Harnessing the Power of Trained Immunity

Corporate Presentation / May 2024

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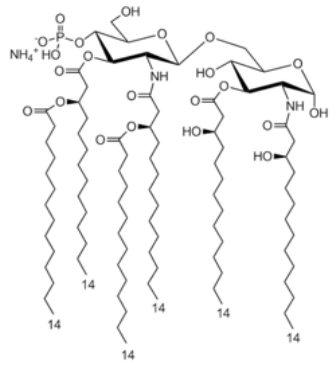
Forward-Looking Statements

This presentation contains forward-looking statements as defined in the Private Securities Litigation Reform Act of 1995, as amended. Forward-looking statements are statements that are not historical facts. These forward-looking statements are generally identified by the words "anticipate", "believe", "expect", "estimate", "plan", "outlook", and "project" and other similar expressions. We caution investors that forward-looking statements are based on management's expectations and are only predictions or statements of current expectations and involve known and unknown risks, uncertainties and other factors that may cause actual results to be materially different from those anticipated by the forward-looking statements. Revelation cautions investors not to place undue reliance on any such forward-looking statements, which speak only as of the date they were made. The following factors, among others, could cause actual results to differ materially from those described in these forward-looking statements: the ability of Revelation to meet its financial and strategic goals, due to, among other things, competition; the ability of Revelation to grow and manage growth profitability and retain its key employees; the possibility that the Revelation may be adversely affected by other economic, business, and/or competitive factors; risks relating to the successful development of Revelation's product candidates; the risk that our preclinical studies will not demonstrate sufficient positive data to support commencement of clinical trials; the risk that we may not fully enroll our clinical studies or enrollment will take longer than expected; risks relating to the occurrence of adverse safety events and/or unexpected concerns that may arise from data or analysis from our clinical studies; changes in applicable laws or regulations; expected initiation of the clinical studies, the timing of clinical data; the outcome of the clinical data, including whether the results of such study is positive or whether it can be replicated; the outcome of data collected, including whether the results of such data and/or correlation can be replicated; the timing, costs, conduct and outcome of our other clinical studies; the anticipated treatment of future clinical data by the FDA, the EMA or other regulatory authorities, including whether such data will be sufficient for approval; the success of future development activities for Gemini or any other product candidates; potential indications for which product candidates may be developed; the ability of Revelation to maintain the listing of its securities on NASDAQ; the expected duration over which Revelation's balances will fund its operations; the ability of Revelation to obtain further financing and other risks and uncertainties described herein, as well as those risks and uncertainties discussed from time to time in other reports and other public filings with the SEC by Revelation.

Gemini™ Development Pipeline

Gemini is Revelation's proprietary formulation of phosphorylated hexaacyl disaccharide (PHAD®)



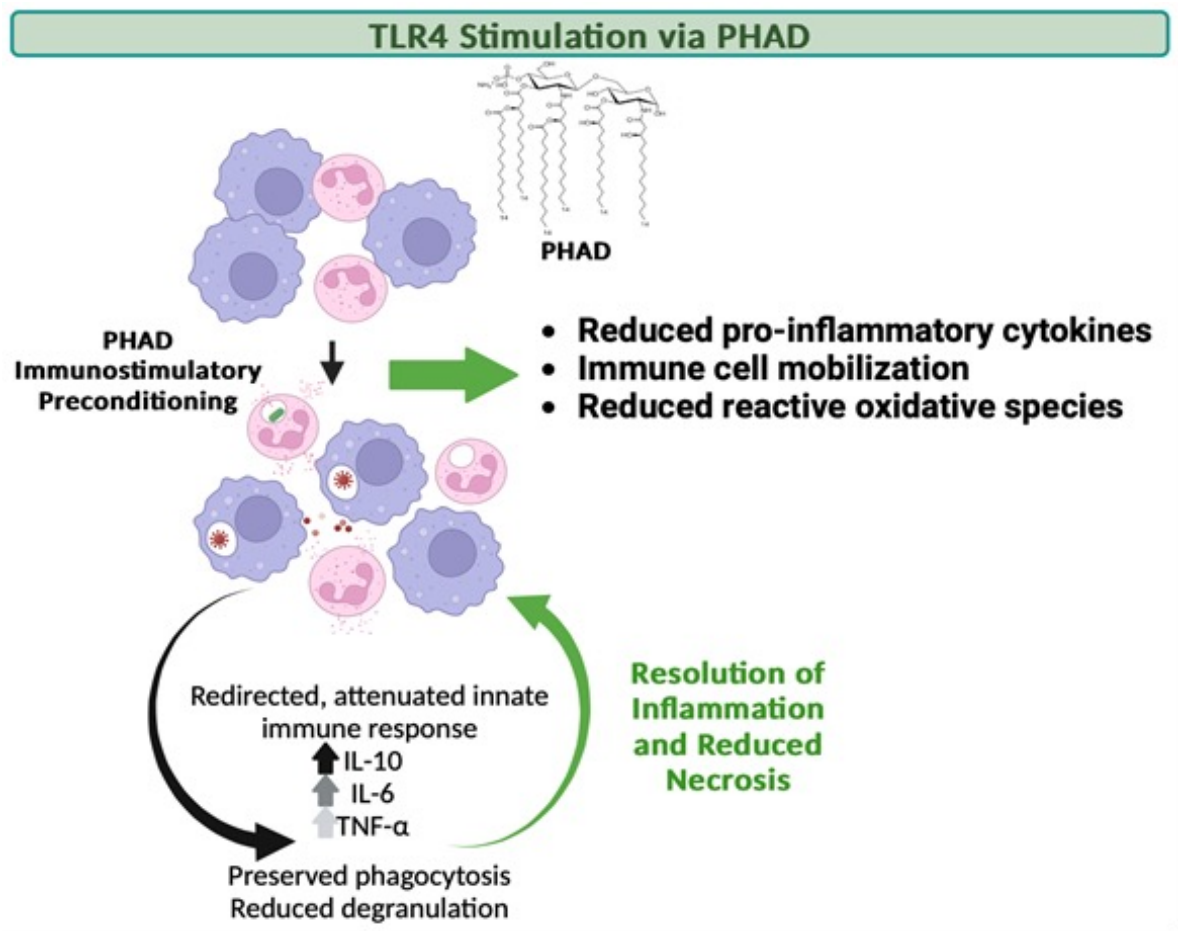
Program Name *(Indication)*	2023	2024	2025
GEM-AKI *(For the prevention of AKI [1])*	Preclinical		Phase 1b[5] / Phase 2
GEM-CKD *(For the prevention of CKD [2])*	Preclinical	Phase 1[4]	
GEM-PSI *(For the prevention of PSI [3,4])*	Preclinical		

