Understanding Stem Cell Therapy for Type 1 Diabetes

A Patient Guide from Auragens

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Advancing Regenerative Medicine:
Treatments & Empathy Amplified by Science and Technology

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Welcome

Living with Type 1 diabetes means navigating a daily reality that most people never see — the carbohydrate calculations, the glucose checks, the insulin adjustments, the constant vigilance. It means carrying the knowledge that your own immune system turned against you, and that no matter how carefully you manage your condition, the underlying biology has not been addressed. It is a demanding, lifelong burden, and the emotional weight of it is real.

If you are reading this guide, you may be curious about whether regenerative medicine — and specifically, stem cell therapy using human umbilical cord-derived mesenchymal stromal cells — might offer something that current treatments cannot. You deserve honest, evidence-based answers to that question.

This guide was prepared by the Auragens medical and scientific team to give you exactly that: a clear, accurate, and compassionate overview of what the research currently shows about stem cell therapy for Type 1 diabetes. It explains the biology of the disease, how hUC-MSCs may interact with the immune system and the pancreas, what clinical trials have found, and what you can realistically expect if you choose to pursue a consultation.

We believe that informed patients make better decisions. We believe that hope and scientific honesty can coexist. And we believe that every person living with Type 1 diabetes deserves access to the most current knowledge available — presented in language that respects both your intelligence and your time.

This guide does not replace your physician, your endocrinologist, or the Auragens medical team. It is a starting point — a resource to bring to your next conversation.

1. Understanding Type 1 Diabetes and Current Treatment Limitations

What Is Type 1 Diabetes?

Type 1 diabetes (T1D) is a chronic autoimmune disease in which the body's own immune system mistakenly identifies and destroys the insulin-producing beta cells located in the islets of Langerhans within the pancreas. Insulin is the hormone that allows glucose — the primary fuel derived from the food we eat — to enter cells throughout the body. Without sufficient insulin, glucose accumulates in the bloodstream, producing the elevated blood sugar levels that define diabetes.

Unlike Type 2 diabetes, which is primarily driven by insulin resistance and metabolic dysfunction, Type 1 diabetes is fundamentally an immune disease. It can develop at any

age, though it most commonly presents in childhood, adolescence, or young adulthood. Approximately 1.6 million Americans live with Type 1 diabetes, and global prevalence continues to rise.

The Autoimmune Mechanism

At the core of T1D is a failure of immune self-tolerance. Under normal circumstances, the immune system is equipped with regulatory mechanisms that prevent it from attacking the body's own tissues. In T1D, this system breaks down. Autoreactive CD4+ helper T cells and CD8+ cytotoxic T cells are activated and directed against beta cell antigens, progressively destroying the cells responsible for insulin production [1].

A critical part of this breakdown involves regulatory T cells (Tregs) — specialized immune cells whose job is to suppress autoimmune responses and maintain tolerance. Research has demonstrated that in people with Type 1 diabetes, Treg populations are functionally impaired, meaning the immune system's natural "brakes" are weakened. This loss of regulatory control allows the autoreactive attack on beta cells to proceed largely unchecked [1, 2].

By the time a diagnosis is made, a significant proportion of beta cell mass has already been lost. However, many patients — particularly those diagnosed as adults or in the early weeks following diagnosis — retain some residual beta cell function. This residual function, measurable through a molecule called C-peptide (a byproduct of natural insulin production), is clinically meaningful. Even small amounts of endogenous insulin production are associated with better glycemic control, reduced hypoglycemia risk, and fewer long-term complications.

How the Disease Progresses

Type 1 diabetes is typically a progressive condition. Without intervention targeting the immune process, the autoimmune destruction of beta cells continues over months to years. In children and younger patients, progression may be more rapid. In adults, the process can be slower — a presentation sometimes called Latent Autoimmune Diabetes in Adults (LADA).

The clinical consequences of progressive beta cell loss include:

- Increasing insulin requirements over time
- Greater difficulty achieving stable blood glucose levels
- Higher risk of hypoglycemic episodes (dangerously low blood sugar)
- Increased long-term risk of complications affecting the eyes, kidneys, nerves, and cardiovascular system

Current Standard Treatments and Their Limitations

Insulin replacement therapy remains the cornerstone of T1D management. Delivered through injections or continuous infusion via an insulin pump, insulin is life-sustaining and has saved millions of lives since its discovery. Advances in continuous glucose monitoring (CGM), hybrid closed-loop systems, and ultra-rapid insulin formulations have meaningfully improved quality of life and glycemic outcomes for many patients.

