



Eledon
Pharmaceuticals

Corporate Overview

March 2026



Forward-Looking Statements

This presentation contains forward-looking statements that involves substantial risks and uncertainties. Any statements about the company's future expectations, plans and prospects, including statements about its strategy, future operations, development of its product candidates, and other statements containing the words "believes," "anticipates," "plans," "expects," "estimates," "intends," "predicts," "projects," "targets," "could," "may," and similar expressions, constitute forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, although not all forward-looking statements include such identifying words. Forward-looking statements include, but are not limited to statements regarding: expectations regarding the timing for the commencement and completion of product development or clinical trials; the rate and degree of market acceptance and clinical utility of the company's products; the company's commercialization, marketing and manufacturing capabilities and strategy; the company's intellectual property position and strategy; the company's ability to identify additional products or product candidates with significant commercial potential; the company's estimates regarding expenses, future revenue, capital requirements and needs for additional financing; developments relating to the company's competitors and industry; and the impact of government laws and regulations.

Actual results may differ materially from those indicated by such forward-looking statements as a result of various important factors, including: the ability to develop commercially viable product formulations; the sufficiency of the company's cash resources; the ability to obtain necessary regulatory and ethics approvals to commence additional clinical trials; whether data from early clinical trials will be indicative of the data that will be obtained from future clinical trials; whether the results of clinical trials will warrant submission for regulatory approval of any investigational product; whether any such submission will receive approval from the United States Food and Drug Administration or equivalent foreign regulatory agencies and, if we are able to obtain such approval for an investigational product, whether it will be successfully distributed and marketed. These risks and uncertainties, as well as other risks and uncertainties that could cause the company's actual results to differ significantly from the forward-looking statements contained herein, are discussed in our annual report on Form 10-K for the year ended December 31, 2024, and other filings with the SEC which can be found at www.sec.gov. Any forward-looking statements contained in this presentation speak only as of the date hereof and not of any future date, and the company expressly disclaims any intent to update any forward-looking statements, whether as a result of new information, future events or otherwise.



Eledon Company Highlights



Optimized & Differentiated Lead Asset: Tegoprubart

- **Novel anti-CD40L antibody with significant advantages vs. approved immunosuppressants to protect transplanted organs and cell**
 - 150+ subjects of safety data, including **100+ patients with transplant efficacy data** in kidney, islet cell, xeno-kidney, and xeno-heart transplantation
 - Data supports potentially **unprecedented safety profile** vs. currently available medicines
 - In kidney transplantation, among **highest levels of kidney function (eGFR) ever reported**
- In pancreatic islet transplantation, 6 of 6 patients with Type 1 Diabetes transplanted to date functionally cured of their T1D



Strong Financial Profile

- **\$93.4M in cash**, cash equivalents and short-term investments as of September 30, 2025
- **Completed \$57.5M financing in November 2025**
- Forecasted **sufficient to fund operations into 2Q 2027**



Expected 12 Month Milestones

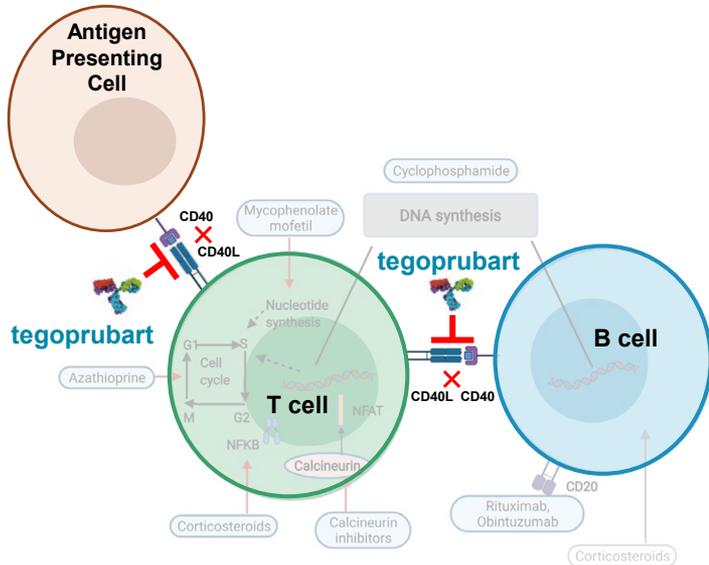
- i. Agreement with FDA on Phase 3 design / path to market for kidney transplantation, islet cell transplantation & xenotransplantation
- ii. Report long-term data in kidney transplantation
- iii. Report 12 patients of data in investigator sponsored Phase 2 trial in islet cell transplantation for Type 1 diabetes
- iv. Launch of multiple new investigator sponsored studies in liver transplantation, islet cell transplantation, xenotransplantation, and tolerance induction

Tegoprubart is a Pipeline in a Product Opportunity

INDICATIONS	DEVELOPMENT STAGE				NOTES
	PRE-CLINICAL	Early Human Trials/ PHASE 1	PHASE 2	PHASE 3	
ALLOTRANSPLANTATION					
Kidney					<ul style="list-style-type: none"> Phase 2 BESTOW completed. Phase 1b & Long-Term Extension trials ongoing
Islet Cell					<ul style="list-style-type: none"> U. Chicago investigator sponsored trial Received U.S. FDA Orphan Drug Designation
Islet Cell in Patients with Impaired Renal Function					<ul style="list-style-type: none"> Multi-site investigator sponsored trial
Kidney Tolerance					<ul style="list-style-type: none"> Mass. General Hospital investigator sponsored trial
Liver					<ul style="list-style-type: none"> IND-ready
XENOTRANSPLANTATION					
Kidney					<ul style="list-style-type: none"> eGenesis sponsored Phase 1/2/3 study
Adult Heart					<ul style="list-style-type: none"> Performed under U.S. FDA Expanded Access Protocol (EAP)
Pediatric Heart					
Amyotrophic Lateral Sclerosis (ALS)					<ul style="list-style-type: none"> Seeking non-equity dilutive financing to advance program to Phase 3

Mechanism Overview of CD40L Inflammatory Signaling

CD40/CD40L Pathway and Tegoprubart Site of Action



- Interaction of CD40 with CD40L on immune cells **mediates activation of the co-stimulatory immune pathway**, controlling "cross talk" between the adaptive and innate immune systems
- Maximal activation of inflammatory system is a 3-step process requiring co-stimulatory signaling
 - **Step 1:** Major histocompatibility complexes (MHC) and CD3/TCR engagement
 - **Step 2:** CD40 and CD40L binding resulting in cell division and clonal expansion
 - **Step 3:** Pro-inflammatory response by polarized T cells expressing inflammatory chemokines and cytokines
- **Blocking CD40L shifts polarization away from pro-inflammatory signaling** to T cell anergy, apoptosis, and polarization **to a Treg environment**
 - Blocking CD40L thus **does not generally result in lymphopenia** often seen with immunosuppressive agents

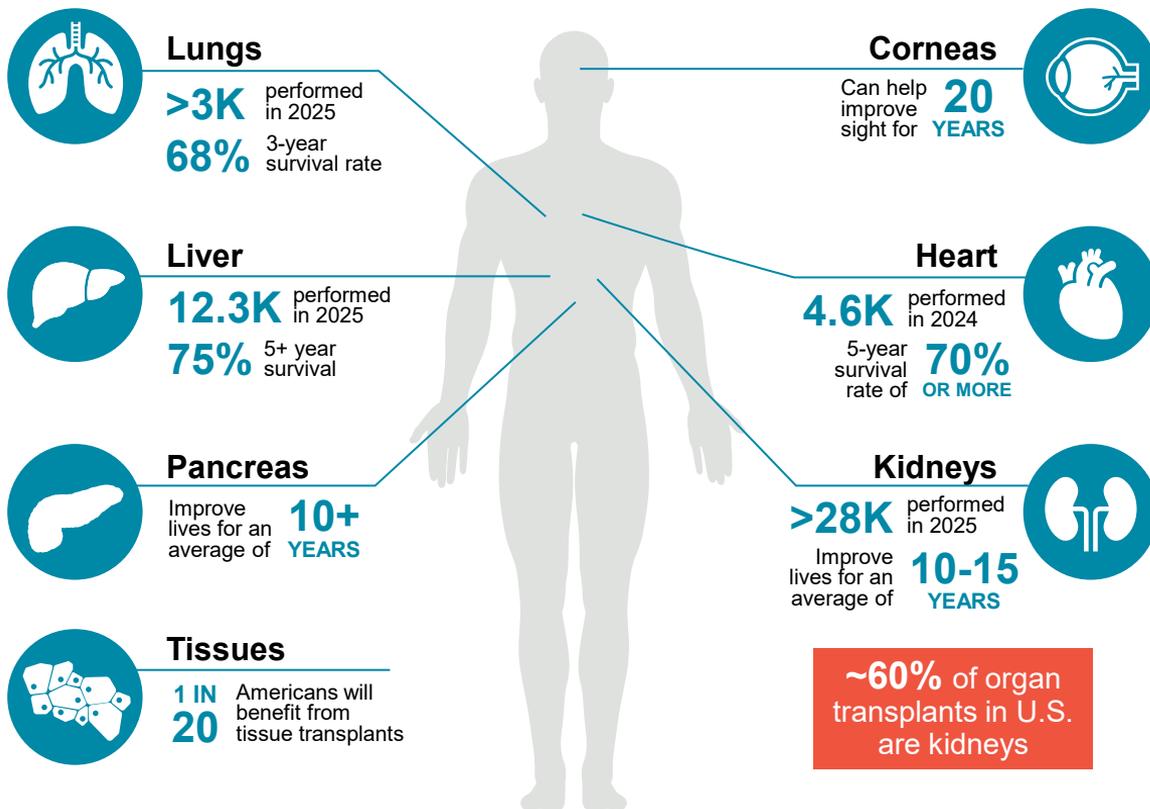
Tegoprubart: Potential Best-in-Class Anti-CD40/CD40L Antibody

