Indaptus THERAPEUTICS

November 15, 2021

Investor Presentation

Forward Looking Statements

This presentation contains forward-looking statements with the meaning of the Private Securities Litigation Reform Act. These include statements regarding management's expectations, beliefs and intentions regarding, among other things, our product development efforts, business, financial condition, results of operations, strategies, plans and prospects. Forward-looking statements can be identified by the use of forward-looking words such as "believe", "expect", "intend", "plan", "may", "should", "could", "might", "seek", "target", "will", "project", "forecast", "continue" or "anticipate" or their negatives or variations of these words or other comparable words or by the fact that these statements do not relate strictly to historical matters. For example, forward-looking statements are used in this presentation when we discuss Indaptus's future plans and expected timeline of its development pipeline.

Forward-looking statements relate to anticipated or expected events, activities, trends or results as of the date they are made. Because forward-looking statements relate to matters that have not yet occurred, these statements are inherently subject to risks and uncertainties that could cause our actual results to differ materially from any future results expressed or implied by the forward-looking statements. In addition, historical results or conclusions from scientific research and clinical studies do not guarantee that future results would suggest similar conclusions or that historical results referred to herein would be interpreted similarly in light of additional research or otherwise. Many factors could cause actual activities or results to differ materially from the activities and results anticipated in forward-looking statements, including, but not limited to, the following: Indaptus's plans to develop and potentially commercialize its technology; the timing and cost of Indaptus's planned investigational new drug application and any clinical trials; the completion and receiving favorable results in any clinical trials; Indaptus's ability to obtain and maintain regulatory approval of any product candidate; Indaptus's ability to protect and maintain its intellectual property and licensing arrangements; Indaptus's ability to develop, manufacture and commercialize its product candidates; the risk of product liability claims; the availability of reimbursement; the influence of extensive and costly government regulation; and Indaptus's estimates regarding future revenue, expenses, capital requirements and the need for additional financing following the merger. These risks, as well as other risks are discussed in the proxy statement/prospectus that was included in the registration statement on Form S-4 filed with the SEC in connection with the merger.

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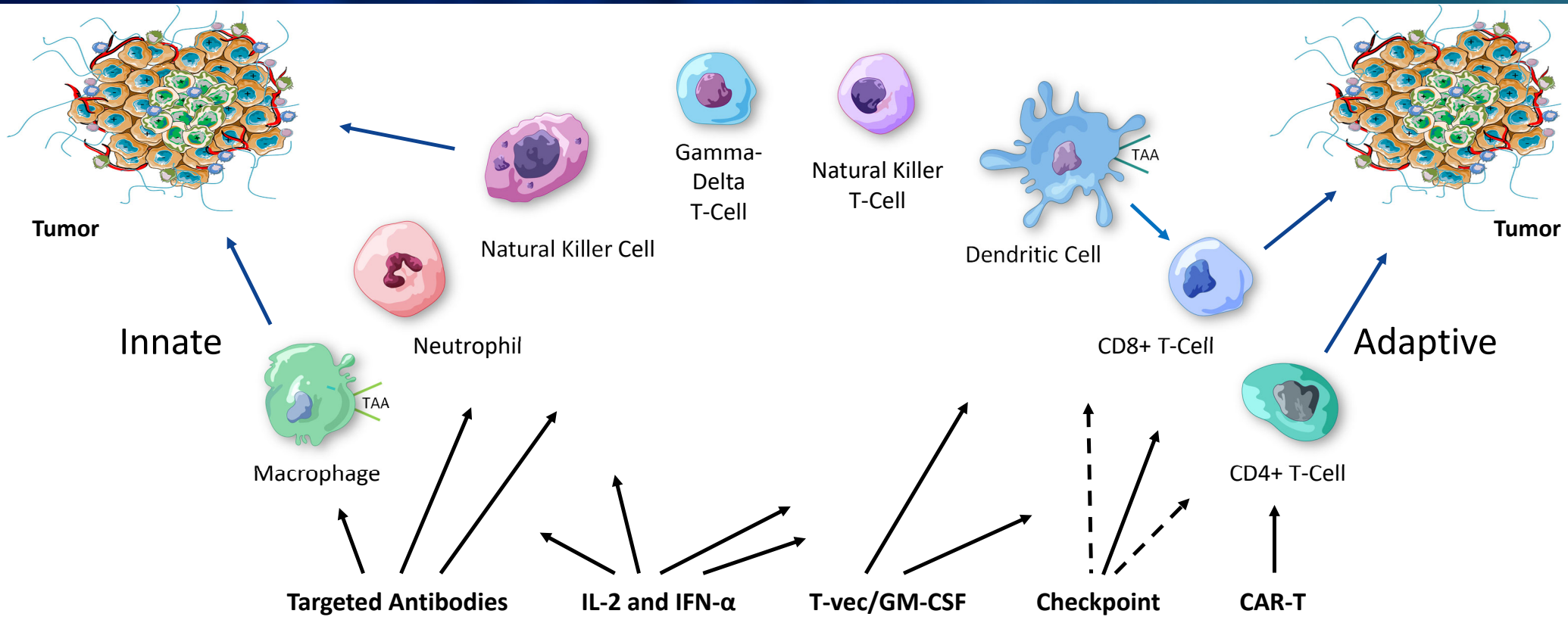
Current Cancer Immunotherapies: Low Percentage Cures for Most Advanced Cancers