However, insulin therapy has fundamental limitations that current technology cannot overcome [4]:

It does not address the underlying autoimmunity. Insulin replaces the hormone that beta cells can no longer produce, but it does nothing to stop the immune system from continuing to destroy those cells. The root cause of the disease remains active.

It does not restore natural insulin production. Even with optimal insulin therapy, the body's own finely tuned, moment-to-moment insulin secretion — responsive to meals, stress, exercise, and countless physiological signals — cannot be perfectly replicated by injected insulin.

Glycemic control remains imperfect. Despite significant technological advances, achieving and maintaining target blood glucose levels requires constant effort. Hypoglycemia and hyperglycemia remain common, and both carry health risks.

The burden is continuous. Managing Type 1 diabetes is a full-time responsibility that affects nearly every aspect of daily life — diet, exercise, sleep, travel, employment, and relationships.

Long-term complications persist. Even with good glycemic management, the cumulative risk of complications — diabetic retinopathy, nephropathy, neuropathy, and cardiovascular disease — remains elevated compared to the general population.

Immunotherapy approaches — including teplizumab, an anti-CD3 monoclonal antibody recently approved in the United States — have shown promise in delaying the onset of clinical T1D in at-risk individuals. However, these approaches target only one arm of the immune response and do not offer a comprehensive solution to immune dysregulation or beta cell restoration.

It is against this backdrop — of a disease well-managed but not fundamentally treated — that regenerative medicine and cellular therapy are being explored as potentially complementary approaches.

2. What Are hUC-MSCs from Wharton's Jelly?

Mesenchymal Stromal Cells: An Overview

Mesenchymal stromal cells (MSCs) — also referred to as mesenchymal stem cells — are a class of multipotent cells found in various tissues throughout the body, including bone marrow, adipose (fat) tissue, dental pulp, and the umbilical cord. They are not the same as embryonic stem cells or induced pluripotent stem cells. MSCs do not form embryos, and their collection raises no ethical concerns comparable to those associated with embryonic sources.

MSCs have attracted significant scientific and clinical interest not primarily because of their ability to differentiate into other cell types, but because of their powerful ability to communicate with and modulate the immune system, reduce inflammation, support tissue repair, and deliver protective signals to stressed or damaged cells — all through a sophisticated network of secreted molecules, proteins, and extracellular vesicles (including exosomes).

Wharton's Jelly: A Unique Source

The umbilical cord — the lifeline connecting a developing baby to the placenta — contains a gelatinous connective tissue called Wharton's Jelly. This tissue is exceptionally rich in MSCs. Human umbilical cord-derived mesenchymal stromal cells from Wharton's Jelly (hUC-MSCs) are considered among the most therapeutically promising MSC sources for several reasons:

Abundance and accessibility. The umbilical cord is routinely discarded after birth. Its collection requires no invasive procedure, no harm to the donor, and raises no ethical concerns. It represents a readily available source of high-quality cells.

Youth and potency. hUC-MSCs are neonatal cells — among the youngest adult-type stem cells available. They retain high proliferative capacity, robust paracrine signaling activity, and strong immunomodulatory properties compared to MSCs derived from older donors.

Low immunogenicity. hUC-MSCs express very low levels of the surface molecules (MHC class II antigens) that typically trigger immune rejection. This means they can generally be administered to recipients who are not genetically matched — an allogeneic (donor-to-patient) approach — without triggering significant immune rejection. This is a critical advantage for clinical scalability.

Potent immunomodulatory capacity. hUC-MSCs are capable of suppressing excessive immune activation, promoting regulatory immune cell populations, and rebalancing inflammatory signaling — properties of direct relevance to autoimmune conditions like Type 1 diabetes [11].

From Donation to Treatment: Quality and Safety at Every Step

At Auragens, hUC-MSCs are derived from umbilical cords donated by healthy, consenting mothers following full-term deliveries. The process includes:

Rigorous donor screening. All donors undergo comprehensive medical history review and infectious disease testing in accordance with international safety standards. Cords from donors who do not meet all safety criteria are excluded.

