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. 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.
Building a *Next Generation* immunology company

- **Immunology focused - platforms in immuno-oncology and neurodegenerative diseases**
- **Target “ignored” elements of immune system**
  - Innate immune system
  - TME (tumor microenvironment)
  - Neuroinflammation is a cause of neurodegenerative disease
- **Multiple clinical programs allows for robust news flow**
- **Platform technologies provide opportunities for therapeutic expansion as resources become available**
- **Experienced management team**
- **Multiple programs provide sum-of-parts value**
Financial Highlights

- **Nasdaq:** INMB
- **Cash 6/30/19:** $9.4 million
- **Debt:** $0
- **Common Shares outstanding:** 10.8 million

**Insider ownership:** ~50%

**Notable Shareholder:** Xencor (XNCR)

**XNCR outstanding licensing fee:** buy 10% of INMB at $100m pre
Our Vision: Become the Leader in Reprograming the Innate Immune System for the Treatment of Diseases
<table>
<thead>
<tr>
<th>INMB Pipeline &amp; Clinical Status</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>INB03</strong> for resistance to immunotherapy</td>
</tr>
<tr>
<td><strong>INKmune</strong> to eliminate <strong>Minimal Residual Disease (ovarian cancer)</strong></td>
</tr>
<tr>
<td><strong>XPro1595</strong> for Neuro-inflammation <strong>(Alzheimer’s disease)</strong></td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>
Pipeline Market Opportunity

• **XPro1595 - Alzheimer’s/dementia market** *
  – 5.8 M patients in US
  – 2019 est cost to care for US AD patients $280B

• **INKmune - Cancer residual disease market** **
  – 1,735,350 new cases and 609,640 people will die
  – Initial targets: Ovarian Cancer and High Risk MDS

• **INB03 - Resistance to Cancer Immunotherapy market** ***
  – Cancer immunotherapy market est. >$119B in 2021
  – Primary and secondary resistance to cancer immunotherapy common
  – Combination therapy with INB03 should decrease the number of resistant patients.
  – Initial targets: Breast or Renal Cell Cancer

INB03 for treatment of cancers resistance to immunotherapy
Resistance to immunotherapy case study: CPI

- Small cell Lung
- Melanoma
- RCC
- Bladder
- Ureothelial
- Merkel Cell
- NSCLC
- Gastric
- HNC
- HCC
- Microsatellite instability

Eligibility vs Benefit CPI
US patients 2018

INMB non-confidential3Q19.v2
INB03: novel inhibitor of soluble TNF

- 17 kDal mutated protein +10 kDal linear PEG
- Identical to human TNF except for 6 amino acid substitutions
- Once a week dosing by subQ injection
Targeting sTNF: fibrosarcoma model

**↓MDSC in tumor**
- Decreased sTNF decreases differentiation and proliferation of MDSC

**↑CD8+ lymphocytes in tumor**
- tmTNF promotes NK/DC crosstalk that recruits CD+8 TILs

**↑112 day survival**
- ↓MDSC with ↑CD8+TIL results in better survival

---

from Vujanovic 2016
INB03 reverses trastuzumab resistance

JIMT-1 breast cancer
into nude mouse

T=trastuzumab
DN=INB03

from Schillaci 2018

INMB non-confidential3Q19.v2
Open label, biomarker directed, dose-escalation trial in patients with advanced solid tumors and elevated markers of inflammation

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
Goals:
1. Determine safety
2. Determine Phase II dose
3. Demonstrate PD effect
4. Determine Phase II strategy

“Top-Line” Results
1. Safe and well tolerated
2. Phase II dose: 1mg/kg
3. Pharmacodynamic effect confirmed
4. Planning Phase II study

11 of 12 patients treated

Demographics: age (median): 74 yo gender: 6M/5F

Disease: ovary; mesothelioma; RCC; lung; prostate; colon, cholangio, 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
1 to 1 ratio DN-TNF to TNF

4 DNs + 4 TNFs =

Flag  His  Flag  +
       His

[wTNF] = 50 μg/ml; [DN-TNF] = 50 μg/ml
Exchange at equilibrium in PBS + 0.1 mg/ml BSA
Separation & quantitation by native PAGE

Varying ratios DN to wt (native PAGE)