[1]Acute Kidney Injury , [2]Chronic Kidney Disease, [3]Post Surgical Infection [4]Formerly known as GEM-SSI (Surgical Site Infection)
[4]Phase 1 single dose escalation study to support development of all Gemini programs
[5]Phase 1b to support GEM-AKI and CKD programs

Gemini Reprograms Toll-Like Receptor 4 Signaling

Introduction

- Gemini is a proprietary formulation of phosphorylated hexaacyl disaccharide (PHAD), a synthetic, detoxified version of lipopolysaccharide (LPS).
- PHAD stimulates TLR4 to precondition immune cells to stress, including early, robust induction of IL-10, IL-1RA and attenuated induction of IL-6, and TNF-α.
- Following injury (e.g. ischemia), preconditioning contributes to rapid mobilization of innate immune cells, a reduced pro-inflammatory response, and a reduction in the generation of reactive oxidative species.
- Preconditioning was shown to provide significant improvement in function, reduced tissue damage, and accelerated resolution of injury in multiple models of kidney injury (I/R and UUO[1]).
- Preconditioning has been shown to provide significant protection from gram-negative and gram-positive bacterial infection.



1. I/R = ischemic reperfusion, UUO = unilateral urinary obstruction

GEM-AKI and GEM-CKD Programs

Gemini For the Prevention of Acute Kidney Injury and Chronic Kidney Disease

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GEM-AKI and GEM-CKD Program Highlights

Scientific Rationale

- Significant protection from multiple factors contributing to AKI observed in ischemia reperfusion model
- Significant anti-fibrotic activity observed in preclinical AKI and CKD model (UUO) with PHAD treatment

Intellectual Property

- Patent applications covering formulations and methods of treating and preventing acute and chronic organ disease filed

Regulatory

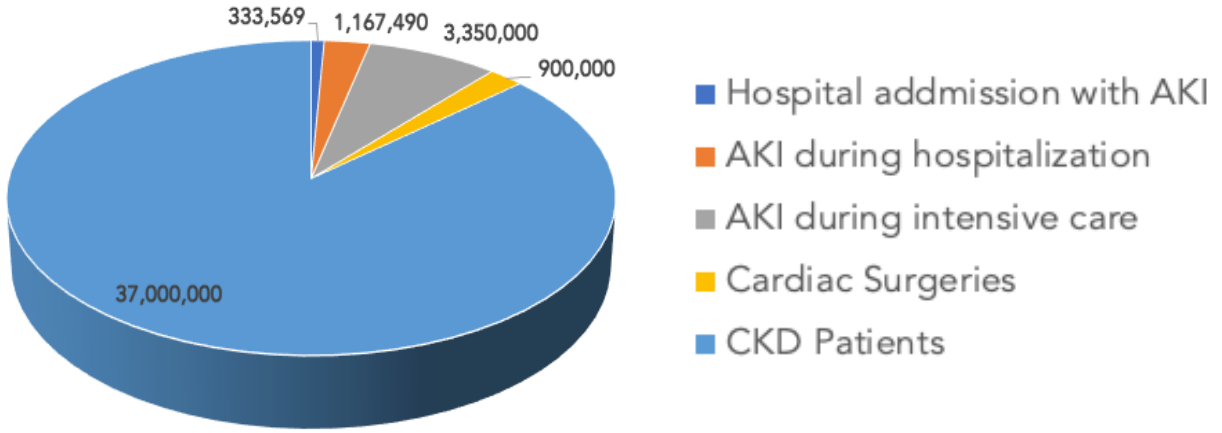
- US IND in 2024

Clinical Plan

- Phase 1 clinical study in healthy volunteers underway (Australia). Readouts include safety and biomarker activity data.
- Phase 1b planned to initiate in Q4 2024

