



Pioneering Gene Therapies for Patients in Need

February 2024



www.genprex.com | NASDAQ: GNPX

Forward-Looking Statements

www.genprex.com

Statements contained in this presentation regarding matters that are not historical facts are “forward-looking statements” within the meaning of the Private Securities Litigation Reform Act of 1995. Because such statements are subject to risks and uncertainties, actual results may differ materially from those expressed or implied by such forward-looking statements. Such statements include, but are not limited to, statements regarding our expected operating results, our ability to maintain compliance with the continued listing requirements of The Nasdaq Capital Market and to continue as a going concern and to obtain capital to meet our long-term liquidity needs on acceptable terms, or at all, including the additional capital which will be necessary to complete the clinical trials that we have initiated or plan to initiate, achievement of key milestones, our ability to advance the clinical development, manufacturing and commercialization of our product candidates in accordance with projected timelines and specifications, and the effects of our product candidates, alone and in combination with other therapies, on cancer and diabetes. Risks and uncertainties that contribute to the uncertain nature of the forward-looking statements include our ability to achieve key milestones, the timing and effect of our achieving those milestones, the competition we face from other biotechnology and pharmaceutical companies, the effects of Fast Track and/or Orphan Drug Designations, and of other factors, on the clinical development, manufacturing and commercialization of our product candidates, as well as the presence and level of our product candidates’ effect on cancer and diabetes, the timing of our IND filings and amendments, the timing and outcome of FDA action with respect to our IND filings and amendments, the timing and our ability to contract with clinical sites and to enroll patients in our clinical trials, including the impact of the COVID-19 pandemic and competition for patients on such timing, the timing and performance of our third party manufacturers, vendors and suppliers, the timing and success of our clinical trials and planned clinical trials of our product candidates, the timing and success of obtaining FDA approval of our product candidates, costs associated with developing our product

candidates, and whether patents will ever be issued under patent applications filed by us or that are the subject of our license agreements or that others may be able to develop competing products that do not infringe our patent rights, such that our product candidates may not have an exclusive market position. These and other risks and uncertainties are described more fully under the caption “Risk Factors” in our annual report on form 10-K for the year ended December 31, 2022 and our other filings and reports with the United States Securities and Exchange Commission. While we believe we have identified material risks, these risks and uncertainties are not exhaustive. Moreover, we operate in a very competitive and rapidly changing environment. New risks and uncertainties emerge from time to time, and it is not possible to predict all risks and uncertainties, nor can we assess the impact of all factors on our business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in any forward-looking statements. All forward-looking statements contained in this press release speak only as of the date on which they were made. Except as required by law, we undertake no obligation to update such statements to reflect events that occur or circumstances that exist after the date on which they were made.

This presentation highlights basic information about our company. Because it is a summary, it does not contain all of the information you should consider before investing in our company. Further information about our company may be found in our public filings and reports with the United States Securities and Exchange Commission.

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Our Mission

Advancing novel gene therapies for **patients**
afflicted with cancer or diabetes.



Program Highlights



ONCOLOGY

- ★ Non-viral gene therapy platform
- ★ Novel approach using systemic gene therapy to replace tumor suppressor genes for cancer in humans
- ★ Three FDA Fast Track Designations, one Orphan Drug Designation and three lung cancer trials
- ★ Clinical achievement in Ph 1 and Ph 2 studies
- ★ Near-term data readouts

DIABETES

- ★ Addressing both Type 1 and Type 2 diabetes with AAV gene therapy
- ★ Novel infusion process delivers genes to pancreas
- ★ Demonstrated ability to stabilize glucose levels and reduce insulin requirements shown in Non-Human Primate (NHP) studies
- ★ Poised for FDA guidance in 2024

Research and Development Pipeline

	Delivery System	Drug Candidate	Indication	Clinical Trial Program Name	Regulatory Designation	Discovery	Preclinical	IND-Enabling	Clinical Phase 1	Clinical Phase 2	Clinical Phase 3
ONCOLOGY	ONCOPREX® NANOPARTICLE DELIVERY SYSTEM (NON-VIRAL AND SYSTEMIC)	REQORSA® IMMUNOGENE THERAPY	NSCLC	Acclaim [®] 1 (ONC-003)	Fast Track Designation	REQORSA® + Tagrisso					
		REQORSA® IMMUNOGENE THERAPY	NSCLC	Acclaim [®] 2 (ONC-004)	Fast Track Designation	REQORSA® + Keytruda					
		REQORSA® IMMUNOGENE THERAPY	SCLC	Acclaim [®] 3 (ONC-005)	Fast Track, Orphan Drug Designation	REQORSA® + Tecentriq					
		OTHER ONCOLOGY TARGETS	—	—							
		REQORSA® IMMUNOGENE THERAPY	NSCLC	(ONC-001)		Monotherapy					
		REQORSA® IMMUNOGENE THERAPY	NSCLC	(ONC-002)		REQORSA® + Tarceva					
DIABETES	AAV Vector	GPX-002	T1D	DIA-001							
		GPX-003	T2D	DIA-002							
		OTHER DIABETES TECHNOLOGIES	—	—							



ONCOLOGY

REPROGRAMMING THE COURSE OF CANCER



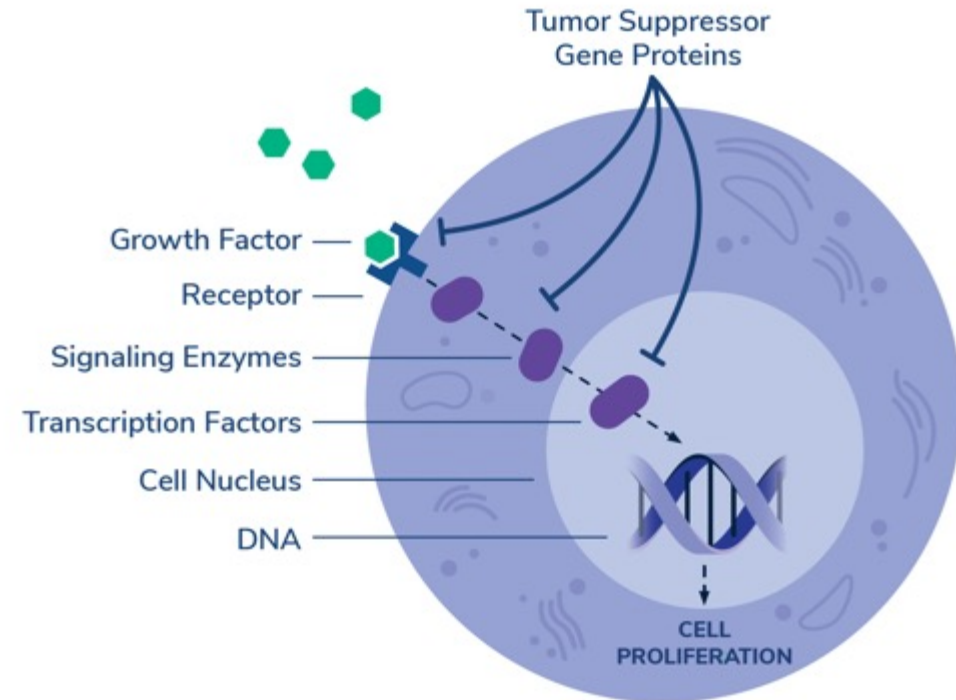
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Tumor Suppressor Genes Deleted During Cancer Development

Why TUSC2?

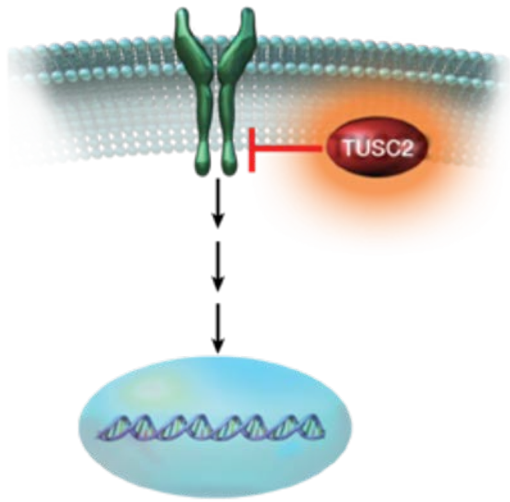
- Tumor suppressor genes are deleted early during cancer development
- 82% of all non-small cell lung cancers and 100% of all small-cell lung cancers express decreased amounts of TUSC2 tumor suppressor protein
- Loss or reduction of TUSC2 expression is associated with significantly reduced overall survival
- Led to the hypothesis that reintroduction of tumor suppressor genes may be a new method of treating cancer

Tumor Suppressor Genes Act Like a Brake Pedal



Reqorsa[®] Targets Cancer At Its Core

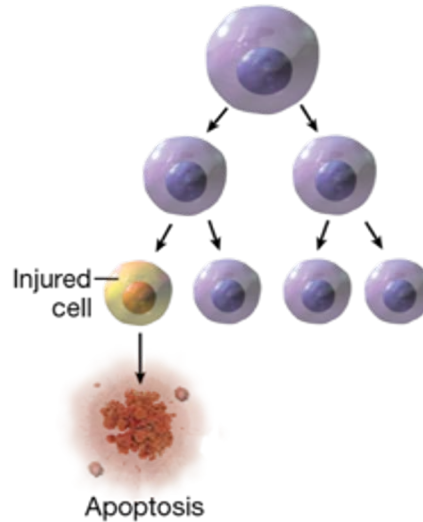
Multiple anti-cancer
mechanisms of action.



1

Controls Cell Signaling

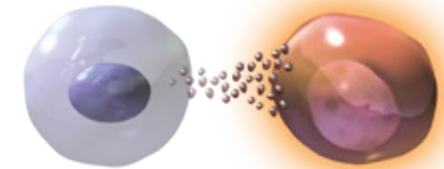
Pan-kinase inhibition
decreases cancer cell
proliferation



2

Stimulates Apoptotic Pathways

Leads to programmed
cancer cell death



3

Modulates Immune Response

Promotes immune
activity against cancer

Our Cancer Treatment Approach

Tumor suppressor genes are deleted early during cancer development.

Our method of treating cancer is to reintroduce tumor suppressor genes to patients.



Tumor Suppressor Gene in a DNA Plasmid

We have rights to tumor suppressor genes that may have cancer-fighting functions. These genes are expressed in a DNA plasmid.



Non-Viral Lipid Nanoparticles

The gene expressing DNA plasmid is then encapsulated into our ONCOPREX® Nanoparticle Delivery system, which consists of non-viral lipid nanoparticles made from lipid molecules.



Systemic Patient Administration

The final drug product is delivered systemically through intravenous injection and specifically targets cancer cells.

Novel Platform to Treat Cancer

Systemic Gene Therapy Platform: **ONCOPREX® Nanoparticle Delivery System**



Genes

Allows for delivery of TUSC2 and NPRL2 genes and potentially a variety of other genes

Synergies

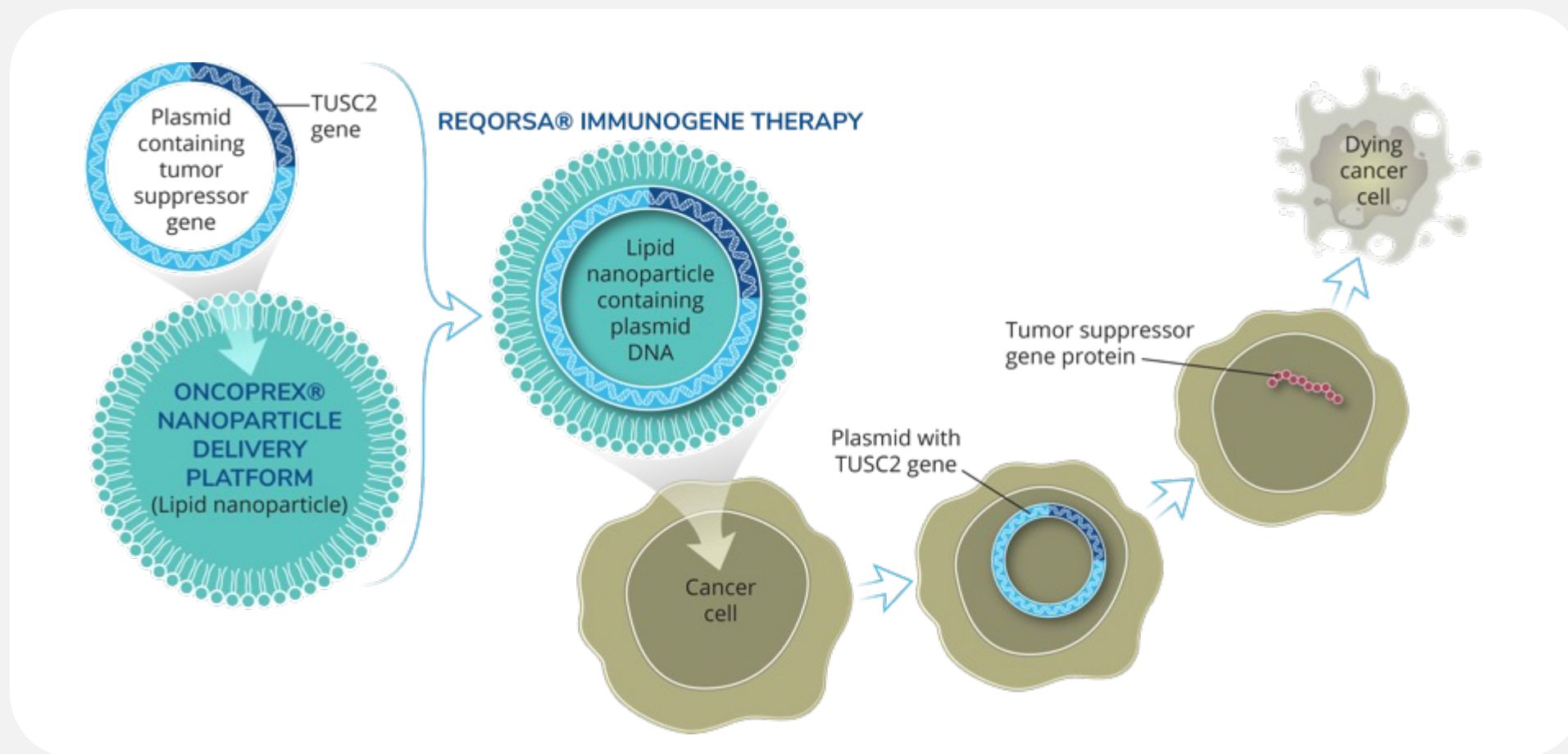
Can be used in combination with other cancer therapies such as Tagrisso®, Keytruda® and Tecentriq®

Cancers

Could combat multiple cancers including NSCLC, SCLC, mesothelioma, glioblastoma, breast, kidney and thyroid

Oncoprex® Nanoparticle Delivery System

Non-viral, positively-charged lipid nanoparticle (LNP) is **systemically delivered**.



