HARNESSING THE POWER OF THE INNATE IMMUNE SYSTEM
Modulating an Innate Immune Response Against Diseases

INMB
Nasdaq

CORPORATE PRESENTATION  Dec 2020
FORWARD LOOKING STATEMENTS

This presentation contains “forward-looking statements” Forward-looking statements reflect our current view about future events. When used in this presentation, the words “anticipate,” “believe,” “estimate,” “expect,” “future,” “intend,” “plan,” or the negative of these terms and similar expressions, as they relate to us or our management, identify forward-looking statements. Such statements, include, but are not limited to, statements contained in this presentation relating to our business strategy, our future operating results and liquidity and capital resources outlook. Forward-looking statements are based on our current expectations and assumptions regarding our business, the economy and other future conditions. Because forward-looking statements relate to the future, they are subject to inherent uncertainties, risks and changes in circumstances that are difficult to predict. Our actual results may differ materially from those contemplated by the forward-looking statements. They are neither statements of historical fact nor guarantees of assurance of future performance. We caution you therefore against relying on any of these forward-looking statements. Important factors that could cause actual results to differ materially from those in the forward-looking statements include, without limitation, our ability to raise capital to fund continuing operations; our ability to protect our intellectual property rights; the impact of any infringement actions or other litigation brought against us; competition from other providers and products; our ability to develop and commercialize products and services; changes in government regulation; our ability to complete capital raising transactions; and other factors relating to our industry, our operations and results of operations. There is no guarantee that any specific outcome will be achieved. Investment results are speculative and there is a risk of loss, potentially all loss of investments. Actual results may differ significantly from those anticipated, believed, estimated, expected, intended or planned. Factors or events that could cause our actual results to differ may emerge from time to time, and it is not possible for us to predict all of them. We cannot guarantee future results, levels of activity, performance or achievements. Except as required by applicable law, including the securities laws of the United States, we do not intend to update any of the forward-looking statements to conform these statements to actual results.
INMUNE BIO HIGHLIGHTS

Clinical-stage biopharmaceutical company developing a new class of therapeutics that target dysfunction of the innate immune system.

Third party validation:
- Over 65 publications from multiple universities worldwide on both platforms with extensive in vivo data.
- More than $4M in grants from AA, ALS and NIH.

Two platforms:
- DN-TNF: First selective TNF inhibitor targeting inflammation without demyelination or immunosuppression.
- NK Cell Priming: Primes patient’s NK cells to eliminate residual.

Highlight of upcoming catalysts:
- P1b AD data January
- P1 HR-MDS Start 1H 2021
- P2 AD Initiation Mid-2021
- P2 TRD Initiation Mid-2021
- P2 CV-19 data 2021
## ACTIVE PIPELINE
Therapies that Target the INNATE IMMUNE Response

<table>
<thead>
<tr>
<th>DN-TNF PLATFORM</th>
<th>DISEASE FIELD</th>
<th>PRE-CLINICAL</th>
<th>PHASE I</th>
<th>PHASE II (POC)</th>
<th>PIVOTAL</th>
<th>EST. NEXT MILESTONE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Quellor</strong></td>
<td>COVID-19 Cytokine Storm</td>
<td></td>
<td></td>
<td></td>
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<td>P2 underway – data 2021</td>
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<tr>
<td><strong>XPro1595</strong></td>
<td>Treatment Resistant Depression</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>P2 Mid-2021</td>
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<tr>
<td><strong>XPro1595</strong></td>
<td>Alzheimer’s Disease CNS</td>
<td></td>
<td></td>
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<td>Jan 2021 P1b data P2 start mid-2021</td>
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INB03 for treatment of MUC4 positive cancer and LIVNate for NASH Phase II trials begin after resolution of pandemic

<table>
<thead>
<tr>
<th>NK PRIMING PLATFORM</th>
<th>DISEASE FIELD</th>
<th>PRE-CLINICAL</th>
<th>PHASE I</th>
<th>PHASE II (POC)</th>
<th>PIVOTAL</th>
<th>NEXT MILESTONE</th>
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<tr>
<td><strong>INKmune</strong></td>
<td>Myelodysplastic Syndrome ONCOLOGY</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1H21 initiate P1</td>
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</table>

INKmune for treatment of ovarian cancer Phase I trial begins after resolution of pandemic
Innate immune system plays a critical and often overlooked role in host protection and normal function.