Targeting CD40 Ligand vs. CD40 Receptor	
CD40L and CD40	CD40L only
<p>Targeting both anti-CD40L and anti-CD40 inhibits B cell polarization and class switching, as well as inhibits the pro-inflammatory polarization of CD4⁺ Helper T cells</p>	<p>✓ Blocking anti-CD40L also inhibits CD11 costimulatory receptors on antigen presenting cells, thus blocking the pro-inflammatory polarization of CD8⁺ Cytotoxic T cells</p>
	<p>✓ Blocking CD40L also polarizes CD4⁺ lymphocytes to FoxP3⁺ Regulatory T cells (Tregs), thus creating a potentially more tolerogenic environment</p>
	<p>✓ CD40L is more selectively expressed, providing the potential for additional safety and PK/PD/dosing advantages</p>

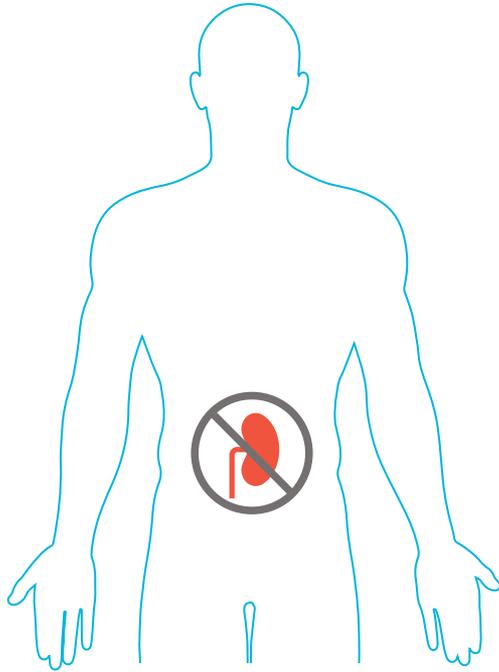
IgG1 vs. fusion protein or pegylated FAB
<p>✓ Up to over 2x times longer half-life</p>
<p>✓ Manufacturing advantages</p>
<p>✓ Less anti-drug antibodies</p>

Kidney Transplantation: Overview

Over 49,000 Organs Were Transplanted in the United States in 2025



Life-long Maintenance Immunosuppression is Necessary to Protect Transplanted Organs



Without immunosuppression, the **host** sees **donor** kidney as “foreign” and attacks (i.e., rejects) it

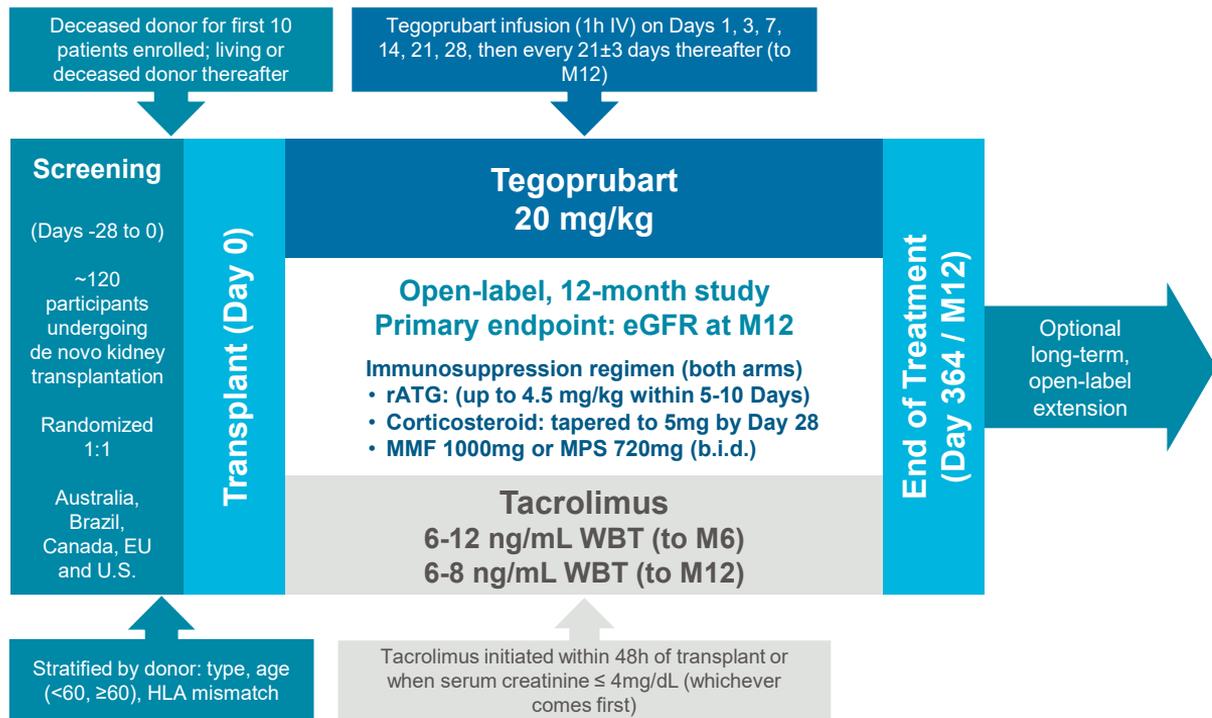
Induction immunosuppression (e.g., ATG) **is given at the time of transplant** to rapidly suppress the graft recipient’s immune system

Chronic, maintenance immunosuppression must then be taken for life or the organ will be rejected even years after the transplant

Kidney Transplantation: Phase 2 BESTOW

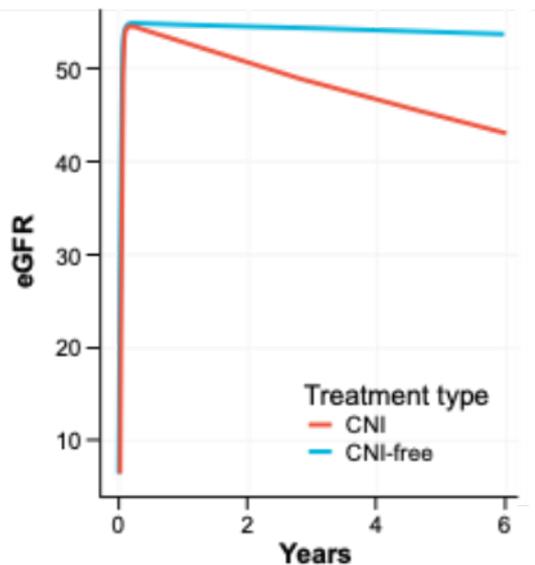
BESTOW Trial Overview

- **BESTOW** is the **first-ever** clinical trial comparing head-to-head tacrolimus to CD40L antibody (tegoprubart) in de novo kidney transplantation recipients
- A **prospective**, multicenter, **active-controlled, randomized, open-label** Phase 2 trial
- **Aim:** to determine the efficacy and safety of tegoprubart as the **core immunosuppressant regimen** in de novo kidney transplant recipients



BESTOW Key Efficacy Endpoints

Primary Endpoint of eGFR as a Proxy of Potential Long-Term Graft Function



Historical Mean eGFR of ~53 mL/min/1.73m² After 12 Months Using CNIs

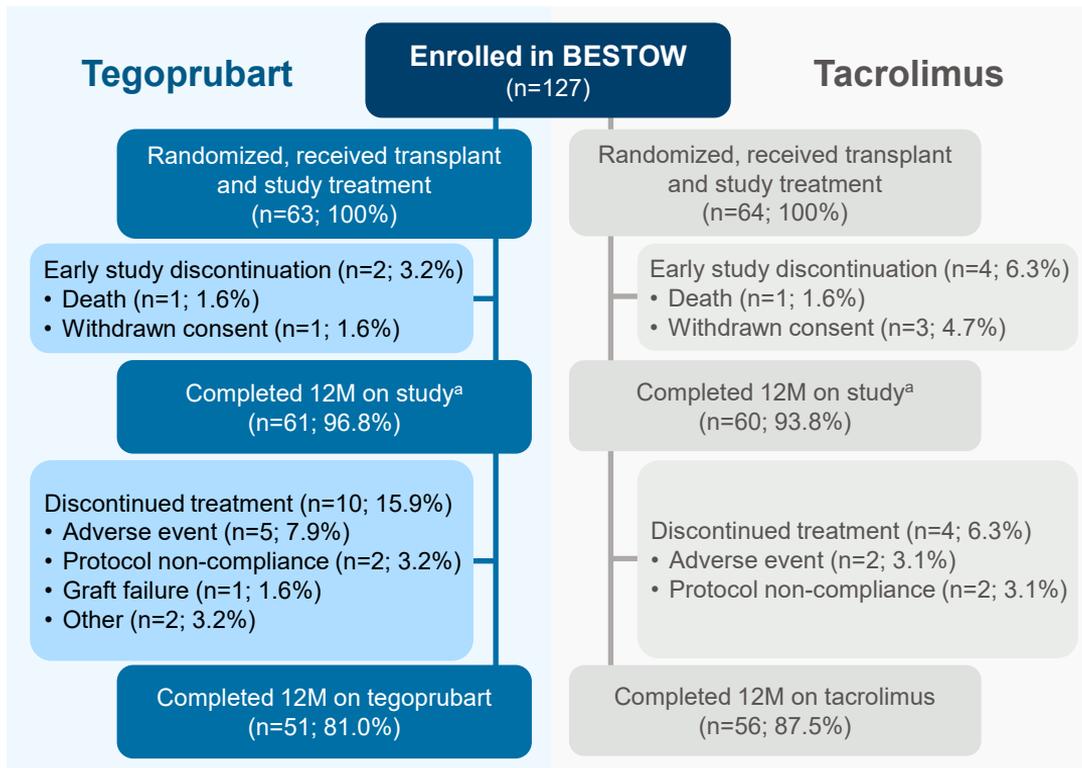
Secondary Endpoints Include Historical Approvable Endpoint of Non-Inferiority

