Genprex®

Pioneering Gene Therapies for Patients in Need

April 2026

www.genprex.com | NASDAQ: GNPX

Forward-Looking Statements

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 effects of any R&D prioritization initiatives or any other strategic alternatives or other efforts we make take in the future, 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 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, 2025 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 presentation 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.

Our Mission

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

Genprex

Program Highlights

ONCOLOGY

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

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
- Received FDA guidance in Q1 2026 on toxicology studies

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® DELIVERY SYSTEM (NON-VIRAL AND SYSTEMIC)	REQORSA® GENE THERAPY	NSCLC	Acclaim·1 (ONC-003)	Fast Track Designation	REQORSA® + Tagrisso					
		REQORSA® GENE THERAPY	SCLC	Acclaim·3 (ONC-005)	Fast Track, Orphan Drug Designation	REQORSA® + Tecentriq					
		REQORSA® GENE THERAPY	Ras Inhibitor Resistant Lung Cancer	—							
		REQORSA® GENE THERAPY	ALK-EML4 Positive Translocated Lung Cancer	—							
		REQORSA® GENE THERAPY	Mesothelioma	—							
		OTHER ONCOLOGY TARGETS	—	—							
DIABETES	AAV Vector	GPX-002	T1D	DIA-001							
		GPX-002	T2D	DIA-002							
		OTHER DIABETES TECHNOLOGIES	—	—							

ONCOLOGY

REPROGRAMMING THE COURSE OF CANCER

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Why TUSC2?

Discovery

- Tumor Suppressor Candidate 2
- Chromosome 3p21.3 deleted gene
- Previously called FUS1
- NPRL2 is in the same area of the chromosome

Tumor Suppressor Gene

- TUSC2 restoration in cancer cells in vitro inhibits cell growth and induces apoptosis
- TUSC2 is encoded by nuclear DNA but TUSC2 protein is located in inner membrane of mitochondria
- Plays a key role in mitochondrial Ca2+ regulation
- Plays a key role in mitochondrial energy metabolism
  - TUSC2 restoration decreases glycolysis
  - Decreases glucose uptake by cancer cells

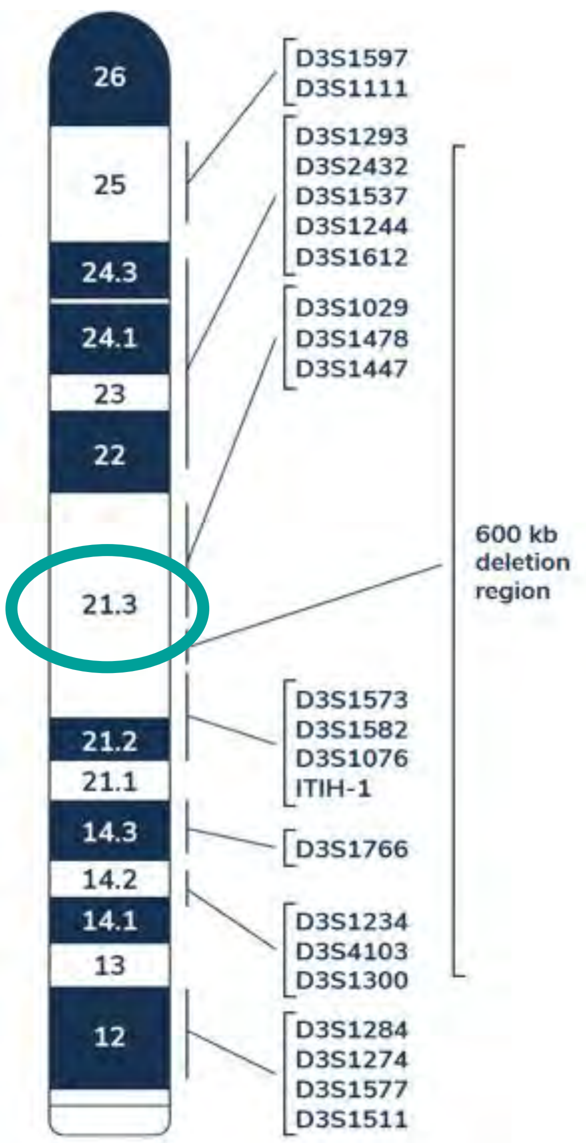

Illustration of Part of Short Arm of Chromosome 3

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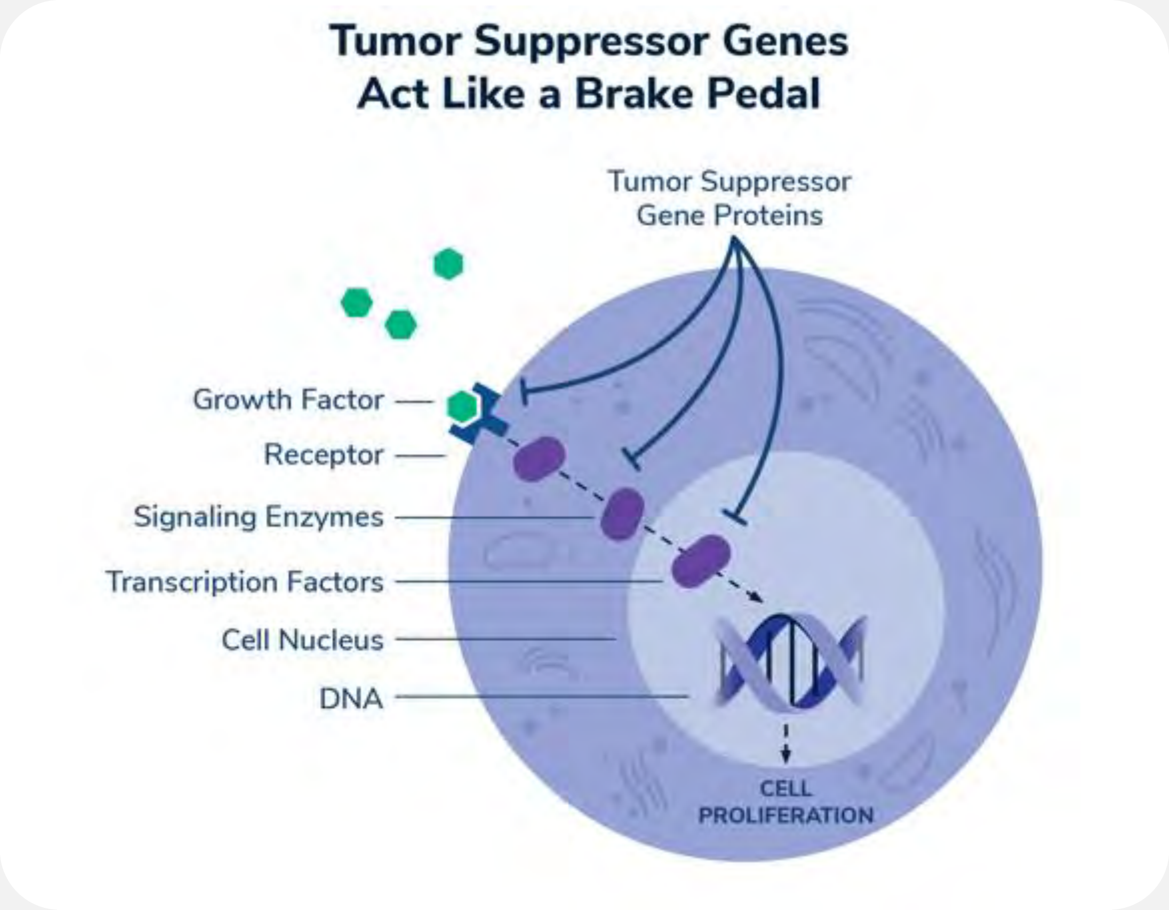
Tumor Suppressor Genes Deleted During Cancer Development

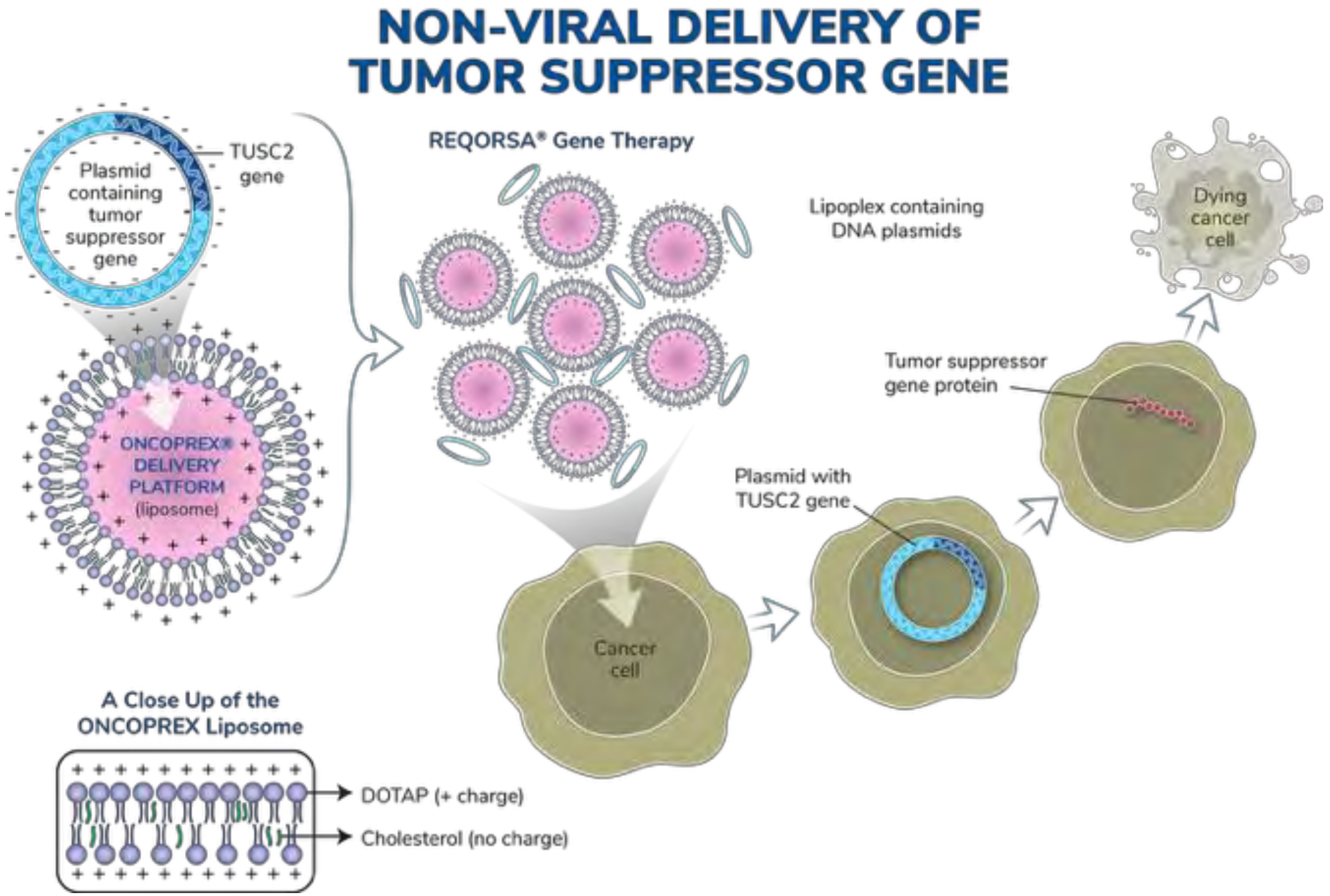
- Tumor suppressor genes are deleted early during cancer development
- 82% of all non-small cell lung cancers and 100% of all small-cell lung cancers express decreased amounts of TUSC2 tumor suppressor protein
- Loss or reduction of TUSC2 expression is associated with significantly reduced overall survival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



Genprex®

Oncoprex® Delivery System

Non-viral, positively-charged lipid-based nanoparticle in a lipoplex form is **systemically delivered.**



Cationic lipoplex carries drug to tumors.

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.

1

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.

2

Non-Viral Lipid Nanoparticles in a Lipoplex

The gene expressing DNA plasmid is then encapsulated into our Oncoprex® Delivery System, which consists of non-viral lipid-based nanoparticles in a lipoplex form.

3

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® Delivery System

Genes

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

Synergies

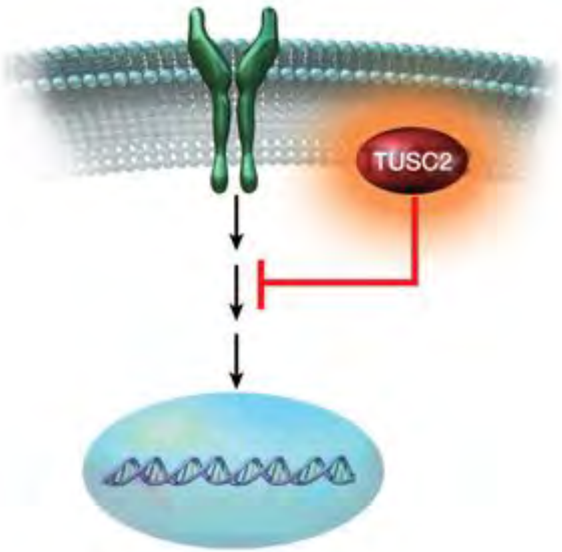
Can be used in combination with other cancer therapies such as Tagrisso® and Tecentriq®

Cancers

Data indicate it could combat mesothelioma, NSCLC with Ras mutations and NSCLC with ALK rearrangements

Reqorsa® Targets Cancer At Its Core

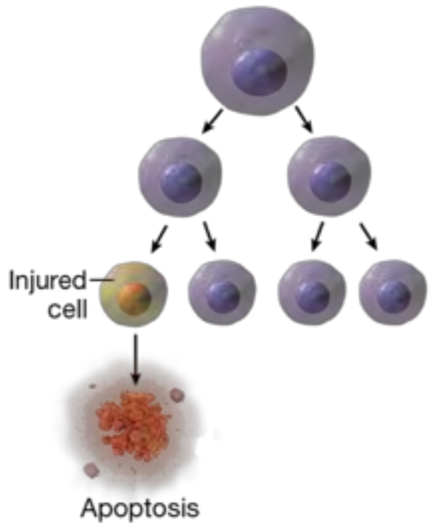
Multiple anti-cancer mechanisms of action.