Sterile laboratory processing. Wharton's Jelly tissue is processed under strict sterile conditions in a controlled laboratory environment. MSCs are isolated, identified, and characterized to confirm their identity and functional properties.

Controlled expansion. Cells are expanded in culture to produce therapeutically relevant quantities while maintaining quality and potency standards.

Quality control and release testing. Before any cell preparation is released for clinical use, it undergoes rigorous testing for sterility, identity, viability, purity, and potency. Only preparations meeting all specifications are approved.

Cryopreservation. Cells are cryopreserved (frozen) in a controlled manner to maintain viability and functional integrity until the time of treatment. Upon thawing, preparations are assessed for quality before administration.

This end-to-end quality system ensures that every patient receives a safe, characterized, and consistent cell preparation.

🏆 Why Auragens: AABB Accreditation — The Gold Standard in Cellular Therapy

Auragens Stem Cell Center is proud to be AABB accredited — a critical distinction that sets us apart from unaccredited clinics.

What is AABB Accreditation?

AABB (Association for Advancement of Blood and Biotherapies) is the internationally recognized leader in setting quality and safety standards for cellular therapies. AABB is a deemed status organization by the FDA and AABB accreditation represents the gold standard in the field — the most rigorous, comprehensive certification available for facilities that process and administer cellular products.

What Does AABB Accreditation Mean for You?

When you choose an AABB-accredited center like Auragens, you can trust that we meet the highest international standards in:

- **Cell Processing Excellence:** Our laboratory protocols follow strict, validated procedures ensuring every cell product meets pharmaceutical-grade quality specifications.
- **Donor Screening & Safety:** Comprehensive screening protocols ensure all donor tissue is thoroughly tested and meets stringent safety criteria.
- **Laboratory Practices:** Our facilities, equipment, and processes undergo regular inspection and must meet exacting standards for sterility, environmental control, and quality assurance.
- **Patient Safety:** From cell arrival through administration and follow-up, every step is designed and monitored to protect your safety.
- **Staff Expertise:** Our team maintains specialized training and competency requirements specific to cellular therapy.
- **Traceability & Documentation:** Complete tracking and documentation of every cell product ensures full accountability.
- **Continuous Quality Improvement:** Regular audits, proficiency testing, and quality reviews.

Why This Matters

The field of stem cell therapy is rapidly growing, but not all clinics meet the same standards. Many facilities operate without independent accreditation or oversight. AABB accreditation means an external, U.S., FDA, expert organization has thoroughly inspected and validated our practices — giving you independent verification, proven quality systems, and genuine peace of mind. When you choose Auragens, you're choosing a center that has been independently verified to meet the most stringent international standards for cellular therapy quality and safety.

AABB accreditation is not easy to achieve or maintain — it requires significant investment in infrastructure, training, quality systems, and ongoing compliance. We've made this commitment because you deserve nothing less than the highest standard of care.

3. How hUC-MSCs May Help in Type 1 Diabetes

Type 1 diabetes is primarily a disease of immune dysregulation — the immune system attacking the pancreas. This makes it a particularly compelling target for hUC-MSC therapy, which exerts its most well-characterized effects precisely in the domain of immune modulation and inflammation. Below, we describe the key mechanisms by which hUC-MSCs may be relevant to T1D, drawing on the current scientific literature.

3.1 Immune Rebalancing and Regulatory T Cell Support

The central immune defect in Type 1 diabetes is a loss of self-tolerance driven by dysfunctional regulatory T cells (Tregs) and overactive autoreactive T cells. hUC-MSCs have been shown in multiple preclinical and clinical studies to promote the expansion and function of Tregs, helping to restore the immune balance that is disrupted in T1D [1, 2].

In animal models of autoimmune diabetes, umbilical cord MSC infusions were associated with increased populations of CD4+CD25+ regulatory T cells in the peripheral blood, alongside reductions in autoreactive T cell activity [9]. In human clinical studies, shifts in immune cell populations consistent with Treg induction and Th1/Th17 suppression have also been observed following UC-MSC administration [6, 7].