50% of all deaths are attributable to inflammation-related diseases.


INNATE IMMUNE DYSFUNCTION

Chronic Inflammation drives Innate Immune Dysfunction are the Causes of Many Diseases

- Neurological Disease
- Cardiovascular Disease
- Infectious Disease
- Autoimmune Disease
- Oncology
- Pulmonary Disease
- Metabolic Disease
- Neurological Disease
- Cardiovascular Disease
- Infectious Disease
- Autoimmune Disease
- Oncology
- Pulmonary Disease
- Metabolic Disease
XPro1595™

Selective sTNF Neutralizer for the Treatment of Neuroinflammation in ALZHEIMER’S DISEASE
ALZHEIMER’S DISEASE (AD)
Driven by Neuroinflammation

- Characterized by chronic inflammation which drives synaptic loss and neurodegeneration
- As of today, there is no treatment for the cognitive decline of AD

1 Alzheimer’s Association

Adapted from Alzheimer’s Association “Changing the Trajectory of Alzheimer’s Disease: How a Treatment by 2025 Saves Lives and Dollars.”
Inflammaging wreaks havoc on patients over time and is a central component of aging and age-related diseases\(^1\).

Dysregulated innate immune responses are widely recognized as playing a major role in the development of neurodegeneration including AD.

Targeting glial activation may prevent much of the damage associated with CNS innate immune dysregulation.

\(^1\)Dumont et al, Front Aging Neurosci. 2019.
White matter (WM) changes are an early event in the development of AD\(^1\).

An elevated WMFW index is a biomarker of neuroinflammation.

Higher WMFW is associated with worse scores on a clinical dementia rating (CDR) based upon numerous studies\(^1\).

Diffusion MRI is used to identify WM changes.

Free water (FW) index measures the diffusion signal explained by unrestricted water to control partial volume effects.

\(^1\) Dumont et al. 2019.
**PHASE 1 AD TRIAL**
Phase IB trial in Alzheimer’s patients with biomarkers of inflammation

**DESIGN**
- 18 patients in 3 cohorts
- Weekly XPro1595™ SubQ injections for 3 months
- Biomarkers of inflammation at 0, 6, 12 weeks

**INCLUSION**
Must have 1 inflammatory biomarker (local lab):
- hsCRP > 1.5
- ESR > 10
- HbA1C > 6%
- APOE4 positive

**ENDPOINTS**
- Safety
- Change in inflammation (blood, CSF, Brain, breath, behavior)
- Change in neuropsychiatric symptoms, cognition, QOL
XPro1595 Reduces Neuroinflammation Within the Arcuate Fasciculus

The arcuate plays a key role in language processing – the ability to understand or express speech – critical to AD

Anticipating Phase 2 clinical study (U.S.) in AD to launch in 2H-2021

% Change in Neuroinflammation

<table>
<thead>
<tr>
<th>% Change</th>
<th>ADNI (N=25)</th>
<th>0.3 mg/kg XPro1595 (N=3)</th>
<th>1.0 mg/kg XPro1595 (N=3)</th>
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<tbody>
<tr>
<td>5.13%</td>
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<td>1.72%</td>
<td>2.33%</td>
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- Premotor cortex
- Broca’s area
- Wernicke’s area

XPro1595 Reduces Whole Brain Neuroinflammation

XPro1595 Reduces Neuroinflammation within the AF

XPro1595 Reduces Neuroinflammation

4.58% XPro1595 (N=6)

- ADNI cohort (N=25)

-40.3%
TNF and NEUROINFLAMMATION ARE VALIDED TARGETS IN AD
Correlation with Changes to Amyloid

RISK OF ALZHEIMER'S DISEASE IN MAN

+800%
Rheumatoid Arthritis

Risk of general population

-60%
Rheumatoid Arthritis + anti-TNF

Adapted from Chou et al. 2016
8.5M claims

Dumont et al Descoteaux Frontiers in Aging Neuroscience 2019
TNF BIOLOGY: Good vs Bad: tmTNF vs sTNF

sTNF is well known, tmTNF is unknown, they do very different things...