1

Controls Cell Signaling

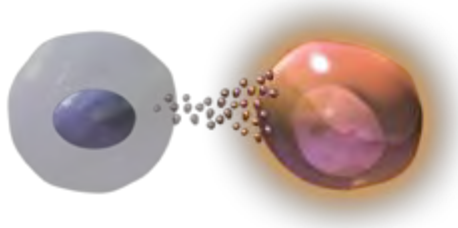
Tyrosine kinase inhibition decreases cancer cell proliferation



2

Stimulates Apoptotic Pathways

Leads to programmed cancer cell death



3

Modulates Immune Response

Promotes immune activity against cancer

Reqorsa® Reduces Glycolysis in Cancer Cells

REQORSA Reverses Fundamental Characteristic of Cancer

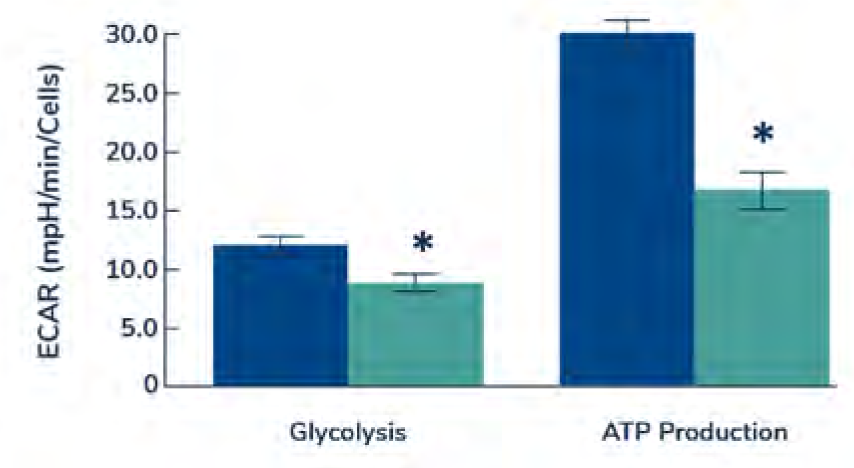
Cancers are detected by PET scans

- PET scanning is based on increased glucose uptake in cancer cells
- Due to high rate of glycolysis in cancer cells
- Increased glycolysis is found in virtually all cancers.

A549 cells are a NSCLC line virtually lacking TUSC2.

Transfected with TUSC2 gene

- Decreased glycolysis
- Decreases glucose uptake
- Leads to decreased ATP production
- Starves cancer cell of energy
- May lead to negative PET scans



* indicates $p<0.05$

Lung Cancer: By the Numbers

124K

2M+

$104B

229K

28%

U.S. Annual Mortality

More than 124,000 deaths per year in the U.S[1].

Global Lung Cancer Incidence

More than two million new cases per year worldwide[2].

Global Market

Global market is projected to grow from $18.3 billion in 2018 to $104 billion by 2032[3].

U.S. Lung Cancer Incidence

More than 229,000 new cases per year in the U.S[1].

U.S. Average Lung Cancer Survival Rate

The lung cancer five-year survival rate in the U.S. is 28%[4].

Reqorsa® Monotherapy

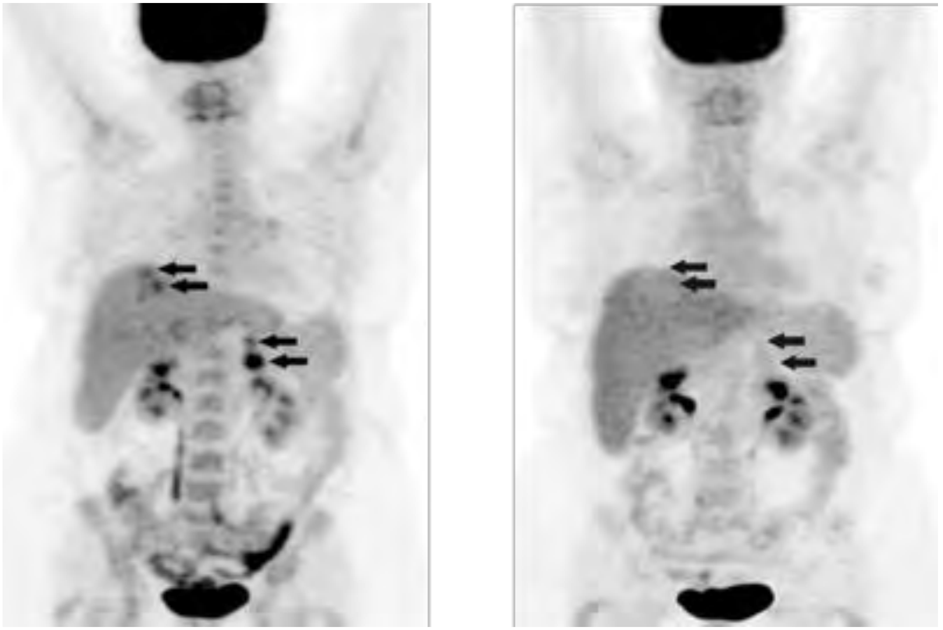
ONC-001 Trial

DOSE ESCALATION STUDY

Explored toxicity and tolerability in patients.

Phase 1 monotherapy results:

- 31 Stage IV lung cancer patients
- 0.01 – 0.09 mg/kg
- 23 patients evaluable
  - 5 patients had stable disease
  - 2 patients had tumor shrinkage
- Generally well-tolerated



Metabolic responses in late-stage metastatic lung cancer patient

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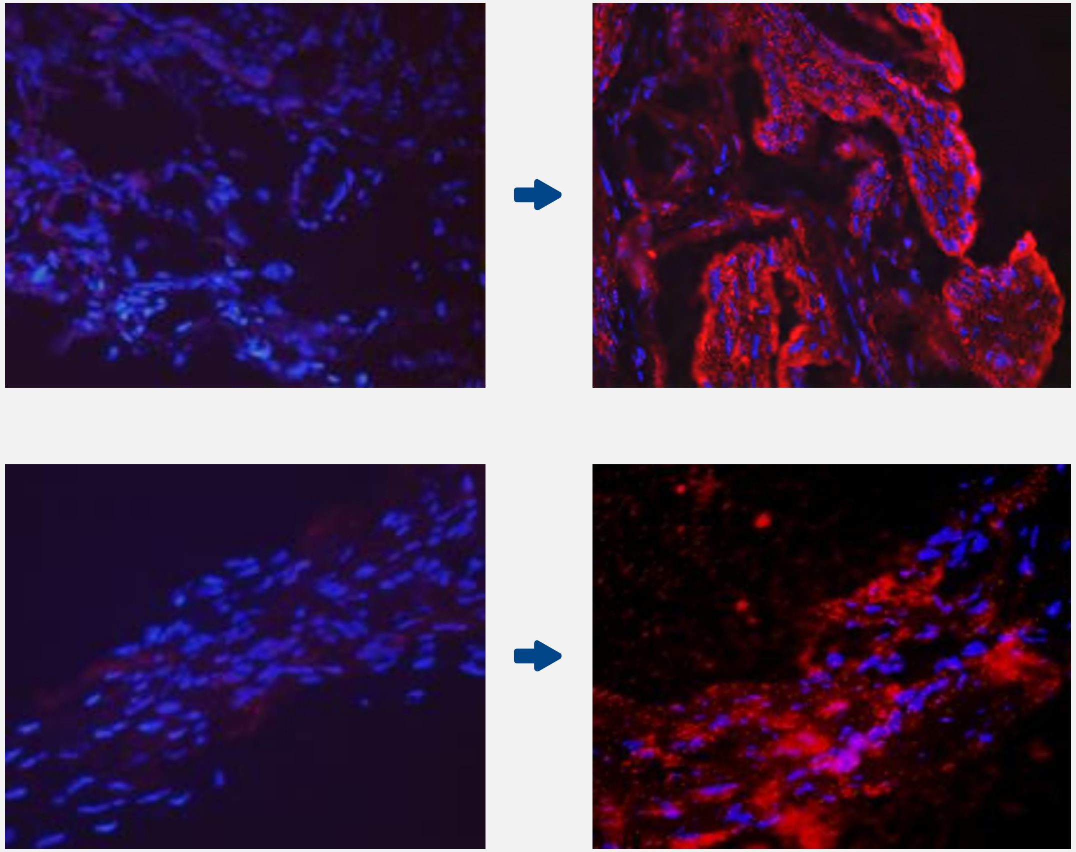
Selective Uptake of Reqorsa®

REQORSA Targets Cancer Cells

Pretreatment Biopsies | **Posttreatment Biopsies**



Patient 1
(.02 mg/kg)

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.

Reqorsa® + Tarceva

ONC-002 Phase 2 Trial

Met Simon 2-stage criteria to enroll the full 39 subjects, but was discontinued to start study with Tagrisso.

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

Genprex®

For most patients, **drug resistance** to Tagrisso® and Tecentriq® **is inevitable.**[1,2,3]

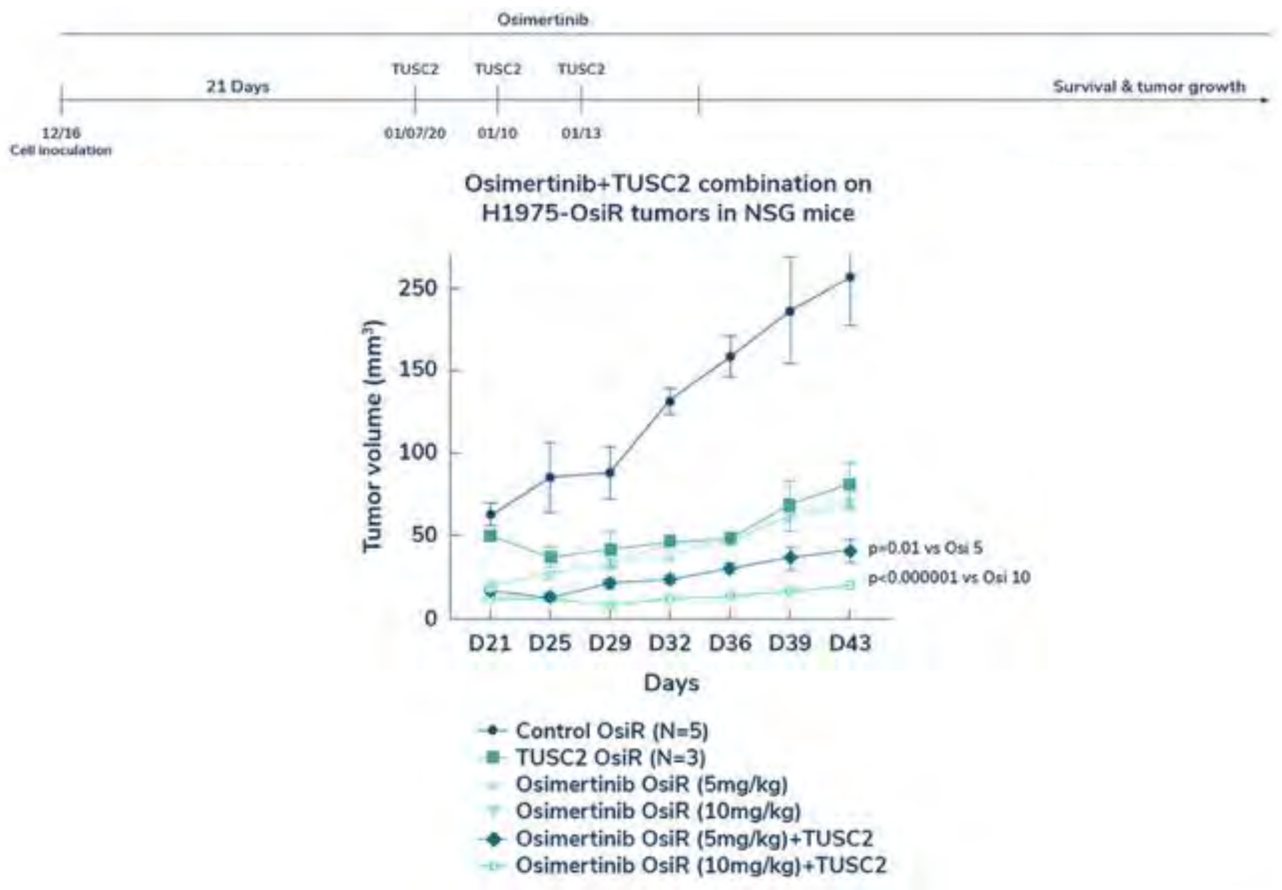
Our approach is designed to address drug resistance.