Current immunotherapies only cure a very small percentage of advanced cancer patients, because they activate only one or a few innate or adaptive immune cell types

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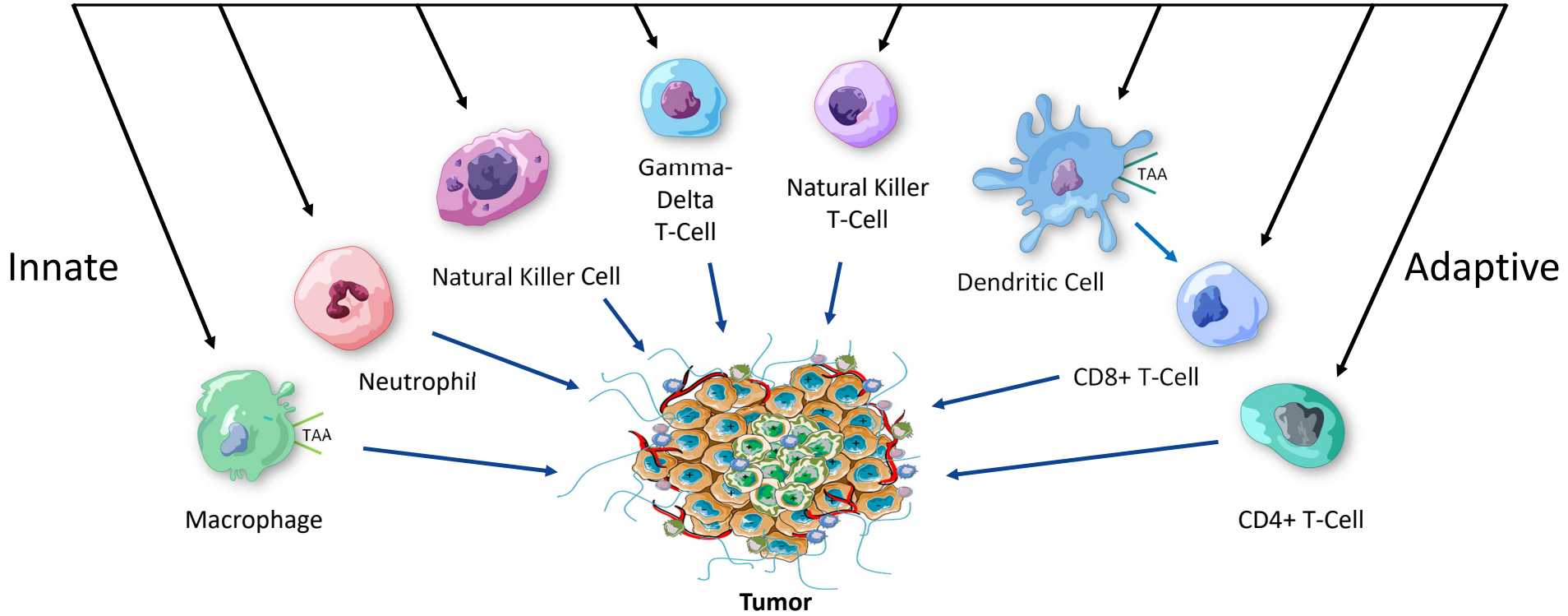
Improving Cancer Immunotherapy - Indaptus Assumptions