- **Soluble TNF (sTNF):** “bad” TNF that is known to cause acute and chronic inflammation and cell death
- **Transmembrane TNF (tmTNF):** “good” TNF improves the immune response, is neuroprotective and promotes remyelination

Adapted from MacEwan et al 2002
XPRO1595: A PRECISION TNF INHIBITOR:

XPro1595 neutralizes sTNF while allowing tmTNF to provide the neuroprotective benefits without the safety risks of current anti-TNF therapies

DIFFERENCES:
Selective vs. Non-Selective sTNF Inhibition

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<th>Selective sTNF Inhibition</th>
<th>Non-Selective sTNF Inhibition</th>
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<tr>
<td>Decreases inflammation</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>Increased risk of infection</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>Increased risk of cancer</td>
<td>Y</td>
<td>N</td>
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<tr>
<td>Causes demyelination</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>Neuroprotective</td>
<td>N</td>
<td>Y</td>
</tr>
<tr>
<td>Enhances neuroplasticity</td>
<td>N</td>
<td>Y</td>
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</table>

Only “GOOD” tmTNF continues to bind to TNFR1 and TNFR2

“BAD” sTNF with DN-TNF does not bind to receptors

DN-TNF neutralizes ONLY sTNF and leaves tmTNF and TNF receptors functional

Adapted from MacEwan et al 2002
HOW XPRO1595 NEUTRALIZES sTNF

Non-glycosylated protein expressed in E. coli

Receptor binding, "small" domain (subunit A)
Receptor binding, (subunit B)
Mutation Y87H
Mutation R31C (for 10kD pegylation)
Mutation A145R

"large" domain (subunit C)
Receptor binding,

see Steed et al., Science, 301, 2003

Inflammatory soluble TNF eliminated
No paracrine signaling through receptors

\[
\text{INFLAMMATORY SOLUBLE TNF ELIMINATED} \rightarrow \text{No Paracrine Signaling Through Receptors}
\]

\[\text{soTNF} \rightarrow \text{Exchange} = \text{No paracrine signaling}\]

DN-TNF neutralizes sTNF without affecting tmTNF
Immunoprotective transmembrane TNF unaffected – Allows juxtacrine cell-cell signaling

\[
\text{DN-TNF NEUTRALIZES sTNF WITHOUT EFFECTING tmTNF} \rightarrow \text{Immunoprotective Transmembrane TNF Unaffected – Allows Juxtacrine Cell-Cell Signaling}
\]

\[\text{tmTNF (ΔTACE site or TACE-inhibitor)} \rightarrow \text{No Exchange = Normal juxtacrine signaling}\]
CURRENT TNF INHIBITORS AND XPRO1595 ARE DIFFERENT

Demyelination is an off-target safety side effect of currently approved anti-TNF therapies

- **NORMAL**
- **CUPRIZONE**
  Model of Multiple Sclerosis
- **ETANERCEPT**
  Anti-inflammatory AND immunosuppressive
- **DN-TNF**
  Anti-inflammatory NOT immunosuppressive

**EXACERBATED DEMYELINATION**

**REMYELINATION**

XPro1595 promotes REMYELINATION

Cuprizone model of demyelination in mice: Prober 2017
UNIQUE MECHANISM EXPANDS MARKET OPPORTUNITY
Currently approved TNF inhibitors contraindicated in most markets

Global TNF inhibitor drug market is estimated to reach $42 billion by 2026

Estimates from ¹GBI Research, ²BCC Research, ³MarketWatch, and ⁴ResearchandMarkets.com.
Targeting Neuroinflammation for the Treatment of TREATMENT RESISTANT DEPRESSION (TRD)
TREATMENT RESISTANT DEPRESSION (TRD)

The Subset of People Who Have Failed At Least Two Anti Depressants

<table>
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<tr>
<th>1 out of 3 MDD patients or 33% of the estimated 7M patients</th>
<th>Economic toll: $64B/year</th>
<th>Higher comorbidity</th>
<th>Chronic course of MDD</th>
<th>TNF biology in TRD</th>
<th>POC studies with anti-TNF blood CRP predicts response</th>
</tr>
</thead>
</table>

Frustrations of psychiatric drug development

- Drug development in psychiatry is a “one size fits all” approach
  - 200+ ways to be diagnosed with depression
  - There are 200+ symptom combinations that will lead to a depression diagnosis
  - No Biomarkers
  - High placebo response

- Drug development failure rate in psychiatry
  - in Phase II: 35% in Phase III: 65%

- Effectiveness of approved drugs
  - 50% do not achieve remission
  - 33% have no response (treatment resistant)

THE PROBLEM
MDD, TRD and Inflammation

1 of 5
21 million people at any given time will have depression at some point in their life (MDD).