- 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.

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.



TUSC2 = Reqorsa
Osimertinib is the generic name for Tagrisso.

Data presented at AACR 2021.

- Patients with advanced, EGFR mutant NSCLC whose disease progressed after Tagrisso® monotherapy or Tagrisso® combination therapy
- FDA Fast Track Designation
- ~10-15 U.S. sites
- ~119 patients
    - Phase 1 Dose Escalation: 12 patients (completed)
    - Phase 2a Expansion: ~33 patients (**currently enrolling**)
    - Phase 2b: ~74 patients
- Phase 2a Expansion interim analysis at 19 patients
- 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

Genprex®

Phase 1 Dose Escalation

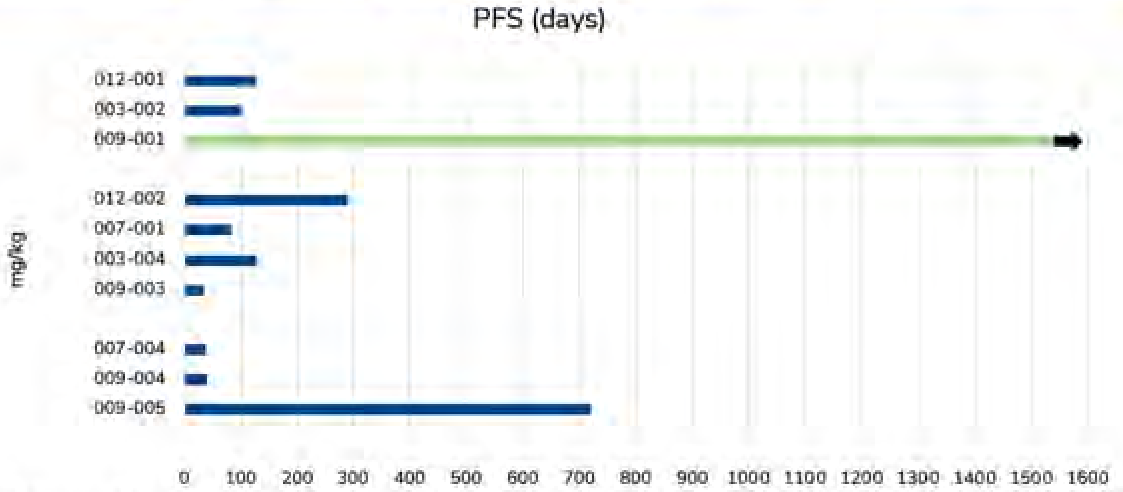
Excellent safety profile and efficacy in relapsed patients.

Enrollment and Dose Limiting Toxicities

	0.06 mg/kg	0.09 mg/kg	0.12 mg/kg	Total
# Patients	3	4◊	5^	12
M/F	0/3	2/2	1/4	3/9
Median Age (range)	59 (50-60)	51 (38-69)	59 (57-74)	59 (38-74)
DLTs	0	0	0	0

◊ 1 patient received quaratusugene ozeplasmid in 1st cycle but was excluded from RP2D assessment for reasons not related to DLT.
^ 1 patient withdrew and 1 lost to follow up before completing 1st cycle

Progression Free Survival by Dose Group



1 patient withdrew and 1 patient was lost to follow up before completing first cycle and therefore were not evaluable for PFS
➡ = Patient's study treatment ongoing
▮ = Patient with partial remission

Delayed Infusion-Related Reaction

- No symptoms during 30 min infusion
- Fever, chills, and muscle aches
- Symptoms generally start 3-6 hours after infusion
- Generally lasts 2-4 hours
- Prophylaxis with dexamethasone, acetaminophen, and diphenhydramine
- Attenuated with repeat dosing

3/12 progressing on Tagrisso containing regimens had prolonged PFS

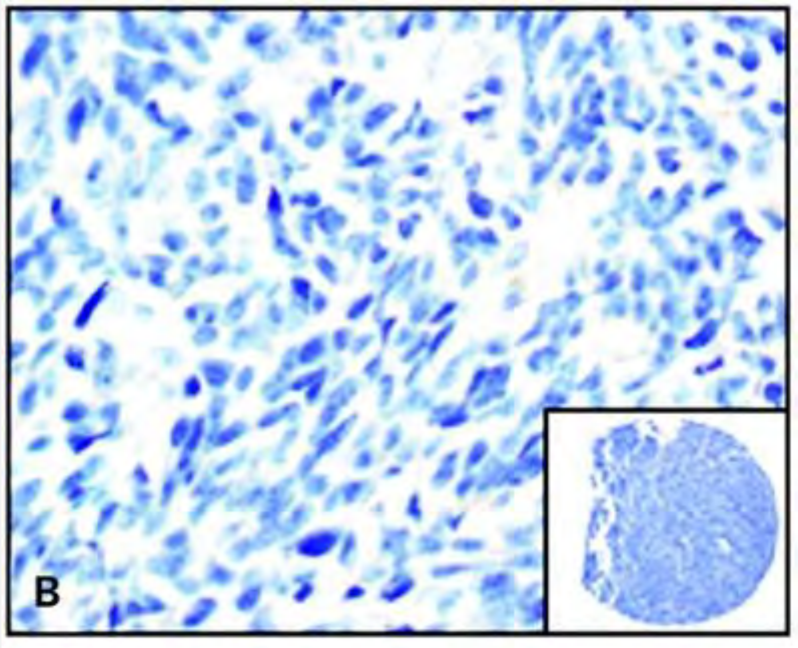
- 1 continuing treatment after 52 cycles (36 mos)
- 1 progressed after 32 cycles (24 mos)
- 1 progressed after 14 cycles (10 mos)

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			
			Lost (negative) *n* (%)	Reduced (low + intermediate) *n* (%)	Preserved (high) *n* (%)	*P* value, Fus1 levels
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)	

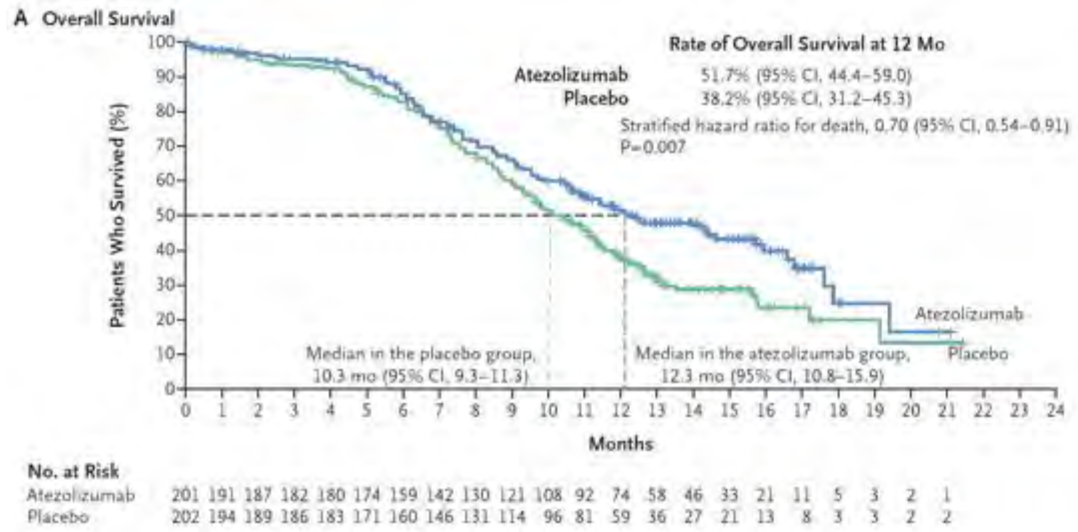
Genprex

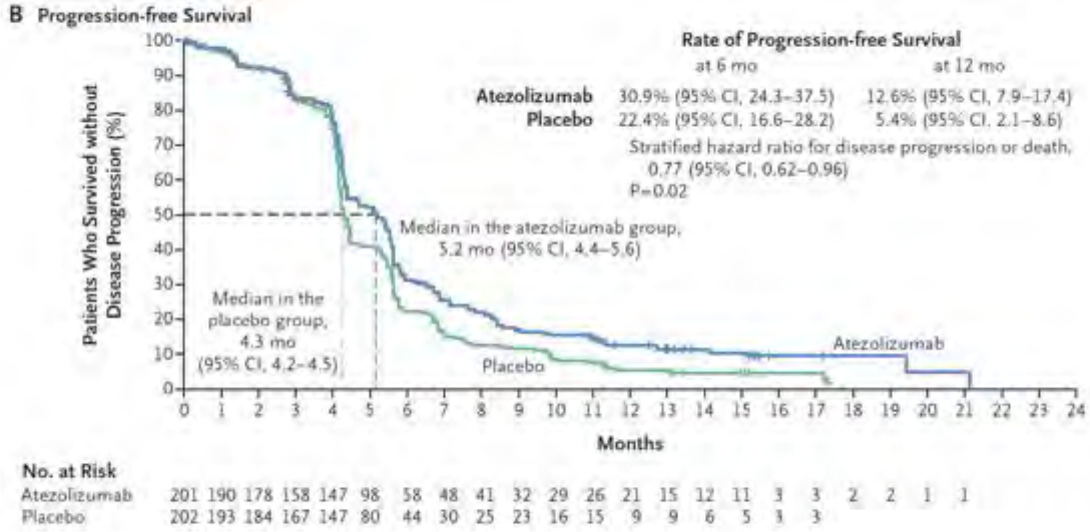
Tecentriq® (atezolizumab) SCLC Approval Trial

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)

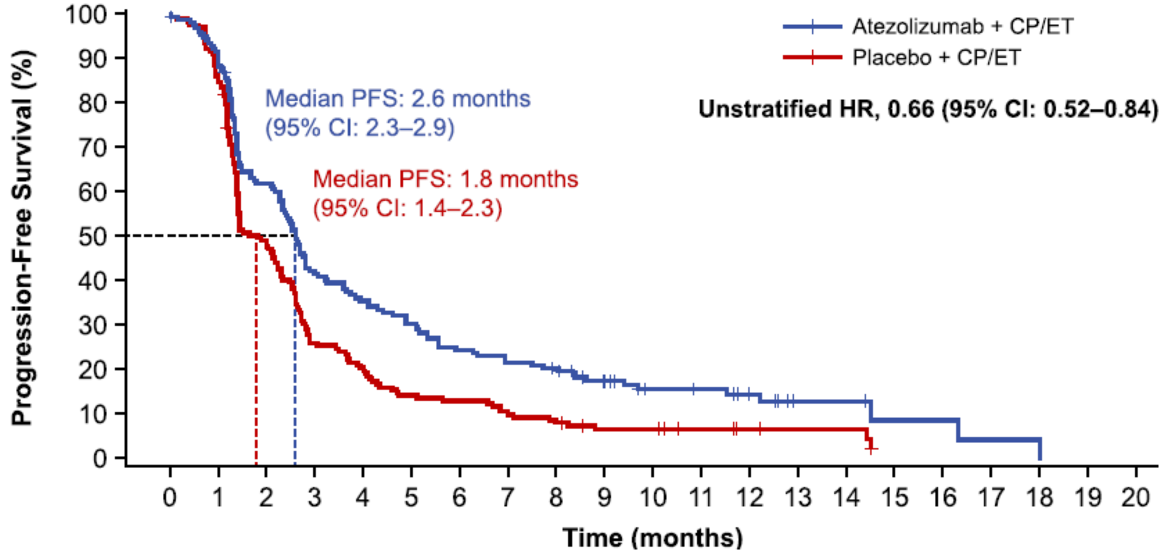




Atezolizumab Maintenance Therapy

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)

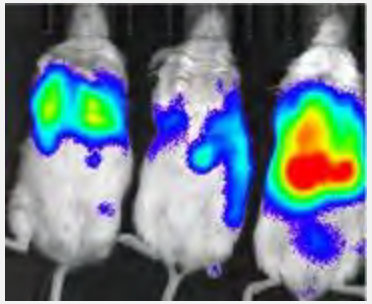


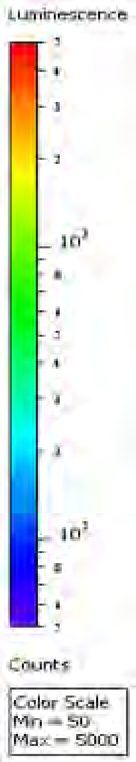
Atezolizumab is the generic name of Tecentriq.