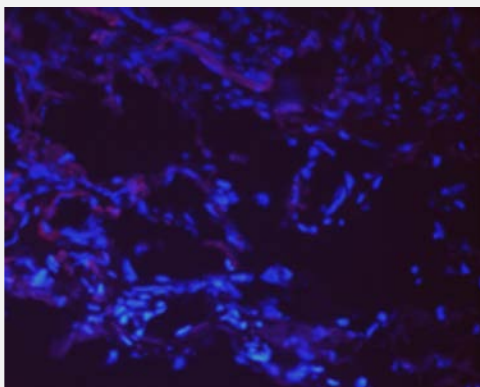
Cationic lipid nanoparticle carries drug to tumors.

Selective Uptake of Reqorsa[®]

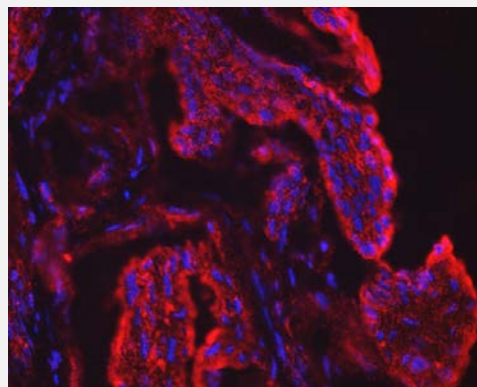
REQORSA Targets Cancer Cells

Patient 1
(.02 mg/kg)

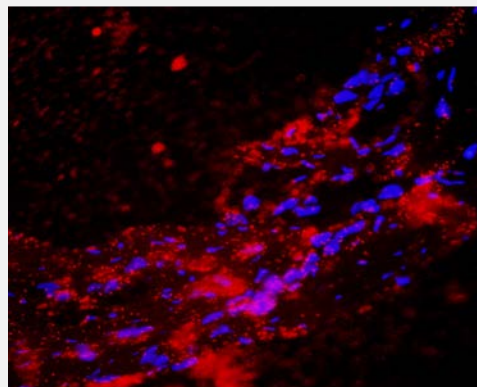
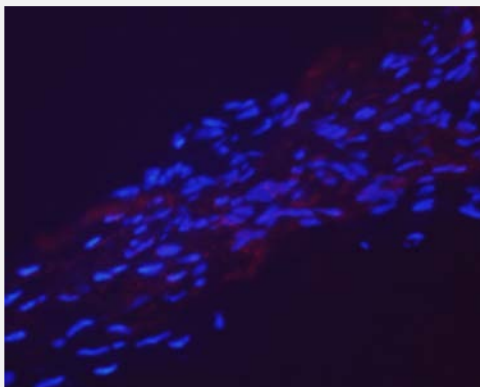
Pretreatment Biopsies



Posttreatment Biopsies



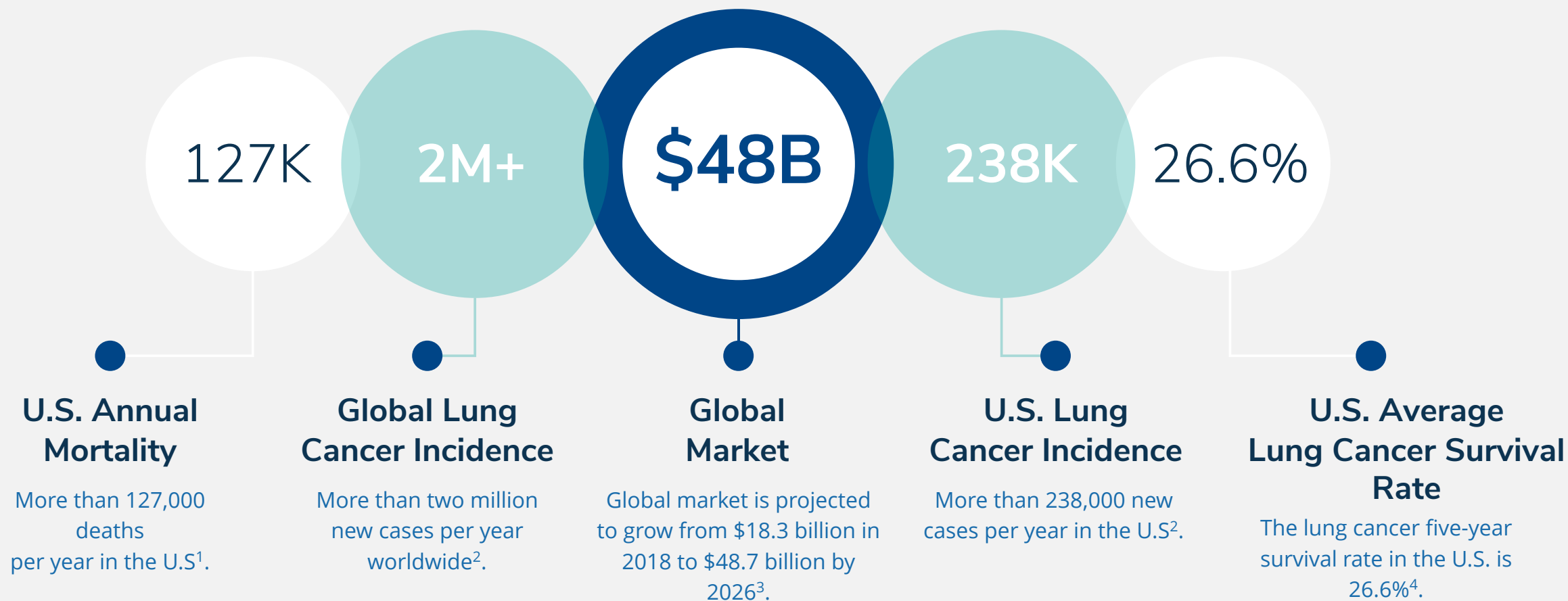
Patient 2
(.06 mg/kg)



REQORSA is designed to deliver the functioning TUSC2 gene to cancer cells while minimizing its uptake by normal tissue.

Tumor biopsy studies show that, in three patients, the expression of TUSC2 was markedly increased 1 day after REQORSA treatment.

Lung Cancer: By the Numbers



For most patients, **drug resistance** to Tagrisso®, Keytruda® and Tecentriq® is **inevitable**.^{1,2,3}

Our approach is designed to address drug resistance.

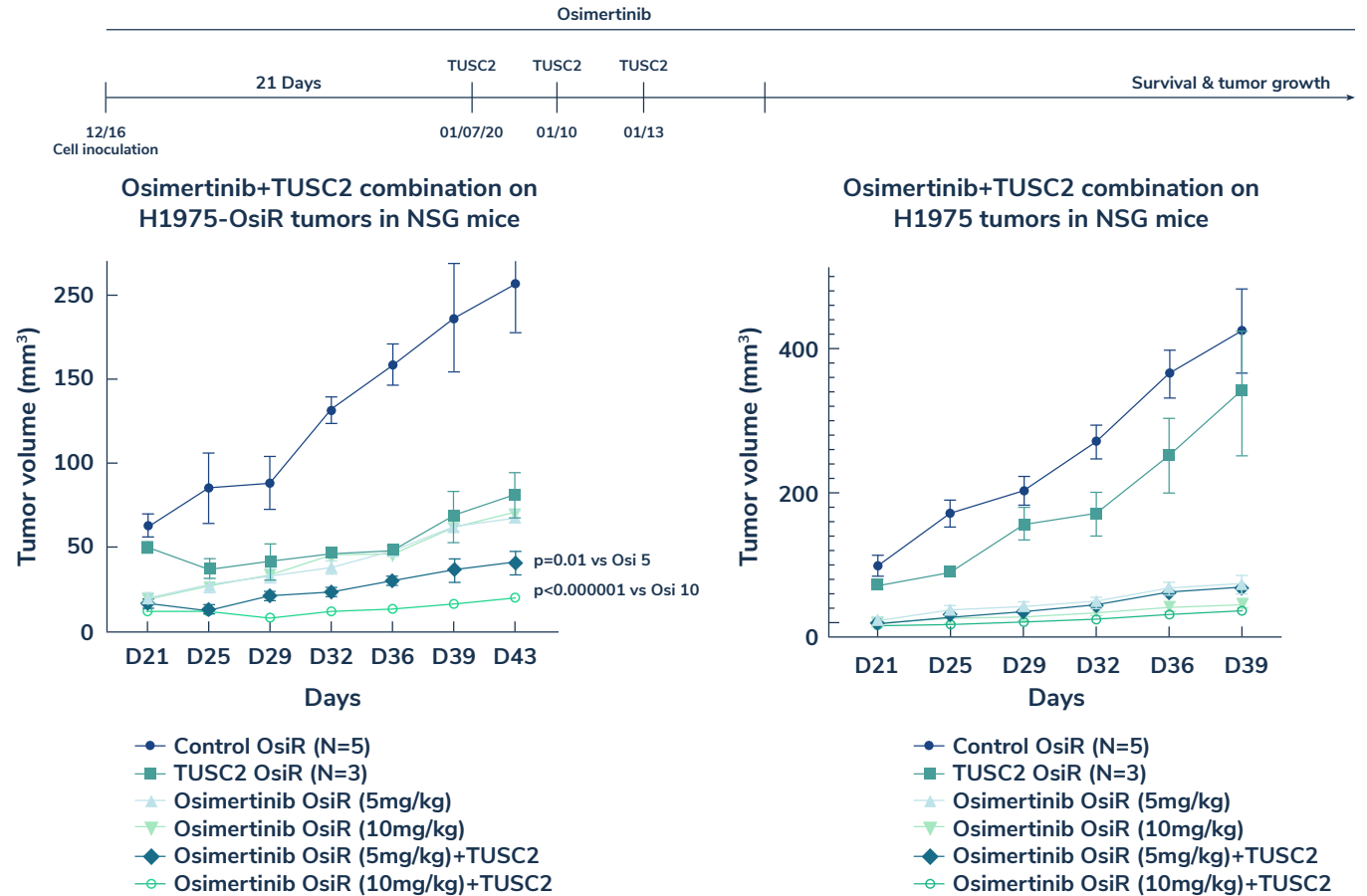
- REQORSA Immunogene Therapy may be complementary with targeted drugs and immunotherapies.
- REQORSA's multimodal activity may block emerging bypass pathways, thereby potentially reducing the probability that drug resistance develops.



AACR 21: Reqorsa[®] + Tagrisso Reduce Tumor Growth in Tagrisso Resistant Tumors

Enhanced Anti-Tumor Activity

REQORSA in combination with Tagrisso demonstrated significantly increased anti-tumor efficacy in EGFR mutant Tagrisso resistant NSCLC tumors in H1975-OsiR mouse xenografts.



Osimertinib is the generic name for Tagrisso.