- Since the 1990's the primary US FDA approvable endpoint has been non-inferiority based on a composite of efficacy failure using:
 - Biopsy Proven Acute Rejection
 - Graft Loss
 - Death
 - Loss to follow-up
- All transplant immunosuppressants are approved for “prophylaxis of organ rejection”
- Acute rejection is generally treatable and not predictive of long-term graft survival

BESTOW Conclusions

1. Observed composite endpoint (death, graft loss, BPAR, LTFU) failure rate in BESTOW was within the range reported in previous late-phase trials of approved immunosuppressive agents and demonstrated non-inferiority for tegoprubart vs. tacrolimus, using a 20% non-inferiority margin
2. Tegoprubart demonstrated excellent graft function. Kidney graft function, as assessed by eGFR, stabilized after the first month and remained higher on tegoprubart across all timepoints, in the range of ~69 mL/min/1.73 m² through month 12, vs. ~66 mL/min/1.73 m² on tacrolimus
3. Tegoprubart demonstrated a favorable safety profile with significant reductions observed in prominent side effects associated with tacrolimus including new onset diabetes, tremor, hypertension, and delayed graft function
4. Results support advancement into Phase 3 and position tegoprubart as the potential next-generation cornerstone of kidney transplant immunosuppression

Participant Flow



Donor Characteristics

Mean (SD), unless stated otherwise	TEGO N = 63	TAC N = 64
Donor age, years	43.5 (15.3)	43.0 (14.64)
Donor type		
Living, n (%)	18 (28.6)	17 (26.6)
Deceased, n (%)	45 (71.4)	47 (73.4)
KDPI score	44.0 (24.3)	35.2 (25.5)
KDPI <35, n (%)	17 (37.8)	27 (57.4)
KDPI ≥35, n (%)	27 (60.0)	20 (42.6)
Cold ischemia time, (h)	13.4 (8.5)	12.6 (8.1)
HLA mismatches, n (%)		
≤3	29 (46.0)	20 (31.3)
>3	34 (54.0)	44 (68.8)
CMV risk, n (%)		
High	16 (25.4)	13 (20.3)
Intermediate	33 (52.4)	43 (67.2)
Low	13 (20.6)	8 (12.5)

Recipient Characteristics

Mean (SD), unless stated otherwise	TEGO N = 63	TAC N = 64
Recipient age, years	50.1 (14.0)	48.3 (12.8)
Recipient sex, n (%)		
Male	42 (66.7)	44 (68.8)
Female	21 (33.3)	20 (31.3)
ESRD, n (%)^a		
Diabetes	16 (25.4)	20 (31.3)
Hypertension	41 (65.1)	32 (50.0)
Glomerulonephritis	24 (38.1)	13 (20.3)
Dialysis		
Yes at screening, n (%)	46 (73.0)	54 (84.4)
Duration in months	36.8 (33.4)	45.0 (41.5)
cPRA score, n (%)		
0%	44 (69.8)	43 (67.2)
>0%, ≤20%	7 (11.1)	8 (12.5)
>20%, ≤80%	11 (17.5)	11 (17.2)
>80%	1 (1.6)	1 (1.6)

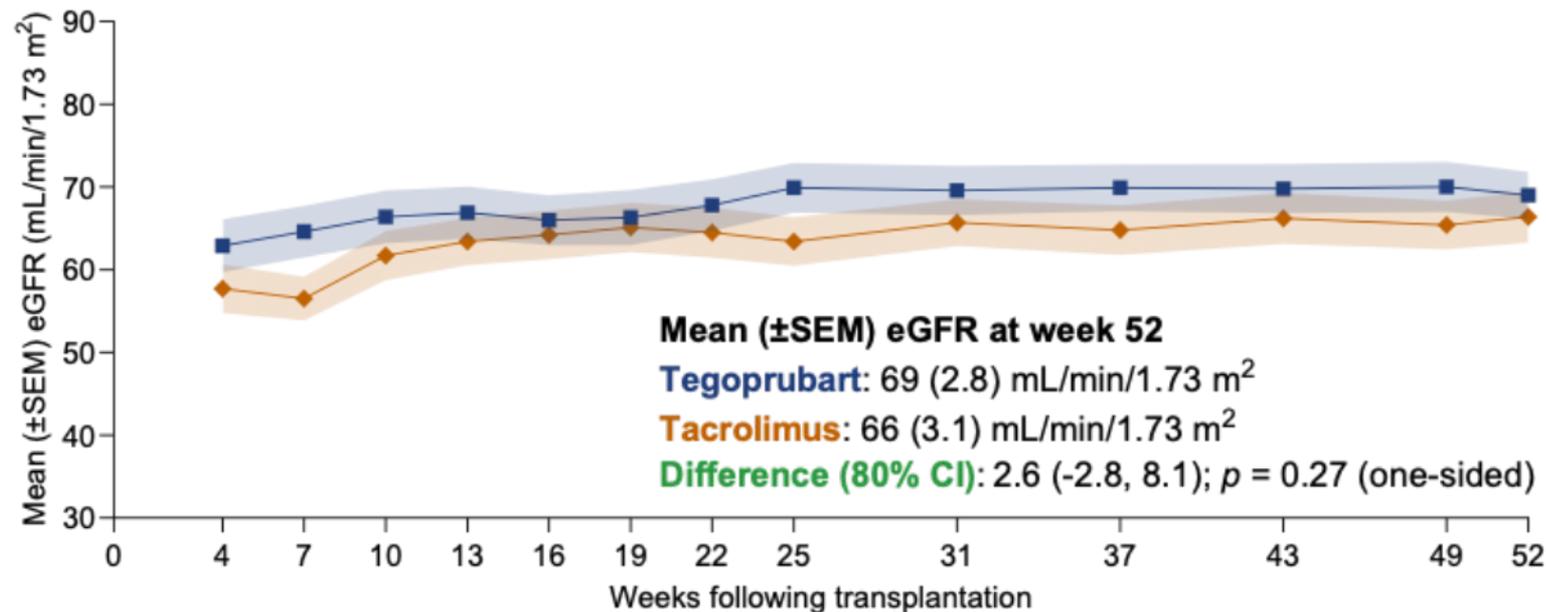
Additional Immunosuppression

Mean (SD), unless stated otherwise	TEGO N = 63	TAC N = 64
Days on treatment	324.8 (98.5)	331.8 (91.1)
Total mg/kg rATG at induction, median (min, max)	4.3 (2.9, 8.7)	4.2 (2.0, 6.0)
Percentage of days on-treatment with full dose MPA	59.4 (35.3)	54.3 (37.22)
Days to first MPA reduction below full dose	92.2 (72.49)	91.9 (86.9)
Corticosteroids		
Total induction bolus, mg	758.3 (313.8)	848.9 (320.1)
Days to stable 5mg dose	23.1 (13.5)	31.5 (41.6)
Corticosteroid discontinuation, n (%)	3 (4.8)	5 (7.8)

^aparticipants can be counted in more than one category.

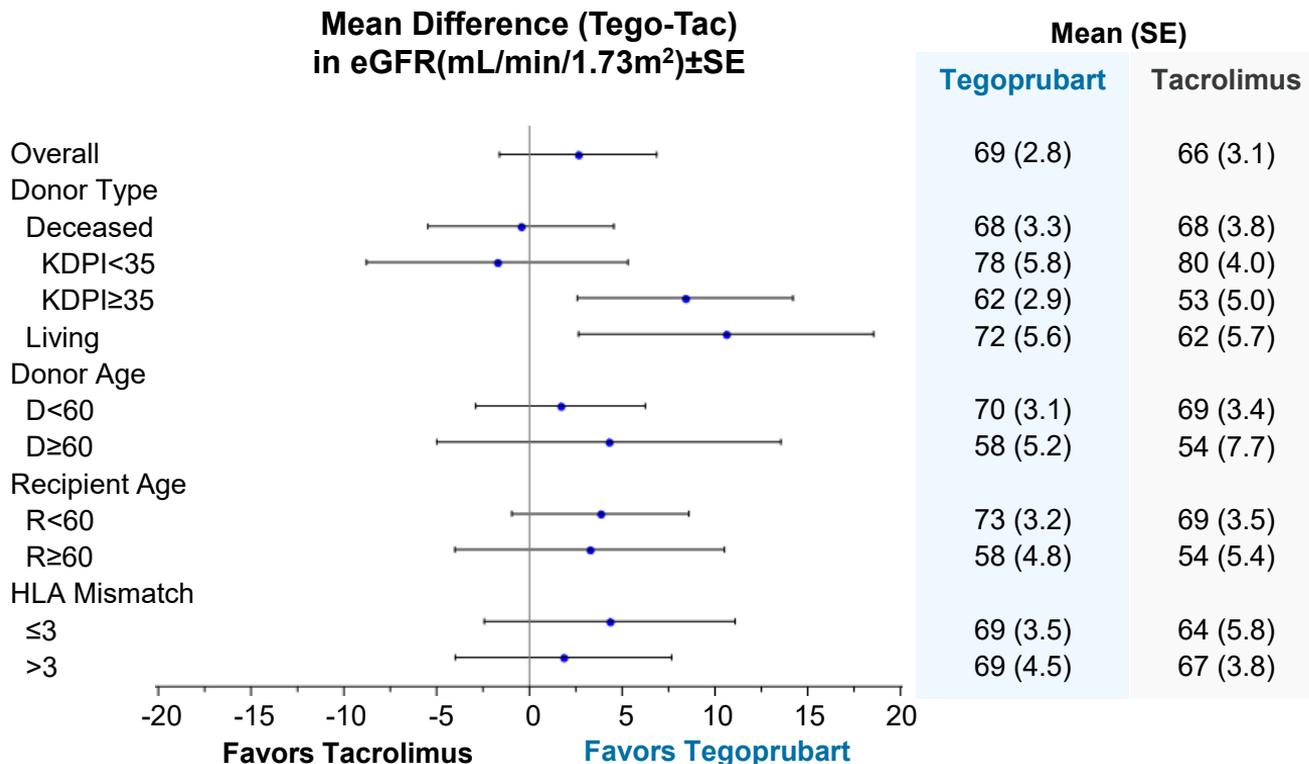
cPRA, calculated panel reactive antibody; CMV, cytomegalovirus; ESRD, end stage renal disease; h, hours; HLA, human leukocyte antigen; KDPI, kidney donor profile index; MPA, mycophenolic acid; rATG, rabbit anti-thymocyte globulin; SD, standard deviation.