Reqorsa® Adds Significantly to Tecentriq Treatment

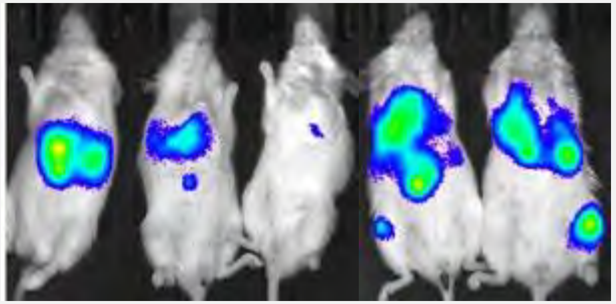
H841 SCLC cell xenografts in humanized mice

CONTROL

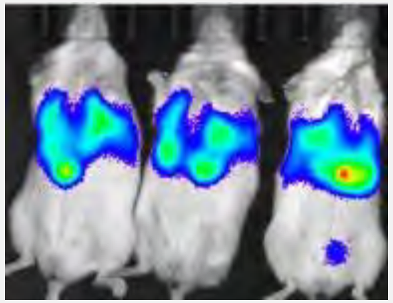




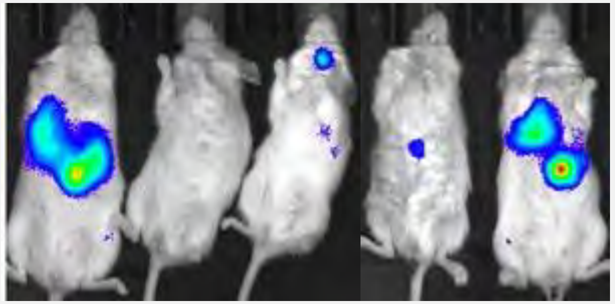
REQORSA

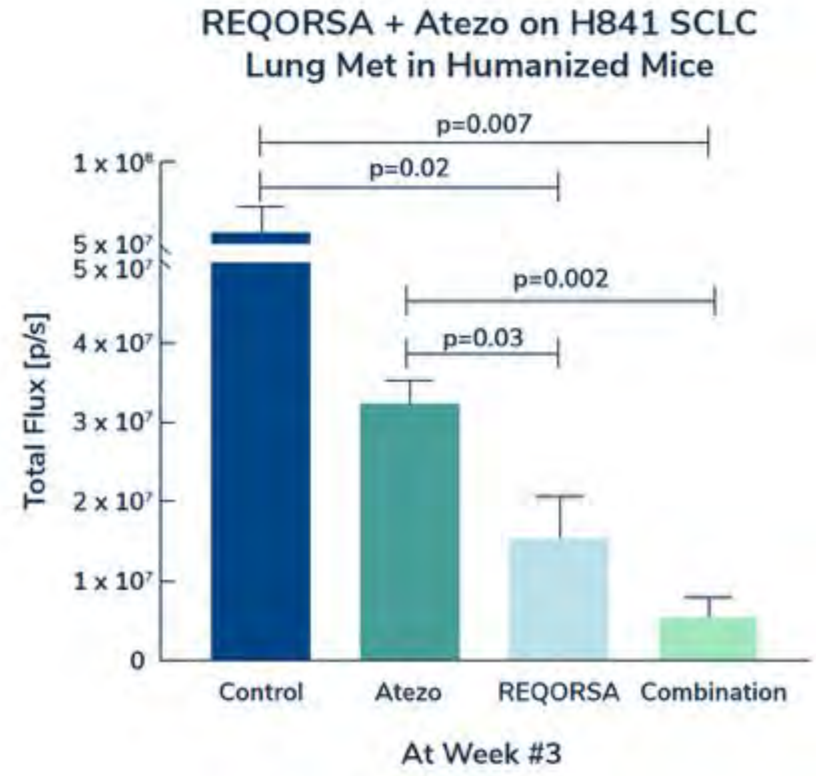


TECENTRIQ



REQORSA + TECENTRIQ

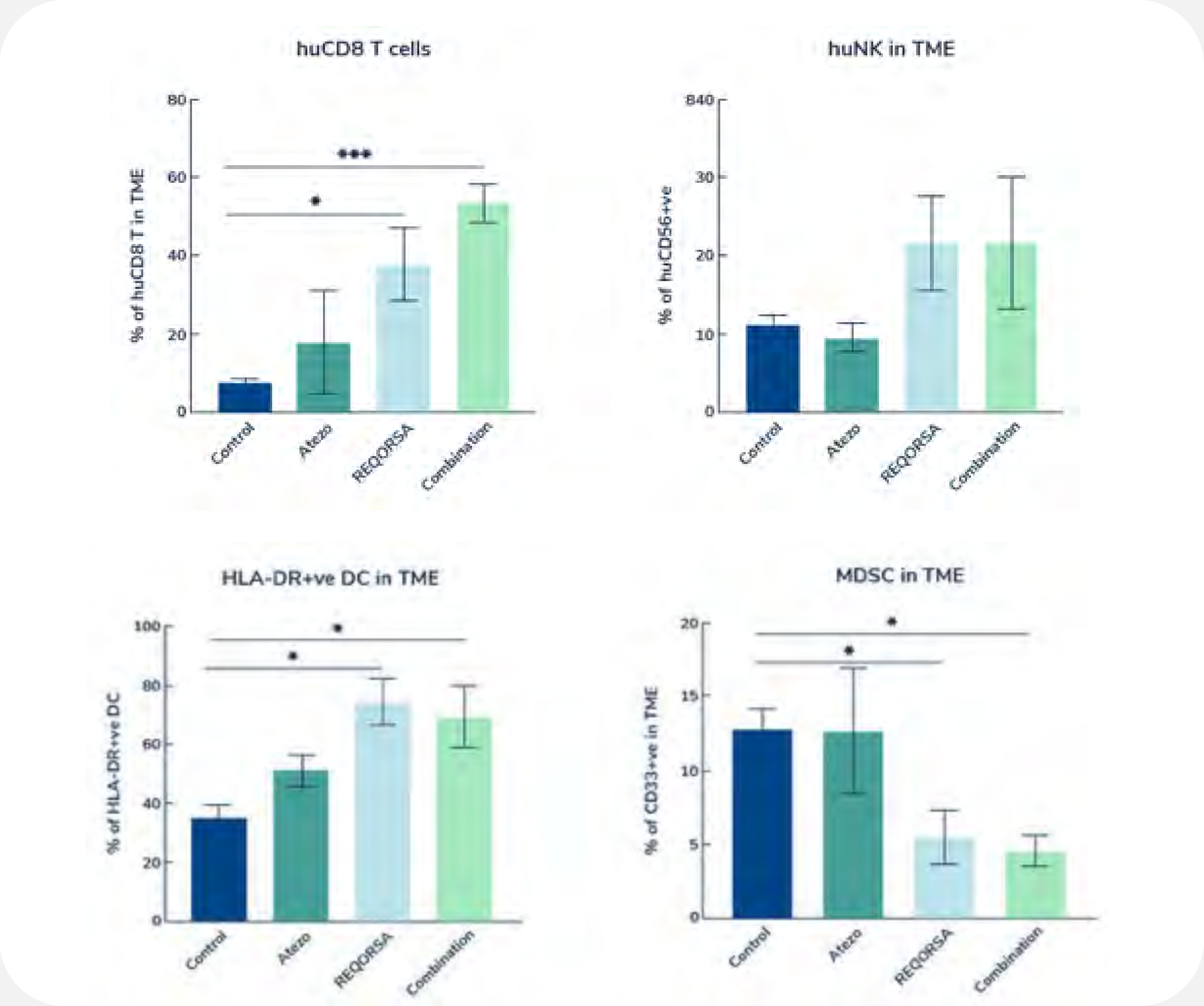




Atezolizumab is the generic name of Tecentriq.

Genprex®

Increased Immune Response with Reqorsa® and Tecentriq



Atezolizumab (Atezo) is the generic name of Tecentriq.
huNK = human natural killer cells
DC = dendritic cells
MDSC = myeloid derived suppressor cells
TME = tumor microenvironment

Genprex®

Acclaim-3

- Patients with ES-SCLC who did not develop tumor progression after receiving Tecentriq® and chemotherapy
- Fast Track Designation and Orphan Drug Designation
- ~10-15 U.S. sites
- ~62 patients
    - Phase 1 Dose Escalation: (completed)
    - Phase 2: ~50 patients (**currently enrolling**)
- Phase 2 interim analysis after 25th patient enrolled and treated reaches 18 weeks of follow up

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

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

Reqorsa® in Ras Inhibitor Resistant Lung Cancer

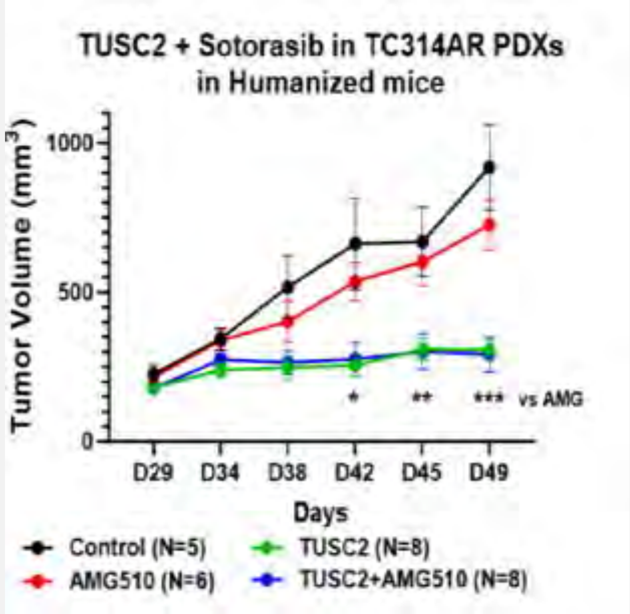
REQORSA effectively overcomes sotorasib AR in KRASG12Cmutant NSCLC mouse xenografts

Published Results:

- TUSC2 transfection significantly reduced colony formation in two Acquired Resistant (AR) cell lines
- Transfection of TUSC2 also markedly increased apoptosis in AR cells
- Treatment with REQORSA alone or in combination with sotorasib was highly effective in controlling H23AR tumor growth in mouse xenografts
- REQORSA alone also exhibited significantly strong antitumor effect on TC314AR patient-derived xenografts (PDXs) where sotorasib alone showed no significant antitumor activity
- A synergistic antitumor effect was observed when TC314AR PDX tumors were treated with the combination of REQORSA and sotorasib

Data presented at 2024 EROTC-NCI-AACR Symposium on Molecular Targets and Cancer Therapeutics.

- Researchers demonstrated that REQORSA, alone or in combination with sotorasib inhibited colony formation, induced apoptosis, and showed significant antitumor efficacy in KRASG12C mutant acquired resistant xenografts and in PDX tumor xenografts



Another clinical opportunity for **REQORSA**

Genprex

Reqorsa® in ALK-ELM4 Positive Translocated Lung Cancer

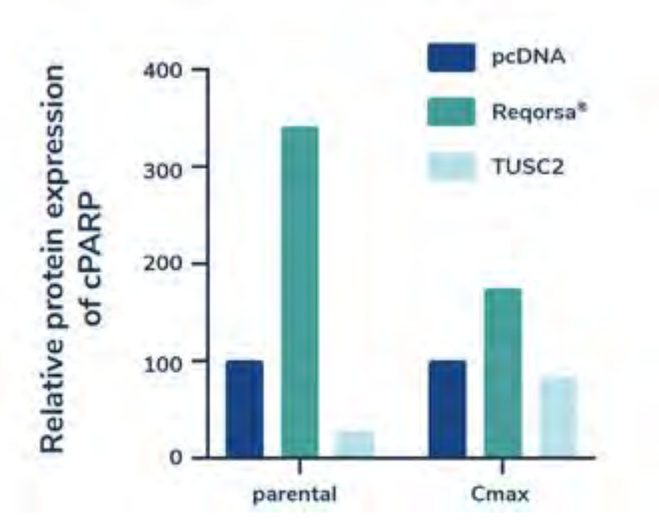
License Agreement and SRA with The University of Michigan Rogel Cancer Center and Collaboration with ALK-Positive Advocacy Group

Published Results:

- REQORSA induced apoptosis in alectinib resistant EML4-ALK positive NSCLC cell lines
- Researchers found that overexpressing TUSC2 using REQORSA treatment in ALK+ lung cancer cell lines inhibited the ability of the cells to form colonies
- REQORSA in ALK+ NSCLC cell lines was effective in decreasing cell growth and proliferation through the activation of apoptotic pathways

ALK-Positive Collaboration:

- Non-profit patient-driven research organization
- Dedicated to improving the life expectancy and quality of life for ALK+ lung cancer patients
- Both Genprex and ALK Positive share the cost of the Sponsored Research Agreement (SRA) with the University of Michigan Rogel Cancer Center



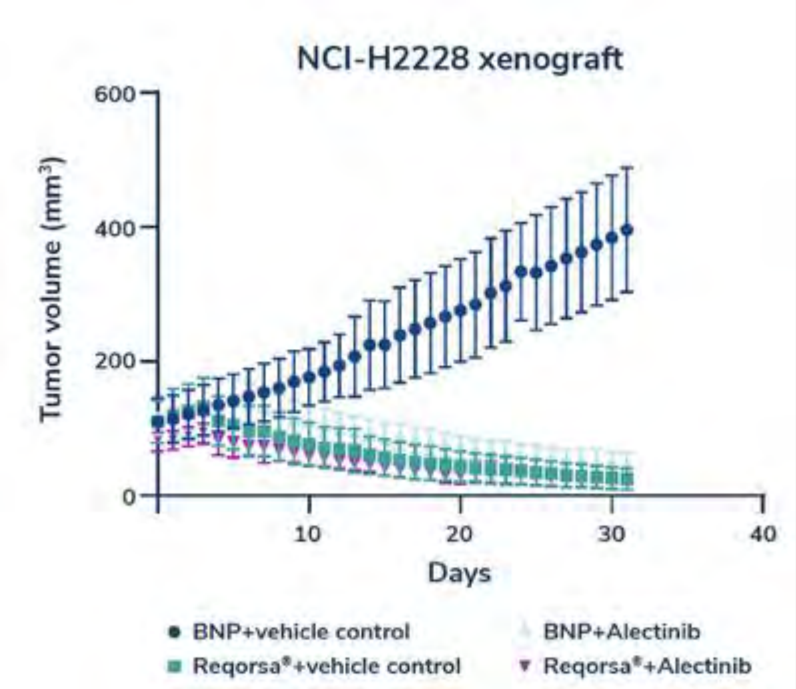
Another clinical opportunity for **REQORSA**

Data presented at AACR 2024.