Potentially Large Markets

- Conservatively, if we treat 20% of the cardiac surgery AKI market at a price of $7.5k per patient: 900K x 20% = 180,000 x $7.5k = $1.35 billion annual revenue potential
- Conservatively, if we treat 5% of the CKD market at a price of $2.5k per patient: 37M x 5% = 1.85M x $2.5k = $4.6 billion annual revenue potential



Gemini Preserves Kidney Function and Reduces Injury in Ischemia/Reperfusion Model

Kidney Function at 24 and 72 hours

Figure 1a. Change in Creatinine[1]

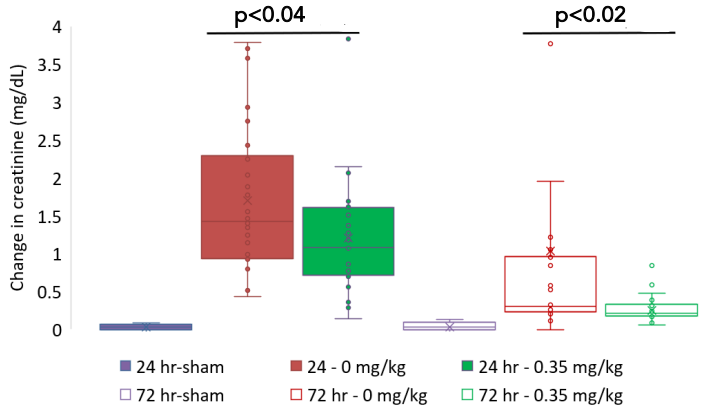
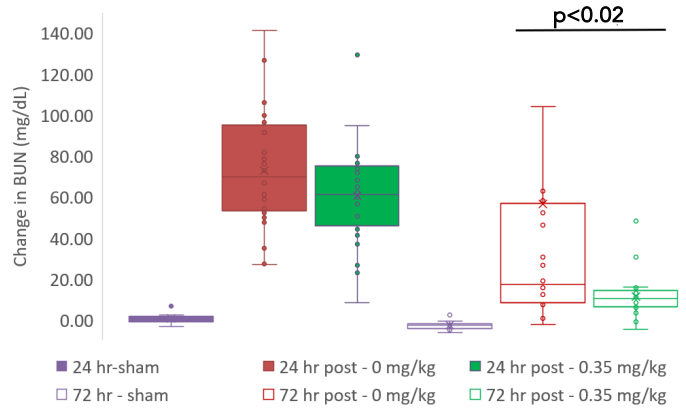


Figure 1b. Change in BUN[1]



- Gemini pretreatment (0.35 mg/kg) significantly preserved kidney function with return to base line faster relative to untreated control animals (Figure 1a and 1b). A similar trend was observed for 0.07 mg/kg (data not shown).
- Additional renal function findings (data not shown) included improved creatinine clearance and excretion relative to the untreated group

Kidney Injury at 72 hours

Figure 2a. Acute Cortical Tubular Necrosis[2]

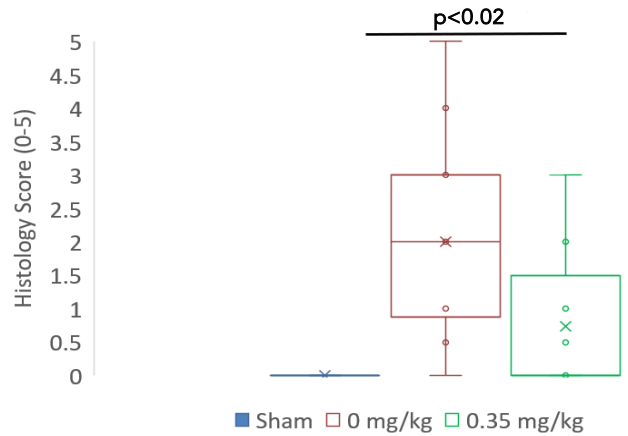
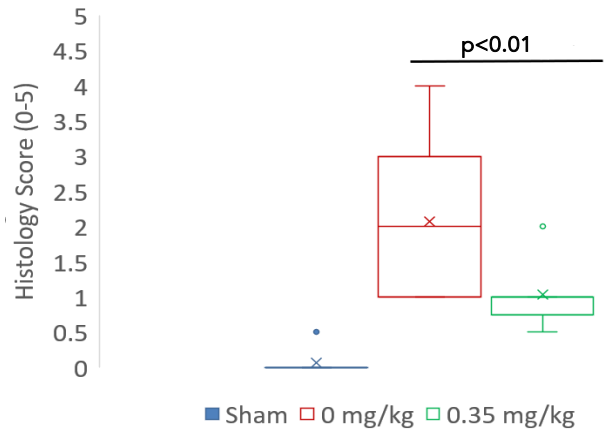


Figure 2b. Medullary Tubular Necrosis[2]



- Gemini reduced injury to the cortical and medullary tubules as measured by histopathology (Figure 2a and 2b). A similar trend was observed for 0.07 mg/kg (data not shown).
- Additional findings: Gemini did not significantly lower cortical or medullary tubular degeneration or tubular protein casts included (data not shown) relative to the untreated group.