Mean eGFR Over 52 Weeks in Patients Who Completed Treatment

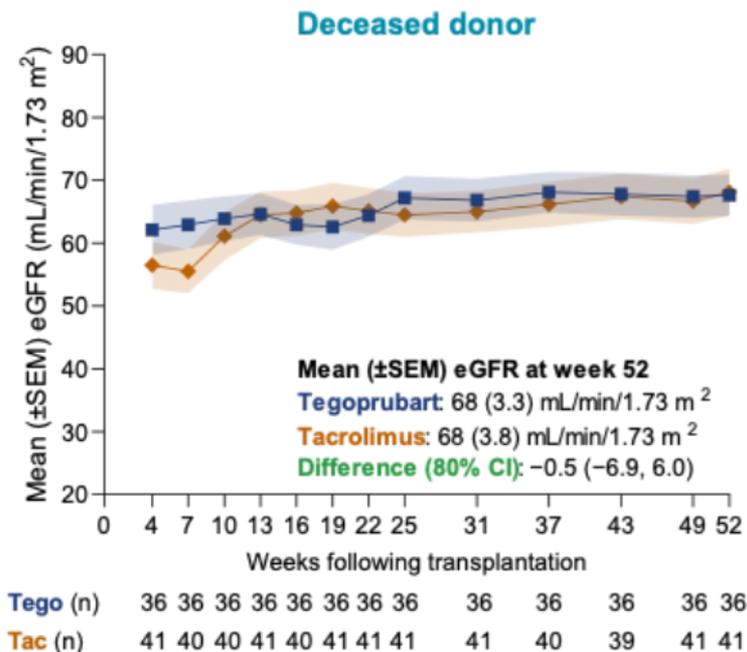
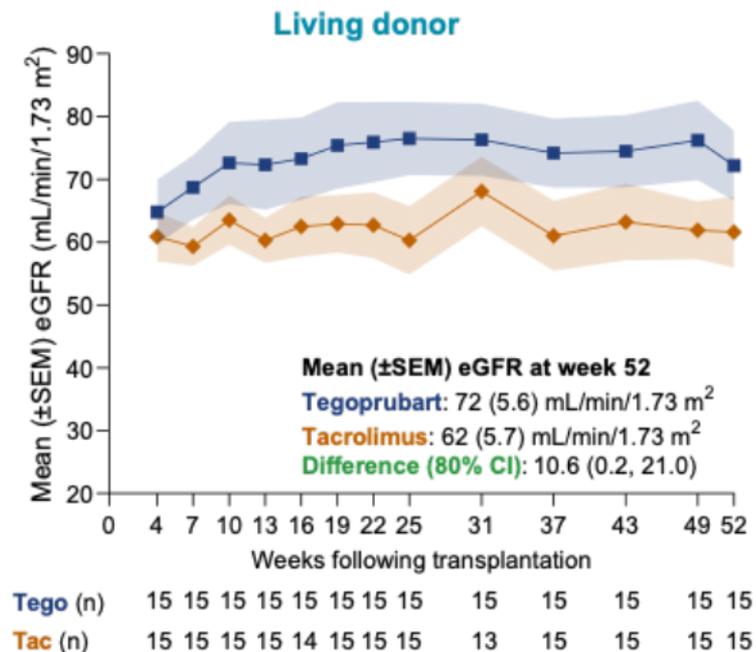


Tego (n)	51	51	51	51	51	51	51	51	51	51	51	51	51
Tac (n)	56	55	55	56	54	56	56	56	54	54	54	54	56

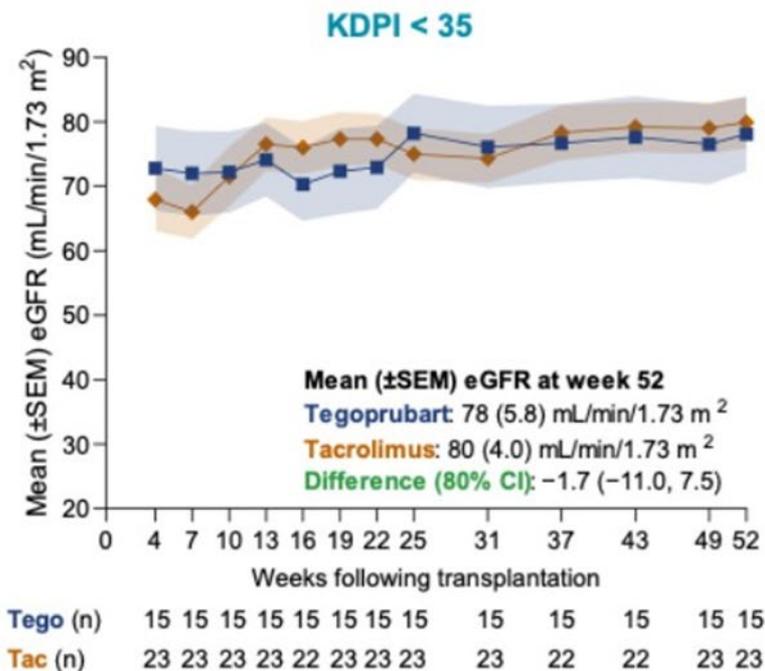
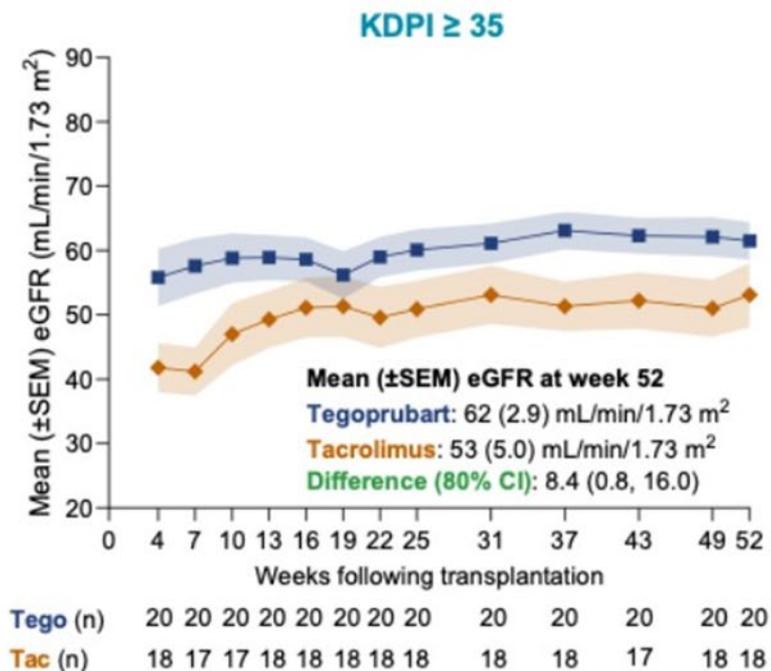
Subgroup Analysis of eGFR at 12 Month in Patients Who Completed Treatment