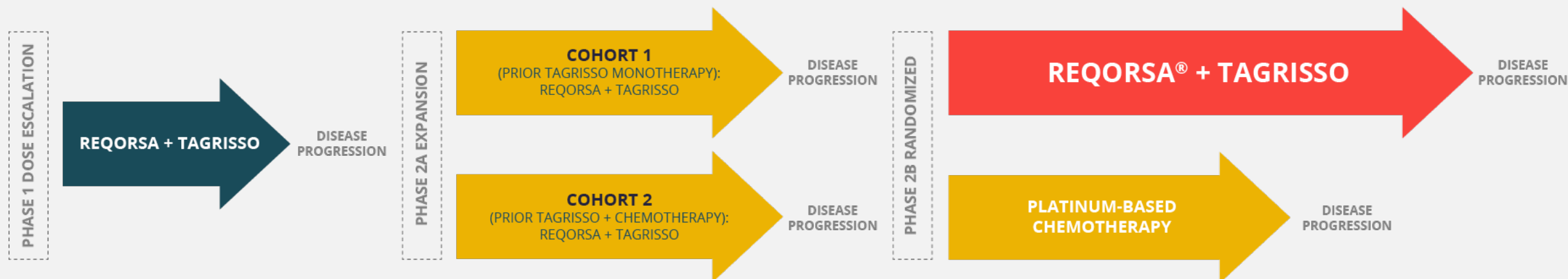
- FDA Fast Track Designation
- Patients with advanced, EGFR mutant NSCLC whose disease progressed after Tagrisso®
- ~15-20 U.S. sites
- ~158 patients
 - Phase 1 Dose Escalation: 12 patients (completed)
 - Phase 2a Expansion: ~66 patients in 2 cohorts (opening for enrollment in Jan. 2024)
 - Phase 2b: ~74 patients
- Phase 2a Expansion cohort interim analysis at 19 patients (each cohort)
- Phase 2b interim analysis at 28 events (i.e., disease progression or death)



Acclaim · 1

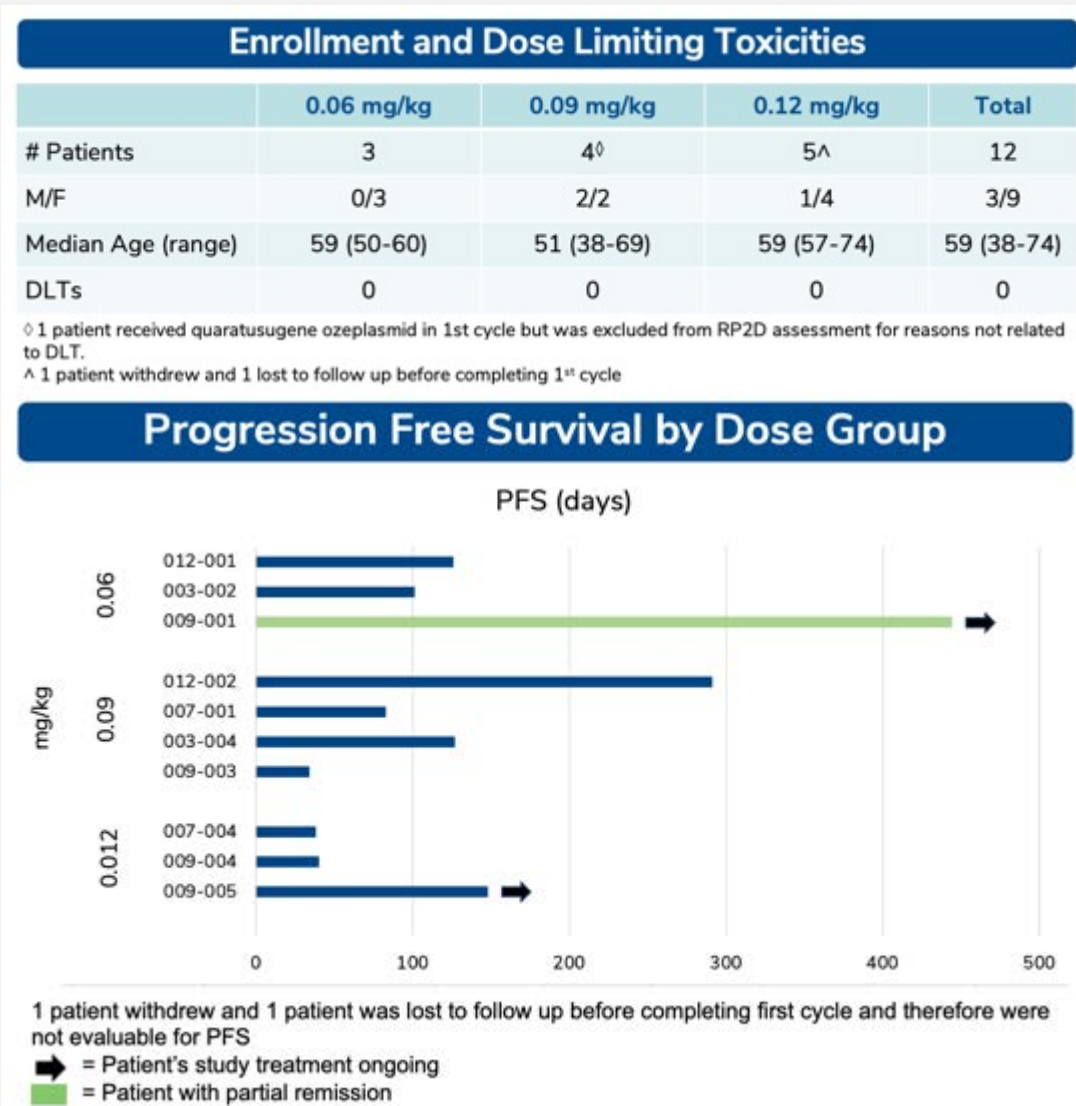
Reqorsa® in combination with AstraZeneca's Tagrisso® for NSCLC

Phase 2b: Comparing Progression Free Survival of REQORSA + Tagrisso vs. Platinum-Based Chemotherapy



Phase 1 Dose Escalation

Excellent safety profile and efficacy in relapsed patients.



Acclaim 1

Reqorsa® in combination with AstraZeneca's Tagrisso® for NSCLC

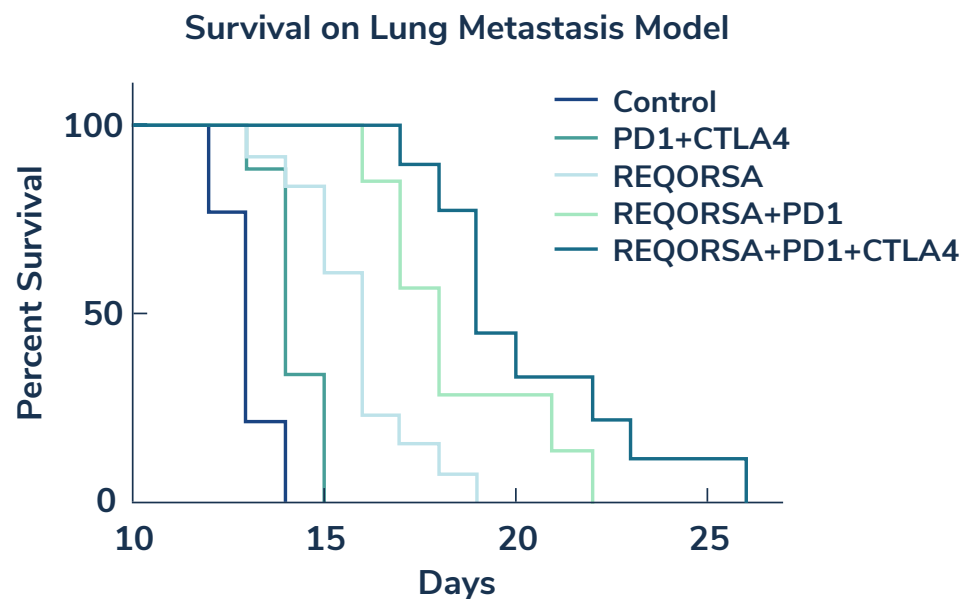
REQORSA was generally well tolerated with no DLTs.

3/12 patients progressing on Tagrisso containing regimens had prolonged PFS on REQORSA + Tagrisso combination therapy

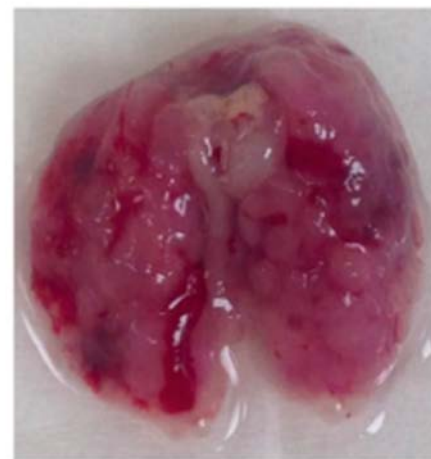
- 1 continuing treatment with PR after 26 cycles
- 1 progressing after 14 cycles of treatment
- 1 continuing treatment with SD after 12 cycles

RP2D is 0.12 mg/kg based on the lack of DLTs.

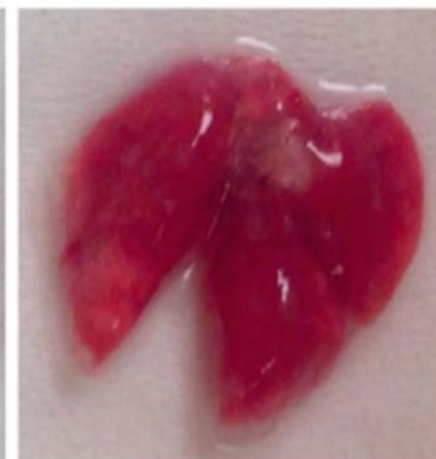
Reqorsa[®] with Immunotherapies



Non-Treated



REQORSA Treated



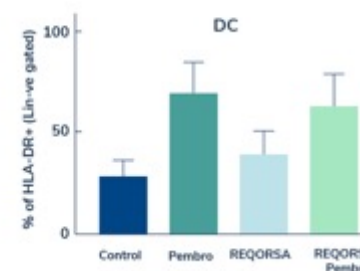
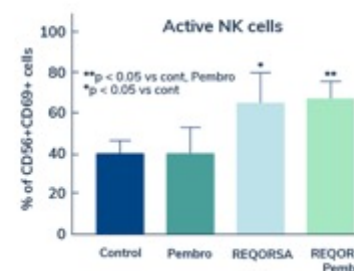
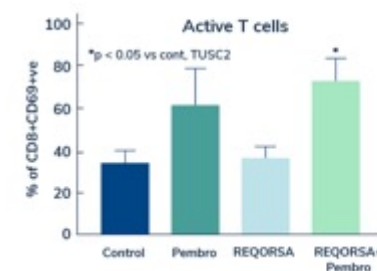
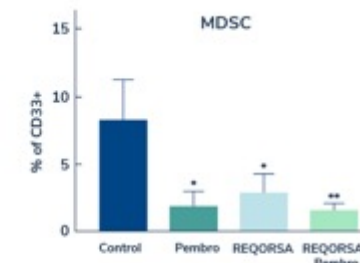
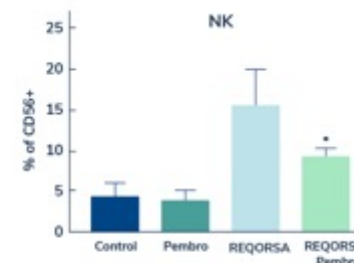
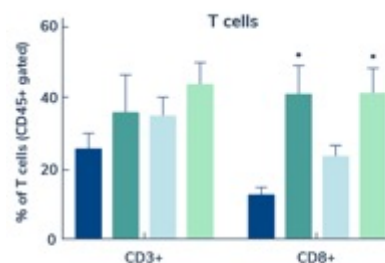
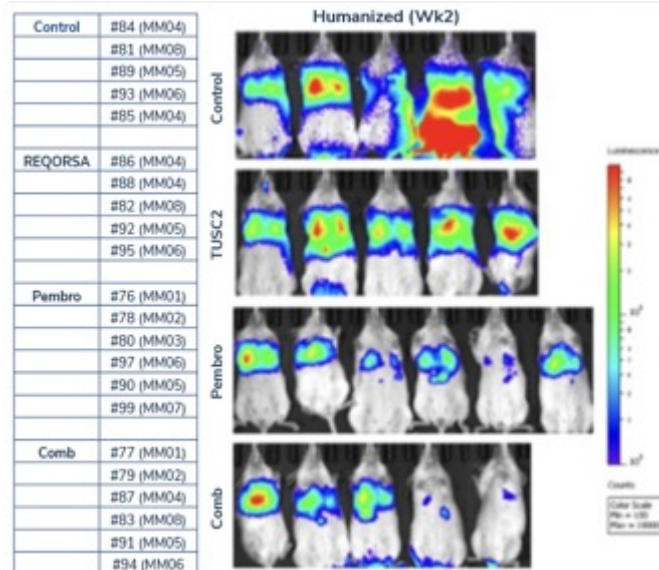
REQORSA+anti-PD1 **exhibit greater anti-tumor activity** than either agent alone or control.

REQORSA+anti-PD1 combination **significantly prolonged survival** in a lung metastasis model refractory to checkpoint blockade alone.

Reqorsa® + Keytruda® Significantly Reduced Tumor Growth

REQORSA increases immune response
against lung cancer xenografts

REQORSA + Keytruda®



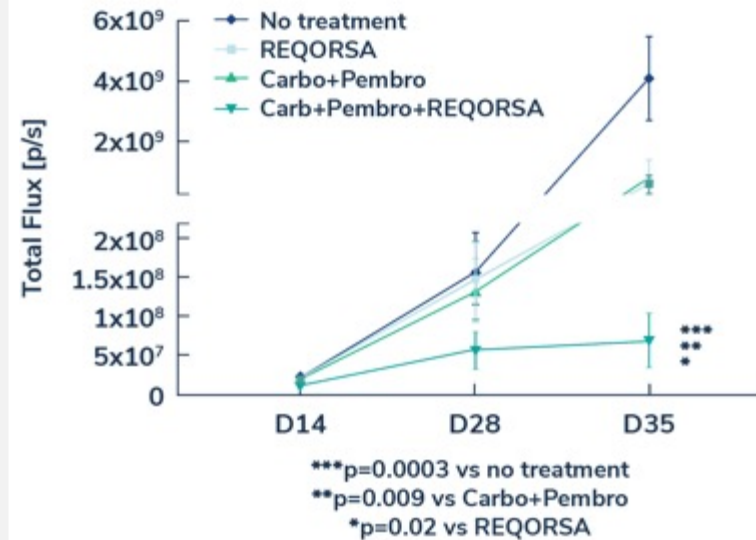
Pembrolizumab is the generic name of Keytruda.