At equilibrium (binomial distribution):
XPro1595 = TNF: Eliminates 75% TNF (1:3:3:1)
2x XPro1595 > TNF: Eliminates ~88.9% TNF
5x XPro1595 > TNF: Eliminates ~97.2% TNF
10x XPro1595 > TNF: Eliminates >99.2% TNF
100x XPro1595 > TNF: Eliminates >99.99% TNF
**Goal:** blood trough level at least 2 logs greater than blood TNF level

**Conclusion:**
- 1mg/kg dose provides adequate drug level
- Above 3 log target 8 hours after first dose

Patients below 3 log target drug level:

<table>
<thead>
<tr>
<th>Day</th>
<th>0.3mg</th>
<th>1.0mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>day 8</td>
<td>2 of 3</td>
<td>0 of 3</td>
</tr>
<tr>
<td>day 15</td>
<td>2 of 3</td>
<td>1* of 3</td>
</tr>
<tr>
<td>day 22</td>
<td>2 of 3</td>
<td>0 of 3</td>
</tr>
<tr>
<td>day 29</td>
<td>1 of 2</td>
<td>0 of 3</td>
</tr>
<tr>
<td>day 43</td>
<td>1 of 2</td>
<td>0 of 2</td>
</tr>
<tr>
<td>day 57</td>
<td>0 of 1</td>
<td>0 of 2</td>
</tr>
</tbody>
</table>
**Phase I summary**

- Safe at all doses tested
  - No SAE, No DLT, Well tolerated
- 1mg/kg once a week will be carried into Phase II
  - Dose provides robust trough drug levels
- Pharmacologically active at all dose levels
  - >50% decreases IL6

**Phase II combination trial**

- Design variables:
  1. Combination with what!
  2. Which tumor type
  3. Interaction of efficacy and safety
  4. Partnering opportunities
  5. First line vs second line therapy
XPro1595 (for the treatment of Alzheimer's Disease/dementia)
AD: the problem...

Nerve cell death → Synaptic dysfunction → Cognitive Decline
Does eliminating TNF prevent AD?

1. Risk of AD in RA patients 8 times higher*
2. Risk of AD in RA patients treated with anti-TNF therapy is 0.4* (60% lower)

* In RA patients compared to patients without RA

from Chou et al. 2016
INMB non-confidential3Q19.v2
**The good:** confirmation of findings from database of 63M patients

<table>
<thead>
<tr>
<th>TNF inhibitor</th>
<th>Odds Ratio</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Enbrel</td>
<td>0.31</td>
<td>21,280</td>
</tr>
<tr>
<td>Humira</td>
<td>0.35</td>
<td>20,130</td>
</tr>
<tr>
<td>Remicade</td>
<td>0.54</td>
<td>10,320</td>
</tr>
</tbody>
</table>

Adapted from Xu, Zhu and Gurney AAIC 2019

**The bad:** ↑ risk of cancer, infection and MS increased when treated with non-selective TNF inhibitor
Alzheimer’s Association awards INmune Bio Part the Cloud Grant - $1M USD translational funding to study XPro1595 in Alzheimer's disease
Phase I biomarker directed trial of patients with inflammation and proven Alzheimer’s Diseases

18 patients in 3 cohorts

• Weekly XPro1595 subQ for 3 months
• Biomarkers of inflammation at 0, 6 and 12 weeks
 • Endpoints:
  – Safety
  – ↓ neuro-inflammation measured in: blood, cerebrospinal fluid, brain, breath and behavior
  – Measures of cognition, neuro-psychiatric symptoms and QOL
Immunologic biomarkers: AD

- MRI
- CSF
- Breath
- Blood

Free Water content of white matter

Behavioral biomarkers of inflammation
- Sleep disorders
- Depression
- Psychosis
- Apathy
- Aggression

Equilibrium & Neurodegeneration

Inflammation & Neurodegeneration

Volatile Organic Compounds
INKmune (for the treatment of Ovarian Cancer and high risk MDS)
Problem: Minimal Residual Disease (MRD) causes relapse

Difference between survivors and relapsers is MRD
INKmune Primed NK Cells Kill Cancer

Human ovarian cancer cells plus patient’s own NK cells

Human ovarian cancer cells with patient’s NK cells after INKmune treatment
**INKmune Clinical Trial**

**INKmune causes in vivo priming so patient’s NK cells can kill their cancer by targeting naturally occurring tumor antigens**