1 of 3
7 million people don’t respond to current treatments (TRD).

1 of 3
2.3 million people will have elevated levels of inflammation.
20 million adults have major depressive disorder (MDD) per year

One third (~7 million patients) have Treatment Resistant Depression (TRD)\(^1\)

Cost of TRD is nearly $64 billion\(^2\)

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\(^1\) Mrazek, et al. Psychiatry Serv 2014.
INMB APPROACH: TNF PREDICTS TRD

Anti-TNF Therapy Improves Depression in Patients With Elevated Inflammatory Biomarkers

Anti-TNF treatment reduces depressive symptoms in patients with elevated biomarkers of inflammation

Inflammation-associated deficits in reward circuitry mediate the relationship between CRP and anhedonia in patients with depression

Treatment Response (≥50 reduction in HAM-D-17 at any point during treatment) Miller 2011
PHASE 2 TRD TRIAL
Supported by Small Business Innovation Research (SBIR) grant from the National Institutes of Health

DESIGN
- Six-week double blind placebo-controlled study of XPro1595
- 45 patients in XPro1595 at 1 mg/kg
- 45 patients in placebo control group
- Biomarkers of inflammation at 0, 2, 6 weeks

INCLUSION
- Failed 2 courses of antidepressants
- C-reactive protein (CRP) levels >3 mg/L
- Anhedonia

ENDPOINTS
- Primary: Improve functional connectivity and reduce biomarkers of inflammation
- Secondary: Improve clinical measures of motivation

BIOMARKERS OF INFLAMMATION

MRI
WMFW as measure of neuroinflammation

BLOOD
Inflammation (Roche NeuroTool kit)

fMRI
Functional connectivity between motivation and reward centers in brain

BEHAVIORAL
Motivation
Clinical scales
Targeting CYTOKINE STORM may Prevent Catastrophic Complications of COVID-19
WHAT BRINGS PATIENTS TO THE HOSPITAL WITH COVID-19?

At time of hospitalization, the Cytokine Storm is making patients sick, not the virus...

Focusing on the Cytokine Storm, not the virus, should make patients better.

Targeting the Master Cytokine with QUELLOR should tame the Cytokine Storm.

The dysregulated innate immune response to the virus causes Cytokine Storm.
TNF is the “MASTER” of the Cytokine Storm

- Block sTNF and downstream pro-inflammatory cytokines should decrease
- Synergism of TNF-α and IFN-γ triggers inflammatory cell death, tissue damage, and mortality

Adapted from Karki et al 2020
TNF CAUSES ENDOTHELIAL CELL ACTIVATION
The Real “Villain” in COVID-19

- TNF activates endothelial cells
- Up-regulation of Tissue Factor promotes coagulopathy
- Blood clots contribute to pulmonary, renal, CNS and cardiovascular disease

McGonagle et al. Lancet Rheumatol., 2020
PHASE 2 TRIAL: QUELLOR™
Treating Pulmonary Complications of COVID-19

DESIGN
- SOC vs SOC+Quellor™ 1mg/kg subQ day 1 and 7 (if in hospital)
- Patient discharged based on clinical status or final study visit day 28
- 366 patients randomized 1:1

INCLUSION
- COVID-19 infection with room air SaO2<94%
- One or more medical/demographic comorbidities: age≥60; hypertension, cardiovascular disease, BMI≥30; diabetes, Black or Hispanic race

ENDPOINTS
- Primary: Need for mechanical ventilation in 28 days
- Secondary: Transfer to ICU, new onset neurologic, cardiovascular or thromboembolic disease, development of renal failure or death

GOAL: PREVENT PROGRESSION TO CATASTROPHIC COMPLICATIONS

First 100 patients “proof-of-concept” to GO/NOGO decision
If DSMB says “GO”, follow-on study 266 patients
Provides essential signals to patients’ NK cells to target and kill tumor cells. INKmune™ is not antigen-specific so there is no need to identify the specific tumor antigen.
RESIDUAL DISEASE IS THE CAUSE OF CANCER RELAPSE

Difference Between Survival and Relapse is NK Failure to Eradicate Residual Disease

- Prevention of relapse can be the difference between survival and death in patients with cancer
- Relapse is caused by hidden residual cancer cells
- The innate immune system’s natural killer (NK) cells are responsible for finding and targeting residual cancer cells to provide long lasting remission, not T cells