In simple terms: hUC-MSCs may help turn down the immune system's attack on the pancreas by strengthening the very regulatory cells that are weakened in T1D.

3.2 Anti-Inflammatory Effects

The destruction of beta cells in T1D is accompanied by a local inflammatory environment within the pancreatic islets — a process called insulitis. Inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α) and interferon-gamma (IFN-γ), play key roles in driving beta cell death.

hUC-MSCs secrete a range of anti-inflammatory mediators, including interleukin-10 (IL-10) and transforming growth factor-beta (TGF-β), which can suppress this inflammatory environment. Studies in animal models and human clinical observations have reported decreases in pro-inflammatory cytokines and increases in anti-inflammatory signals following MSC therapy [7, 9]. By reducing the inflammatory milieu around the islets, hUC-MSCs may help create conditions more favorable to beta cell survival.

3.3 Beta Cell Preservation Through Paracrine Signaling

hUC-MSCs do not appear to work primarily by directly differentiating into new beta cells. Instead, their effects are largely mediated through paracrine signaling — the secretion of a diverse array of growth factors, protective proteins, and signaling molecules that act on surrounding tissues. These secreted factors may promote beta cell survival, reduce apoptosis (programmed cell death) of remaining beta cells, and support the microenvironment of the pancreatic islets [11].

This paracrine protection of residual beta cells is clinically significant: even modest preservation of beta cell mass and function translates into measurable improvements in C-peptide levels, reduced insulin requirements, and improved glycemic stability.

3.4 Exosome and Extracellular Vesicle Signaling

A particularly exciting area of research involves the role of extracellular vesicles — including exosomes — secreted by MSCs. Exosomes are tiny membrane-enclosed particles that carry a cargo of proteins, lipids, and genetic material (including microRNAs) from the cells that produce them to target cells throughout the body.

MSC-derived exosomes have been shown to carry specific microRNA molecules — including miR-23a-3p — that can directly target and suppress inflammatory signaling

pathways involved in autoimmune insulitis [13]. In animal models, MSC-derived exosomes have also been shown to promote beta cell regeneration through pathways involving the transcription factor Pdx-1, a master regulator of beta cell development and function [14].

Research is also exploring whether exosome preparations — cell-free products derived from MSCs — could one day offer a practical alternative or complement to cell-based therapy, potentially with enhanced safety and scalability [15].

3.5 Modulation of the Immune Microenvironment

Beyond their direct effects on individual immune cell populations, hUC-MSCs appear to exert broader effects on the immune microenvironment — the complex ecosystem of cells, signals, and structural factors that govern immune activity in and around the pancreas and its draining lymph nodes.

Preclinical studies have shown that MSC therapy can alter immune cell composition in pancreatic lymph nodes, reducing the presence of autoreactive cells and modifying the local conditions that sustain the autoimmune attack [10]. This systemic and local microenvironmental remodeling may contribute to durable effects beyond the immediate post-treatment period.

3.6 Oxidative Stress Reduction

Beta cells are particularly vulnerable to oxidative stress — cellular damage caused by reactive oxygen species (free radicals). The inflammatory environment of T1D generates substantial oxidative stress within the islets, contributing to beta cell death. MSCs have been shown to secrete antioxidant factors and to reduce markers of oxidative stress in preclinical models, potentially offering an additional layer of protection to surviving beta cells [11].

4. Clinical Evidence and Research Findings

The State of the Evidence

Research into stem cell therapy for Type 1 diabetes has progressed meaningfully over the past decade, moving from animal models and small pilot studies toward randomized controlled trials. The evidence base, while still developing, includes several well-designed human studies that provide important signals about the potential of hUC-MSC therapy — particularly for preserving residual beta cell function in patients with recent-onset T1D.

It is important to understand the current evidence in its proper context. Stem cell therapy for T1D remains investigational. The studies conducted to date have generally involved small numbers of patients, short to medium follow-up periods, and varying patient

populations and treatment protocols. Larger, longer-term, multicenter randomized trials are needed before definitive conclusions about efficacy can be drawn. The findings summarized here represent the best available evidence, presented honestly and without exaggeration.