**Phase 1/2 study in relapsed/refractory CaOva**

Platinum resistant/refractory patients with residual disease

- Treatment: INKmune -6 doses
- Endpoints:
  - Safety
  - ↑ NK activation and tumor killing
  - ↓ tumor burden*

INKmune therapy weekly times three then monthly

*when in Phase II
INmune Bio = Innate Immunity
Milestones

- February 1, 2019 – IPO closed
- February 4, 2019 – NASDAQ Capital Markets listing
- Feb19 – Part the Cloud award – Alzheimer’ Assoc
- 3Q19 – initial data on INB03 Phase I
- 3Q19 – first patient enrolled XPro1595 AD trial
- 4Q19 – first patient enrolled INKmune CaOva
- 1H20 – initiate INB03 combination Phase II trial
- 1H20 – initial data from INKmune Phase I
- 2H20 – Start INKmune Phase II CaOVA
- 2H20 – XPro1595 AD Phase II trial
Experienced Leadership

Management

RJ Tesi, MD
Chief Executive Officer & Chief Medical Officer

Mark Lowdell, PhD
Chief Scientific Officer

David Moss
Chief Financial Officer

Board of Directors

J. Kelly Ganjei
CEO of Cognate

Timothy Schroeder
CEO and Founder of CTI

David Szymkowski, PhD
VP of Cell Biology

Scott Juda
Founder and Managing Member, Fossick Capital

Edgardo (Ed) Baracchini

For complete biographies, visit www.inmunebio.com
INB03/XPro1595

- 2021 – composition-of-matter (licensed from Xencor)
- 2032 – Methods for treatment of neurologic disease
- 2035 – use for treatment of cancer (licensed from University of Pittsburgh)

INKmune

- 2035 – use for treatment of cancer
- Multiple filings in process

Continuing to expand patent portfolio for both platforms
How XPro159/INB03 Neutralizes sTNF

Normal sTNF activity

Active TNF + XPro1595 → Heterotrimers or No sTNF activity

Active TNF + Inactive heterotrimers → Heterotrimers cannot bind TNFR

sTNF bind TNFR → Inflammation

NO Inflammation
Why is Xpro1595/INB03 Different?

**Normal TNF Biology**

- TNFR1
- TNFR2

sTNF (bad)  ---------->  tmTNF (good)

**INB03 does not neutralize tmTNF**

INB03

tmTNF binds TNFR with normal function

**Approved TNF inhibitors**

TNFR1  ---------->  TNFR2

Premax (red cross)  sTNF (bad)  ---------->  tmTNF (good)

**XPro1595/INB03**

TNFR1  ---------->  TNFR2

Prevent function of sTNF and tmTNF

TNFR1  ---------->  TNFR2

sTNF (bad)  ---------->  tmTNF (good)

Neutralizes sTNF allowing normal of TM TNF
Precision Therapy: Targets sTNF not tmTNF

Inflammatory soluble TNF eliminated:
No paracrine signaling through receptors

Immunoprotective transmembrane TNF unaffected:
Allows juxtacrine cell-cell signaling

Inflammatory soluble TNF eliminated:
No paracrine signaling through receptors

Immunoprotective transmembrane TNF unaffected:
Allows juxtacrine cell-cell signaling

Log [anti-TNF (µg/ml)]

Caspase activity

INB03 etanercept infliximab adalimumab vehicle

INB03 (recombinant or physiological)

Exchange = No paracrine signaling

Normal juxtacrine signaling

Zalevsky et al., J. Immunol. 2007
Repeat Dose Modeling of XENP1595 in Humans
1mg/kg Administered SC Every 5, 7, or 10 Days

Allometric scaling of cyno PK data suggests weekly dosing of 1 mg/kg SC in humans will sustain >1 μg/ml trough serum levels of INB03

V = 494.9 ml
k_{01} = 3.552/day
k_{10} = 0.25/day

Volume and k_{ab} are the same as for cyno. The k_{el} term is reduced by ~2x
• INB03 was engineered for single, site-specific clean pegylation (10kD PEG/monomer x 3 monomers)
• Unpegylated XENP550 is produced through E. coli expression
• INB03 is a nonglycosylated small (17 kD monomer) & stable homotrimeric protein, with superior manufacturing vs. mAbs & Fc fusions
• Current production yield = ~300mg/liter
• Yd improvement and new batch manufacturing planned for after IPO
Eligibility vs Response to CPI - US