[1]N=16-28, dosed 24 and/or 48 hours prior to surgery. [2]N=8-14, dosed 24 hours prior to surgery.

Gemini Significantly Attenuates Inflammatory Response in an Ischemia/Reperfusion Model of AKI

Figure 3a. Urinary CRP at 24 hours[1]

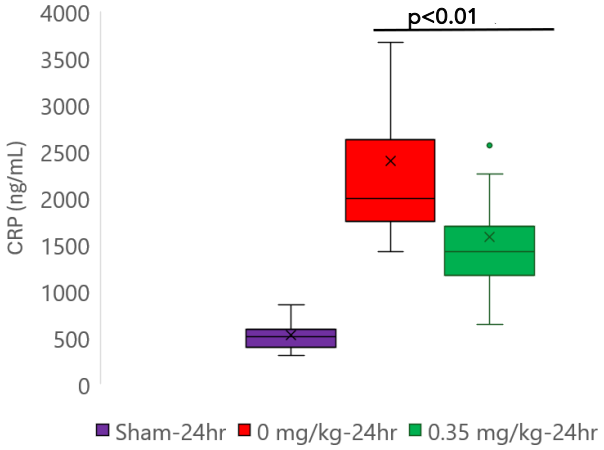


Figure 3b. Urinary IL-6 at 24 hours[1]

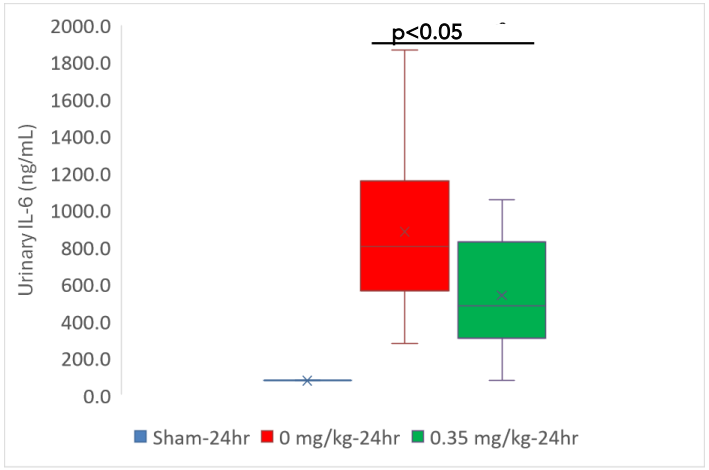


Figure 3c. Serum CRP at 72 hours[1]

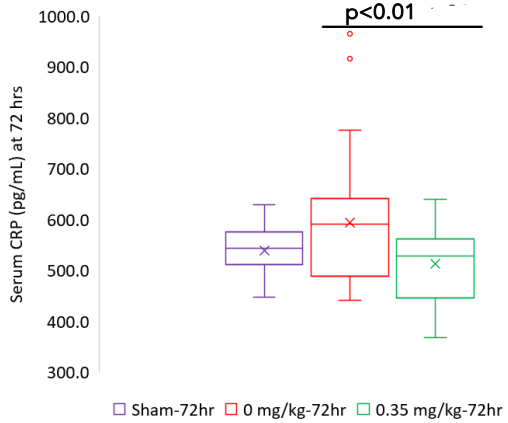
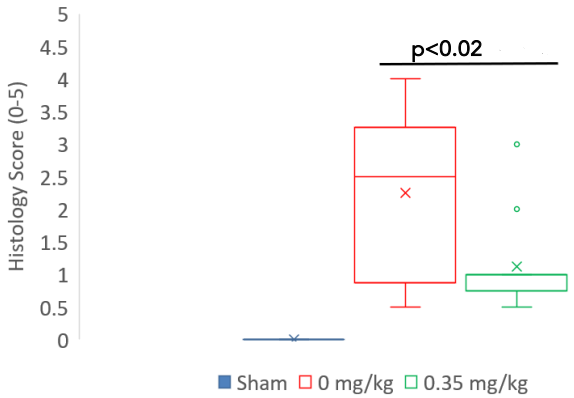