The ProTrans Trial: The Most Rigorous Evidence to Date

The most methodologically rigorous clinical evidence for Wharton's Jelly-derived MSC therapy in Type 1 diabetes comes from the ProTrans trial, a Phase I/II randomized, double-blind, placebo-controlled study conducted at Uppsala University Hospital in Sweden and published in *Diabetologia* in 2023 [5].

Study design. Fifteen adult patients with recent-onset Type 1 diabetes were randomized to receive either two intravenous infusions of allogeneic umbilical cord-derived MSCs (the ProTrans product) or placebo, administered three months apart. Patients were followed for 12 months.

Primary endpoint: C-peptide preservation. The primary measure of beta cell function was the C-peptide area under the curve (AUC) during a mixed-meal tolerance test (MMTT) — a standardized way of measuring the pancreas's ability to produce insulin in response to a meal.

Key findings: - In the placebo group, C-peptide AUC declined by approximately **47%** over 12 months — consistent with the natural history of progressive beta cell loss in T1D. - In the MSC-treated group, C-peptide AUC declined by only approximately **10%** — a statistically significant difference ($p < 0.05$). - Insulin requirements in the placebo group increased by a median of 10 units per day over 12 months. In the MSC-treated group, insulin requirements remained stable. - No serious adverse events related to treatment were reported.

These findings represent a compelling signal that Wharton's Jelly-derived MSC therapy can meaningfully slow the loss of endogenous insulin production in adults with recent-onset T1D. The randomized, double-blind, placebo-controlled design provides a level of rigor that distinguishes this study from much of the earlier literature.

The Lu et al. Clinical Study (2021)

A larger open, parallel-controlled clinical study published in *Stem Cell Research & Therapy* in 2021 enrolled 53 patients with Type 1 diabetes (27 receiving UC-MSC therapy, 26 controls) and followed them for 12 months [6].

Key findings: - 40.7% of MSC recipients met the primary endpoint of a ≥10% increase in fasting or postprandial C-peptide levels at 12 months, compared to the control group. - Three MSC recipients achieved periods of insulin independence lasting between 3 and 12

months — a remarkable outcome, though not universal. - Overall, MSC recipients in the adult-onset subgroup showed reductions in insulin requirements compared to controls. - In younger patients (juvenile-onset), differences between MSC and control groups were less pronounced, suggesting that age at onset and residual beta cell mass at the time of treatment may influence response. - No severe adverse events were reported over the 12-month follow-up period.

The Zhang et al. Comparative Study (2022)

A study published in *Stem Cell Research & Therapy* in 2022 compared the effects of umbilical cord-derived MSCs (UC-MSCs) with bone marrow-derived MSCs (BM-MSCs) in both Type 1 diabetes patients and animal models [7].

Key findings: - Both UC-MSC and BM-MSC therapy were associated with preserved beta cell function, reduced insulin requirements, and improved HbA1c in patients compared to conventional insulin therapy alone. - The therapeutic effects of UC-MSCs and BM-MSCs were broadly comparable, suggesting that the MSC class — rather than the specific tissue source — drives the key therapeutic effects. - Immunologic analysis showed increases in regulatory T cell populations and decreases in Th17 cells following MSC therapy, consistent with immune rebalancing.

Preclinical and Mechanistic Evidence

Complementing the human clinical data, a substantial body of preclinical research has explored the mechanisms and potential of hUC-MSC therapy in animal models of autoimmune diabetes. Key findings include:

- Umbilical cord MSC infusion in NOD (non-obese diabetic) mice — a well-established model of autoimmune diabetes — resulted in increased Treg populations, reduced TNF-α, increased IL-10, and improved glycemic control [9].
- Microencapsulated Wharton's Jelly hUCMSCs were shown to reverse hyperglycemia and induce Treg responses in NOD mice, and to modulate immune responses in human peripheral blood mononuclear cell co-cultures, suggesting a potential cell-protective strategy [8].
- MSC-derived exosomes carrying specific microRNA cargo (including miR-23a-3p) attenuated autoimmune insulitis in rodent models by targeting inflammatory signaling pathways [13].
- Stem cell-derived exosomes promoted beta cell regeneration through Pdx-1-dependent pathways in rat models of Type 1 diabetes [14].