- 44% US cancer patients eligible for CPI
- 29% of cancer patients who receive CPI respond
Resistance to Immunotherapy: CPI

Cancers affected by immunology drugs
Respond to immunology drugs

Percentage vs. Year from Haslam 2019

- 2011: 0%
- 2012: 0%
- 2013: 0%
- 2014: 0%
- 2015: 41%
- 2016: 47%
- 2017: 47%
- 2018: 12%

Haslam 2019
Different Approach to Immuno-oncology

- Effector cells: T and NK cells
- Protector cells: MDSC, TAM, TAN, Treg, CAF
MDSC Predict Resistance to CPI

- INB03 targets MDSC
- CPI + INB03 should overcome MDSC based resistance by eliminating MDSC so CPI become effective
INB03 decreases MDSC by “starvation”

No pSTAT3 stops differentiation and proliferation of myeloid cells to MDSC

↑ NK/DC recruits CD8+ TIL to tumor

downward arrow MDSC causes downward arrow TAM and Treg

tmTNF required for NK/DC crosstalk

INMB non-confidential3Q19.v2
INB03 HER2/MUC4 Appendix
• ~30% of women with breast cancer are HER2+
• 30% - 50% patients have 1° or 2° resistance to trastuzumab
• MUC4 expression predicts trastuzumab resistance

INMB non-confidential3Q19.v2
INB03 Reverses Trastuzumab Resistance

INB03 overcomes trastuzumab resistance in JIMT-1 tumors

MUC4 blocks trastuzumab binding

INB03 decreases MUC4 expression

INB03 overcomes trastuzumab resistance in JIMT-1 tumors

T-trastuzumab
DN- INB03

Schillaci-SABCS2018
Alzheimer’s Disease Appendix
Cancer vs. Alzheimer’s disease/Dementia

U.S. HEALTHCARE COST VS. U.S. VENTURE CAPITAL FUNDING OF NOVEL R&D FOR HIGHLY PREVALENT CHRONIC DISEASES

AD is an expanding, expensive and intractable disease that defies a therapeutic solution

- **Problem:** synaptic pruning and nerve cell death
- **Solution:** reduce neuroinflammation
- **Drug:** XPro1595
- **Target:** activated glial cells in the brain
- **Status:** entering Phase I trial in patients with mild to moderate Alzheimer’s disease
Majority of genes associated with AD expressed in innate immune cells

<table>
<thead>
<tr>
<th>Cell type</th>
<th>-log10(pvalue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Microglia</td>
<td></td>
</tr>
<tr>
<td>Hippocampal CA 1 pyramidal neurons</td>
<td></td>
</tr>
<tr>
<td>Endothelial cells</td>
<td></td>
</tr>
<tr>
<td>Prefrontal glutamergic neurons 2</td>
<td></td>
</tr>
<tr>
<td>Prefrontal glutamergic neurons 1</td>
<td></td>
</tr>
<tr>
<td>Oligodendrocytes</td>
<td></td>
</tr>
<tr>
<td>Astrocytes 2</td>
<td></td>
</tr>
<tr>
<td>Oligodendrocyte precursor cells 1</td>
<td></td>
</tr>
<tr>
<td>GABAergic interneurons 1</td>
<td></td>
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<tr>
<td>Neuronal stem cells</td>
<td></td>
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<tr>
<td>GABAergic interneurons 2</td>
<td></td>
</tr>
<tr>
<td>Oligodendrocyte precursor cells 2</td>
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<tr>
<td>Hippocampal CA 3 pyramidal neurons</td>
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</tr>
<tr>
<td>Astrocytes 1</td>
<td></td>
</tr>
<tr>
<td>Dentate gyrus granule neurons</td>
<td></td>
</tr>
</tbody>
</table>

Jansen2019
Genetics implicates every aspect of the immune cell is dysfunctional

McQuade & Blurton-Jones, 2019
Chronic inflammation: real disease….bad name

Acute inflammation
fast and furious, coming and going quickly for the benefit of the patient.