Adapted from Lowdell et al Br J Haematology. 2002.
INKmune™ IS A BIOLOGIC SYSTEM TO DELIVER ESSENTIAL PRIMING SIGNALS TO PATIENTS’ RESTING NK CELLS

INKmune™ CELLS PRIME NK CELLS TO ELIMINATE RESIDUAL DISEASE

RELAPSE DUE TO RESIDUAL DISEASE

Resting NK Cell

Cancer Cell

Tumor cells down-regulate surface molecules which prime NK cell activity, thus evading NK cell killing allowing the cancer to grow

INKmune™ ADVANTAGES:

✓ Universal—Potentially effective in blood cancers and solid tumors
✓ Off-the-shelf therapy
✓ Easy to use

Provides the innate signals necessary to prime a patient’s resting NK cells, empowering the immune system to eliminate residual cancer cells
HIGH-RISK MYELODYSPLASTIC SYNDROME (MDS)

- MDS is an **incurable disease**
- One-third of all MDS cases evolve to become acute myeloid leukemia (AML)
- **Survival for patients with high-risk MDS is dismal**
- Patients with high-risk MDS have functionally defective NK cells
- Level of NK dysfunction is predictive of overall survival
- By 2022, global MDS drug market is expected to reach **$2.4 billion** USD

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1 Kiladjian et al 2006
2 Tsirogianni et al 2019
3 Grand View Research
INKmune™ PHASE 1 STUDY
Phase I Trial in High-Risk MDS

DESIGN
- Open-label dose escalation study of intravenous INKmune enrolling 9 patients

INCLUSION
- Patients with MDS with excess blasts

ENDPOINTS
- Primary: Evaluate the safety and tolerability of INKmune when given intravenously
- Secondary:
  - Assess the change in [or effect upon] number and percentage of blasts in peripheral blood and bone marrow
  - Assess the overall response rate in subjects administered INKmune™ using WHO criteria
  - Assess the duration of response
MILESTONES AND FUTURE CATALYSTS

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<tr>
<th>Q1</th>
<th>Q2</th>
<th>Q3</th>
<th>Q4</th>
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<tbody>
<tr>
<td>INB03°</td>
<td>INB03° Phase 1 Readout</td>
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<tr>
<td>2019</td>
<td>$1 Million Park the Cloud Award – Alzheimer’s Association</td>
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<tr>
<td>$500,000 ALS Association award</td>
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| 2020 | |
| XPro1595 Interim readout showing decreases neuroinflammation |
| $25 M Capital Raise |

| 2021 | |
| XPro1595 Expanded P1 readout |
| INKmune° First Patient high-risk MDS Trial |
| XPro1595 TRD Phase 2 |

NASDAQ Listing

- INB03°
  - Initial Data on INB03 Phase 1
  - XPro1595 First Patient Enrolled in Phase 1 AD Trial
  - XPro1595

- $25 M Capital Raise

- $2.9M Grant from NIMH for TRD Phase II

- IND for CV-19

- INKmune°
  - First Patient Ovarian Cancer

- XPro1595
  - Phase 2 AD initiation

- LIVNate°
  - INB03° Phase 2
DN-TNF PATENTS*

2024
pegylated DN-TNF (licensed from Xencor)

2032
Methods for treatment of neurologic disease

2035
use for treatment of cancer (issued in US)

2039
use for treatment of NASH

2040
use for immune mediated complications from COVID-19/CRS

NK PATENTS*

2035
use for treatment of cancer

2039
INB16 composition-of-matter

FUTURE

Broad Platforms allow for continual R&D and new IP

* Subject to issuance by patent granting authority
MANAGEMENT TEAM

Raymond J. Tesi, M.D.
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CFO

Mark W. Lowdell, Ph.D., FRCPath, FRSB, CSO

Christopher J. Barnum, Ph.D.
Director of Neuroscience

Joshua S. Schoonover, Esq.
Assoc. General Counsel
Free water = edema/swelling = inflammation
NEUROINFLAMMATION – FREE WATER WHITE MATTER

Measures Whole brain inflammation

Safe White Matter Mask

Measure Inflammation in specific white matter tracts

Arcuate fasciculus
Uncinate fasciculus
Inferior fronto-occipital fasciculus
Splenium of the corpus callosum
Inferior longitudinal fasciculus
INB03™