The independent immunologic effects of REQORSA and Keytruda markedly decrease tumor growth by increasing the immunologic attack on the tumor compared to PD-1 inhibition alone.

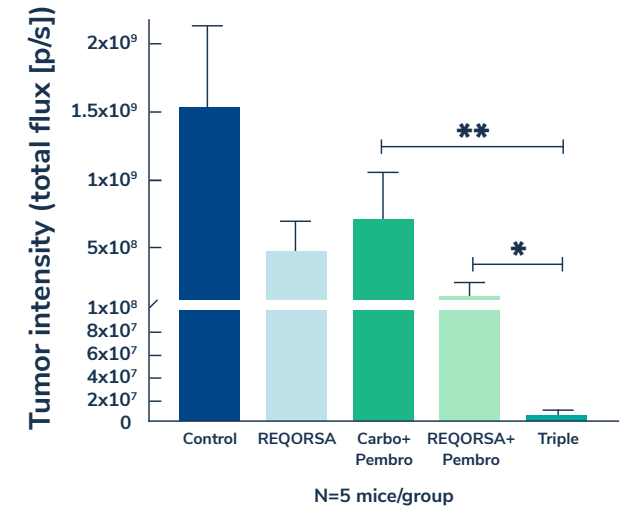
AACR 21: Reqorsa® May Enhance First-Line Standard of Care

Reqorsa® + Keytruda® + Chemo

- REQORSA enhances the efficacy of chemo-immunotherapy on KRAS-LKB1 (KL)-mutant lung metastases in humanized mice.
- Triple combination demonstrated strong antitumor efficacy and induced robust antitumor immunity in KL-mutant NSCLC in clinically relevant humanized mice models.



Triple treatment on A549 lung met in humanized mice Tumor intensity (Wk4) post treatment



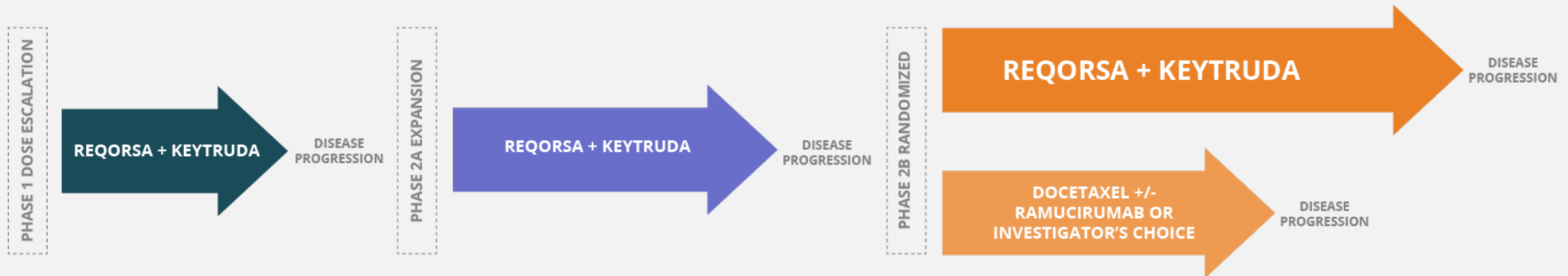
Pembrolizumab is the generic name of Keytruda.

- FDA Fast Track Designation
- Patients with advanced NSCLC whose disease progressed after treatment with Keytruda®
- ~10 U.S. sites
- ~180 patients
 - Phase 1 Dose Escalation: Up to 18 patients
 - Phase 2a Expansion: 36 patients
 - Phase 2b Randomized: 126 patients
- Phase 2b interim analysis at 50 events (i.e., disease progression or death)



Reqorsa® in combination with Merck & Co's Keytruda® for NSCLC

Phase 2b: Comparing Progression Free Survival of REQORSA + Keytruda vs. docetaxel +/- ramucirumab or Investigator's Choice

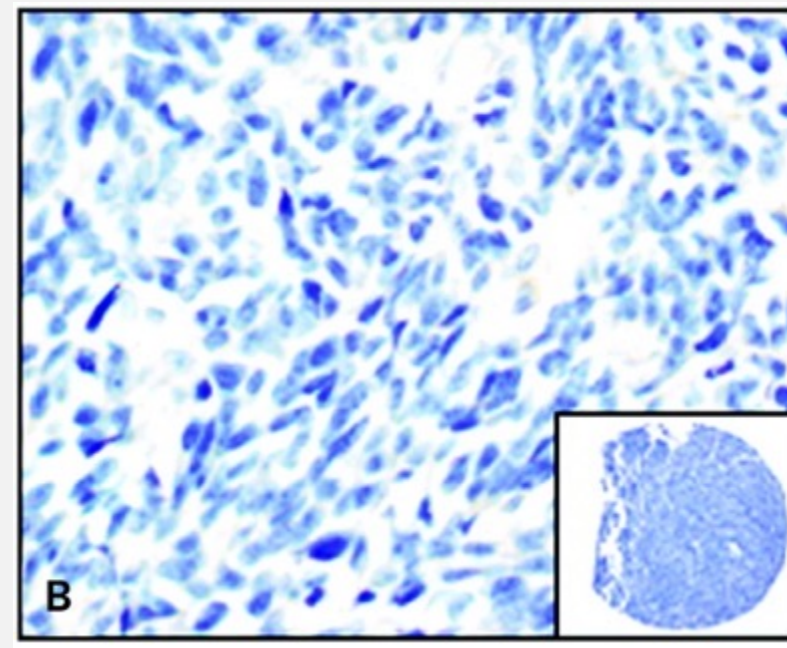


Reqorsa® in Small Cell Lung Cancer

Targeting Small Cell Lung Cancer (in addition to NSCLC) **allows Genprex® to address virtually the entire lung cancer market.**

Small Cell Lung Cancer:

- Consistently has low TUSC2 protein levels
- Documented to often have deletion of at least one TUSC2 gene allele.
- Extensive stage SCLC has very poor prognosis – a median PFS of 5.2 months.



Small cell lung cancer with negative TUSC2 expression.

Image source: Clin Cancer Res 2008;14:41-7.

Another clinical opportunity to combine
REQORSA with checkpoint inhibitors

SCLCs Express Low Levels Of TUSC2 Protein

IHC analysis of tumor specimens

- 41% of SCLC have no TUSC2 protein expression
- 100% of SCLC have reduced or no TUSC2 protein expression

Since all SCLCs have reduced or no TUSC2 protein expression, re-expressing TUSC2 protein may lead to clinical efficacy.

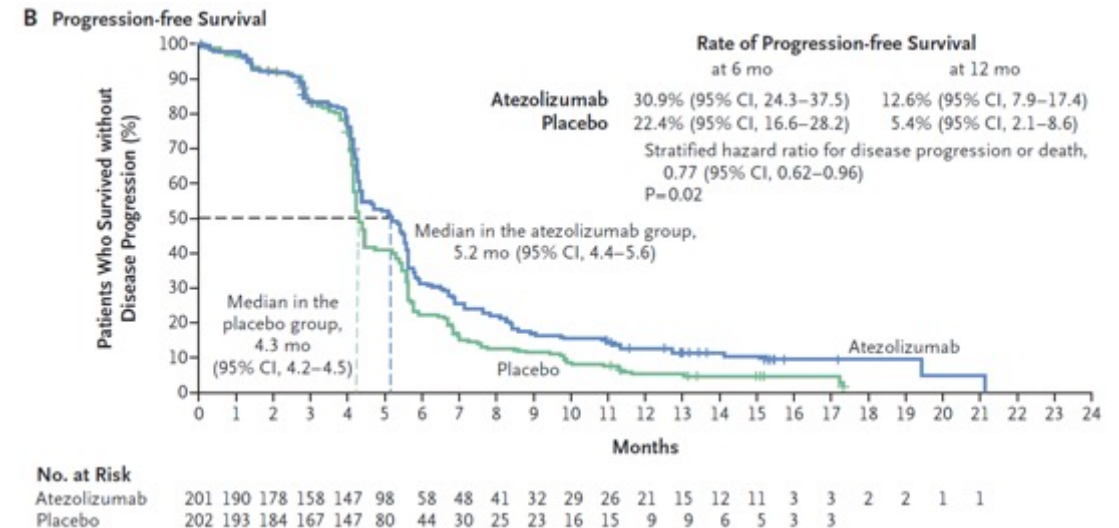
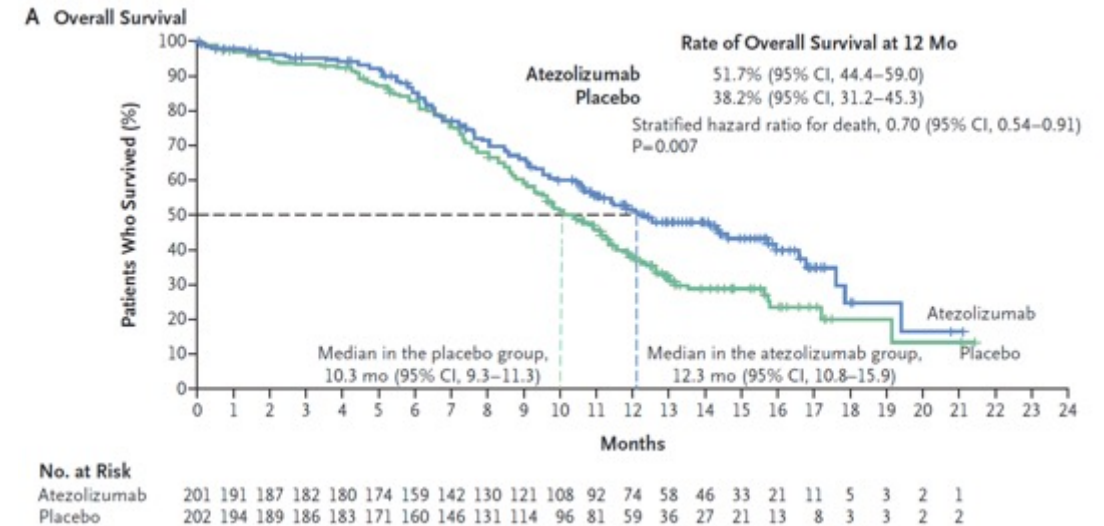
Histology of samples	No. of samples	Fus1 score, mean (SD)	Fus1 score levels			P value, Fus1 levels
			Lost (negative) n (%)	Reduced (low + intermediate) n (%)	Preserved (high) n (%)	
Cancer specimens						Comparison between tumors
SCLC	22	57 (67.4)	9 (41)	13 (59)	0	0.0008
NSCLC	281	121 (87.3)	36 (13)	194 (69)	51 (18)	
Adenocarcinoma	172	127 (91.8)	25 (15)	110 (64)	37 (22)	0.07
Squamous cell carcinoma	109	111 (79.1)	11 (10)	84 (77)	14 (13)	

Atezolizumab (Tecentriq®) SCLC Approval Trial

IMpower133 Study

Adding Tecentriq to standard therapy improves survival in SCLC and establishes a new standard therapy for ES-SCLC.