Reqorsa® in ALK Xenograft Model

REQORSA markedly decreases growth of ALK-sensitive xenografts in mice
- Both as single agent and in combination with alectinib

Treatment	Tumor Shrinkage (%)
Reqorsa + vehicle control	78.8
BNP + alectinib	60.14
Reqorsa + alectinib	78.60



BNP = Blank Nano Particles
QO = Quaratusugene Ozeplasmid (REQORSA)
Alectinib is the generic name for Alecensa®

Data presented at AACR 2025.

Genprex®

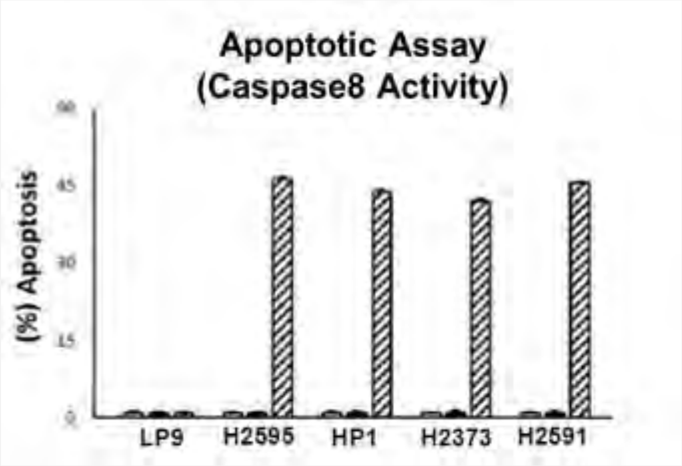
Reqorsa® in Mesothelioma

License Agreement and SRA with New York University Langone Health

Published Results:

- Researchers investigated whether TUSC2 transfection could modulate MPM aggressive properties
- Four MPM cell lines and tert-transformed mesothelial LP9 cells were treated with REQORSA and control liposomes for 48h. Treated cells were then evaluated for TUSC2 expression by semi quantitative RT-PCR, Western blot analysis, and functional assays including cell proliferation, invasion, and apoptosis.

- Researchers demonstrated that REQORSA treatment resulted in:
  - Significant decrease in cell proliferation, cell invasion, and a significant increase in cell apoptosis in all four MPM cell lines
  - Potent tumor suppressive activity of the TUSC2 gene delivered by REQORSA



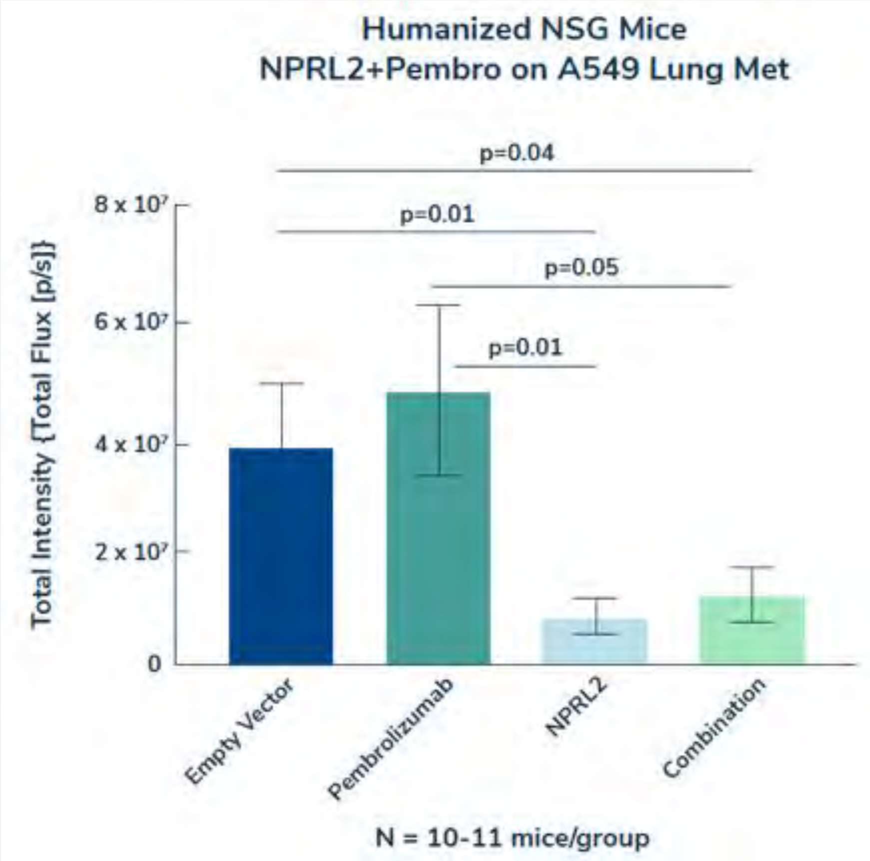
Another clinical opportunity for **REQORSA**

Data presented at 2024 EROTC-NCI-AACR Symposium on Molecular Targets and Cancer Therapeutics.

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



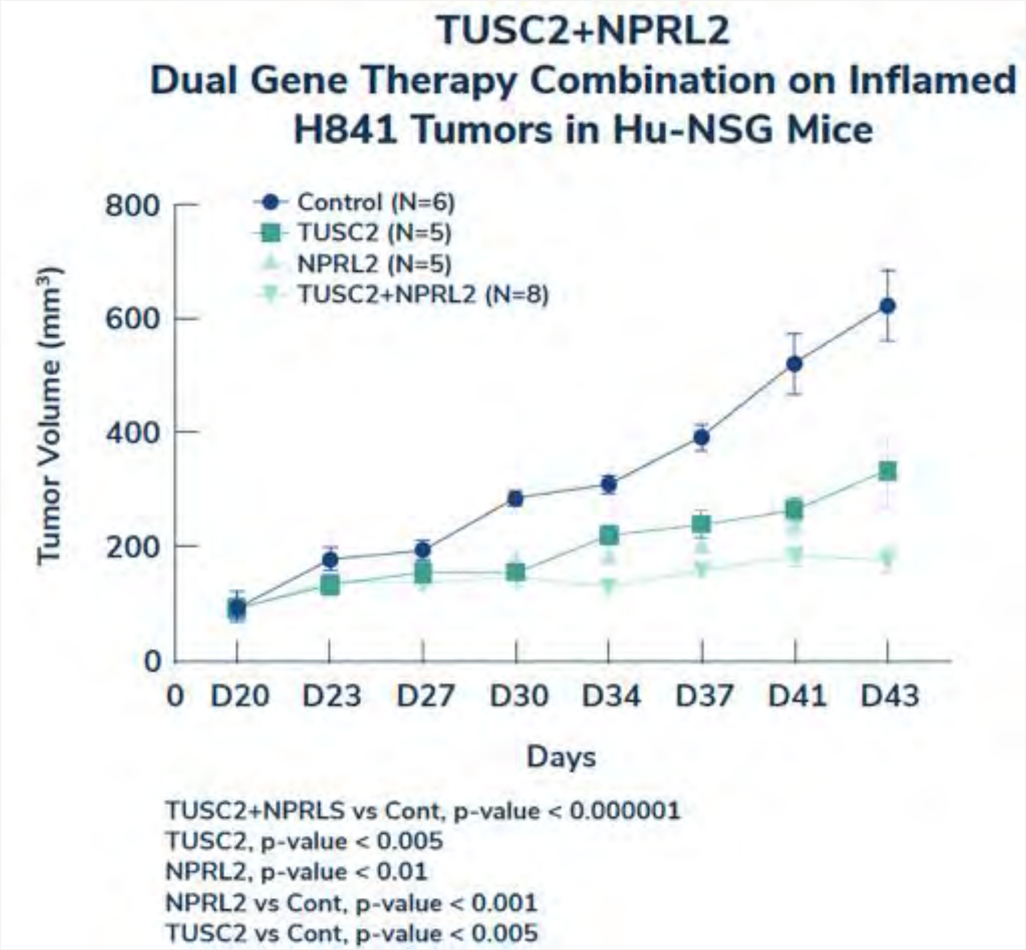
Provides preclinical validation of the ONCOPREX Delivery System, **which may provide a multitude of potential pipeline opportunities beyond lung cancer.**

Combined TUSC2 and NPRL2 Re-Expression in SCLC

H841 lacks both TUSC2 and NPRL2 protein

Increased control of xenograft growth compared to:

- Control
- TUSC2 re-expression alone
- NPRL2 re-expression alone




DIABETES
www.genprex.com

Diabetes: By the Numbers

40M

Diabetes U.S. Prevalence[1]

115M U.S. adults have prediabetes.

589M

Diabetes Global Prevalence[2]

Expected to rise to 853M by 2050.

$1T

Global Diabetes Expenditures[3]

Diabetes caused at least USD 1 trillion dollars in health expenditure – a 338% increase over the last 17 years.

$43B

T2D Global Market (7.05% CAGR[4])

$79B expected U.S. market sales in 2035.

$37B

T1D Global Market (17.2% CAGR[5])

$70B expected U.S. market sales by 2035.

Diabetes can cause serious complications.

In 2024, there was approximately **1 death every 9 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.

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.

Chronic Kidney Disease

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

Hearing Loss

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

Nerve Damage

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

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.

Foot Health (Diabetic Neuropathy)

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

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

Potential for disease modification **for long-term effectiveness.**

1901
Islets of Langerhans produce insulin and the destruction of these cells resulted in diabetes

1922
Insulin first introduced to Type 1 diabetes patient

1978
Recombinant DNA techniques produce synthetic "human" insulin

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

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.

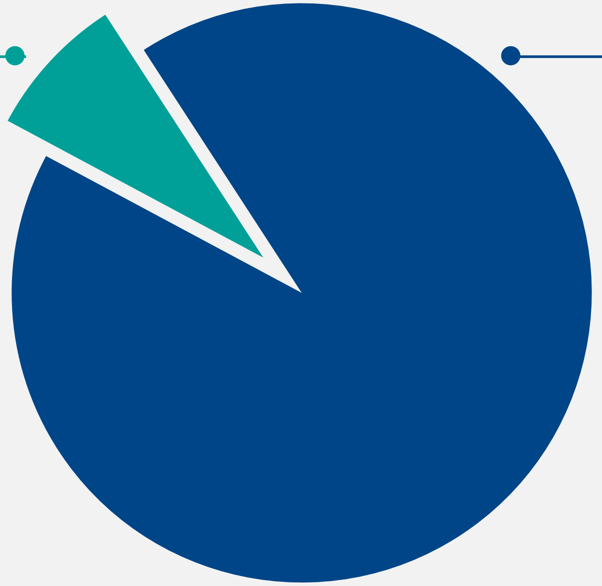
Novel Gene Therapy Diabetes Program

License Agreement and SRA with University of Pittsburgh **intends to address both T1D and T2D.**

38M or 11.6% of Americans Have Diabetes[1]

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.

Genprex is positioned as an **innovator in emerging diabetes therapies.**

GPX-002 Replenishes Insulin Producing Cells

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.