DN-TNF Options in Immuno-Oncology for Treatment of Resistance to Immunotherapies
~20% of women with breast cancer are HER2+
~25% have metastatic disease
~30% will develop brain metastasis
MUC4 expression predicts trastuzumab resistance

**PROBABILITY OF DISEASE-FREE SURVIVAL**

- **TRASTUZUMAB RESISTANCE IN BREAST CANCER**
- **INBO3 CHANGES TME IN VIVO**

**JIMT-1 BREAST CANCER INTO NUDE MOUSE**

**FEWER MYELOID CELLS**

**MORE ACTIVATED NK CELLS**

InmuneBio 41
INB03 REVERSES TRASTUZUMAB RESISTANCE

1st – It Modifies immunology of TME and DECREASES MYELOID CELLS, INCREASES NK CELLS IN VIVO
2nd – It Decreases MUC4 Expression – MUC4 Expression resistance to Trastuzumab and promotes metastasis

Model of Metastasis: Downregulation of MUC4 prevents wound closure in JIMT-1 TUMORS

MUC4 BLOCKS TRASTUZUMAB BINDING
Soluble TNF and MUC4 blocking Trastuzumab binding

MUC4 BLOCKS FUNCTION OF TRASTUZUMAB CONJUGATES

Using MUC4 silencing siRNA confirms MUC4 causes resistance to trastuzumab based immunotherapy – naked or as part of conjugate (Schillaci 2017)
CONCLUSIONS FROM INB03™ PHASE I DATA

Open Label, Biomarker Directed, Dose-escalation Trial In Patients With Advanced Solid Tumors And Elevated Markers Of Inflammation

PHASE I SUMMARY
INB03 has been tolerated across multiple dose levels
- No SAE, No DLT
- Well tolerated
- 1mg/kg once a week will be carried into Phase 2
- Dose provides robust trough drug levels
- Pharmacologically active: >50% decrease of IL6 in blood
- Results informed Phase 2 design

ENROLLMENT CRITERIA:
Advanced solid tumors
hsCRP > 4 mg/L
Treatment: INB03 subQ once a week
3 cohorts of 3: 0.3, 1.0, 3.0 mg/kg

% change of inflammatory biomarkers after 28 days of INB03 treatment

CRP
51.42 mg/L
SD (29.42)
-29%

CCL2
773.527 mg/mL
SD (761.299)
-50%

IL6
5.3 kg/mL
SD (1.3)
-41%

SAMPLE
11 patients treated

DEMOGRAPHIC
Age (median): 54 Gender: 6M/5F

DISEASE
Ovary, Mesothelioma; RCC, Lung; Prostate; Colon, Cholangial, other

PREVIOUS LINES OF THERAPY
3 (range:2-5)

INB03 DURATION
74 days (21-119d)
- No drug related SAE
- All discontinuation due to disease progression

ENROLLMENT CRITERIA:
Advanced solid tumors
hsCRP > 4 mg/L
Treatment: INB03 subQ once a week
3 cohorts of 3: 0.3, 1.0, 3.0 mg/kg
GOAL: IMPROVE RESPONSE TO IMMUNOTHERAPY

Modifying the TME To Reverse Resistance To Immunotherapy

- Metastatic HER2+ breast cancer resistant to immunotherapy
  - Trastuzumab resistant
  - Immune checkpoint resistant

- MUC4 expression is a biomarker for trastuzumab resistance and an immunosuppressive TME (immune desert)

- Hypothesis: elimination of MUC4 expression improves response to trastuzumab and “improves” TME

- Two step plan:
  - Demonstrate INB03 decreases expression of MUC4 and eliminates immune desert in metastatic HER2+ breast cancer
  - Addition of immune checkpoint inhibitors will improve outcome

Twofer1: PHASE II IN HER2+
METASTATIC BREAST CANCER

Step 1: Eliminating MUC4 and Modifying the TME

- Metastatic MUC4+/HER2+ breast cancer on trastuzumab based immunotherapy

- Biopsy proven MUC4 expression with quantification of myeloid and lymphoid populations of TME

- n=25 patients on ANY trastuzimab based therapy

- Add INB03 to treatment regimen
  - INB03 1mg/kg/week subcutaneous injection

- Repeat biopsy at 4 weeks

- Predicted outcome:
  - Decrease MUC4 expression
  - Decrease myeloid population of TME
  - Increase CD8+ lymphoid population of TME
Addressing the Link Between Liver Fibrosis, the Innate Immune Cells of the Liver and Inflammation, a Pleiotropic Approach
THREE CYCLES OF INFLAMMATION LEAD TO NASH