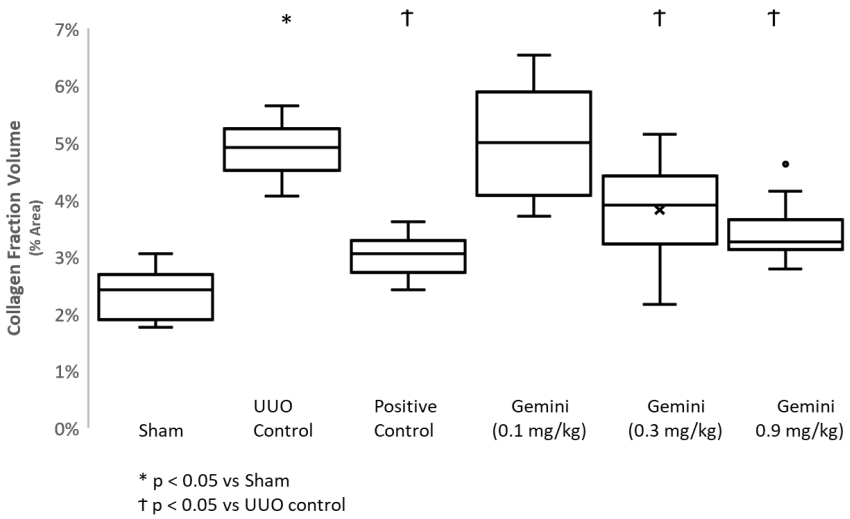
Figure 3d. Neutrophilic Inflammation at 72 hours[2]

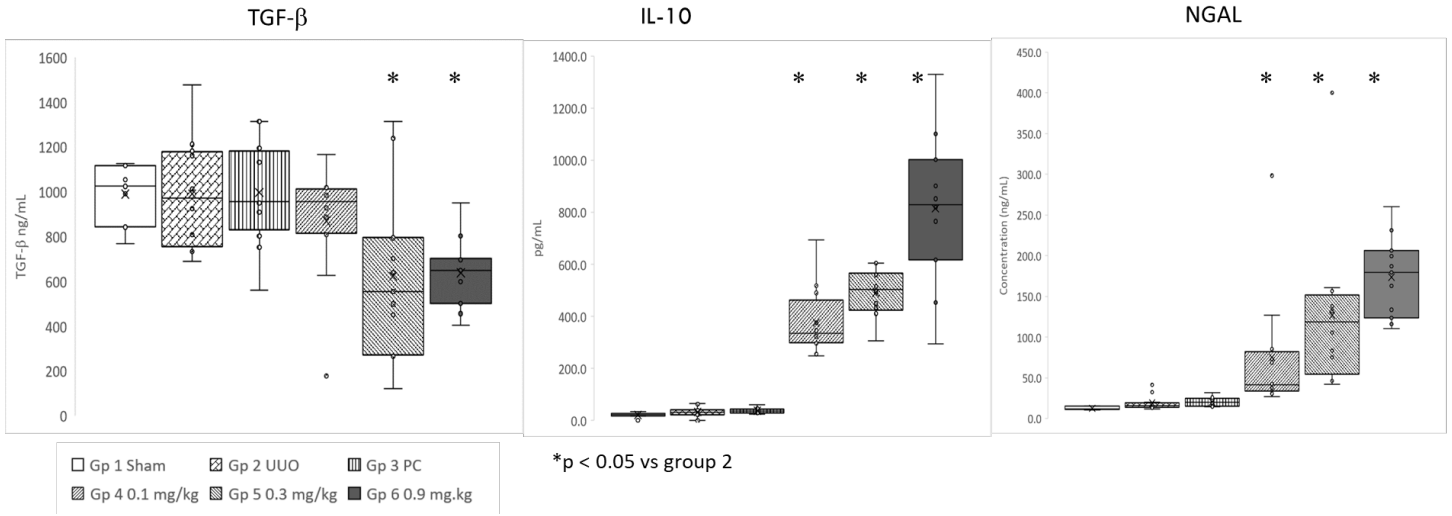


- Pretreatment with Gemini (0.35 mg/kg) significantly reduced multiple markers of local inflammation at 24 and/or 72 hours in urine (Figure 3a and 3b, 72-hour data not shown).
- Pretreatment with Gemini (0.35 mg/kg) significantly reduced a key marker (CRP) of systemic inflammation at 72 hours in serum (Figure 3c).
- Pretreatment with Gemini also significantly reduced markers of cellular inflammation as observed via reduced neutrophilic inflammation (Figure 3d).
- Additional markers of reduced cellular inflammation observed (data not shared).

[1]N=16-28, dosed 24 and/or 48 hours prior to surgery. [2]N=8-14, dosed 24 hours prior to surgery.

Gemini Treatment Reduces Fibrosis in Acute and Chronic Kidney Model* (UUO in Rats)





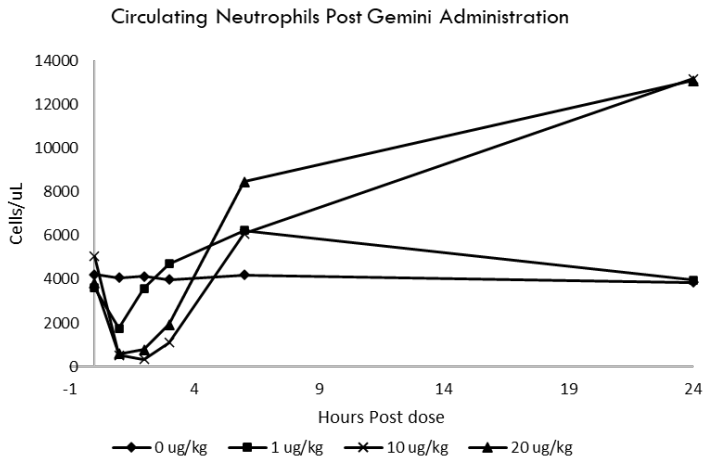
- Treatment with Gemini resulted in a significant dose-dependent reduction in fibrosis (all results normalized to sham group, n=6)