- Untreated, extensive stage SCLC
- Carboplatin & etoposide chemotherapy + atezolizumab or placebo
 - 4 cycles, then atezolizumab maintenance therapy or placebo until progression
 - Atezolizumab 1200 mg every 3 weeks
- PFS 5.2 vs 4.3 mos (HR 0.77)
- OS 12.3 vs 10.3 mos (HR 0.70)



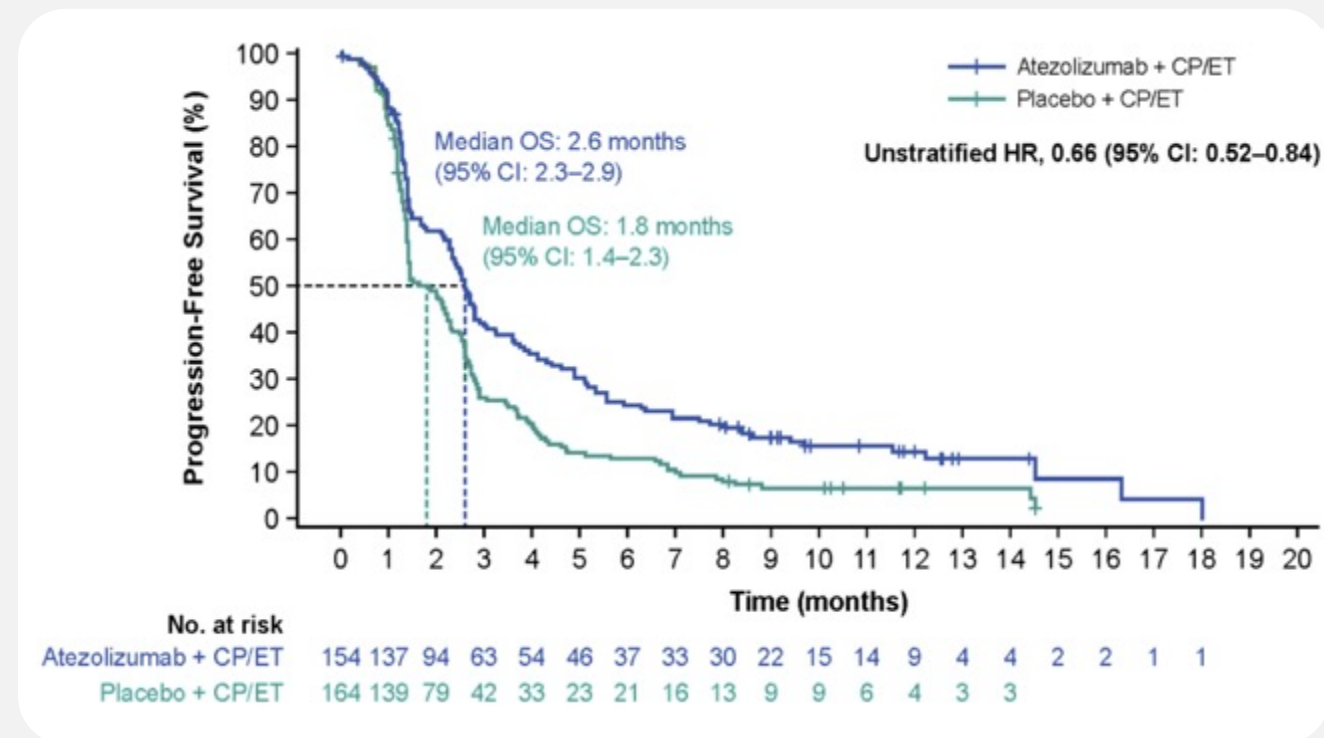
Atezolizumab Maintenance Therapy

Once patients begin maintenance therapy with Tecentriq, Progression Free Survival is very short (2.6 mos).

- Atezolizumab vs placebo
 - All CR, PR, and SD patients received maintenance therapy
 - Endpoints measured from the start of maintenance therapy

- PFS 2.6 vs 1.8 mos (HR 0.63)

- OS 12.5 vs 8.4 mos (HR 0.59)



- Fast Track Designation and Orphan Drug Designation
- Patients with ES-SCLC who did not develop tumor progression after receiving Tecentriq® and chemotherapy
- ~10 U.S. sites
- ~62 patients
 - Phase 1 Dose Escalation: Up to 12 patients (open for enrollment in Jan. 2024)
 - Phase 2: ~50 patients
- Phase 2 futility analysis after 25th patient enrolled and treated reaches 18 weeks of follow up



Acclaim 3

Reqorsa® in combination with Genentech, Inc.'s Tecentriq® for SCLC

Phase 2: Determine 18-week Progression Free Survival Rate of REQORSA + Tecentriq Maintenance Therapy



AACR 23: NPRL2 Induces Anti-tumor Activity in NSCLC

Further Evidence of Oncoprex® Delivery System as a Platform for Treatment Using Tumor Suppressor Genes

- Study investigated the antitumor responses to NPRL2 gene therapy on anti-PD1 resistant KRAS/STK11 mutant NSCLC in a humanized mouse model
- Humanized mice were treated with NPRL2 gene therapy, Keytruda®, or the combination
- A dramatic antitumor effect was observed by NPRL2 treatment, whereas Keytruda was largely ineffective
- NPRL2 gene therapy induces antitumor activity on KRAS/STK11 mutant anti-PD1 resistant NSCLC through DC mediated antigen presentation and cytotoxic immune cell activation



Provides preclinical validation of the ONCOPREX Nanoparticle Delivery System, **which may provide a multitude of potential pipeline opportunities beyond lung cancer.**

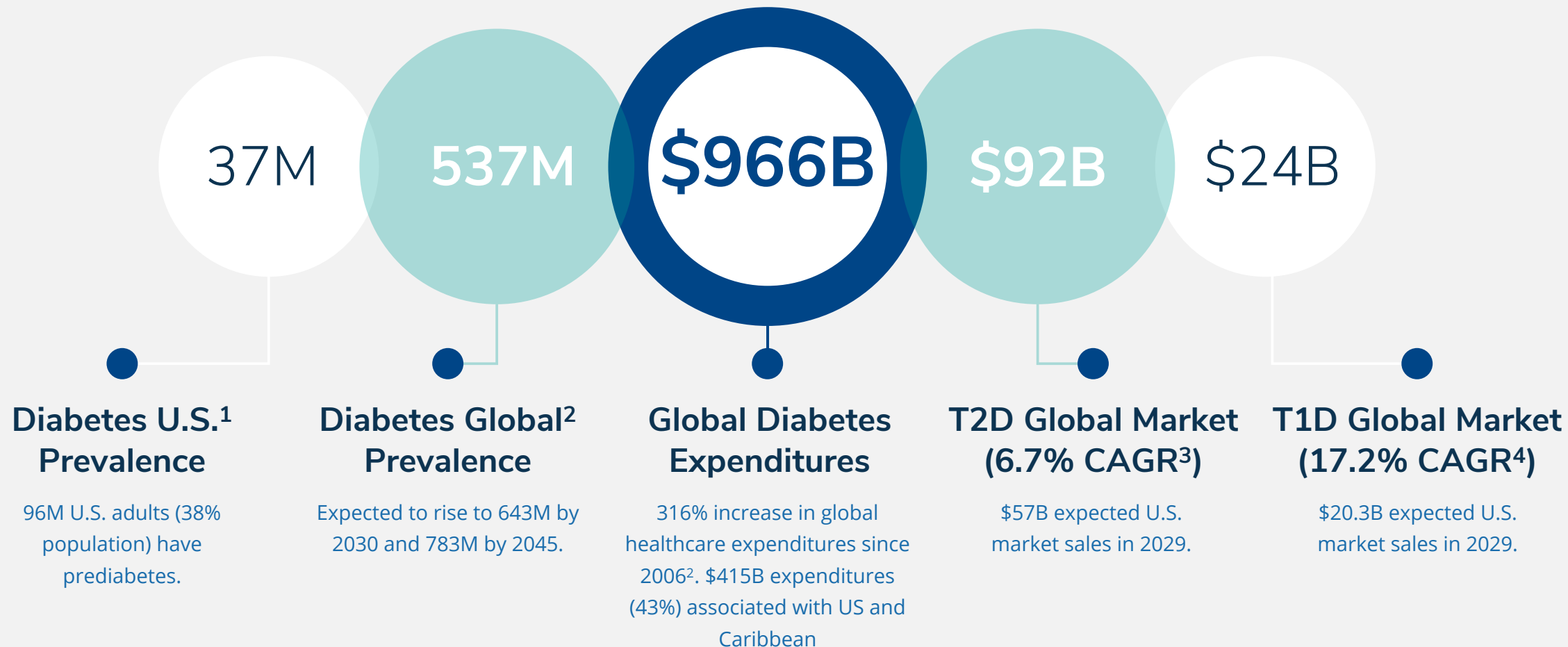


DIABETES



www.genprex.com

Diabetes: By the Numbers



Diabetes can cause serious complications.

In 2021, there was
approximately
1 death every 5 seconds
caused by diabetes
worldwide.



Diabetes Causes Serious Complications



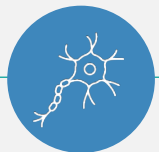
Heart Disease

Leading cause of death for men and women in U.S.
Diabetics are 2x as likely to have heart disease or a stroke.



Chronic Kidney Disease

Approximately 1 in 3 adults with diabetes have CKD.
Kidney diseases are the 9th leading cause of death in U.S.



Nerve Damage

High blood sugar can lead to diabetic neuropathy. 50% of people with diabetes have nerve damage.

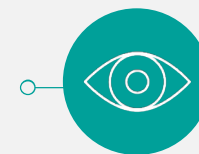


Foot Health (Diabetic Neuropathy)

Feet and legs most affected by diabetic neuropathy. 50% of annual amputations are associated w/ diabetes.

Vision Loss (Diabetic Retinopathy)

Diabetic retinopathy affects almost 1/3 of adults over 40 years old.
Diabetes is leading cause of new blindness cases in adults.



Hearing Loss

Hearing loss is 2x as common in diabetics. Prediabetes have a 30% higher rate of hearing loss.



Oral Health

Gum disease can be more severe and take longer to heal. 25% of U.S. diabetics over 50 years old have severe tooth loss.

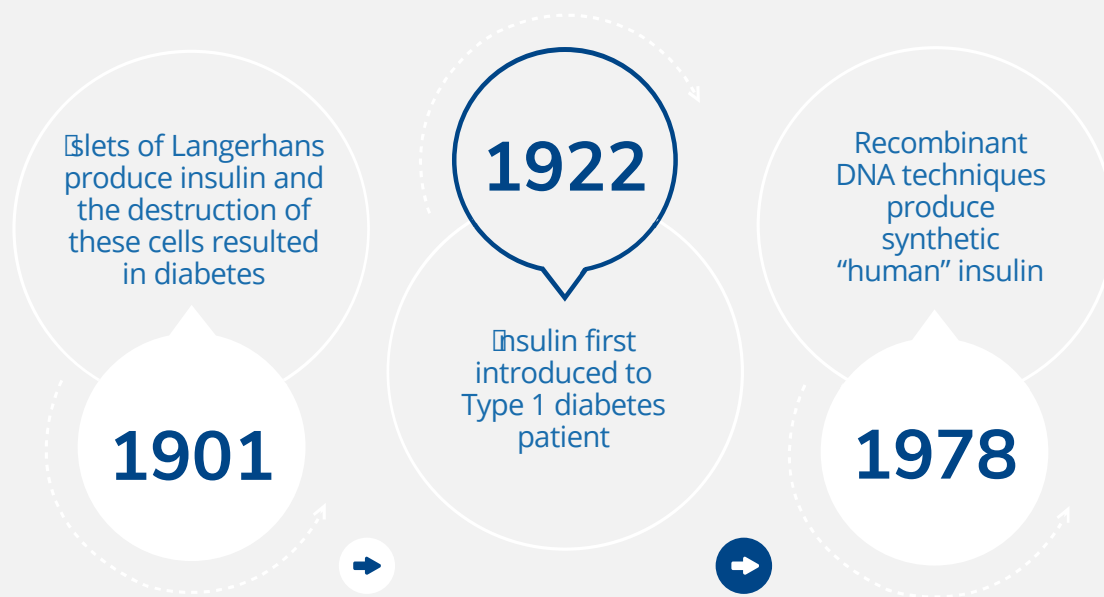


Mental Health

Blood sugar levels are affected by stress. Diabetics are 2-3x more likely to have depression.



Diabetic Patients Are In Need of Advanced Therapy



The most significant advancement in the treatment of diabetes happened in 1922
– more than 100 years ago.

Potential for disease modification
for long-term effectiveness.



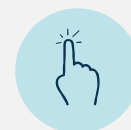
Patients suffer compromised quality of life

Despite certain advancements in treatment, quality of life remains highly compromised for many individuals with diabetes.



Gene therapy has potential to be the key

Diabetes gene therapies hold the potential to provide long-term effectiveness and change the course of the disease.



Potential to improve diabetic's lifestyle

Our treatment may replace the daily burden of blood glucose monitoring and insulin replacement therapy, including finger pricks and insulin injections.

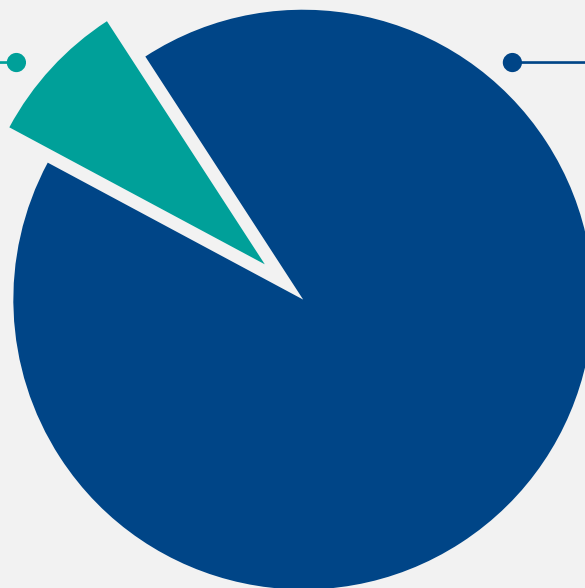
Novel Gene Therapy Diabetes Program

Collaboration with University of Pittsburgh
intends to address both T1D and T2D.

37.3M or 11.3% of Americans Have Diabetes¹

Type 1 Diabetes (5-10%)

An auto-immune condition where the body's immune system destroys pancreatic beta cells that make insulin. Generally occurs in children and adolescents.



Type 2 Diabetes (90-95%)

Inability of the pancreas to produce enough insulin due largely to resistance to insulin function. Generally occurs in adulthood, and highly related to obesity.

Two unique product candidates position Genprex as an
innovator in emerging diabetes therapies.

GPX-002 Replenishes Levels Of Insulin

Reprograms and restores cell function in T1D.