PERIPHERAL INFLAMMATION
- Obesity
- Insulin Resistance

REGIONAL INFLAMMATION
- Intestinal Inflammation
- Mesenteric Fat

LOCAL INFLAMMATION
- Lipotoxicity
- Innate Immune Activation

STEATOHEPATITIS (NASH)
- Healthy Liver
- Fatty Liver

LIVNate™ REDUCES INSULIN RESISTANCE
With No Weight Change
# STAM MODEL

LIVNate™ Reduces Hepatocyte Death and Fibrosis *In Vivo With No Change in Total Body Weight*

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<th>Parameter (mean ± SD)</th>
<th>Control</th>
<th>LIVNate™</th>
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<tr>
<td>Sirius Red positive area (%)</td>
<td>0.96 ± 0.29</td>
<td>0.69 ± 0.23</td>
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<table>
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<td>NAS (mean ± SD)</td>
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NASH TRIAL DESIGN
Phase IIa Biomarker Directed Trial

Phase IIa open label trial
- Patients with F2/3 NASH by non-invasive studies with >10% fat in liver with LIVNate™ 1mg/kg SQ treatment with for 12 weeks
- PIFF and non-invasive biomarkers at 0, 6 and 12 weeks
  - Open label so biomarkers “visible” at 6-week time point

N = 20 patients
Expected results
1. Decrease in liver fat
2. Improvement in liver function tests
3. Improvement in insulin resistance.

Exploratory end-points
- Improvement in breath test
- Decrease in intestinal leak
David Szymkowski, PhD
David E. Szymkowski leads the immunology group as Vice President of Cell Biology at Xencor Inc where he is focused on translational development of Fc-engineered and bispecific antibodies for the treatment of autoimmune diseases, allergic diseases, and cancer. Prior to joining Xencor in 2002, Dr. Szymkowski was a principal scientist in the respiratory group at Roche Bioscience in Palo Alto, CA. With 25 years of drug and biotech R&D experience at Roche and Xencor, Dr. Szymkowski has been instrumental in 10 IND submissions, co-authored over forty papers and reviews, is an inventor on over a dozen patents, and speaks frequently on the development of antibody therapeutics and other biologics. He received his B.A. at Johns Hopkins University and his Ph.D. in molecular and cell biology from Penn State, and completed a postdoc at the Imperial Cancer Research Fund (U.K.).

J. Kelly Ganjei
Mr. Ganjei has been a director since September 2016. He is Chief Executive Officer of Cognate BioServices, Inc., a position he has held since 2011. Mr. Ganjei has over 20 years of executive experience within the life science, venture capital and IT sectors and has lead companies through various stages of development. Prior to joining Cognate, Mr. Ganjei was Principal at an SBA venture capital firm where he instrumental in supporting deal flow with a specific focus on regenerative medicine, immunotherapy and cell therapy investment opportunities. He began his career at the National Institutes of Health. Mr. Ganjei has published numerous scientific, peer-reviewed papers and has been a speaker and presenter at various business forums. Mr. Ganjei received his B.S. in Microbiology from the University of Maryland College Park in 1995.

Timothy Schroeder
Mr. Schroeder has been a director since December 2016. Mr. Schroeder has more than 35 years of clinical and academic industry experience in global drug and device development programs. He is CEO of CTI Clinical Trial and Consulting Services, a multi-national research firm with locations in North America, Europe, Latin America and Asia-Pacific. Prior to founding CTI, Mr. Schroeder was a faculty member of the University of Cincinnati College of Medicine. He previously served as Executive Vice President of Clinical Development at SangStat Medical Corporation, a firm he co-founded. Mr. Schroeder is a board member for more than a dozen corporate and non-profit organizations. He was named as EY Entrepreneur of the Year in 2015 and was recognized as Top Leader by the Enquirer Media in 2016.