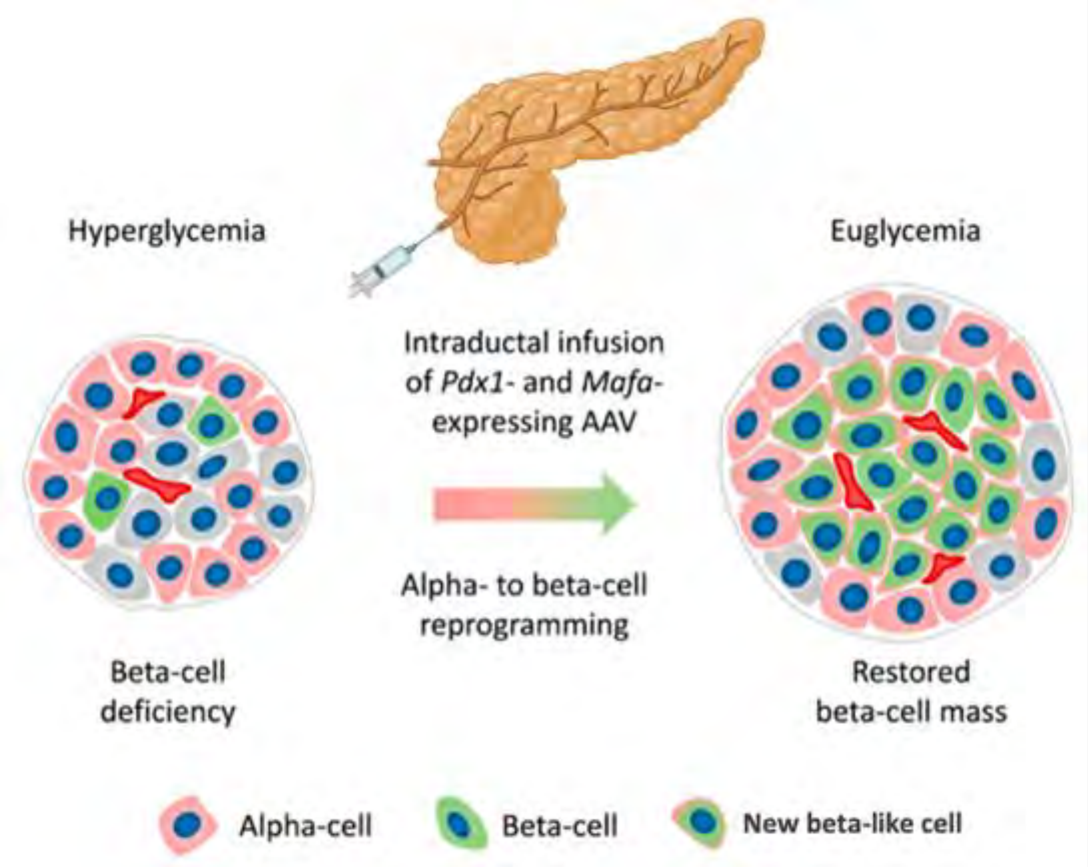
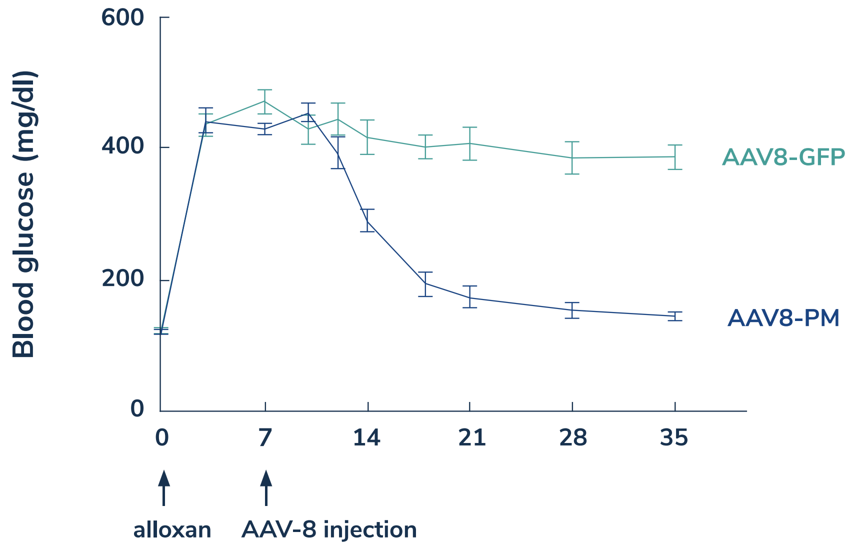


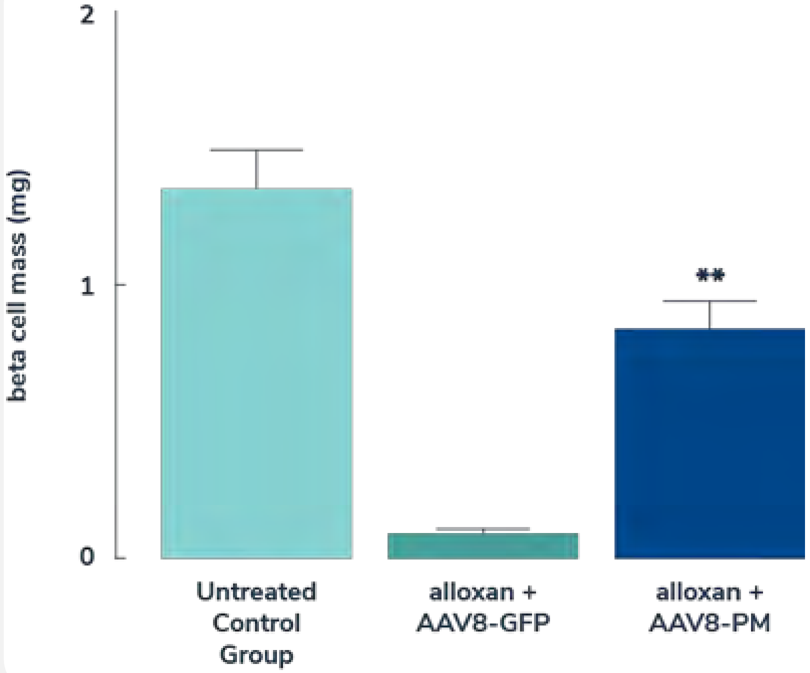
Image source: Osipovich, Anna & Magnuson, Mark. (2018). Alpha to Beta Cell Reprogramming: Stepping toward a New Treatment for Diabetes. Cell Stem Cell. 22. 12-13. 10.1016/j.stem.2017.12.012.

Genprex

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



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

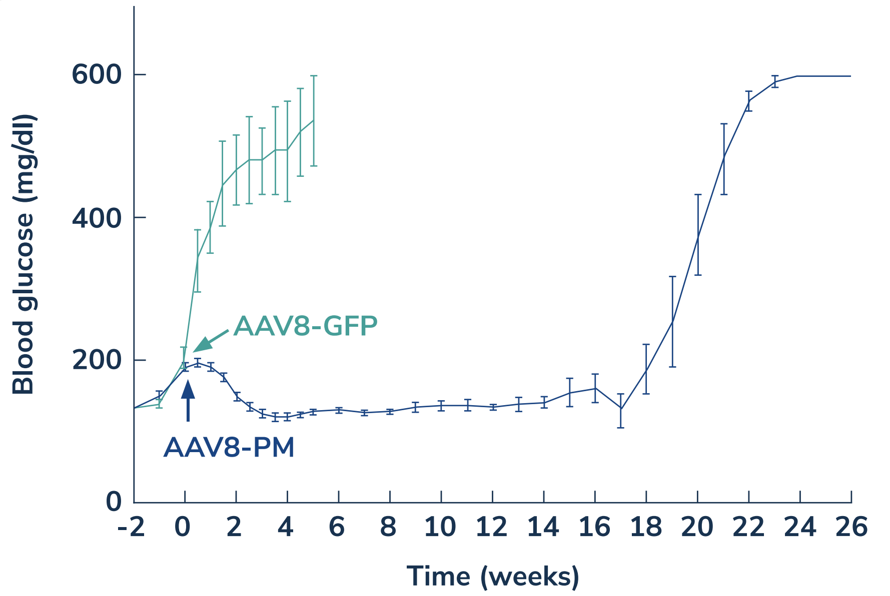


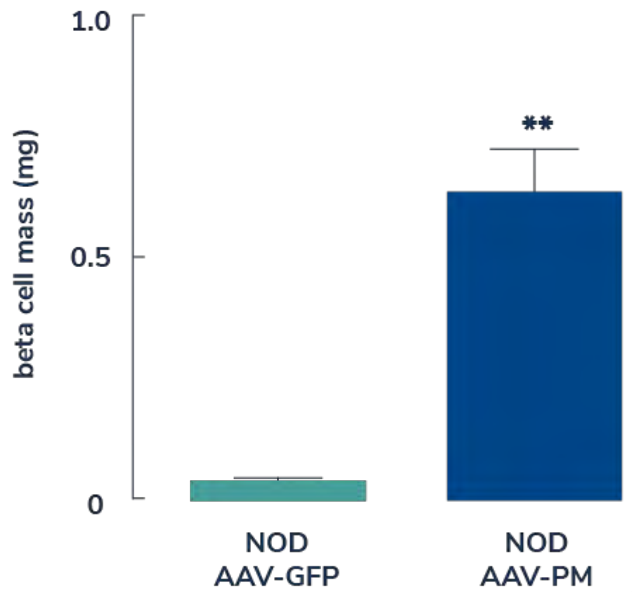
- Reprogramed alpha cells into beta-like cells that appropriately produce insulin in response to glucose levels.
- Normalized blood glucose in beta cell-toxin-induced diabetic mice.

Restored Blood Glucose in T1D In 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.
- NOD mice given syngeneic islet transplants became hyperglycemic a median of 17 days after treatment.



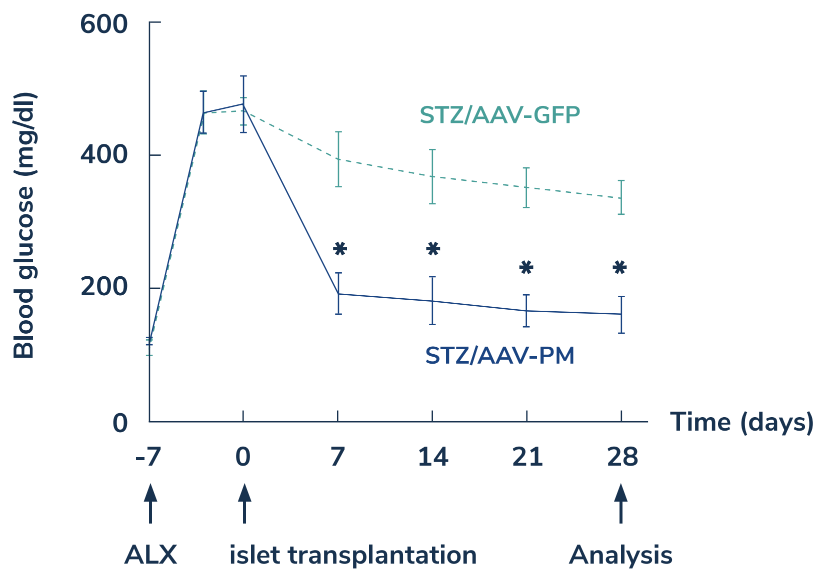


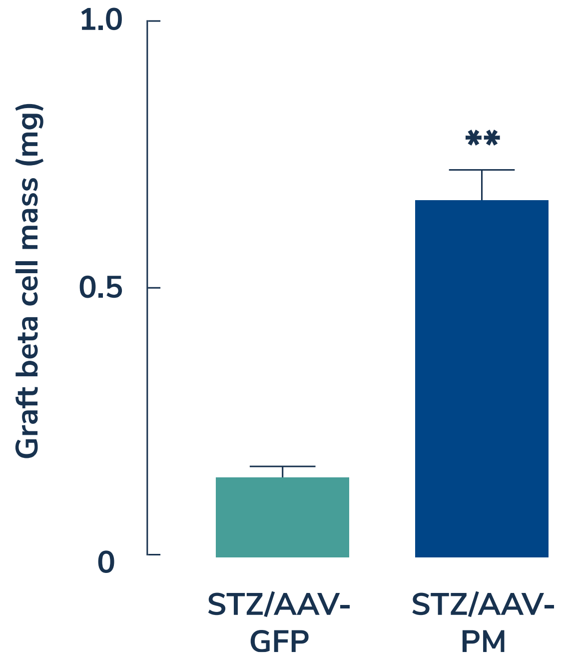
GFP = Green Fluorescent Protein (fluorescent marker) | PM = Pdx1 + MafA

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

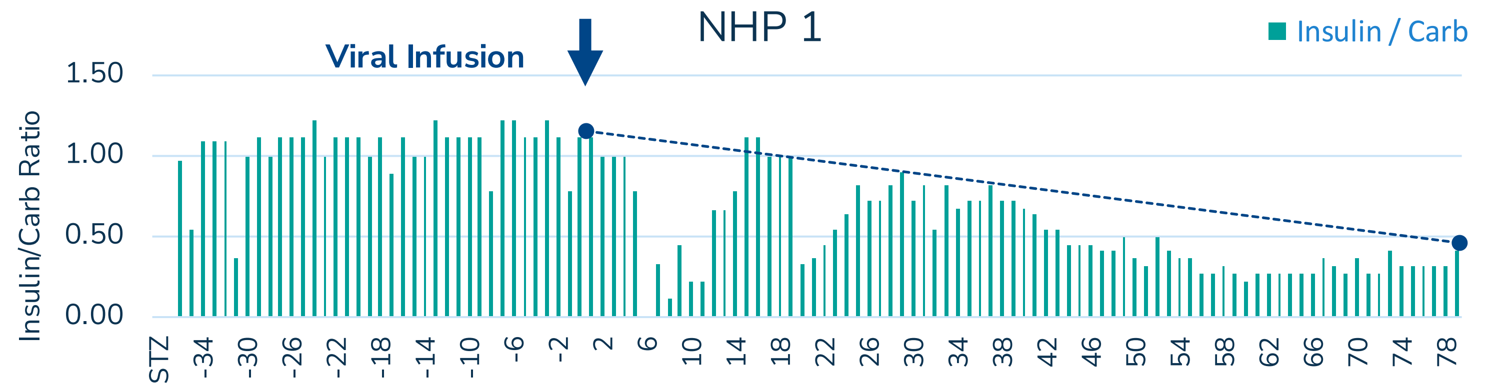
- Human islets treated with streptozotocin to destroy beta-cells, then treated with either AAV-PM or AAV-GFP
- AAV treated islets then transplanted into hyperglycemic NOD/SCID mice, treated with alloxan to destroy beta cells
- NOD/SCID mice receiving AAV-PM islets had significantly lower blood glucose 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**