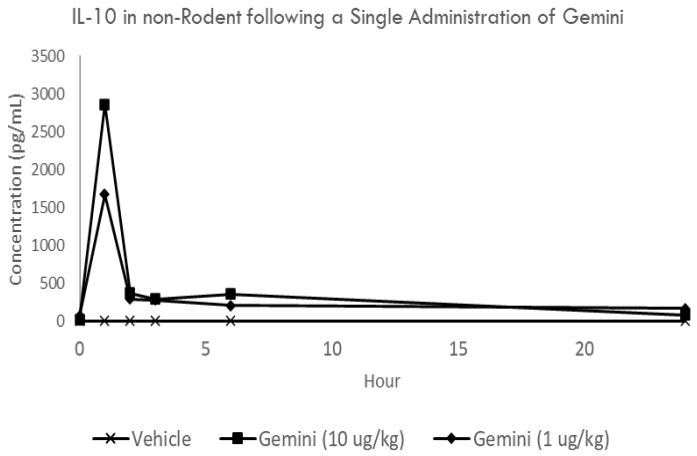
- Reduction in fibrosis driven by reduction in pro-inflammatory cytokines and reduction in protective cytokines:
  - TGF-β is pro-fibrotic and is directly linked to the propagation of fibrosis[1,2,3,4]
  - IL-10 is a key driver for the reduction and resolution of inflammation
  - NGAL is an important defense for preventing excessive oxidative damage resulting from injury/ongoing inflammation

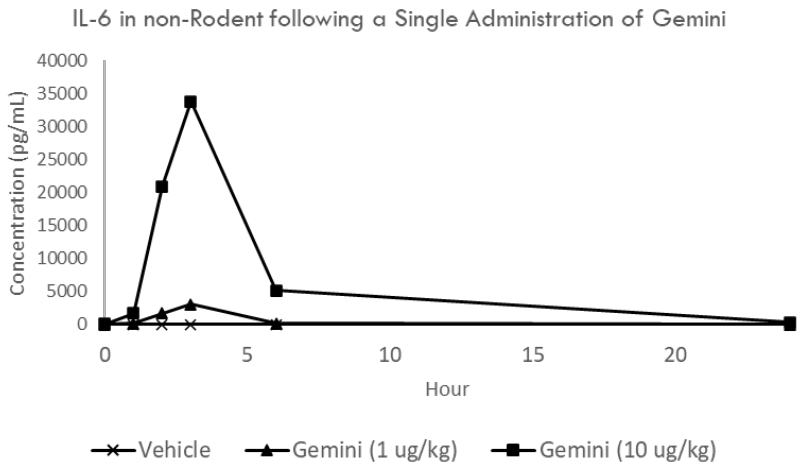
*Rats (n=11-12 per treatment group) were subjected to the unilateral ureteral obstruction (UUO) surgical procedure. Composite data represents the average of 3 anatomically distinct depths (10 images / depth / rat / group = ~60-65% of renal cortical area). Positive control = SB-525334, a TGF-β blocker

[1]Mohy doi: 10.1016/j.mgene.2014.08.002. [2]Tian doi: 10.3892/etm.2019.8355,
[3]Nawar, Elham A. et al. "Clinical value of transforming growth factor beta as a marker of fibrosis in adolescents with Chronic Liver Diseases." (2011). [4]De Heer, E., et. al. Nephrol Dial Transplant (2000) 15 [Suppl 6]: 72–73

Gemini Administration Induces Multiple Markers of TLR4 Stimulation Mediated Innate Immune Activity in Healthy Animals







- Gemini administration in healthy animals results in:
  - White blood cell migration (including neutrophils (data shown), monocytes, and lymphocytes) from circulation and subsequent rebound at 24 hours post-dose
  - Upregulation of IL-10 (shown) and IL-1RA, anti-inflammatory cytokines critical to resolution of inflammation
  - Upregulation of IL-6 (shown), a necessary first step in the establishment of trained immunity
  - Absent or minimal detection of IL-1b and TNF-a, key cytokines associated with chronic inflammation
- These are examples of key biomarkers to demonstrate Gemini-mediated TLR4 stimulatory activity and are being evaluated in the Phase 1 clinical study

Based on biomarkers observed following administration of Gemini to healthy animals. Study Design: Dogs received a single dose of vehicle Gemini and blood samples were collected at various time-points up to 24 hours post dose for analysis (n=3–10 per group)

2024 Clinical Plan

Title

- A Phase 1, Randomized, Placebo Controlled, Single Blind, Single-Ascending Dose in Healthy Volunteers

Design

- Single dose, dose escalation
- 5 cohorts, 8 subjects per cohort 1:4 placebo vs drug
- Follow for 7 days