Delivers Genes to the Pancreas

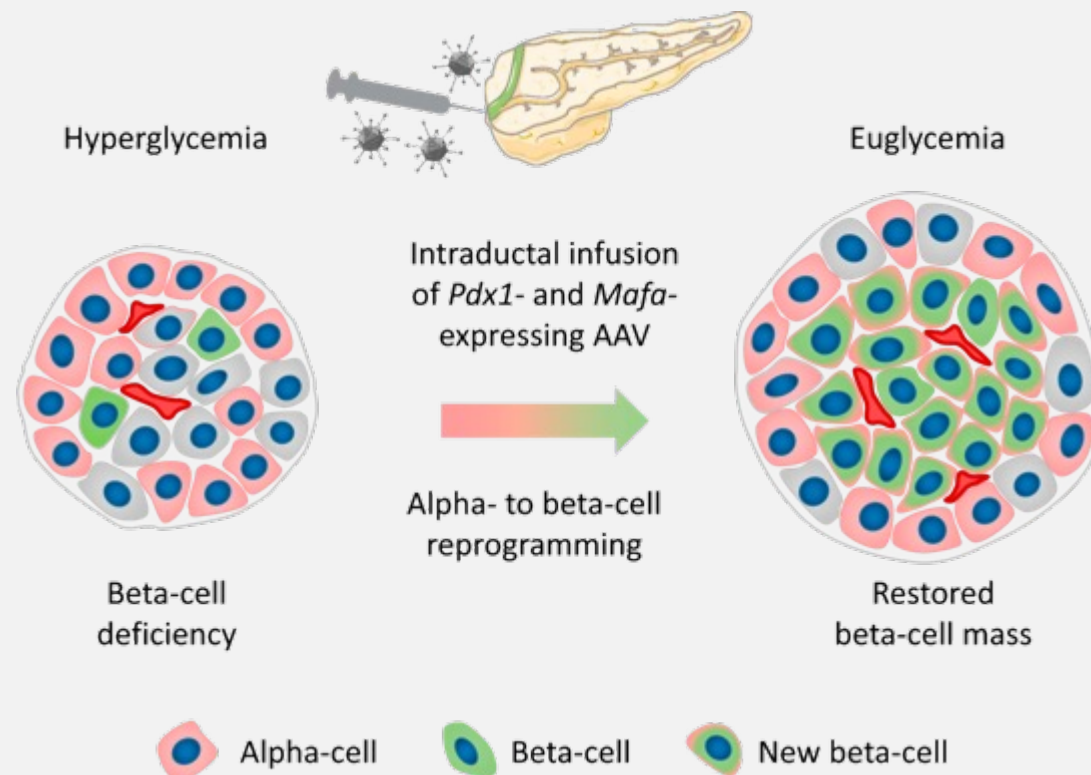
A novel infusion process uses an AAV vector to deliver the Pdx1 + MafA (PM) genes to the pancreas.

Reprograms Alpha Cells

GPX-002 **transforms alpha cells** in the pancreas into functional beta-like cells, which can produce insulin but may be distinct enough from beta cells to evade the body's immune system.

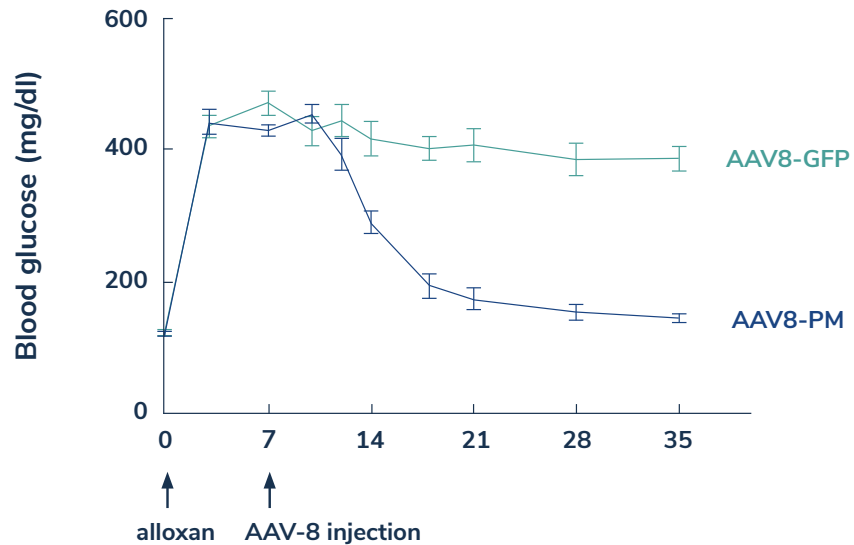
Restores Blood Glucose Levels

In vivo, preclinical studies show that **GPX-002 restored normal blood glucose levels** for an extended period of time.

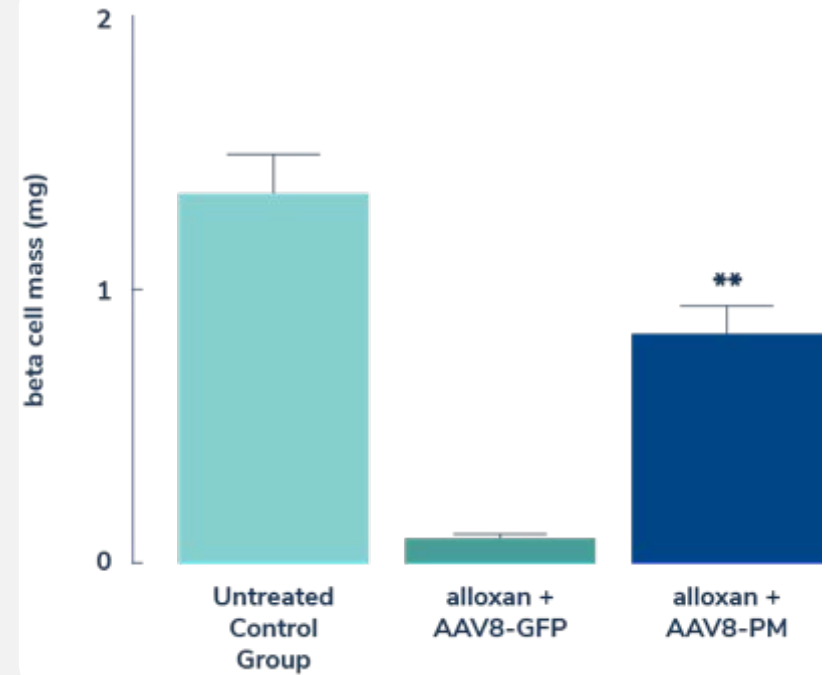


[Image source: Osipovich, Anna & Magnuson, Mark. (2018). Alpha to Beta Cell Reprogramming: Stepping toward a New Treatment for Diabetes. Cell Stem Cell. 22. 12-13. 10.1016/j.stem.2017.12.012.

Reversed Drug-Induced Diabetes in T1D Toxin-Induced Mouse Model



GFP = Green Fluorescent Protein (fluorescent marker) | PM = Pdx1 + MafA

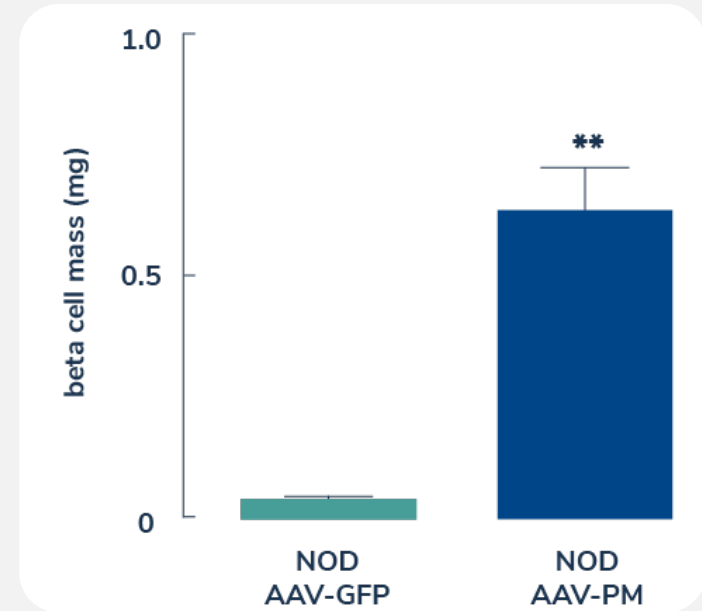
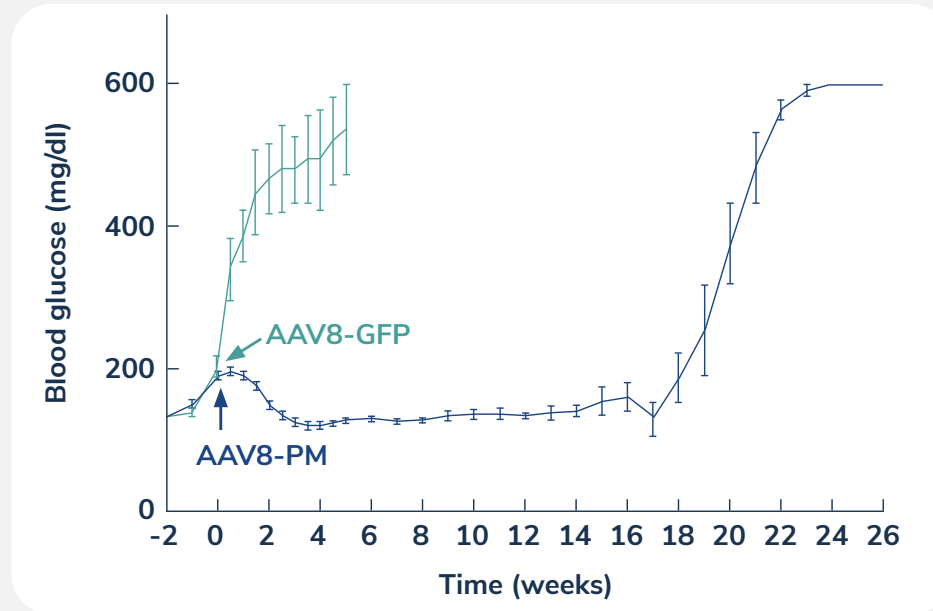


- Reprogrammed alpha cells into beta-like cells that appropriately produce insulin in response to glucose levels.
- Normalized blood glucose in beta cell-toxin-induced diabetic mice.

Restored Blood Glucose in T1D Autoimmune Mouse Model for Four Months

The duration of restored blood glucose levels in mice could potentially translate to decades in humans.

- One week in a mouse tends to correlate to about one year in humans.

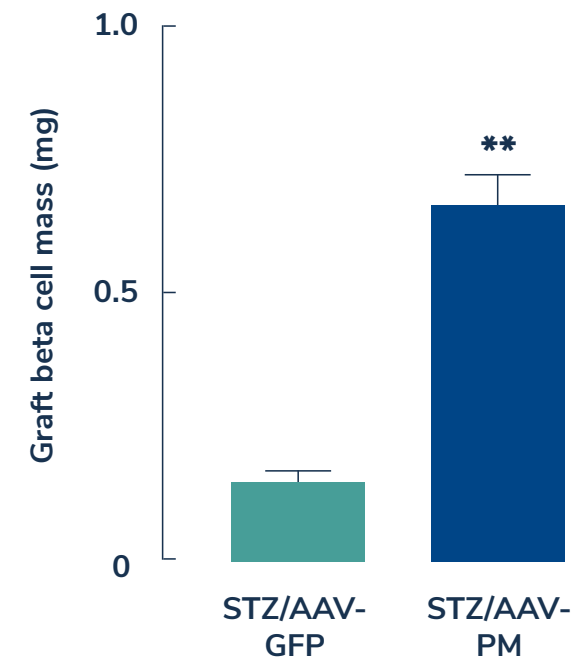
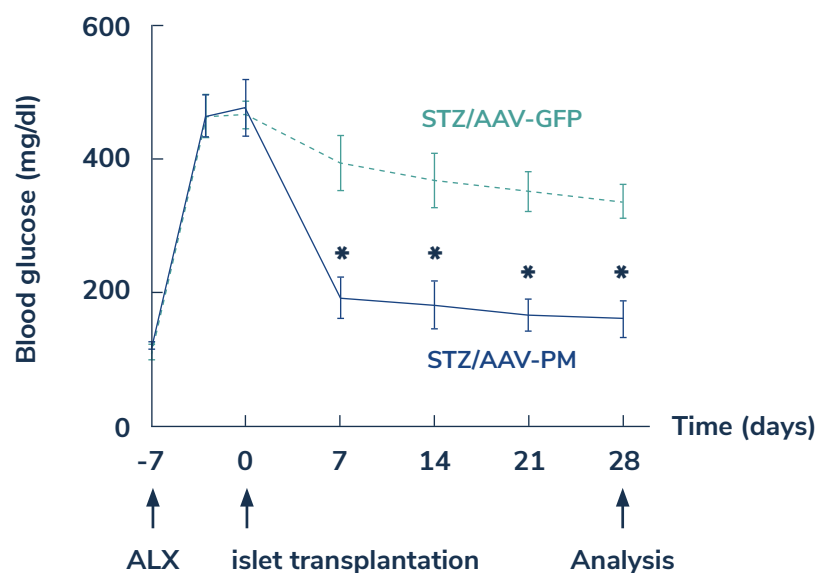