Interpreting the Evidence: What It Means for You

The available evidence supports the following conclusions, stated honestly:

- hUC-MSC therapy has shown **meaningful signals of benefit** in preserving beta cell function and stabilizing insulin requirements in recent-onset Type 1 diabetes in human clinical studies.
- The most rigorous evidence (ProTrans RCT) demonstrates that Wharton's Jelly MSC therapy can significantly slow C-peptide decline compared to placebo over 12 months.
- Some patients in open studies have experienced periods of reduced insulin requirements or transient insulin independence, though this is not a universal outcome.
- Effects appear to be more pronounced in adult-onset patients and those treated early in the disease course, when more residual beta cell function remains.
- The evidence is promising but remains limited by small sample sizes, short follow-up periods, and heterogeneous study designs. Stem cell therapy for T1D is **investigational**, and larger confirmatory trials are ongoing.
- hUC-MSC therapy is not a cure for Type 1 diabetes. It does not eliminate the autoimmune process entirely or guarantee restoration of full insulin independence.

5. Safety Profile: What the Research Shows

Overall Safety Signal

Across the human clinical studies conducted to date, hUC-MSC therapy for Type 1 diabetes has demonstrated an **acceptable short-term safety profile**. No treatment-related serious adverse events have been reported in the major published trials, and the therapy has been generally well-tolerated.

This is an important finding — but it must be understood in context. The studies conducted to date have involved relatively small numbers of patients and follow-up periods of 12 months or less. Long-term safety data — beyond one to two years — remain limited. As with any investigational therapy, ongoing vigilance and systematic safety monitoring are essential.

Findings from Key Clinical Studies

ProTrans Trial (Carlsson et al., 2023): In this randomized, double-blind, placebo-controlled Phase I/II trial, no serious adverse events related to treatment were reported in the MSC-treated group over 12 months. Adverse events that did occur — such as upper respiratory infections — were mild, transient, and comparable in frequency between the treatment and placebo groups [5].

Lu et al. (2021): In this 12-month open parallel-controlled study of 27 UC-MSC recipients, no severe side effects were reported. The treatment was described as well-tolerated across multiple infusions [6].

These safety findings are consistent with the broader literature on allogeneic MSC therapy across multiple conditions, which generally reports a favorable short-term safety profile for intravenous MSC administration.

Considerations Specific to Type 1 Diabetes

Several safety considerations are particularly relevant to patients with Type 1 diabetes:

Hypoglycemia monitoring. Because hUC-MSC therapy may preserve or modestly restore endogenous insulin production, patients receiving this therapy require careful monitoring of blood glucose levels. If beta cell function improves, insulin dose adjustments may be needed to prevent hypoglycemia. This is a manageable consideration with appropriate medical oversight — and is, in fact, a positive indicator of therapeutic effect.

Immune monitoring. Because hUC-MSCs modulate immune function, patients are monitored for any unexpected immune-related effects. To date, no concerning immune adverse events have been reported in T1D trials.

Infusion-related reactions. As with any intravenous infusion, mild infusion-related reactions (such as temporary flushing or chills) are possible. These are typically transient and manageable.

What We Do Not Yet Know

Honest communication requires acknowledging the limits of the current safety data:

- **Long-term safety** beyond 12–24 months has not been systematically studied in T1D populations.
- The safety of **repeated or multiple courses** of hUC-MSC therapy over years has not been fully characterized.
- **Rare adverse events** may not be detectable in studies of the current scale; larger trials are needed to fully characterize the safety profile.

At Auragens, safety monitoring is integrated into every aspect of the treatment program. Patients are followed carefully before, during, and after treatment, and any concerns are addressed promptly and transparently.

6. What to Expect Next at Auragens

A Personalized, Evidence-Based Consultation

If you are considering hUC-MSC therapy for Type 1 diabetes, the first step at Auragens is a comprehensive, individualized consultation with our medical team. We do not offer one-

size-fits-all programs. Every patient's situation is different — their age at diagnosis, duration of disease, residual beta cell function, current glycemic control, overall health status, and personal goals all factor into the evaluation.

Your consultation will include:

Comprehensive medical history review. Our team will review your complete medical history, including your diabetes history, current insulin regimen, recent HbA1c values, continuous glucose monitoring data if available, and any relevant laboratory results.

