target, secretion control, prevention of overheating and immune protection. This is an exploratory engineering branch; an established clinical T1D solution was not identified.

48 Ultrasound and neuromodulation

V for a curative T1D application • P3. Neural regulation and physical interventions can affect secretion and metabolism. Better glycaemia in an insulin-resistance model does not demonstrate beta cell restoration in autoimmune diabetes. Relevant T1D models and independent mechanistic confirmation are required.

49 Photobiomodulation and low intensity lasers

V or VI depending on the claim • P3/P0. Lasers may have measurable tissue effects, but wound healing or changes in local inflammation do not establish reversal of insulin deficiency. A T1D treatment claim requires evidence of restored secretion and control of autoimmunity. Such clinical evidence was not established in the reviewed materials.

50 Wave genetics bioresonance and bioenergy

VI • P0. Claims about rewriting diabetes information, disease frequencies or organ restoration through a wave template were not supported here by reproducible clinical evidence of T1D treatment. Historical narratives, patents, professional titles and patient testimonials do not replace experiments.

A hypothesis remains open to testing if its proponent defines a measurable intervention, mechanism, expected effect and conditions that would falsify it. Controlled blinded experiments and independent replication must follow. Until then it remains outside the therapeutic portfolio, with no assigned implementation date or probability of cure.

11 Bioinformatics and validation methods

51 Bioinformatics and omics

M • P1. Single-cell transcriptomics, spatial methods, proteomics and immune-repertoire analysis help identify targets and disease subtypes. These are established scientific disciplines, not an exotic lower tier. A possible SP Science output is reproducible analysis of open data followed by experimental testing of predictions.

52 Islet biophysics

M • P1. Oxygen diffusion, glucose and insulin transport, membrane mechanics, beta cell electrical activity and blood supply directly affect implant quality. Measurable physical models are useful today and must be distinguished from unproven disease energy fields. [12, 38, 47]

53 AI and mathematical models

M • P1/P2. Applications include candidate discovery, protein design, cellular data analysis, response prediction and manufacturing optimisation. Docking, a high computational molecule score or a digital twin does not demonstrate therapeutic benefit. Model outputs require independent data validation and biological experiments.

54 Biomarkers and patient selection

M • P1. Autoantibodies, HLA, C-peptide, immune features, CGM and clinical history help define intervention targets. Antigen therapy particularly requires a prespecified population rather than a search for any apparent effect. An improved laboratory marker cannot replace an agreed clinical outcome.

Evidence needed for progression

Validation stage	Evidence required	Insufficient evidence
Cells	Function, viability, composition and specificity	An increased marker without function
Animals	Reproducible effect, control group and relevant model	One successful experiment
First human studies	Safety, objective signal and the complete regimen	One patient interview
Confirmatory clinical studies	Prespecified outcomes and adequate follow-up	A favourable subgroup selected after analysis
Implementation	Manufacturing, quality, logistics, regulatory decision and access	A patent, IND or announcement of future plans

Models are not interchangeable. Streptozotocin and alloxan primarily damage beta cells and do not reproduce the entire human autoimmune process. Normalised glycaemia in these models does not prove immune tolerance. Different questions need autoimmune or transplant models, human islets or immune-cell systems. This is a methodological assessment criterion, not an experimental procedure.

12 Adjacent research and the limits of cure claims

55 Automated insulin delivery and CGM

I for several systems • engineering and algorithms. Pumps, sensors and increasingly automated closed-loop systems deliver practical benefits today. They reduce hypoglycaemia and patient burden but retain dependence on external insulin. Better glucose control does not automatically establish preservation of beta cells; that is a separate endpoint. [40]

56 New insulins and delivery routes

I–V depending on the product. Ultrafast, inhaled, oral and glucose-responsive designs have different readiness levels. Approval of one delivery route cannot be extended to the entire class. Smart insulin may reduce continual decision-making but does not itself remove autoimmunity.

57 Adjunctive metabolic medicines

Readiness and indications vary. GLP-1 agonists, amylin approaches, glucagon-pathway interventions, SGLT agents and others are studied for control, reduced insulin dose or additional risks. Lower HbA1c alone does not qualify an approach as a cure. T1D-specific risks, including ketoacidosis with certain classes, are especially important.

58 Complications quality of life and access

Kidney, eye, vascular, neurological and mental-health research, education and treatment access are major fields in their own right. They are essential to a complete T1D picture, but a detailed trial catalogue is beyond this document's focus on functional restoration and modification of disease progression.

13 Priorities for the research centre

The sequence below sets priorities for scientific assessment. It is not a promise of a result or a change to the centre's funding framework.