GFP = Green Fluorescent Protein (fluorescent marker) | PM = Pdx1 + MafA

Induced Generation of Functional Insulin Expressing Cells from Alpha Cells in Human Islets

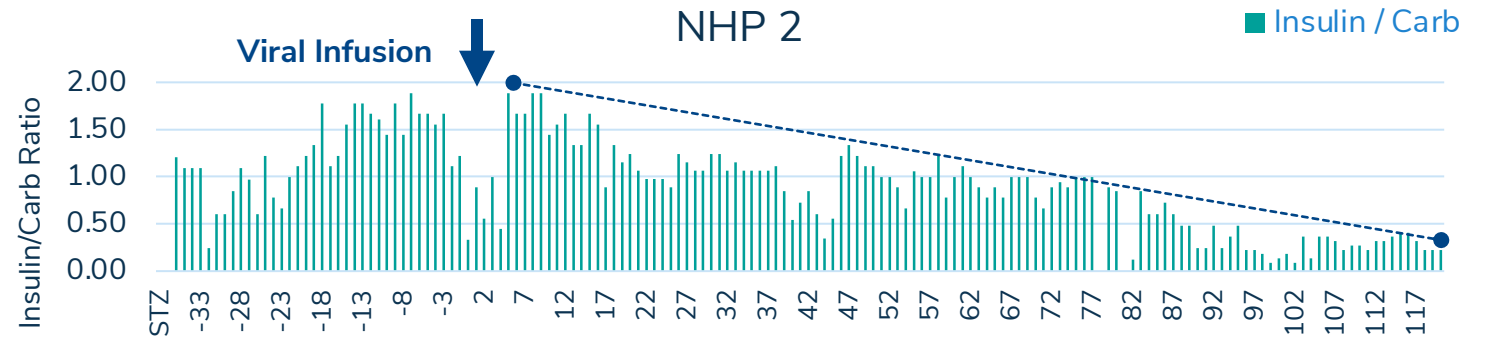
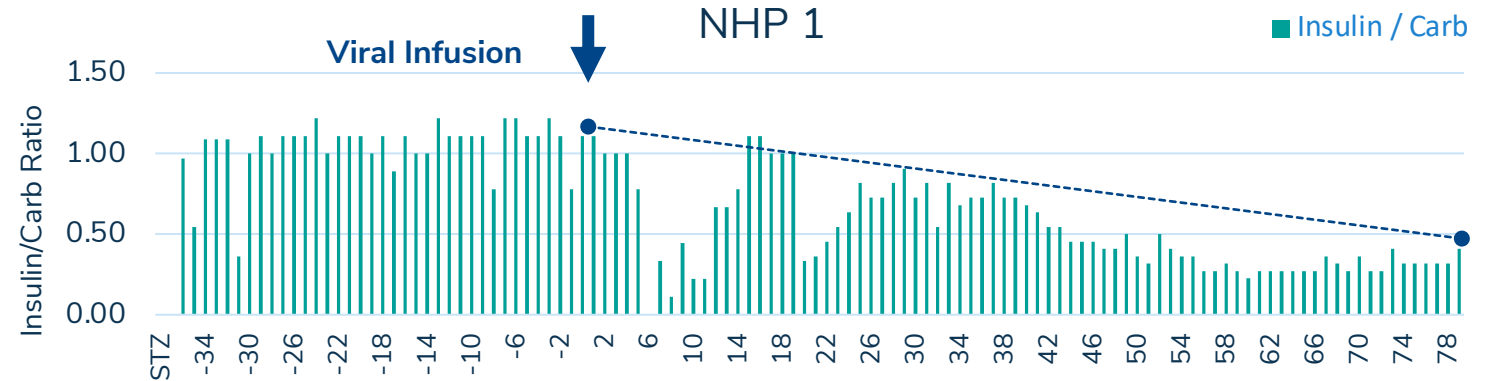
Provides a potential basis for further investigation in human Type 1 diabetes

- Human islets treated with streptozotocin to destroy beta-cells, then treated with either AAV-PM or AAV-GFP
- AAV treated islets then transplanted into hyperglycemic NOD/SCID mice, treated with alloxan to destroy beta cells
- NOD/SCID mice receiving AAV-PM islets had significantly lower blood glucose levels and significantly higher beta cell mass than those receiving AAV-GFP islets
- These data suggest that the **AAV-PM treatment can convert human alpha cells into human beta-like cells that secrete insulin**



GFP = Green Fluorescent Protein (fluorescent marker) | PM = Pdx1 + MafA

Non-Human Primate Model of T1D Reduced Insulin Requirements



NHP = Non-Human Primate

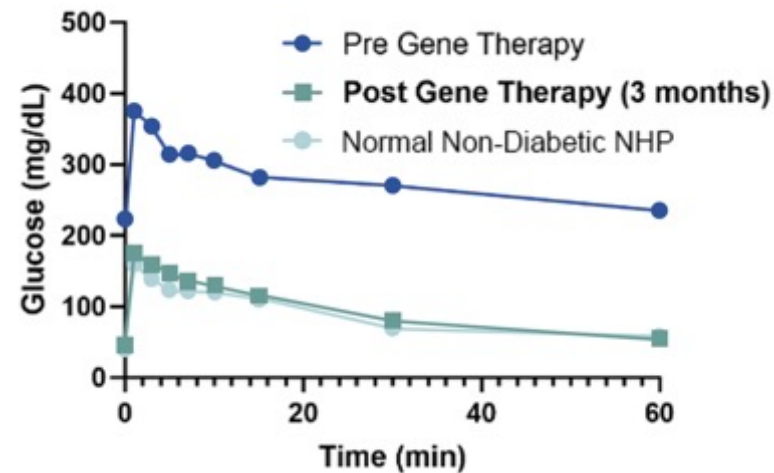
Preliminary data from University of Pittsburgh researchers show a marked reduction in insulin requirements.

ATTD 23: Statistically Significant Decreases In Insulin Requirements in NHPs

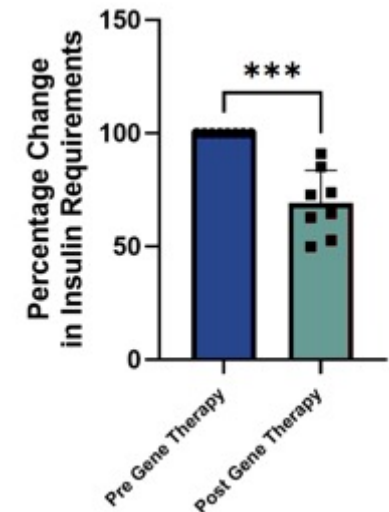
Following the pancreatic intraductal infusion of the AAV engineered construct, the eight NHPs had:

- Decreased insulin requirements ($p < 0.001$)
- Increased c-peptide levels ($p < 0.05$)
- Improved glucose tolerance compared to baseline ($p < 0.05$)
 - One NHP had normal glucose tolerance three months post-gene therapy
- The presence of more insulin-positive cells compared to non-treated diabetic controls based on immunohistochemistry (IHC)

Average Insulin Requirements

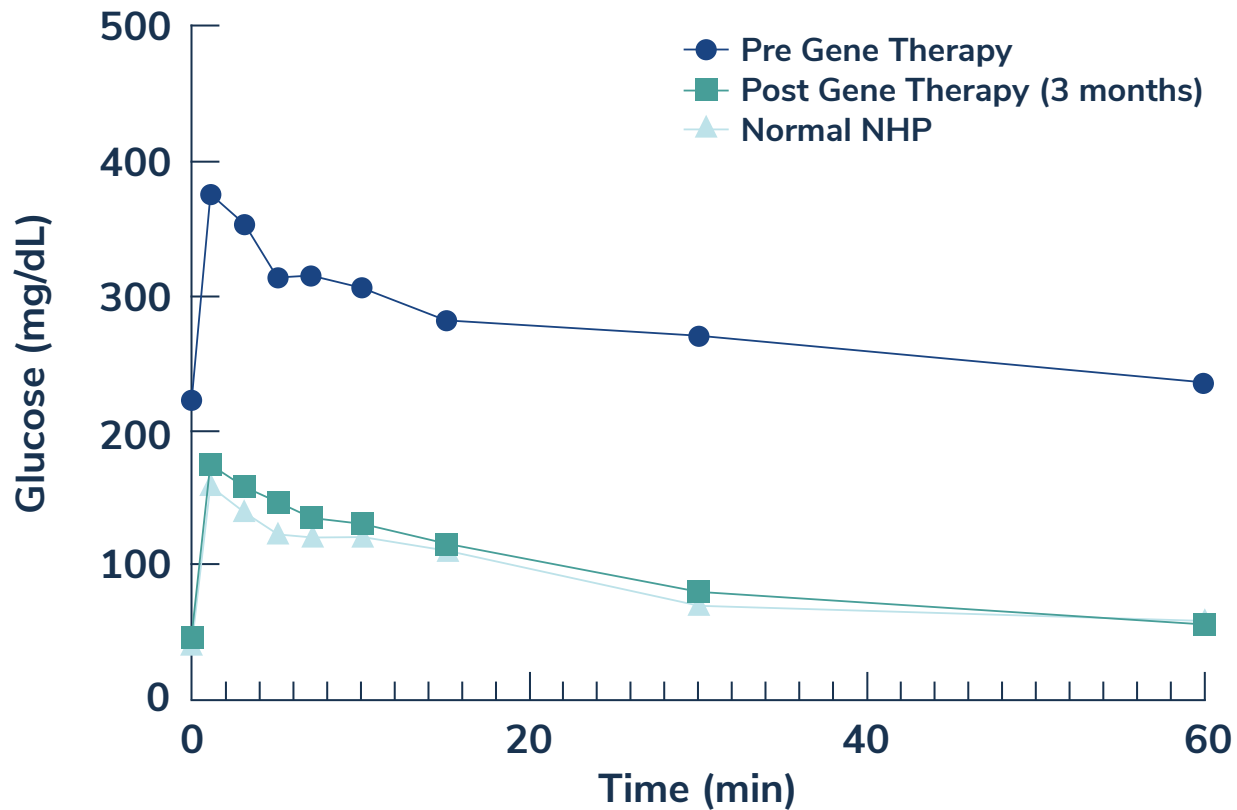


NHP2 Recent Glucose Tolerance Test



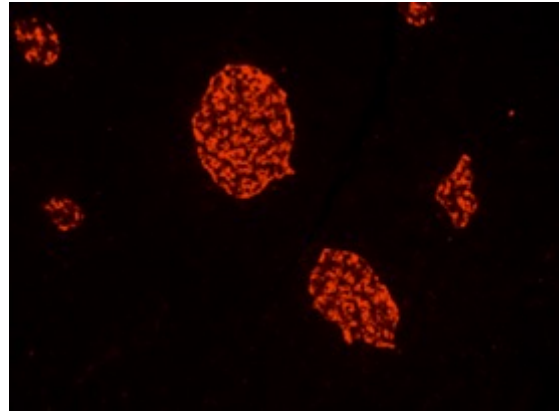
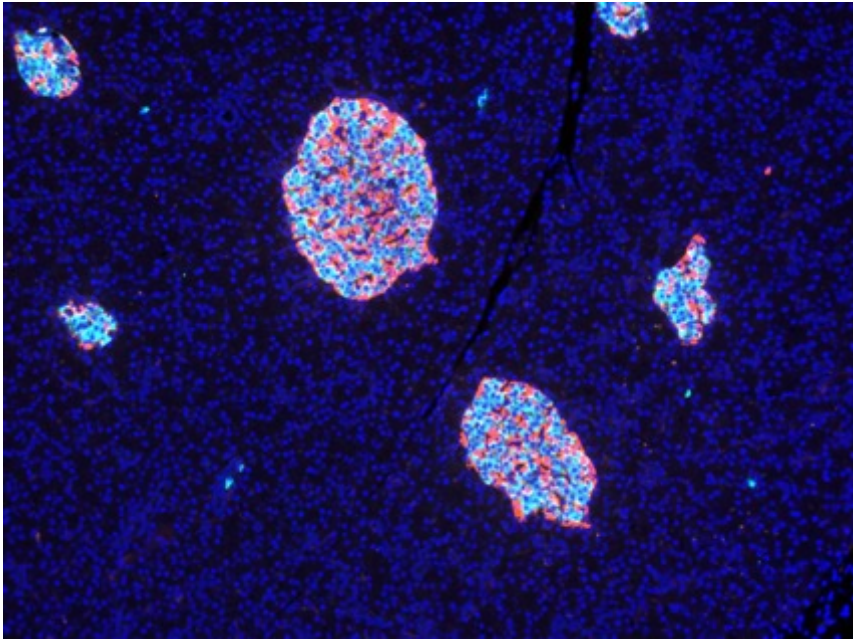
NHP2