Assessment of residual beta cell function. Where appropriate, we may recommend baseline C-peptide testing to assess your current level of endogenous insulin production. This information is important for understanding your potential response to therapy and for tracking outcomes over time.

Discussion of your goals. We will take the time to understand what matters most to you — whether that is reducing insulin requirements, improving glycemic stability, preserving the beta cell function you still have, or simply understanding your options. Your goals guide the conversation.

Transparent review of the evidence. We will walk you through what the research currently shows — including both the promising findings and the important limitations — so that you can make a genuinely informed decision. We do not overstate what the therapy can do.

Personalized treatment planning. If you and the Auragens team agree that hUC-MSC therapy is appropriate for your situation, a personalized treatment plan will be developed, including the proposed protocol, schedule, monitoring plan, and follow-up structure.

Realistic expectations. We will be clear about what can and cannot be predicted. Individual responses to hUC-MSC therapy vary. Some patients experience meaningful preservation of beta cell function and stabilization of insulin requirements; others may experience more modest or less clearly defined effects. We will not promise outcomes we cannot guarantee.

Logistics and Practical Considerations

Our team will guide you through all practical aspects of treatment, including travel arrangements, accommodation, pre-treatment preparation, and post-treatment follow-up. We are committed to making the process as clear and manageable as possible.

7. Next Steps

Is This the Right Time to Explore Consultation?

hUC-MSC therapy for Type 1 diabetes may be most relevant for individuals who:

- Have been recently diagnosed with Type 1 diabetes (within the past few months to a few years) and still have measurable residual beta cell function (detectable C-peptide)
- Are adults or older adolescents (the clinical evidence to date is strongest in adult populations)
- Are seeking to preserve the beta cell function they still have, rather than restore what has already been lost
- Have realistic expectations about the investigational nature of the therapy and are prepared to engage with the process thoughtfully
- Are under the care of an endocrinologist or diabetes specialist with whom they can coordinate their care

Questions to Discuss with Your Healthcare Team

Before pursuing any consultation, we encourage you to bring the following questions to your endocrinologist or diabetes care team:

1. Do I still have measurable C-peptide levels? What does my current beta cell function look like?
2. Am I in a stage of the disease where preserving residual beta cell function is a realistic goal?
3. Are there any aspects of my current health status that would make investigational stem cell therapy inadvisable?
4. How would we monitor my response to treatment, and how would we adjust my insulin regimen if my beta cell function improves?
5. What are your thoughts on me exploring a consultation with a specialized regenerative medicine center?

Contacting Auragens

To schedule a consultation or request additional information, please contact the Auragens team through our website at **auragens.com**. Our patient care coordinators are available to answer your questions, help you understand the consultation process, and guide you through the next steps at your own pace.

There is no obligation. A consultation is an opportunity to gather information and have an honest conversation — nothing more, and nothing less

A Message of Hope

Living with Type 1 diabetes requires a kind of courage that is quiet and daily — the courage to keep managing, keep adjusting, keep going, even when the disease is demanding and the future feels uncertain. If you have come this far in reading this guide, you are already doing something important: you are seeking knowledge, and you are taking your health seriously.

The science of regenerative medicine is advancing. A decade ago, the idea of using stem cells to modulate the immune system and preserve beta cell function in Type 1 diabetes was largely theoretical. Today, we have randomized clinical trial data showing that Wharton's Jelly-derived MSC therapy can meaningfully slow the loss of the body's own insulin production. We have mechanistic research explaining how these cells interact with the immune system. We have safety data showing that the therapy is well-tolerated in the short term. The field is moving forward.

We want to be honest with you: stem cell therapy is not a cure for Type 1 diabetes. It does not reverse established beta cell loss. It does not eliminate the need for insulin in most patients. The evidence, while promising, is still developing. Larger, longer trials are needed. Individual responses vary.

But hope grounded in evidence is real hope. And the possibility that a carefully administered, rigorously quality-controlled cellular therapy might help preserve what your pancreas can still do — might slow the progression, stabilize your management, and improve your quality of life — is a possibility worth exploring with open eyes.

At Auragens, we are here to support that exploration with honesty, expertise, and genuine care for your wellbeing. Whatever you decide, we hope this guide has given you something valuable: a clearer picture of where the science stands, and the confidence to ask better questions.

The Auragens Patient Care Team

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