Edgardo (Ed) Barachchini, PhD
Mr. Eduardo ("Ed") Barachchini has been a director since July 2019. He is also current a member of the board of directors of 4D Pharma PLC. Prior to providing biotech consulting services since September 2018, Ed was chief business officer of Xencor, Inc. Ed was associated with Metabasis Therapeutics, initially as vice president of business development, and later as SVP of business development. Ed holds over 25 years of experience in structuring and negotiating research and development partnerships, mergers and acquisitions, and licensing agreements. He has personally, negotiated more than 80 business transactions with multinational and Asian pharmaceutical firms, biotechnology companies, and prominent academic institutions. Ed serves on the boards of several investment firms, leading to transactions valued in excess of $5.3 billion. Additionally, he has been a key member of executive teams that have raised over $850 million in private and public financing, and that have successfully completed two IPOs. Ed received his MBA from the University of California, Irvine, his PhD in molecular and cell biology from the University of Texas at Dallas, and his B.S. in microbiology from the University of Notre Dame.

Marcia Allen
Marcia Allen has been a director since November 2019. Ms. Allen was a founder and served as CFO and Director of The Movie Group, (AMX) the originating company which is today Lionsgate Entertainment (NYSE). She has more than 25 years with mergers and acquisitions, corporate finance and CFO and CEO experience. Ms. Allen was a Chief Financial Officer and Corporate Development Officer for W.R. Grace & Co. (NYSE) and was based both in Newport Beach, CA with the Restaurant Division and in its New York headquarters. Ms. Allen moved to California from Pennsylvania where she was part of the founding group of Venti-Tuesday, Inc., (NYSE) a national restaurant chain. She relocated to join Taco Bell, Inc. as the Company’s Chief Financial Officer where she structured and facilitated the acquisition of Taco Bell, Inc. by PepsiCo, Inc. She currently serves as the chairperson of the audit committee for Ark Restaurants Corp (Nasdaq). Ms. Allen received a Bachelors, Finance and Accounting degree from the University of Tennessee.

Scott Juda, JD
Mr. Juda has been a director since March 2018. Mr. Juda is Manager and co-founder of Fossick Capital, a technology-focused hedge fund. Mr. Juda co-founded The Juda Group, Inc., an institutional capital markets focused broker-dealer division of CCM, where he served as Chief Executive Officer from 2012 to 2016. Before that, Mr. Juda was at SMH Capital from 2002 to 2011, serving as a Managing Director in the Investment Banking Group and Chief Operating Officer of The Juda Group subsidiary. From 2000 to 2002, Mr. Juda was an institutional sales-trader for Sutro & Co. From 1997 to 2000, Mr. Juda practiced corporate and securities law at Buchalter Nemer LLP. Mr. Juda received his bachelor degree from the University of Southern California and his juris doctor from the University of Pepperdine School Of Law. Mr. Juda is a member of the State Bar of California.
TWO HYPOTHESIS BEHIND NK IMMUNOTHERAPY

Difference Between Survival and Relapse is NK Failure to Eradicate Residual Disease

- Cancer patient NK cells are terminally defective and must be replaced with NK from healthy donors
  - Nanktwest
  - Fate
  - Takeda
  - Glycostem

- Cancer patient NK cells can be enhanced to mediate autologous tumor killing
  - Rituximab / Herceptin etc
  - IL15RA
  - Ex-vivo activated/expanded NK cells
  - INmuneBio INKmune
REFRACTORY OVARIAN CANCER CLINICAL

Phase 1 Open-label Study of Intraperitoneal INKmune™ in Patients with Relapsed Platinum-resistant, Platinum-refractory, or Platinum-Intolerant Ovarian Cancer

- Refractory CaOva is an incurable disease
- Despite optimal surgery and paclitaxel–carboplatin chemotherapy approximately 70% of patients with primary ovarian cancer will relapse in the first 3 years
- Objective response rates to second-line therapies such as weekly paclitaxel, doxorubicin, topotecan and gemcitabine are in the range of 20-30% and median overall survival is <1 year (Gordon et al 2000, Markman et al 2002)
- The ascites of CaOva patients contains plentiful NK but few T cells; suggesting that the principal IR to CaOva is NK mediated (supported by failure of CPIs)
- Our in vitro data show that defective NK cells from CaOva ascites can be primed with INKmune to kill NK-resistant CaOva tumor cells

**Primary**

1. To evaluate the safety and tolerability of INKmune when given intraperitoneally

**Secondary**

1. To assess progression-free survival
2. To assess the overall response rate using RECIST v1.1 and/or GCIG CA 125 criteria
3. To assess the duration of RECIST and/or GCIG CA 125 response

**Exploratory**

1. To assess the pharmacodynamics of INKmune