Readouts

- Safety, tolerability, PK, and activity biomarkers
- Key Biomarkers of Activity[1]
  - Leukocytes (e.g. neutrophil mobilization/upregulation)
  - IL-10, IL-6, IL-1RA

Status

- Recruiting – Target completion Q3 2024

Title

- A Phase 1b, Randomized, Placebo Controlled, Single Blind, Single-Ascending Dose in Stage 3 and Stage 4 CKD Patients
- PRIME – PRotective IMmunostimulatory Evaluation

Design

- Arm 1 - Single dose, dose escalation
- Arm 2 – Two doses at highest tolerated dose
- 5 cohorts, 8 subjects per cohort 1:4 placebo vs drug
- Follow for 14 days

Readouts

- Safety, tolerability, PK, and activity biomarkers
- Key biomarkers of activity: TBD (possibly CRP, IL-10, IL-1RA, TGF-β)

Status

- Planning – Target initiation Q4 2024

[1]Based on biomarkers observed following administration of Gemini to healthy animals.

GEM-PSI

Gemini for the Prevention of Post-Surgical Infection

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GEM-PSI* Program Highlights

Scientific Rationale

- Multiple preclinical studies performed demonstrating consistent reduction or prevention of infection (both gram negative and gram positive)

Intellectual Property

- US 11,389,465 (Licensed from Vanderbilt University). Additional related applications anticipated

Regulatory

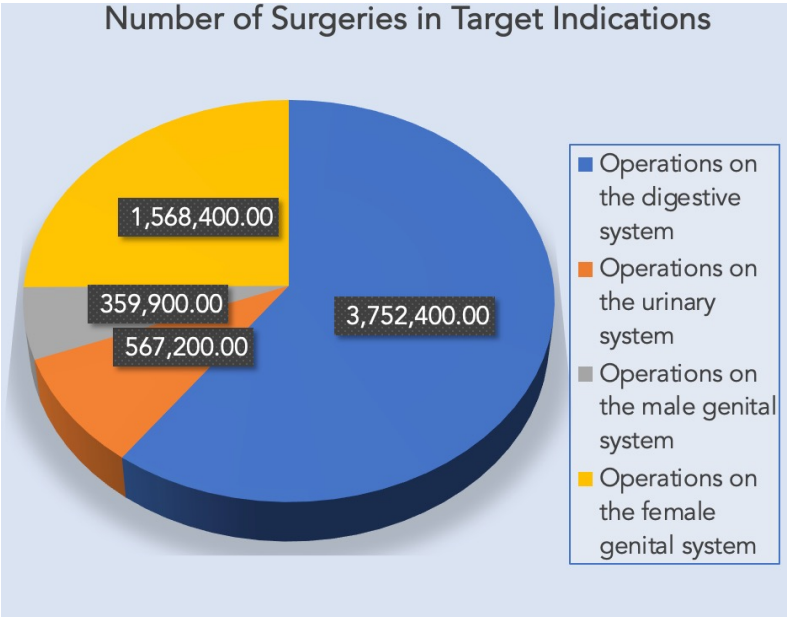
- Potential fast track, breakthrough designations possible. Potential for orphan status for certain indications

Clinical Plan

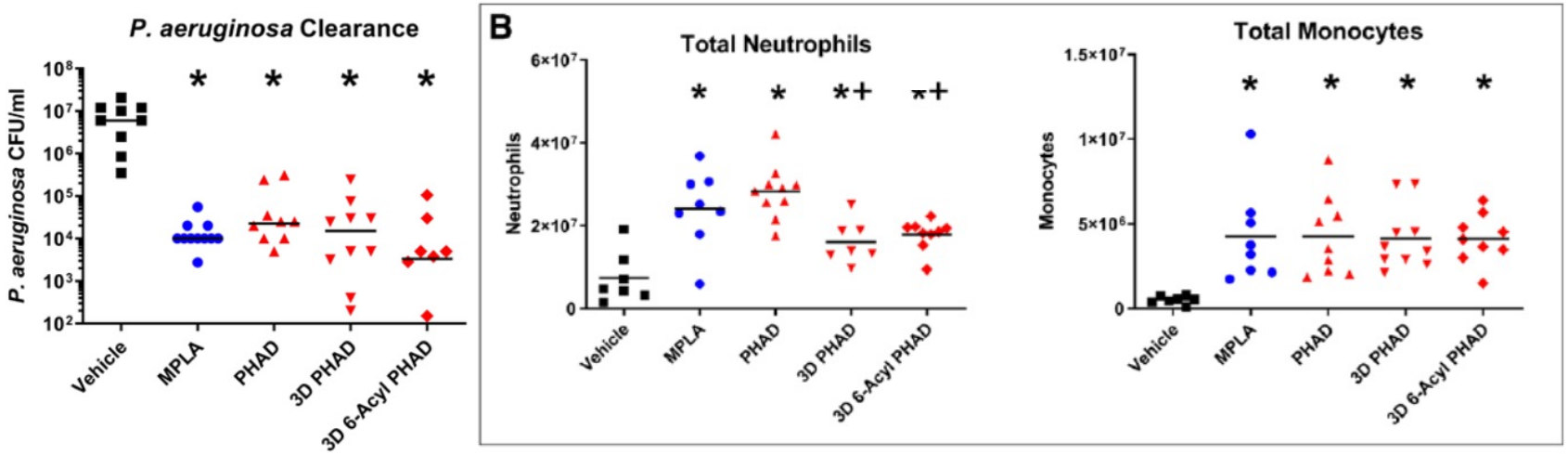
- Phase 1 clinical study in healthy volunteers underway. Readouts to include safety and biomarker activity data