Three-Month Glucose Tolerance Test

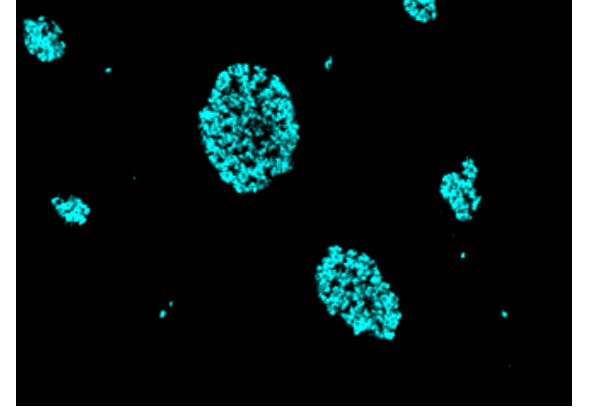


IHC

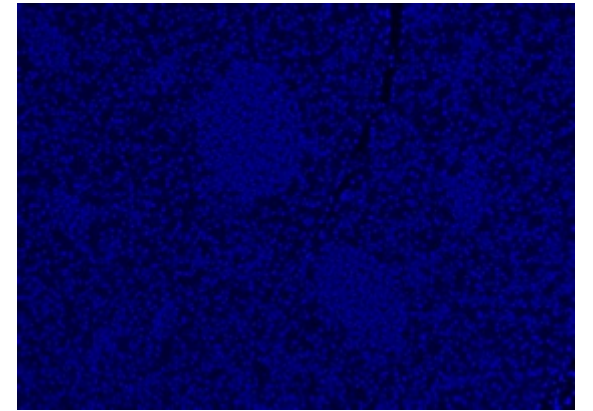
In a Normal NHP



GLUCAGON



INSULIN

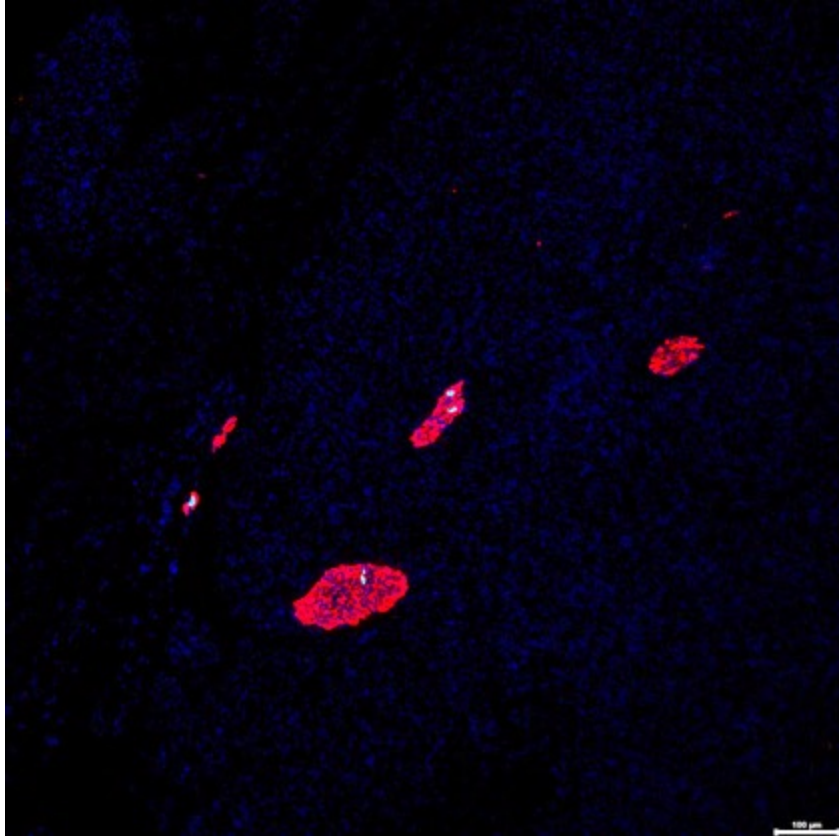


DAPI

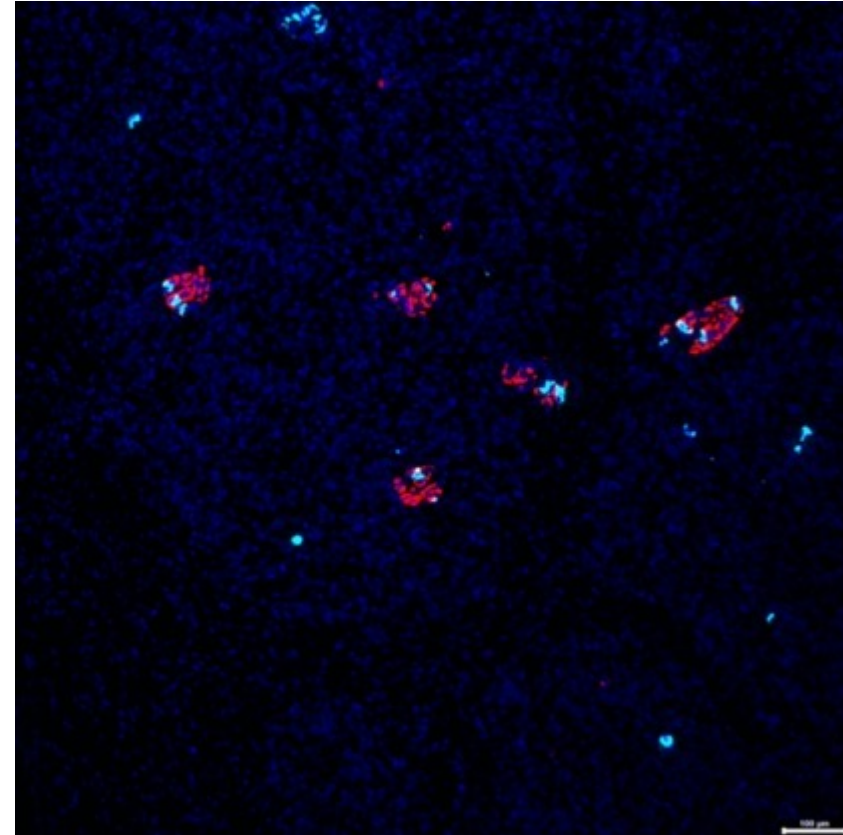
IHC Eight Weeks After Gene Therapy

Need at least 20% of normal beta cell mass to
maintain normoglycemia

DAPI INSULIN GLUCAGON



Diabetic NHP Without Gene Therapy



Diabetic NHP After Gene Therapy



CORPORATE



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Chief Executive Officer

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Former owner of securities broker dealer firm



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Chairman of the Board

30+ years of expertise in corporate law, including corporate governance, in biotech industry

Former owner of securities broker dealer firm



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Chief Executive Officer, Longnecker & Associates

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Member of the National Academy of Sciences and its Institute of Medicine; Professor of Biomedical Sciences, Yale University; Chairman of the Department of Cell Biology, Yale School of Medicine; Director of the Nanobiology Institute, Yale West Campus

Achievements & Upcoming Milestones

Acclaim · 1

- ✓ Present full data from Phase 1 portion of the trial at the 2023 AACR-NCBEORTC meeting
- ✓ Open for enrollment in Phase 2a Expansion portion of the trial in Jan. 2024
- Complete enrollment of 19 patients in each cohort of Phase 2a Expansion portion of the trial by end of year 2024

Acclaim · 2

- Complete enrollment for Phase 1 Dose Escalation portion in 2H 2024

Acclaim · 3

- ✓ Open for enrollment in Phase 1 Dose Escalation portion of the trial in Jan. 2024
- Complete Phase 1 Dose Escalation by 2H 2024
- Start the Phase 2 Expansion portion of the trial in 2H 2024

GPX-002 & GPX-003

- ✓ Report data from ongoing preclinical studies in 1H 2023
- ✓ Finalize AAV constructs in 2023
- ✓ Request to meet with FDA by the end of 2023*
- Poised for FDA guidance on IND-enabling studies in 2024

Corporate

- ✓ Expand global IP portfolio (Ongoing)
- ✓ Engage KOLs in discussions on our oncology and diabetes programs
- Expect collaborators to present preclinical data at the April 2024 AACR meeting

**As a result of the FDA's response, the Company will continue with its planned additional nonclinical studies in 2024 before requesting regulatory guidance for the IND-enabling studies*

We believe in a future of
transformational patient care.

21st Century
Gene
Therapies

Large
Markets &
Unmet Need

Combination
Trials with Top
Selling Drugs

Three FDA
Fast Track
Designations

Exploring New
Indications &
Partnerships



Research References

Slide 12

1. Selective and Preferential Cancer Cell Uptake and Anti-Cancer Activity in Advanced Lung Cancer Patients. Lu et al. PLoS1 (2012).

Slide 13

1. American Cancer Society (2023): <https://bit.ly/3SPcMRi>
2. World Health Organization (2022): <https://bit.ly/3fLGVSQ>
3. Fortune Business Insights: <https://bit.ly/3Ewbnup>
4. American Lung Association (2023): <https://bit.ly/47VkKzr>

Slide 14

1. British Journal of Cancer: <https://bit.ly/3FjaOTF>
2. Frontiers in Oncology: <https://bit.ly/3VOvdqO>
3. Translational Lung Cancer Research: <https://bit.ly/3YbNY8T>

Slide 29

1. Centers for Disease Control: <https://bit.ly/3Vk2gmL>
2. International Diabetes Federation: <https://bit.ly/3SXWohu>
3. Research and Markets: <https://bwnnews.pr/3SS5VGU>
4. Global Data: <https://bit.ly/3RSw1bf>

Slide 30

1. IDF Diabetes Atlas: <https://bit.ly/3vtEUv>

Slide 31

1. Centers for Disease Control: <https://www.cdc.gov/diabetes/managing/problems.html>
2. Amputation Prevention Centers of America: <https://www.apcofamerica.com/diabetic-amputation-neuropathy/>
3. National Institutes of Health: <https://www.niddk.nih.gov/health-information/diabetes/overview/preventing-problems/gum-disease-dental-problems>

Research References

Slide 32

1. American Diabetes Association: <https://diabetes.org/blog/history-wonderful-thing-we-call-insulin>

Slide 33

1. Centers for Disease Control: <https://www.cdc.gov/diabetes/data/statistics-report/index.html>

Slides 34-37

1. Xiao X, Guo P, Shiota C, et al. Endogenous Reprogramming of Alpha Cells into Beta Cells, Induced by Viral Gene Therapy, Reverses Autoimmune Diabetes. Cell Stem Cell. 2018;22(1):78-90.e4. doi:10.1016/j.stem.2017.11.020.

Slide 38

1. Gittes, G. Gene Therapy for Diabetes and Chemical Pancreatectomy for Pancreatitis [video]. YouTube. <https://www.youtube.com/watch?v=cL0tpXvxBqw>
Published July 20, 2022. Accessed October 14, 2022.

Slide 57 (Appendix)

1. Phase I Clinical Trial of Systemically Administered TUSC2(FUS1)-Nanoparticles Mediating Functional Gene Transfer in Humans
Lu C, Stewart DJ, Lee JJ, Ji L, Ramesh R, et al. (2012) Phase I Clinical Trial of Systemically Administered TUSC2(FUS1)-Nanoparticles Mediating Functional Gene Transfer in Humans. PLOS ONE 7(4): e34833. <https://doi.org/10.1371/journal.pone.0034833>.



APPENDIX

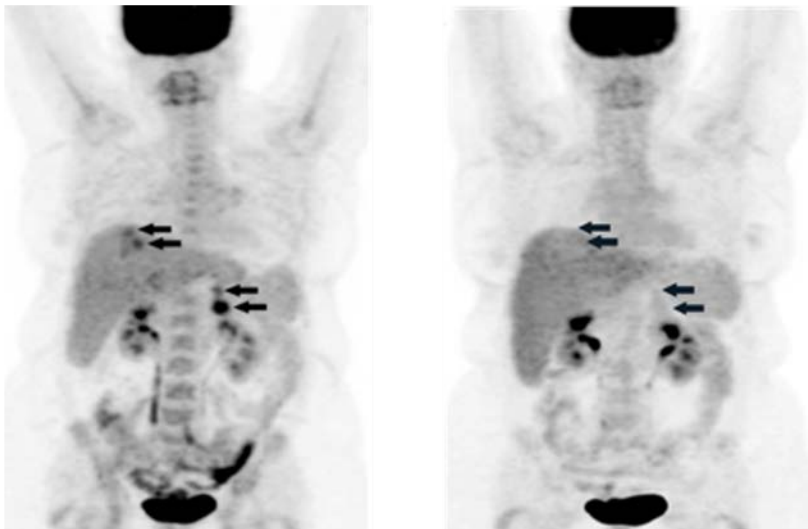


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Metabolic responses in late-stage metastatic lung cancer patient

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REQORSA + TARCEVA (ONC-002)

Ph 2 data in subjects with or without EGFR mutations

BEST OVERALL RESPONSE	NUMBER OF CYCLES	EGFR MUTATION STATUS	PRIOR THERAPY	PRIOR LINES OF THERAPY
CR	11 cycles	Positive (exon 18+20)	Chemo	3
SD 24% Regression target lesion	6 cycles	Unknown	Chemo/anti-PD1	2
SD 30% Regression one target Lesion 17% Regression all target lesions	8 cycles	Negative	Chemo/anti-PD1	6
SD	4 cycles	Positive (exon 21)	Erlotinib (10 cycles)/Chemo	3
SD	4 cycles	Positive (exon 21)	Erlotinib (12 cycles)	2
SD	4 cycles	Negative	Chemo	2
SD	4 cycles	Unknown	Chemo	4