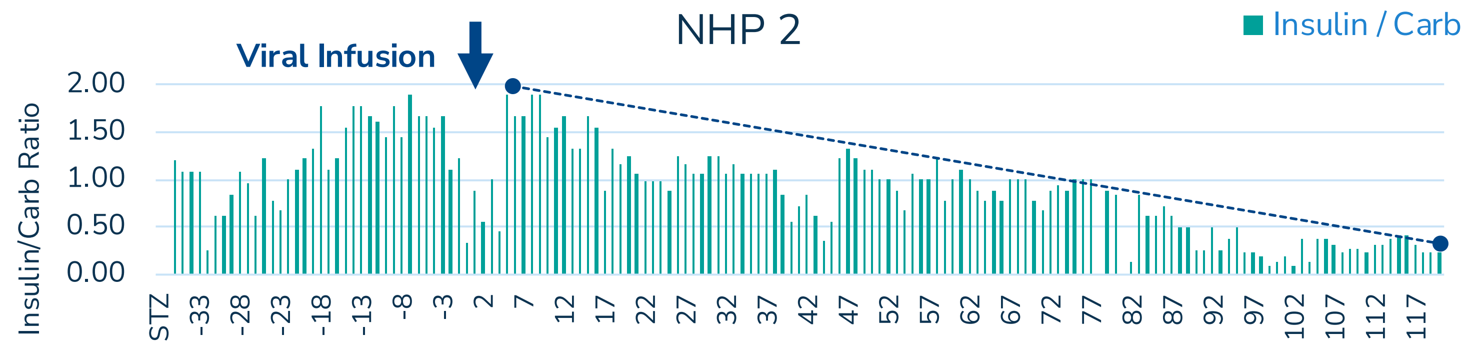




GFP = Green Fluorescent Protein (fluorescent marker) | PM = Pdx1 + MafA

Non-Human Primate Model of T1D Reduced Insulin Requirements





NHP = Non-Human Primate

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

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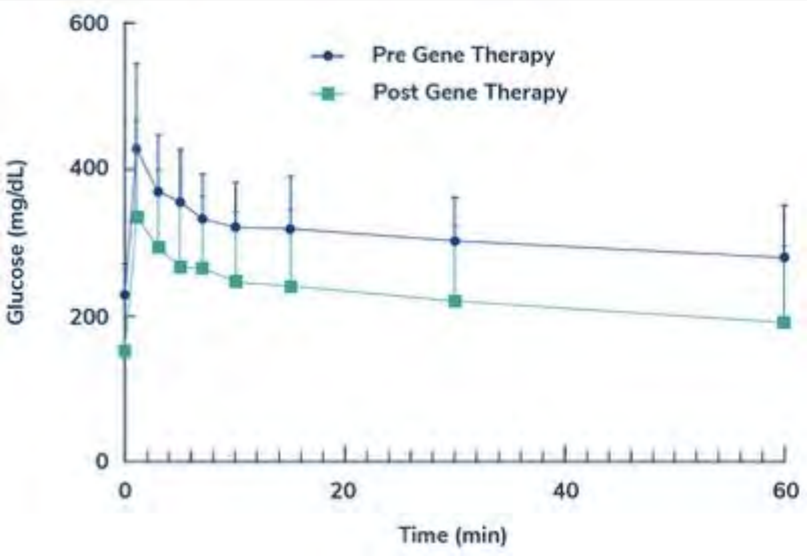
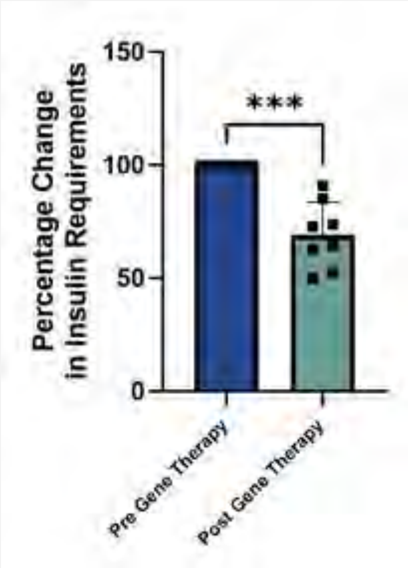
Decreased Insulin Requirements and Improved Glucose Tolerance in NHPs

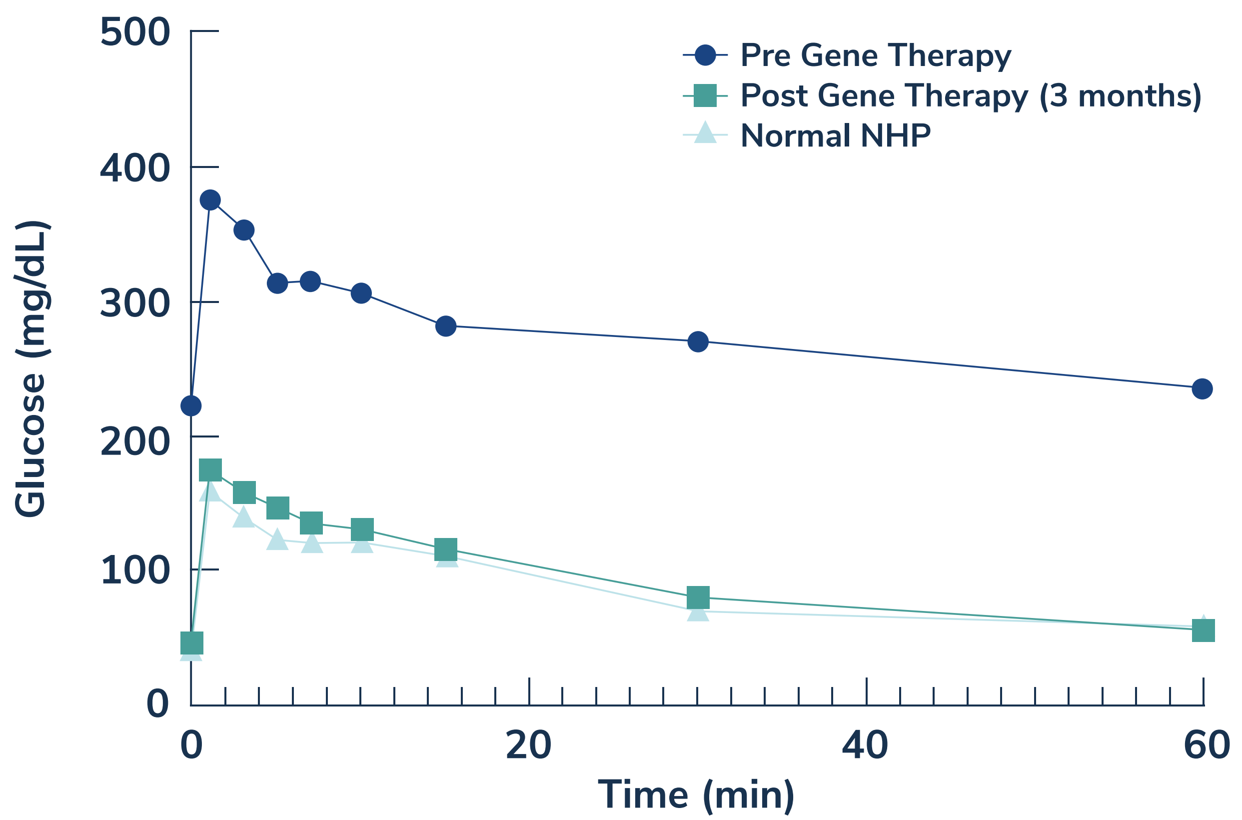
Following the pancreatic intraductal infusion of the AAV engineered construct, the eight NHPs had:

- 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)





NHP2
Three-Month Glucose Tolerance Test



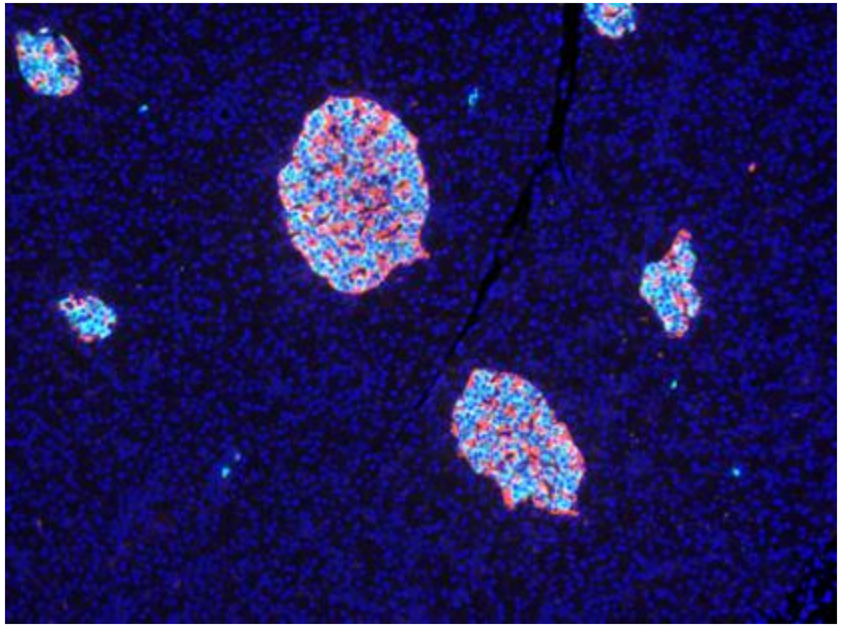
UNPUBLISHED DATA

Genprex

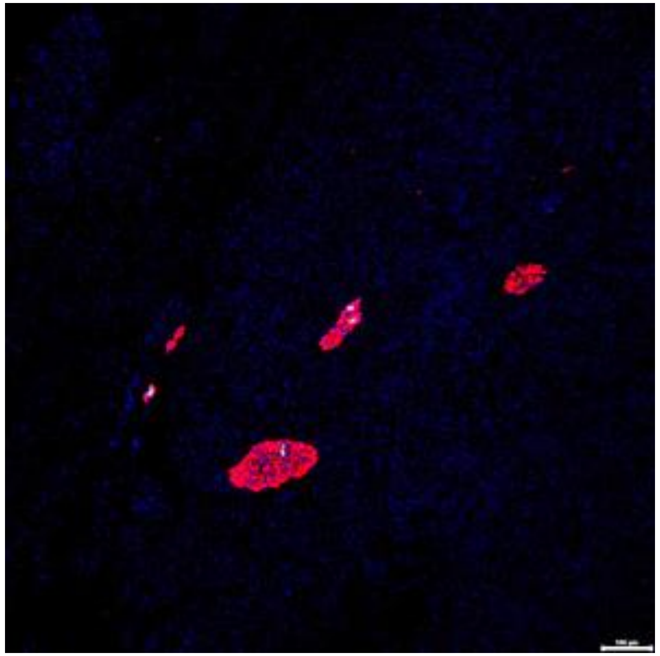
IHC Eight Weeks After Gene Therapy

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

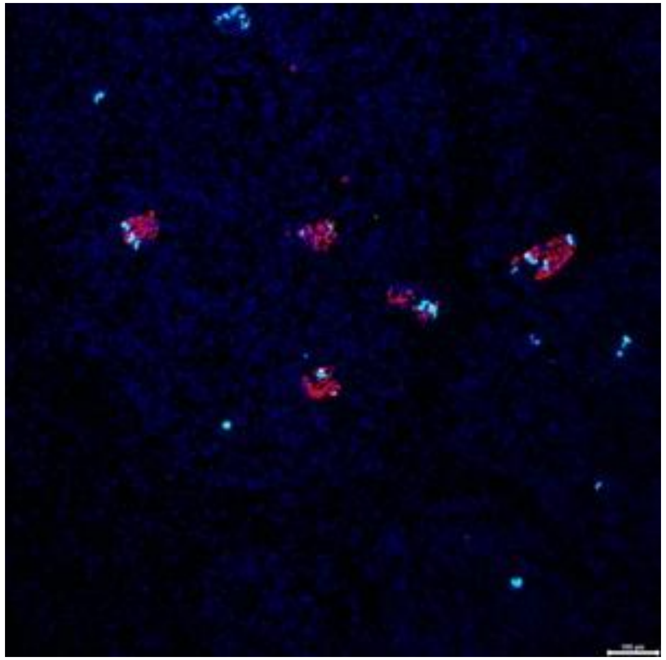
DAPI INSULIN GLUCAGON



Normal NHP



Diabetic NHP Without Gene Therapy



Diabetic NHP After Gene Therapy

Rejuvenates Exhausted Beta Cells in T2D NHP Models

NHP 1

- AAV infusion into the pancreatic duct
- Improved glucose tolerance testing over several months
- At seven months, NHP had normal glucose tolerance testing
- Decrease in insulin requirements
- Intraductal infusion is superior to direct injection

NHP 2

- Direct pancreas injection
- At three months, NHP displayed significant improvements in glucose tolerance testing but did not achieve normal testing results
- Direct injection of GPX-002 shows efficacy against T2D but was inferior to intraductal infusion.

GPX-002 reprograms and rejuvenates exhausted beta cells.