Potentially Large Markets

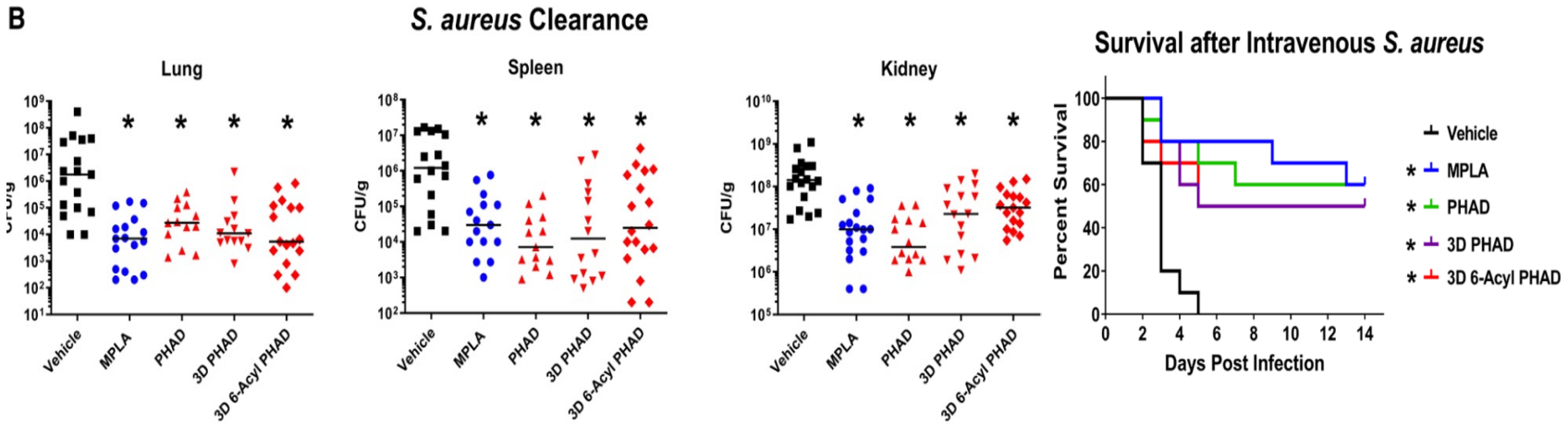
- Large Market potential: Approximately 3% of hospital patients suffer at least one hospital associated infection (HAI) (~687,000 HAI annual cases in acute care settings resulting in ~72,000 deaths)[1]
- Conservatively, if we treat 10% of the digestive system market at a price of $5k per patient: 3.8 M x 10% = 380,000 x $5k = $1.87 billion annual revenue potential



PHAD Pretreatment Reduces Severity of Infection (Gram Negative and Gram Positive)



Study Design: Mice were pre-treated (24 and 48 hours) with vehicle, MPLA (20ug), or PHADs (20ug) prior to infection with *P. aeruginosa*. All given IP. Cell counts assessed from peritoneal lavage 6 hours post infection.
n = 7 to 10 animals per group.
3D and 3D-6-Acyl PHAD are analogs of PHAD.



Study Design: Mice were pre-treated (24 and 48 hours) with vehicle, MPLA (1 mg/kg), or PHADs (1 mg/kg) prior to infection with *S. aureus*. All given IV. Bacterial counts assessed 3 days post infection.
n = 7 to 10 animals per group.

Hernandez, et. al. Phosphorylated Hexa-Acyl Disaccharides Augment Host Resistance Against Common Nosocomial Pathogens. DOI: 10.1097/CCM.0000000000003967

Financial Overview

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Financial Overview

Cap Table	Shares
Common Stock Outstanding	**1,632,935**
Class D common stock warrants w/$4.53 exercise	2,730,000
Class C common stock warrants w/$4.53 exercise	16,239
Public Warrants w/$12,075.00 exercise (REVBW)	10,012
Warrants w/$630.00 weighted avg exercise[1]	11,457
Roll-over RSU's	94
Options granted	1,157
Equity Pool (available for grant)	20,466
Fully Diluted	**4,422,360**

Management	Shares
Total management	**3,613**

1. *Includes (i) 7,937 Private Warrants w/exercise of $630.00, (ii) 155 Roll-over Warrants w/exercise of $2816.92, (iii) 2,809 Common Stock Warrants w/exercise of $3,454.50, and 556 Placement Agent Warrants w/exercise of $787.50.*

Thank You!

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