Order	Scope	Purpose
1 Clinical benchmarks	Zimislecel; donor islets with tegoprubart; Cell Pouch	Understand the conditions under which insulin independence has been achieved
2 Protection without chronic systemic therapy	UP421 and SC451; CTX211; Encellin; iTOL-102	Address the main barrier to wider cell therapy
3 A feasible research niche	Local immune protection; viability and oxygen; model standardisation	Choose a testable task rather than reproduce an entire product
4 Preservation and regeneration	Early immune interventions; DYRK1A; cell stress; rational combinations	Explore an additional route, especially when residual reserve remains
5 Supporting analytics	Bioinformatics, biomarkers and data quality	Support target selection and valid interpretation
6 Longer-term hypotheses	Reproducible electrical, light-controlled and other cellular systems	A small exploratory portfolio with clear stopping criteria
Outside the therapeutic portfolio	Unverified wave and bioenergy claims	Verify the effect before deciding scientific value

Turning the map into project selection

For each direction, define one problem the centre can realistically investigate, such as islet viability, local immune modulation or analysis of treatment-response features. Compare reproducibility, access to materials, partner expertise and the ability to obtain an interpretable result.

Implementation requires separate confirmation of laboratory capacity, access to human islets and animal models, manufacturing capabilities where relevant, and partner agreements. A scientific priority does not itself establish the centre's ability to develop an entire product independently.

Project dossier for internal selection

Minimum fields: product and owner; objective; population and T1D duration; cell source; immune protection; delivery site; phase and status; trial identifier; participant count; follow-up; insulin independence; C-peptide; HbA1c and CGM; severe hypoglycaemia; serious adverse events; manufacturing; source and date; unresolved question.

Record separately whether evidence comes from a paper, registry record, conference abstract or company announcement. This prevents established clinical effects from being presented with the same confidence as plans to start a study.

14 Summary of leading programmes

The order broadly reflects readiness for the intended outcome. A goal is an intended property, not an achieved one. Not confirmed means a limit of this review, rather than proof an event did not occur.

Programme	Level	Evidence described	Main unresolved issue
Lantidra	I	Approval for selected adults [2]	Donors, immunosuppression risks and access
Teplizumab	I	Use in specified early indications [17–18]	Preservation, not universal regeneration
Zimislecel	II	10/12 insulin independent at one year [3]	Immunosuppression, scale and approval
Tegoprubart with islets	III	Company reports 12/12 insulin independent [5]	Confirmation in a broader programme
Sernova Cell Pouch	III	Clinical donor-islet programme [8–9]	Device contribution and transition to new cells
Sana UP421	III	Function through 14 months in one patient [10]	Full dose, reproducibility and scale
Encellin ENCRT	III	Early data from seven participants [11]	Clinical efficacy of the complete system
CTX211	III with incomplete current status	Registered early programme [13]	Current status and results
Autologous CiPSC islets	III	Published clinical case [15]	Immune protection and repeatable manufacturing
OPF-310	III with incomplete current status	Registered programme [14]	Full outcomes and durability
SR-02	Transition to clinical development	New plan anticipates dosing in 2027 [8]	First data from this particular programme
SC451	IV in August update	IND and clinical preparation [10]	First dosing not confirmed

Programme	Level	Evidence described	Main unresolved issue
iTOL-102	IV	Preclinical work and pre-IND interaction [16]	Translation of local protection to humans
DYRK1A with GLP-1	IV	Expansion of human cell mass in mice [31]	Safe regeneration during autoimmunity
Electrogenetic cells	V	Experimental control [36]	Clinical safety and durability
Diamyd DIAGNODE-3	X	Continuation criteria not met [27]	A new testable programme
Wave rewriting of disease	VI	Reliable therapeutic effect not established	Reproducible evidence first

The most substantiated central direction for SP Science is restoration of an adequate mass of functional cells with safe protection. Biophysics and bioinformatics can provide practical tools. Novelty alone is neither a benefit nor a reason for rejection; the quality of testing and reproducibility determine value.

Sources and status verification

Evidence cut-off: 20 September 2026. P: peer-reviewed publication; D: developer or research-centre statement; R: registry; A: regulator or government research institute. Links are active. Publication dates matter: a paper establishes that research was conducted, but not that recruitment remains open today.

- [1] A. NIDDK — Pancreatic Islet Transplantation
- [2] A. FDA — Lantidra prescribing information
- [3] D. Vertex — Zimislecel results at ADA
- [4] D. Vertex — Current type 1 diabetes pipeline
- [5] D. Eledon — Islet transplantation IND submission and 12 patient update
- [6] D. Piotr Witkowski — Tegoprubart study patient information
- [7] Conference abstract. Wojcik et al. — Tegoprubart for Beta Cell Replacement Therapy
- [8] D. Sernova — Merger agreement with Seraxis and updated development plans
- [9] D. Sernova — Pipeline and Cell Pouch results
- [10] D. Sana — UP421 14 month results and SC451 development update
- [11] D. Encellin — Phase 1 progress and seven participant update
- [12] D. Encellin — ENCRT membrane technology
- [13] R. NCT05565248 — CTX211
- [14] R. NCT06575426 — OPF-310 porcine islets
- [15] P. Wang et al. — Autologous chemically reprogrammed islets in a person with T1D
- [16] D. iTolerance and Kadimastem — iTOL-102 pre-IND meeting and animal data
- [17] A. EMA — Teizeild product information and assessment
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