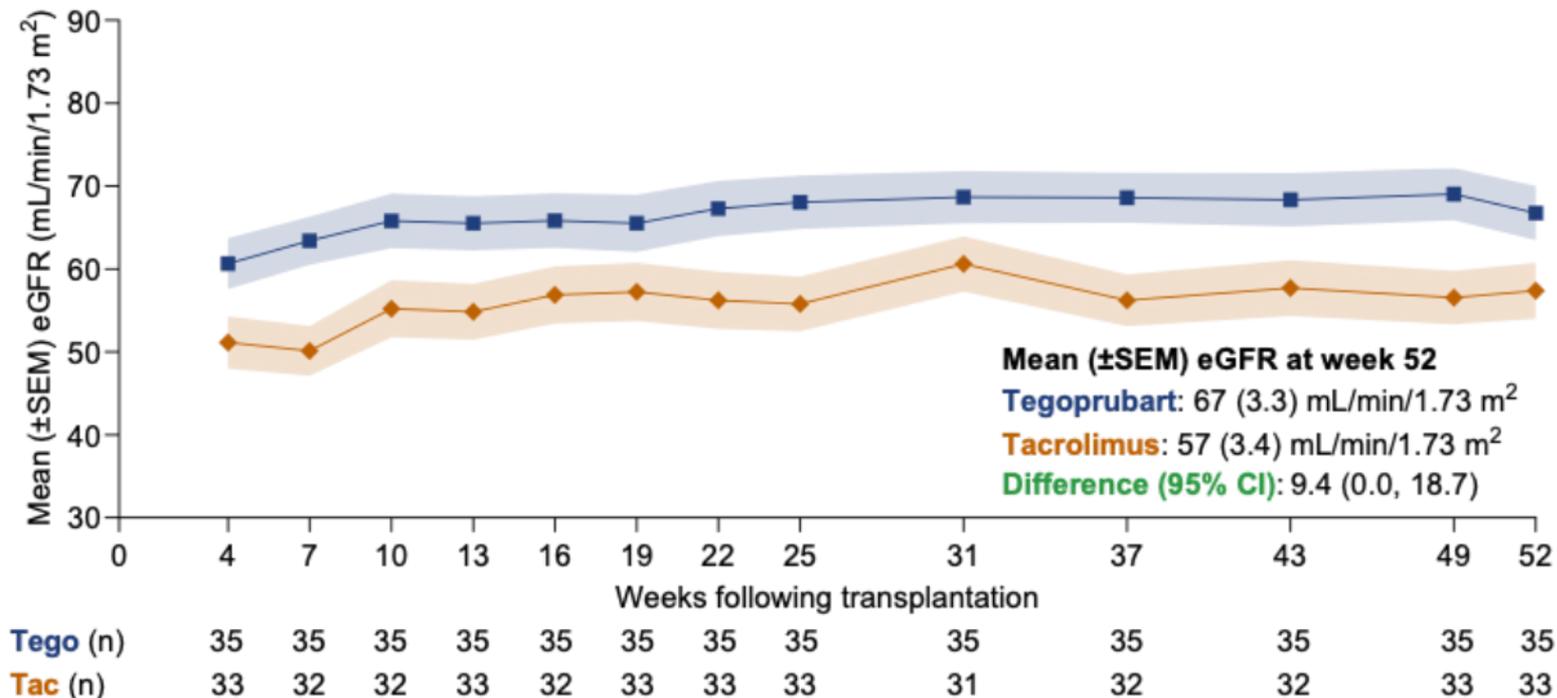
Mean eGFR by Donor Type in Patients Who Completed Treatment



Mean eGFR by KDPI Score in Deceased Donor Recipients Who Completed Treatment



Mean eGFR at 12-months for Subjects Who Received a Living Donor Kidney or a Deceased Donor Kidney With KDPI ≥ 35



Composite Biomarker Panel (iBox)

- **iBox is a predictive model of 5-year kidney graft survival** developed to estimate the risk of **long-term graft loss following kidney transplantation** using kidney function and recipient immunological response, either with a 12-month biopsy (i.e., ‘full iBox’) or without histopathologic assessment (i.e., ‘abbreviated iBox’).
- **Abbreviated iBox** (without biopsy) includes:
 - eGFR
 - anti-HLA DSA
 - proteinuria
 - time since transplantation
- iBox is superior to BPAR as a surrogate for long-term graft loss and excludes BPAR, whose effectiveness as a predictor of long-term graft survival is close to random chance.

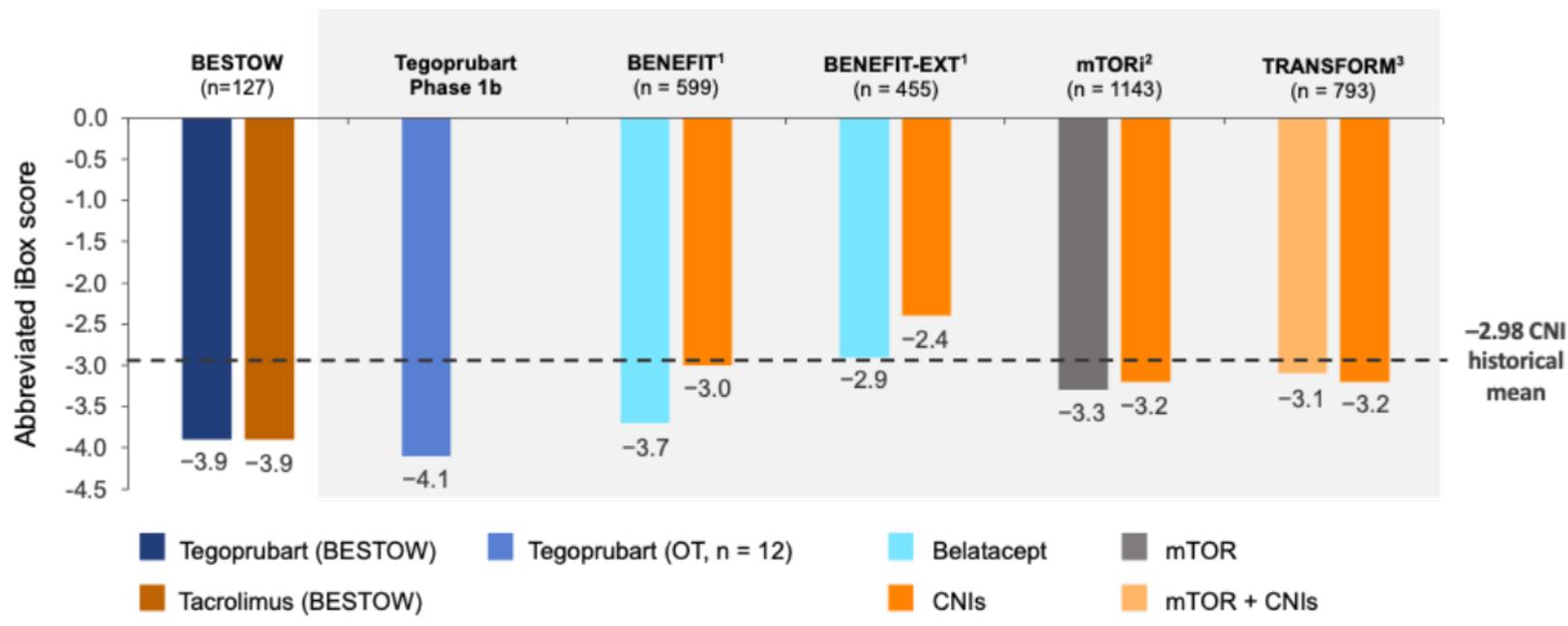
C-statistics (SE) at 1-year post transplantation

	Full iBox	Abbreviated iBox	BPAR
Combined validation (n = 1,534)	0.81 (0.03)	0.80 (0.03)	0.57 (0.03)

C-statistics ≥ 0.80 indicates a strong predictive model

A difference in abbreviated iBox score of -0.40 at 12 months is considered a minimum clinically important difference and is predictive of a 4–5% difference in 5-year graft survival

Comparative Abbreviated iBox Scores at 12 Months



CNI, calcineurin inhibitor; ITT, intent to treat; mTOR, mammalian target of rapamycin; mTOR, mTORi, mammalian target of rapamycin inhibitor; OT, on treatment.

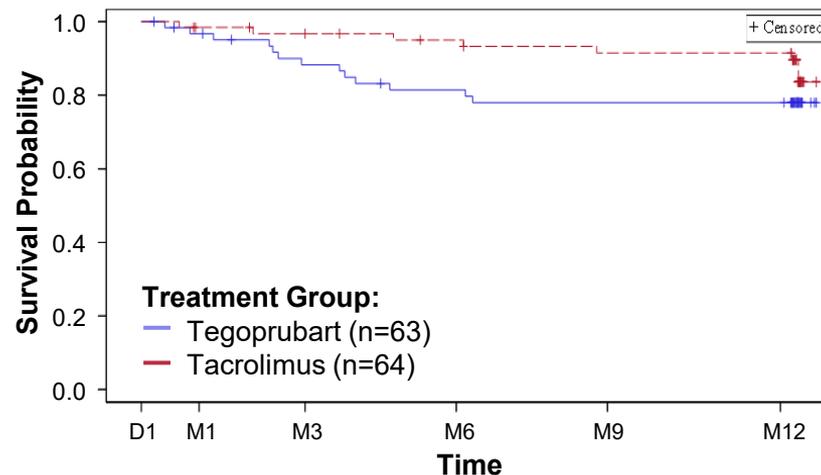
1. Klein A et al. Am J Transplant 2023;23:1496–506. 2. European Medicines Agency (EMA) Scientific Opinion-Qualification. Briefing Dossier.

https://www.ema.europa.eu/en/documents/scientific-guideline/qualification-opinion-applicant-revised-briefing-book-ibox-scoring-system-composite-biomarker-panel_en.pdf (Accessed 24 Jul 2025) 3. Aubert O et al. BMJ Open 2021;11:e052138

BPAR Based on All Biopsies Through 12 Months

Acute Parameters	Tegoprubart 20 mg/kg (N=63) n (%)	Tacrolimus (N=64) n (%)
Total biopsies	58 (92.1)	51 (79.7)
Acute Rejection	13 (20.6)	9 (14.1)
Acute AMR	2 (3.2)	1 (1.6)
Acute TCMR	13 (20.6)	8 (12.5)
Acute TCMR		
IA	2 (3.2)	5 (7.8)
IB	5 (7.9)	2 (3.1)
IIA	4 (6.3)	1 (1.6)
IIB	2 (3.2)	0
III	0	0