Chronic inflammation
low, slow inflammatory response that doesn’t end and harms the patient.
Inflamming and cognitive decline

Atherosclerosis Risk in Communities Study (ARIC)

Honolulu-aging Study

<table>
<thead>
<tr>
<th>Quartiles of hs-CRP</th>
<th>Risk of Dementia</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;0.34mg/L</td>
<td>1.0</td>
</tr>
<tr>
<td>0.34-0.56mg/L</td>
<td>2.9</td>
</tr>
<tr>
<td>0.57-1.0mg/L</td>
<td>3.8</td>
</tr>
<tr>
<td>&gt;1.00mg/L</td>
<td>2.7</td>
</tr>
</tbody>
</table>

hsCRP>0.57mg/dL had a increased risk of dementia over 25 years

from Walker2019 and Schmidt 2002

hsCRP>1.05 mg/L had greater cognitive decline over a 20 year period
Pivotal role of TNF in AD

Microglia dysfunction/activation

TNF

Synaptic dysfunction

Nerve cell death
TNF in humans: the dark passenger of AD

- Elevated plasma TNF (Fillit et al. 1991)
- TNF co-localized with plaques (Dickson 1997)
- TNF levels correlate with disease progression (Paganelli et al. 2002)
- Elevated TNF in CSF (Tarkowski et al. 2003)
**Why XPro1595?**

*Difference between selective vs non-selective TNF inhibition*

<table>
<thead>
<tr>
<th></th>
<th>Non-selective TNF inhibitor</th>
<th>XPro1595</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decreases inflammation</td>
<td>YES</td>
<td>YES</td>
</tr>
<tr>
<td>Causes immunosuppression</td>
<td>YES</td>
<td>No</td>
</tr>
<tr>
<td>Causes demyelination</td>
<td>YES</td>
<td>No</td>
</tr>
<tr>
<td>Neuroprotective</td>
<td>NO</td>
<td>YES</td>
</tr>
<tr>
<td>Enhances neuroplasticity</td>
<td>NO</td>
<td>YES</td>
</tr>
</tbody>
</table>
Bad history of non-selective TNF therapy in neurologic disease

• Lenercept made RRMS worse
• Non-selective TNF inhibitors contra-indicated in neurologic disease

Non-selective TNF inhibitors exacerbates RRMS

Neurology 1999
In retrospect…

**XPro1595 improves, etanercept worsens**

(Brambrilla et al. 2011)
What happened?

XPro1595 promotes remyelination, etanercept promotes demyelination

Probert et al. 2017

INMB non-confidential3Q19.v2
XPro1595 attenuates AD pathology and restores normal function

**Synapse dysfunction**

<table>
<thead>
<tr>
<th>Saline</th>
<th>XPro1595</th>
</tr>
</thead>
<tbody>
<tr>
<td>NTG</td>
<td></td>
</tr>
<tr>
<td>TgCRND8</td>
<td></td>
</tr>
</tbody>
</table>

**Cognitive Impairment**

Spatial Learning

Swim distance (% first block)

**Immune dysfunction**

**Amyloid pathology**

Activated Myeloid cells

Brain T cell infiltration

Microglia

Hippocampus

**Efficacy has been shown in 3xTgAD, 5xFAD, TgCRND8 and aged mice**
Problem XPro1595 Addresses: Neuroinflammation, Linked to Every Aspect of Alzheimer’s Disease Pathology

Abnormal Ab and Tau processing

Synaptic loss

Neuronal cell death

Microgliosis and Astrogliosis

Excitotoxicity

Suppression of growth factors

sTNF

XPro1595 is a protein biologic that targets soluble Tumor Necrosis Factor (sTNF) to decrease neuroinflammation linked to Alzheimer’s Disease.
Phase 1b Biomarker Directed, Proof of Biology Study in Mild to Moderate AD Patients

Key enrollment criterion
- Mild to Moderate Disease (N=18)
- Positive for Amyloid
- Blood hsCRP > 2mg/L

Baseline
- Safety
- Blood inflammation
- Brain inflammation
- Imaging (MRI)
- Cognition
- Psychiatric symptoms
- Quality of Life

Week 6
- Safety
- Blood inflammation
- Brain inflammation
- Imaging (MRI)
- Cognition
- Psychiatric symptoms
- Quality of Life

Week 12
- Safety
- Blood inflammation
- Brain inflammation
- Imaging (MRI)
- Cognition
- Psychiatric symptoms
- Quality of Life

Cohort (n=6) Dose of XPro1595
- Cohort 1: 0.3 mg/kg
- Cohort 2: 1.0 mg/kg
- Cohort 3: 3.0 mg/kg
INKmune Appendix
Residual disease is the cause of relapse and death in a majority of patients

- **Problem:** cancer remains after treatment
- **Solution:** eliminate residual disease
- **Drug:** INKmune
- **Target:** patient’s NK cells
- **Status:** entering Phase I/II trial in women with relapse/refractory ovarian cancer
What is MRD?