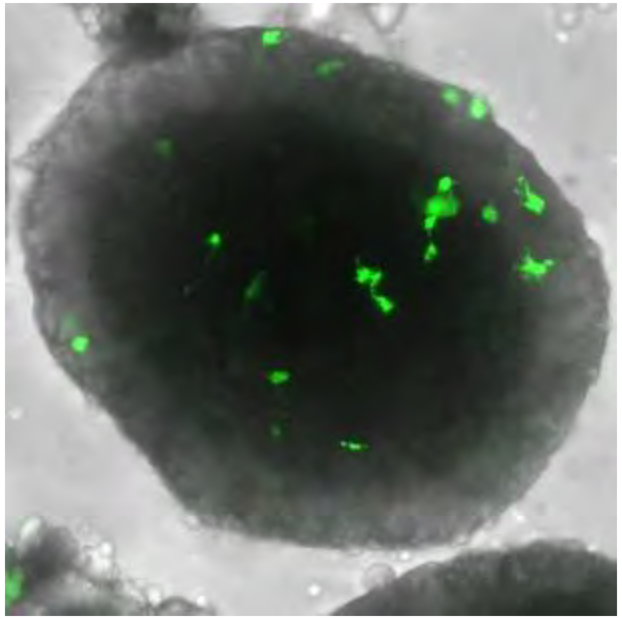
Studying GPX-002 Using a Lipid Nanoparticle

Shows **Potential for Re-Dosing** Using Non-Viral Delivery System

New studies use an alternative second generation approach with a non-viral lipid nanoparticle delivery system

First and only company to research diabetes gene therapy using a lipid nanoparticle in place of a viral AAV

May allow for repeat dosing of patients



LNP with a GFP mRNA payload injected into the mouse common bile duct efficiently transfected mouse Islet of Langerhans cells.

Data presented at AACR 2025.

CORPORATE

www.genprex.com

Our Team:
Company Management

Ryan Confer, MS
President, Chief Executive Officer & Chief Financial Officer

10+ years of C-Level experience in emerging technology companies

Extensive experience in investment management, deal negotiation and technology transfer

Mark S. Berger, MD
Chief Medical Officer

25 years of biotech and pharmaceutical company experience in the development of oncology therapeutics

Successfully brought two drugs through the regulatory process to approval

Thomas Gallagher, JD
Senior Vice President, IP & Licensing

25+ years of expertise in biotech IP law, business development, licensing transactions

Seasoned IP executive and attorney

David Schloss, JD
Senior Vice President, Human Resources

25+ years of experience as human resources executive and employment attorney in life sciences with a focus on biotech and cell and gene therapy

Suzanne Thornton-Jones, PhD
Senior Vice President, Regulatory Affairs

25+ years of experience in drug development and regulatory strategy and affairs for gene therapies

Our Team:
Scientific Advisory Board

Jack A. Roth
MD, FACS, Chairman

Professor and Bud Johnson Distinguished Clinical Chair, Department of Thoracic and Cardiovascular Surgery; Chief, Section of Thoracic Molecular Oncology; Professor of Molecular and Cellular Oncology; UT MD Anderson Cancer Center

Director, W.M. Keck Center for Innovative Cancer Therapies

Tony S. K. Mok
MD, FRCP(C), FHKCP, FHKAM

Professor and Chair of Clinical Oncology, the University of Hong Kong; Co-founder of the Lung Cancer Research Group

Pasi Antero Jänne
MD, PhD

Professor of Medicine, Harvard Medical School; Director of Dana Farber Cancer Institute Lowe Center for Thoracic Oncology; Scientific Director of the Belfer Center for Applied Cancer Science; Director, Chen-Huang Center for EGFR Mutant Lung Cancers; Head of The Jänne Lab

George Simon
MD

Chair, Department of Medical Oncology at Advent Health – Celebration; Executive Director of the Moffitt Cancer Center-Advent Health joint Clinical Research Unit

George K. Gittes
MD

Chief of Pediatric Surgery and Surgeon-in-Chief Emeritus at the UPMC Children’s Hospital of Pittsburgh; Director of the Richard King Mellon Foundation Institute for Pediatric Research; Co-Scientific director at UPMC Children's Hospital

Our Team:
Clinical Advisory Board

Michael Morse
MD, MHS, FACP

Professor of Medicine, Division of Medical Oncology in the Department of Surgery at Duke University

Research expertise in targeted therapies and immunotherapies for cancer

Andrew Becker
MD, PhD

President and Founder, Becker Pharmaceutical Consulting

Experience in consulting biotech and pharma companies on a global basis

Col. George Peoples
MD, FACS

Chief Executive Officer of Cancer Insight, LLC, a boutique cancer immunotherapy CRO

Professor of Surgery at Uniformed Services University; Professor of Surgical Oncology at MD Anderson Cancer Center

Our Team:
Board of Directors

Jose A. Moreno Toscano
Chairman of the Board

Chief Executive Officer, LFB USA Inc

20+ years of experience in pharma and biotech industries

Ryan Confer, MS
Board Director

10+ years of C-Level experience in emerging technology companies

Extensive experience in investment management, deal negotiation and technology transfer

Brent Longnecker
Board Director

Chief Executive Officer, Longnecker & Associates

30+ years of experience consulting with BODs, CEOs, key executives and advisors in many industries

William R. Wilson, Jr.
Board Director

Chief Executive Officer, Wilson Land & Cattle Co.

40+ years of legal experience spanning health care, biotech, clinical trial management

Achievements & Upcoming Milestones

Acclaim·1

- ✓ Open for enrollment in Phase 2a Expansion portion of the trial
- ✓ Expect to open additional sites by the end of 2025
- ☐ Complete enrollment of first 19 patients in Phase 2a Expansion portion of the trial in 1H 2026 for interim analysis
- ☐ Phase 2a interim analysis expected in 2H 2026

Acclaim·3

- ✓ Open for enrollment in Phase 2 Expansion portion of the trial
- ✓ Expect to open additional sites by the end of 2025
- ☐ Complete enrollment of first 25 patients in Phase 2 Expansion Portion in 1H 2026 for interim analysis
- ☐ Phase 2 interim analysis expected in 2H 2026

DIABETES

- ✓ Recently met with FDA in Q1 2026 regarding toxicology studies
- ☐ Research, optimize and finalize constructs
- ☐ Begin clinical scale production in a cGMP compliant facility
- ☐ Initiate toxicology studies

DISCOVERY

- ✓ Present new data on biomarkers at major medical conferences in 2026
- ✓ Execute a new SRA for expanding research with biomarkers
- ☐ Evaluate patient samples for biomarkers to optimize clinical studies
- ☐ Extend understanding of REQORSA biology

We believe in a future of **transformational** patient care.

21st Century Gene Therapies	Large Markets & Unmet Need	Combination Trials with Top Selling Drugs	Two FDA Fast Track Designations	Exploring New Indications & Partnerships

Genprex®

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Slide 39

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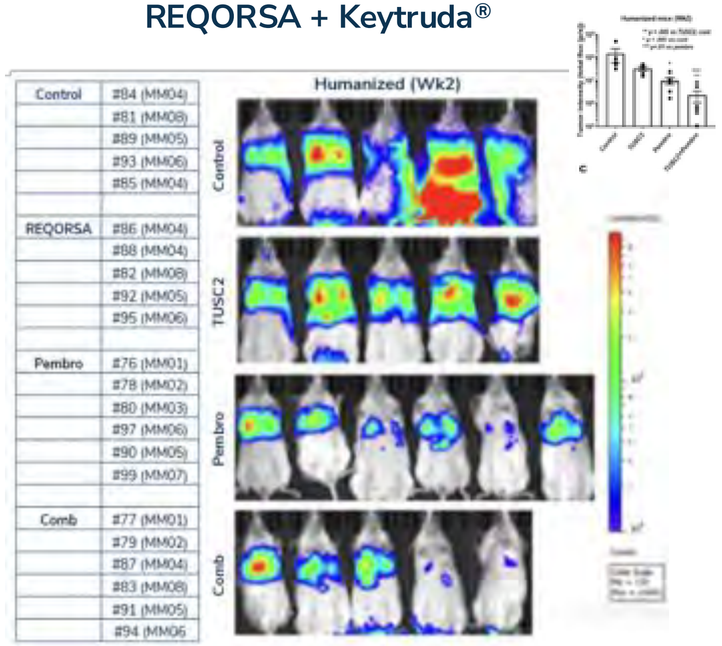
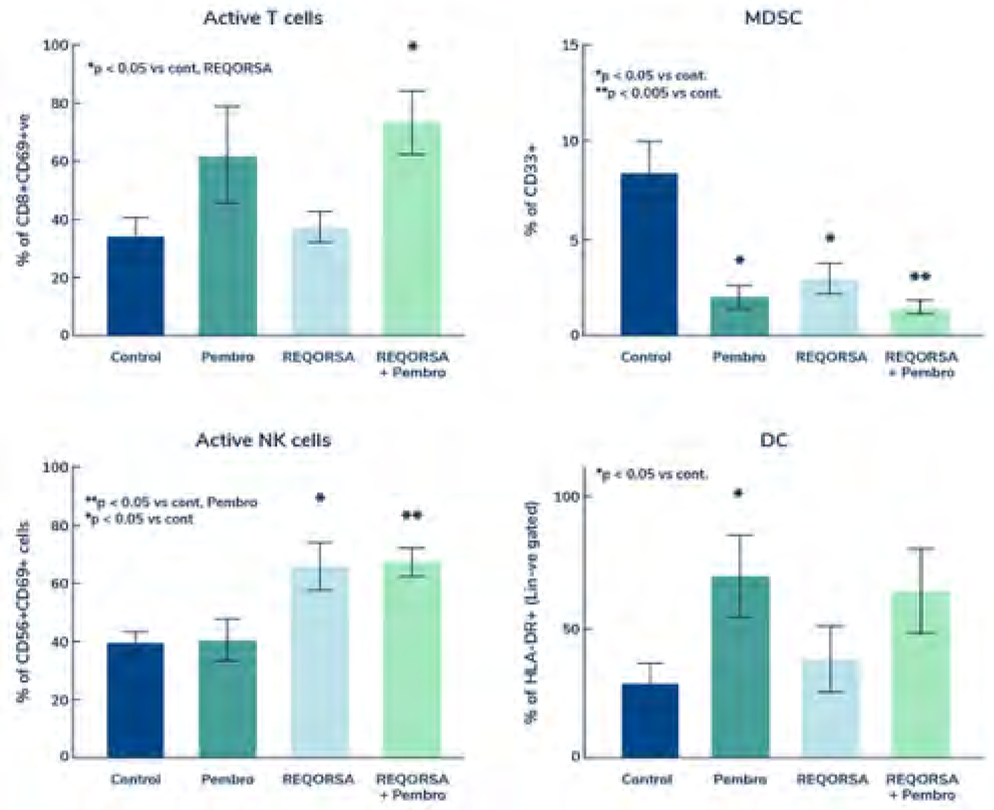
APPENDIX

Reqorsa® + Keytruda® Significantly Reduced Tumor Growth

REQORSA increases immune response against lung cancer xenografts

REQORSA + Keytruda®





Pembrolizumab is the generic name of Keytruda.

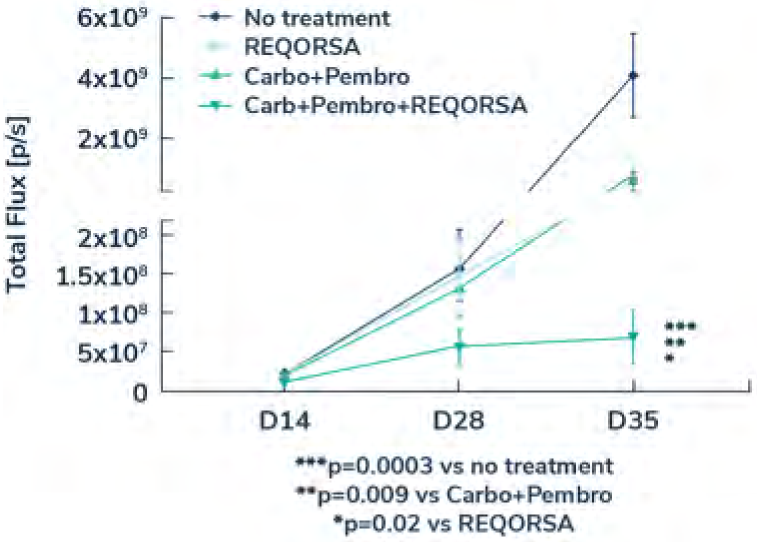
The independent immunologic effects of REQORSA and Keytruda markedly decrease tumor growth by increasing the immunologic attack on the tumor compared to PD-1 inhibition alone.

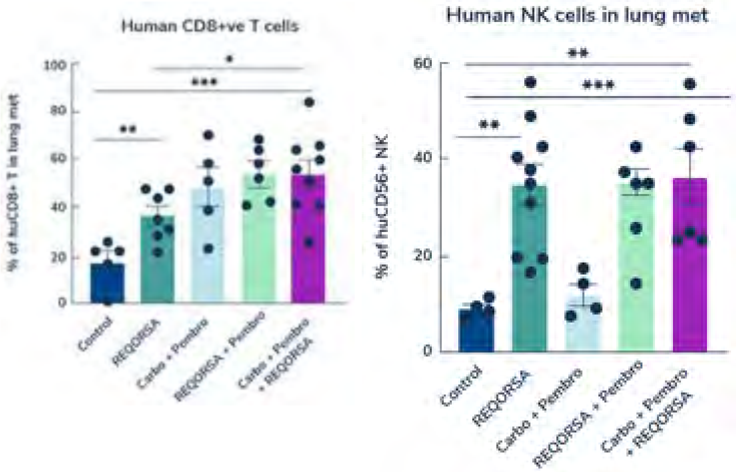
Genprex®

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.





Pembrolizumab is the generic name of Keytruda.

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Acclaim-2 is no longer enrolling new patients.

Overview of the former trial:

- Patients with advanced NSCLC whose disease progressed after treatment with Keytruda®
- FDA Fast Track Designation

Acclaim·2

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