BPAR-Free Time on-Treatment

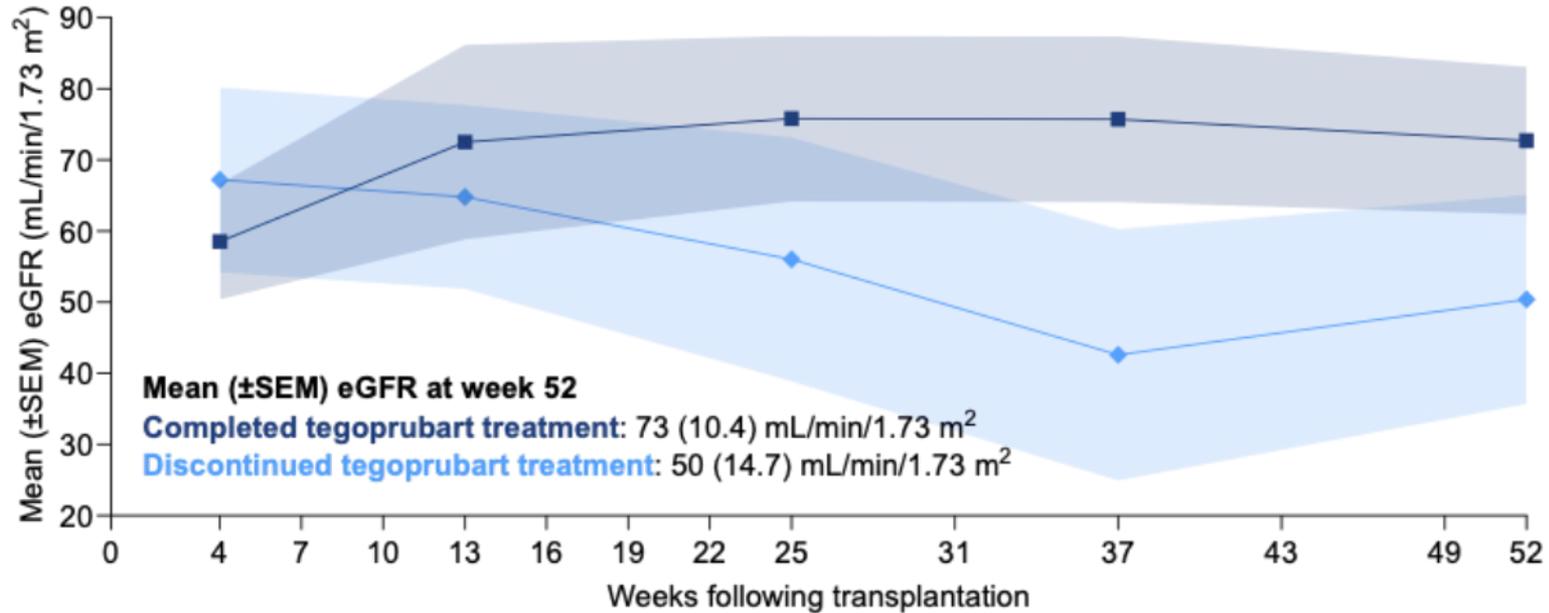


Tegoprubart (n=63)	63	59	52	47	45	45
Tacrolimus (n=64)	63	60	58	54	51	51

Note: All biopsies centrally reviewed by a blinded reference pathologist (Banff criteria). 'All cause' includes acute TCMR \geq 1A and/or active AMR, and uses data from all biopsies.

^dParticipants with TCMR and AMR were only counted once in BPAR calculation. AMR, antibody mediated rejection; BPAR, biopsy proven acute rejection; CI, confidence interval; M, months; Tac, tacrolimus; TCMR, T cell-mediated rejection; Tego, tegoprubart.

Mean eGFR by Tegoprubart Treatment Completion in Patients Who Experienced Rejection



Comp. (n)	6	6	6	6	6
Disc. (n)	5	5	5	5	5

Note: Patients who completed treatment were those patients who completed 52 weeks of the study and received all planned doses of the randomized study drug. Rejection was determined by BPAR (biopsy proven acute rejection); Comp., completed tegoprubart treatment; Disc., discontinued tegoprubart treatment; eGFR, estimated glomerular filtration rate; SEM, standard error of the mean

Composite Efficacy Failure & Donor-Specific Antibodies

All biopsy, aAMR+aTCMR	Tegoprubart (N=63) n (%)	Tacrolimus (N=64) n (%)	Difference (Tegoprubart – Tacrolimus) % (95% CI)
BPAR	13 (20.6)	9 (14.1)	6.6 (-7.0, 20.3)
Graft loss	1 (1.6)	1 (1.6)	0
Death	1 (1.6)	1 (1.6)	0
LTFU	0	0	0
EF rate through 12 months	14 (22.2)	11 (17.2)	5.0 (-9.5, 19.4)

At end of treatment, 1 tegoprubart subject and 2 tacrolimus subjects were positive for Donor-Specific Antibodies

Overall Summary of Treatment-Emergent Adverse Events

	Tegoprubart (N=63) n (%)	Tacrolimus (N=64) n (%)	Risk Difference (95% CI) ¹
Any AE	61 (96.8)	64 (100)	-3.2 (-11.1, 2.9)
AE by Max Severity²			
G1: Mild	5 (7.9)	8 (12.5)	
G2: Moderate	20 (31.7)	23 (35.9)	
G3: Severe	31 (49.2)	28 (43.8)	
G4: Life-threatening	4 (6.3)	4 (6.3)	
G5: Death	1 (1.6)	1 (1.6)	
Related AEs	32 (50.8)	35 (54.7)	-3.9 (-21.4, 14.2)
Serious AEs (SAE)	33 (52.4)	36 (56.3)	-3.9 (21.3, 13.9)
Related SAEs	11 (17.5)	8 (12.5)	5.0 (-7.9, 18.3)
AE of Special Interest	47 (74.6)	49 (76.6)	-2.0 (-17.3, 13.4)
AE leading to study drug withdrawal	4 (6.3)	3 (4.7)	1.7 (-7.9, 11.4)

¹Tegoprubart rate to Tacrolimus rate

²Participants are counted only once at their highest grade of AE, so risk difference is not presented.

On-treatment: first dose through last dose + 30 days (includes early discontinuations).

AEs $\geq 5\%$ with ≥ 2 Times Risk Observed with a Therapy

		Tegoprubart (N=63) n (%)	Tacrolimus (N=64) n (%)	Relative Difference ¹	
Opportunistic Infections	Bacteremia	1 (1.6)	7 (10.9)		6.8x
	Sepsis	2 (3.2)	5 (7.8)		2.4x
Renal	Proteinuria	10 (15.9)	1 (1.6)	9.9x	
Metabolic	Hyperglycemia	6 (9.5)	14 (21.9)		2.3x
	New Onset Diabetes (NODAT)	1 (1.6)	7 (10.9)		6.8x
	Hyperkalemia	7 (11.1)	17 (26.6)		2.4x
CNS	Tremors	1 (1.6)	16 (25.0)		15.6x
	Muscle Spasms	3 (4.8)	10 (15.6)		3.3x
	Pruritus	2 (3.2)	6 (9.4)		2.9x
Cardiovascular	Hypertensive Crisis	1 (1.6)	5 (7.8)		4.9x
Blood	Lymphopenia	4 (6.3)	10 (15.6)		2.5x

Opportunistic Infections

	Tegoprubart (N=63) n (%)	Tacrolimus (N=64) n (%)	Risk Difference (95% CI) ¹
Opportunistic Infections	43 (68.3)	44 (68.8)	-0.5 (-17.2, 15.8)
Viral Infections	33 (52.4)	31 (48.4)	3.9 (-13.8, 21.6)
CMV	25 (39.7)	23 (35.9)	3.7 (-13.3, 20.7)
CMV disease	1 (1.6)	3 (4.7)	-3.1 (-11.7, 4.7)
CMV DNA-emia	24 (38.1)	20 (31.3)	6.8 (-9.9, 23.5)
BKV (Polyomavirus)	14 (22.2)	13 (20.3)	1.9 (-12.7, 16.7)
BK nephropathy	2 (3.2)	3 (4.7)	1.6 (-5.7, 9.6)
Polyomavirus DNA-emia	14 (22.2)	13 (20.3)	1.9 (-12.7, 16.7)
EBV DNA-emia	1 (1.6)	1 (1.6)	0.0 (-7.1, 7.2)
Sepsis and Bacteremia	3 (4.8)	11 (17.2)	-12.4 (-24.9, -1.4)
Bacteremia	1 (1.6)	7 (10.9)	-9.4 (-20.0, -0.7)
Sepsis	2 (3.2)	5 (7.8)	-4.6 (-14.5, 4.3)
Fungal Infections	2 (3.2)	2 (3.1)	0.0 (-8.1, 8.3)
Other Opportunistic Infections	22 (34.9)	26 (40.6)	-5.7 (-22.6, 11.4)
PML	0	0	0.0

AEs ≥5%: Renal & Metabolic

	Tegoprubart (N=63) n (%)	Tacrolimus (N=64) n (%)	Risk Difference (95% CI) ¹
Renal			
Delayed Graft Function	9 (14.3)	16 (25.0)	-10.7 (-25.0, 3.4)
Mean Days on Dialysis for DGF (SD)	4.6 (4.19)	6.1 (7.71)	
Oliguria	0	5 (7.8)	-7.8 (-17.3, -1.4)
Proteinuria	10 (15.9)	1 (1.6)	14.3 (4.7, 25.9)
Metabolic			
Hyperglycemia	6 (9.5)	14 (21.9)	-12.4 (-25.7, 0.6)
New Onset Diabetes (NODAT)	1 (1.6)	7 (10.9)	-9.4 (-20.0, -0.7)
Hyperkalemia	7 (11.1)	17 (26.6)	-15.5 (-29.4, -1.4)
Hypokalemia	7 (11.1)	6 (9.4)	1.7 (-9.6, 13.5)
Hypophosphatemia	19 (30.2)	10 (15.6)	14.5 (-0.4, 29.4)
Metabolic Acidosis	10 (15.9)	6 (9.4)	6.5 (-5.6, 19.0)

Difference in DGF occurrence and time on dialysis correspond to an estimated ~115 additional days on dialysis per 100 deceased donor recipients on tacrolimus vs. tegoprubart