- Residual disease is the cancer that is left behind after treatment
- Two types:
  - Overt: visible by imaging studies
  - Minimal (MRD): invisible by imaging studies
- Natural Killer (NK) cells are responsible for eliminating MRD
- **Problem:** patient’s NK cells are inert
- **Solution:** INKmune primes the patient’s NK cells to attack MRD
• Relapser NK cells *kill* survivors AML.
• Survivor NK cells *can not kill* relapser AML.

*Conclusion*: AML mutates to evade NK cell killing.
Relapse due to Minimal Residual Disease

Relapse due to MRD

Cancer down-regulates S1, “evading” NK cells, allowing cancer to grow

INKmune artifically replaces S1 allowing the NK to engage and kill cancer cells

INKmune primes NK cells to eliminate MRD

aNK Cell = Activated NK Cell
rNK Cell = Resting NK Cell
INKmune TpNK are active against many types of cancers:

- **Blood cancers**: AML, MM, lymphoma
- **Solid tumors**: breast, ovary, prostate, renal, lung

modified from Lowdell2007 and 2011
2 Phase 1 clinical trials; 20 patients with high-risk AML using ex vivo, personalized TpNK therapy

Clinical Results

- Prolonged remissions with excellent quality of life
- Put chemo-resistant disease into remission
- 2 patients remain alive in complete remission after >3 years

Conclusions:
- TpNK biology translated to the clinic
- Most patients relapsed – need multiple treatments
- Personalized therapy cumbersome – need universal off-the-shelf solution
Biomarker – Patient Selection

Autologous INKmune pNK cells lyse primary MM cells irrespective of prior treatment

INKmune pNK target tumor and NOT normal cells

Am J Hematology; 86:967-973
2\textsuperscript{nd}-generation TpNK causes \textit{in vivo} priming so patient’s NK cells can kill their cancer by targeting naturally occurring tumor antigens.

\textbf{Phase 1/2 study in relapsed/refractory CaOva}

Platinum resistant/refractory patients with residual disease

\begin{itemize}
  \item Treatment: INKmune IP
  \item Endpoints:
    \begin{itemize}
      \item Safety
      \item ↑ NK activation and tumor killing
      \item ↓ tumor burden*
    \end{itemize}
\end{itemize}

\textbf{Phase 1/2 dose-finding study in high-risk MDS}

Patients with high-risk MDS and measurable disease

\begin{itemize}
  \item Treatment: INKmune IV
  \item Endpoints:
    \begin{itemize}
      \item ↑ NK activation and tumor killing
      \item ↓ blasts in marrow*
    \end{itemize}
\end{itemize}

*in Phase II

INKmune therapy weekly times three then monthly
NK cells and INKmune in Ovarian Cancer

SKOV3 killing by NK cells from healthy donor (HD) or CaOva patient ascites

- Patient ascites: NK cells kill SKOV3
- IL2 primed NK cells do not kill SKOV3

IL2 primed NK cells do not kill SKOV3
INB16 vs cytokine priming – Differential protein expression @16h

- Paradoxical decrease (not loss) in ligand expression associated with increased function
- INKmune priming fundamentally different than cytokine or antibody priming
INKmune Primed NK Cell Killing Assay

Real-Time Cytolysis Plot

% Cytolysis vs. Time (hr Post-Treatment)

- NK 1:1
- NK 2:1
- NK 5:1
- NK 20:1
- TP-NK 1:1
- TP-NK 2:1
- TP-NK 5:1

INKmune added
NK added
ALCAM – NK cell migration and adhesion
STAT5 – “molecular switch” essential for NK killing
CD70 – CD27L and NCR for B cell tumours
VEGFA – survival signal for NK
CXCL10 – chemoattractant for NK cells