AEs ≥5%: CNS and Cardiovascular

	Tegoprubart (N=63) n (%)	Tacrolimus (N=64) n (%)	Risk Difference (95% CI) ¹
CNS			
Tremors	1 (1.6)	16 (25.0)	-23.4 (-35.8, -11.5)
Muscle Spasms	3 (4.8)	10 (15.6)	-10.9 (-22.7, 0.1)
Neurocognitive & Psychiatric Disorders	14 (22.2)	22 (34.4)	-12.2 (-27.9, 3.8)
Headache	7 (11.1)	14 (21.9)	-10.8 (-24.4, 2.5)
Dizziness	3 (4.8)	6 (9.4)	-4.6 (-15.3, 5.1)
Fatigue	7 (11.1)	5 (7.8)	3.3 (-7.9, 14.8)
Insomnia	6 (9.5)	11 (17.2)	-7.7 (-20.3, 4.8)
Pruritus	2 (3.2)	6 (9.4)	-6.2 (-16.6, 3.2)
Cardiovascular			
Heart Failure	0 (0.0)	3 (4.7)	-4.7 (-13.1, 1.6)
Hypertension	10 (15.9)	16 (25.0)	-9.1 (-23.5, 5.3)
Hypertensive Crisis	1 (1.6)	5 (7.8)	-6.2 (-15.8, 1.8)
Hypotension	8 (12.7)	7 (10.9)	1.8 (-10.2, 14.2)
Peripheral Edema	10 (15.9)	7 (10.9)	4.9 (-7.8, 17.6)
Thromboembolic Events	6 (9.5)	4 (6.3)	3.3 (-7.0, 14.2)

AEs ≥5%: Blood, Cancer & Gastro-Intestinal

	Tegoprubart (N=63) n (%)	Tacrolimus (N=64) n (%)	Risk Difference (95% CI) ¹
Blood			
Anemia	20 (31.7)	19 (29.7)	2.1 (-14.2, 18.8)
Leukopenia	26 (41.3)	15 (23.4)	17.8 (1.4, 33.9)
Lymphopenia	4 (6.3)	10 (15.6)	-9.3 (-21.4, 2.0)
Neutropenia	9 (14.3)	10 (15.6)	-1.3 (-14.3, 11.8)
Cancer			
Non-Melanoma Skin Cancer	3 (4.8)	1 (1.6)	3.2 (-4.3, 12.0)
PTLD	0	0	
Gastro-Intestinal			
Constipation	19 (30.2)	16 (25.0)	5.2 (-11.1, 20.9)
Diarrhea	14 (22.2)	22 (34.4)	-12.2 (-27.9, 3.8)
Dyspepsia	4 (6.3)	7 (10.9)	-4.6 (-15.7, 6.4)

Islet Cell Transplantation

Islet Cell Transplant Opportunity



~2 million Americans live with **Type 1 diabetes (T1D)**

~33% of people with T1D report **Impaired Awareness of Hypoglycemia regardless of CGM or Automated Pump (AID) usage**



~12% of people with T1D **experience recurrent severe hypoglycemic events** annually, putting them at higher risk for adverse outcomes

~5% to 8% of adults with T1D **experience Diabetic Ketoacidosis (DKA)** annually, often as a result of poor glycemic control

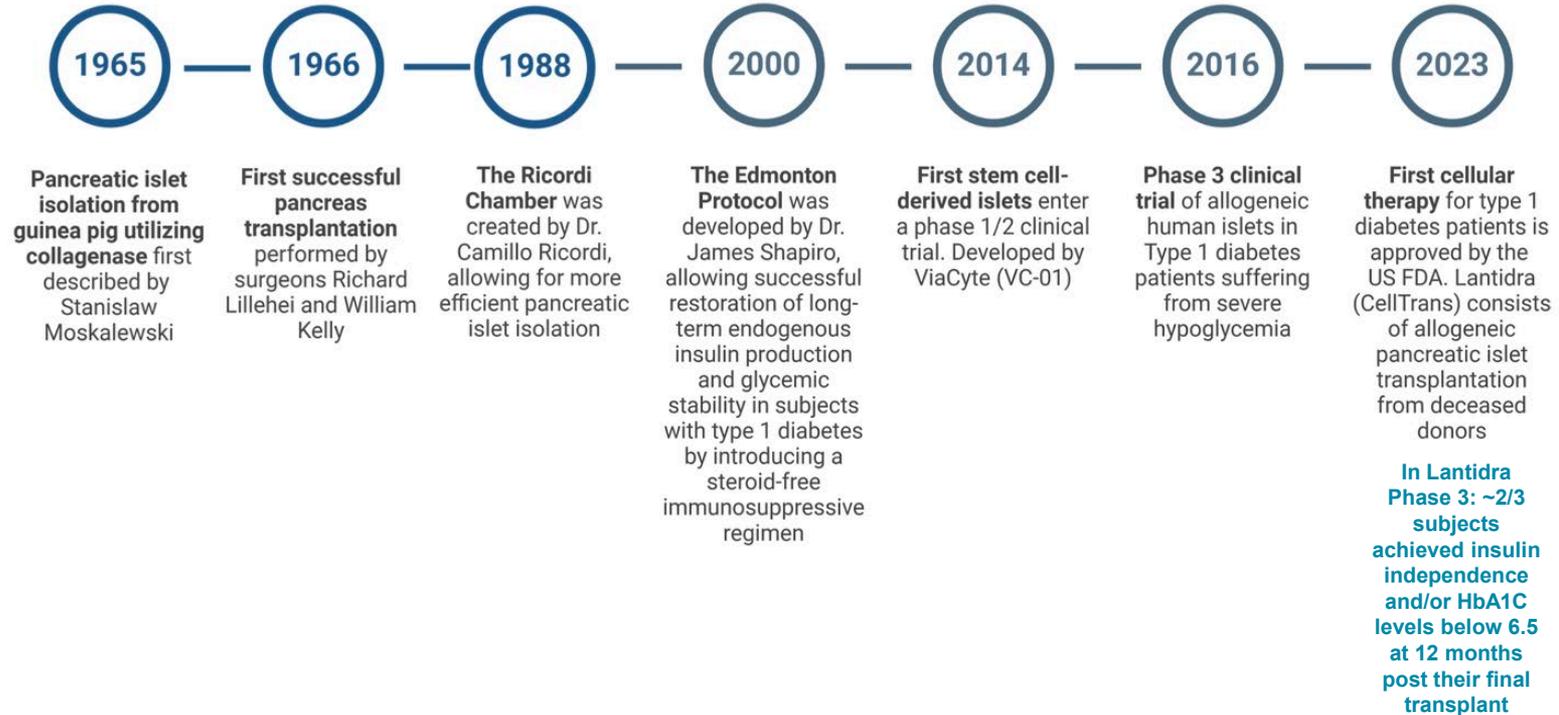


Currently, **islet cell transplantation is underutilized** in part due to immunosuppressive regimens with CNIs that may (i) be toxic to transplanted insulin producing islet cells resulting in the need for repeat transplants and (ii) cause significant side effects

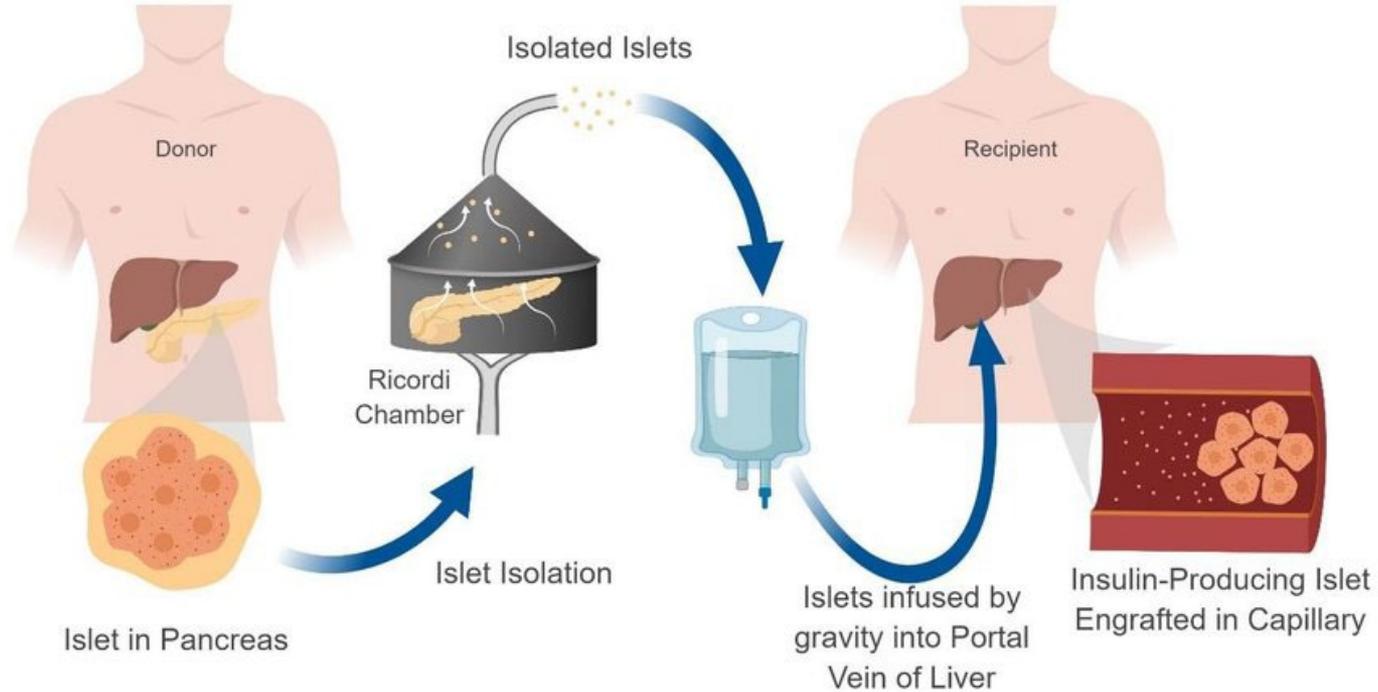
Tegoprubart may unlock the islet cell transplant market by potentially:

1. Improving islet cell graft survival regardless of cell source (e.g., isolated from deceased donor pancreases or stem-cell derived) and/or the use of a pouch
2. Reducing side effects associated with standard of care regimens including islet cell death

60 Years of Milestones in Pancreatic Islet Transplantation But Better Immunosuppression Remains a Key Need



Islet Cell Transplant Procedure



Phase 1/2 Study Assessing the Use of Tegoprubart to Prevent Islet Cell Transplant Rejection in Participants with Type 1 Diabetes (T1D)

DESIGN

- 52-week, open label, single dose level study
- Initial group of 12 patients with T1D transplanted in an investigator sponsored trial at the University of Chicago
- Islet cell transplant combined with induction therapy plus tegoprubart and mycophenolate mofetil (MMF) every third week by IV infusion
- Financing principally from Breakthrough T1D (a.k.a. JDRF) and The Cure Alliance

PLANNED DATA GENERATION

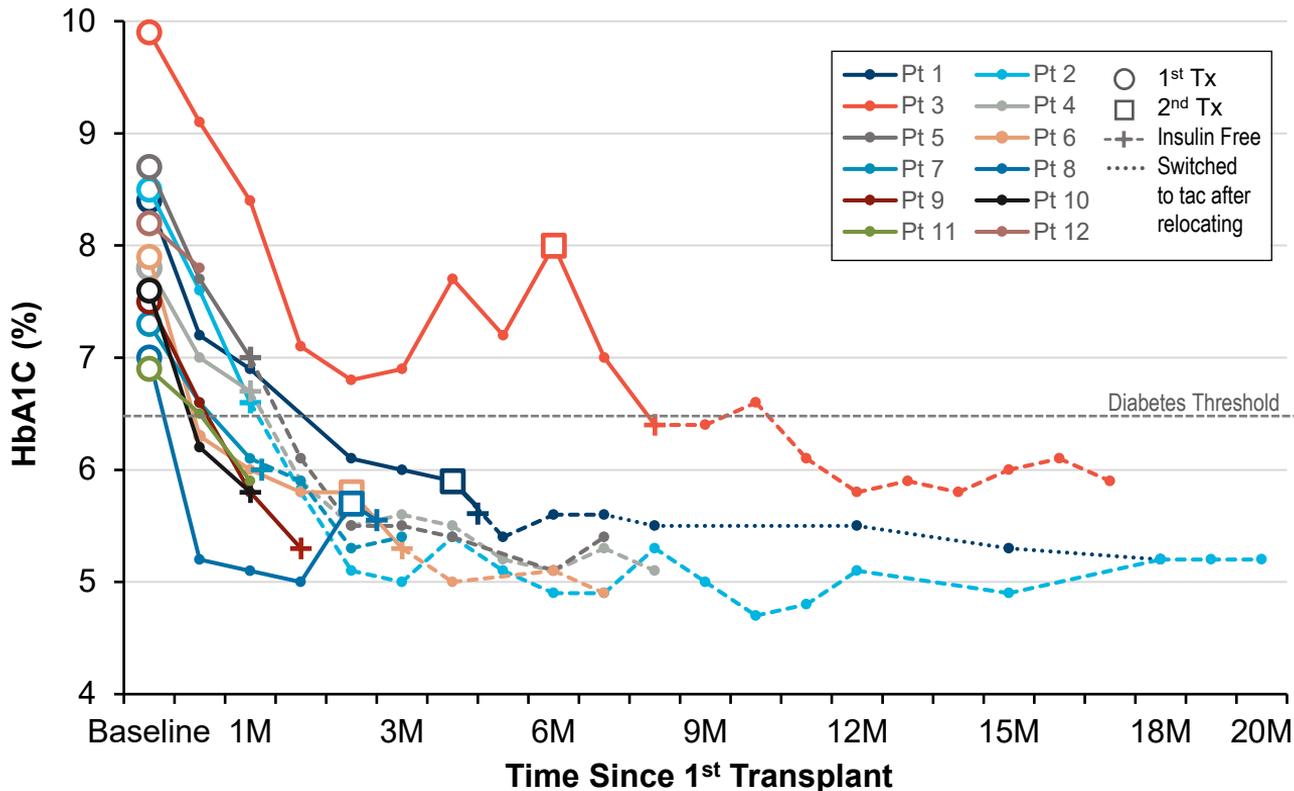
- **Safety & tolerability**
- **Graft function**
 - e.g., HbA1C, C-peptide
- **Number of hypoglycemic events**
- **Insulin independence**
- **Need for repeat islet cell transplant(s)**

Subject Demographics and Transplanted Islet Dose(s)

Subject	Gender	Age at Transplant	Years of T1D	BMI	Baseline HbA1C	Baseline Insulin (u)	1 st Tx (IEQ/Kg)	2 nd Tx (IEQ/Kg)
1	F	42	31	30	8.4	80	4,092	5,501
2	F	30	26	21	8.5	60	6,775	
3	M	37	19	30	9.9	90	4,086	4,211
4	F	51	40	19	7.8	35	5,569	
5	F	49	35	25	8.7	65	5,159	
6	M	19	9	29	7.9	90	5,491	7,096
7	F	59	41	26	7.3	40	6,519	
8	M	35	34	25	7.0	35	6,436	6,928
9	F	39	13	22	7.5	40	6,109	
10	F	49	32	24	7.6	40	6,684	
11	F	41	34	23	6.9	55	6,016	
12	M	57	39	25	8.2	35	7,731	
Average	8F /4M	42	29	25	8.0	55	5,889	5,934

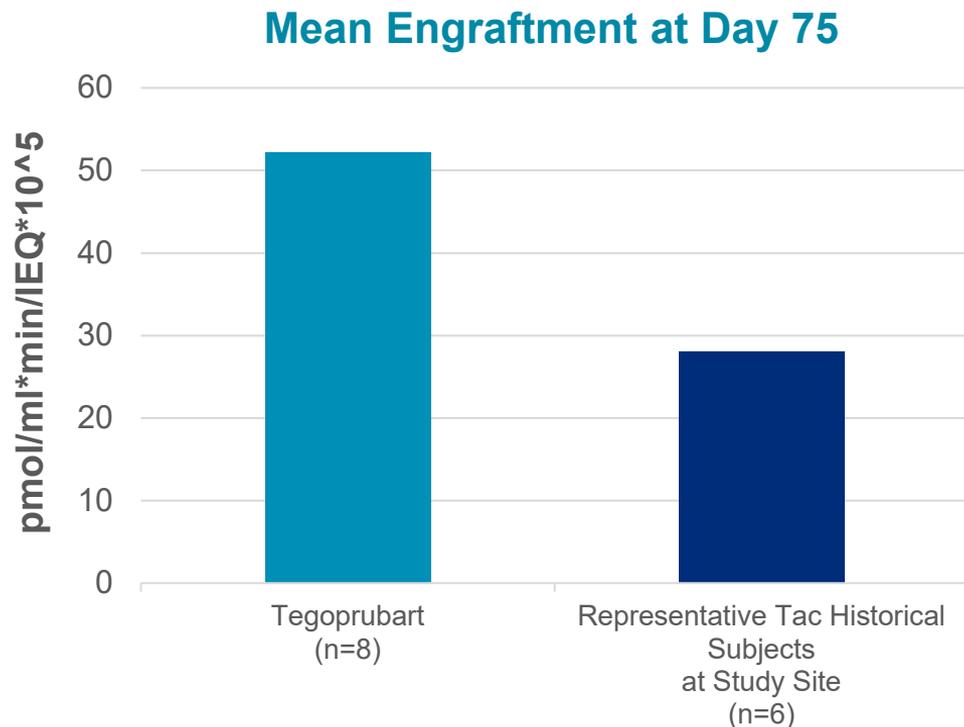
All subjects have a history of recurrent severe hypoglycemic events as well as impaired awareness of hypoglycemia, despite standard of care diabetes management and education

Pre- and Post- Transplant HbA1C Levels & Timing of Independence from Exogenous Insulin



- 10 of 10 subjects >4 weeks post-transplant achieved insulin independence and an average ~2.65% reduction in HbA1c to date
- 9 of 10 subjects >4 weeks post transplant achieved normal HbA1C levels within ~2 months after initial transplant, all within 12 months
- No subjects experienced a severe hypoglycemic event post-transplant

Day 75 Calculated Islet Engraftment



- **Islet engraftment**, calculated based on c-peptide secretion during a Mixed Meal Tolerance Test on day 75 post transplant, **demonstrated higher levels of engraftment with tegoprubart vs. representative historical subjects on tacrolimus**
- **Single donor islet transplants** treated with tegoprubart permitted **lowering insulin requirements by up to ~65u of insulin per day**
- 4 subjects who required a second transplant were able to reduce their insulin doses by an average of ~66% after their initial transplant

Islet Cell Transplant Safety Profile Summary To Date

- **All 12 subjects achieved stable islet graft function with rapidly improved blood glucose control**
- **No severe hypoglycemic events**
- **No signs of rejection or de novo donor specific antibodies (DSAs)**
- **No signs of kidney toxicity, neurotoxicity, GI toxicity, hypertension, or thromboembolic events**
- **No severe infections**
 - 2 subjects experienced transient, low titer CMV viremia that responded to lowered MPA
 - 1 subject experienced a superficial skin infection that responded to lowered MPA
 - 1 subject experienced episode of norovirus diarrhea
- **Non-clinically significant anemia & leukopenia that responded to lowered MPA**



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Thank You!

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