- **We need innate & adaptive pathway activation in lymphoid organs as well as tumor**
  - Tumors promote an immune-suppressive environment
  - Tumors negatively remodel the entire systemic immune system
  - Most steps required for innate and adaptive immune responses take place outside of the tumor

Goal is Activation of Both Innate and Adaptive Cellular Pathways in Multiple Locations

No one has figured out how to do this safely



History provided a clue about how to do this

World's First Immunotherapy: Clinically Validated and Composed of Killed Bacteria

- **Coley's Toxins (CT) – based on observation of regression of cancer in setting of infection**
  - Invented by Dr. William Coley at Memorial Sloan Kettering in NYC in 1894
  - Composed of heat-killed bacteria
- **Coley's Toxins produced durable responses with several hundred advanced cancer patients**
  - Associated with induction of fever by killed, Gram-negative bacteria

https://www.cancerresearch.org/about-cri/cri-history

https://www.mskcc.org/blog/immunotherapy-revolutionizing-cancer-treatment-1891

- **Coley's Toxins worked best i.v., but were too toxic, so given i.t. and s.c.**
  - i.t. and s.c. administration produced highly variable results

Scientific Knowledge Lagged Behind Clinical Validation

- **FDA required to certify old and new drugs in 1962 and decided not to grandfather-in CT as an approved drug in 1963, despite cures, due to variability in clinical response**

- **Pharmaceutical industry abandoned the product**
  - Mechanism of action wasn't known - could not determine source of variability and correct
  - Non-approval meant requirement to carry out expensive clinical trials
  - Very old drug - no patent coverage

Immunological Sciences Caught Up to the Clinic in the 1990's
Bacteria Contain Immune System Stimulating Danger Signals

- **The most prominent danger signal family activates Toll-like receptors (TLR)**

Source	Danger Signal (TLR Ligand / Agonist)	Toll-Like Receptor
Bacteria	Lipoproteins, Peptidoglycans	TLR2 (1/2, 6/2)
Viruses (Bacteria?)	Double Stranded RNA	TLR3
Bacteria	Lipopolysaccharide (LPS / endotoxin)	TLR4
Bacteria	Flagellin	TLR5
Viruses (Bacteria?)	Single Stranded RNA	TLR7/8
Bacteria	Unmethylated CpG DNA	TLR9

- **TLRs directly and indirectly activate essentially all immune cells (innate + adaptive)**
  - Indirect activation occurs via induction of secretion of cytokines and chemokines
- **Cytokines and chemokines are principal inducers of anti-tumor immune responses**
  - Innate - cell recruitment, MΦ activation, NK cell activation, γδT-cell activation, ↓Treg
  - Adaptive - cell recruitment, APC/DC activation, T-cell activation ($CD4_H$/$CD8_{CTL}$), ↓Treg

High Levels of TLR4 Agonist Lipopolysaccharide (LPS) Produce I.V. Toxicity

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Bacteria	Unmethylated CpG DNA	TLR9

- **TLR4 agonist LPS-endotoxin is the most potent and broadly acting danger signal**
- **Constitutes about 75% of the Gram-negative outer cell membrane**
- **Limits the number of bacteria (and other danger signals) that can be administered i.v.**

  Decoy Hypothesis – reduce surface LPS activity by ~90% to produce a safe & effective product

Indaptus has Used Modern Science to Optimize and Re-Invent the Approach

Decoy Product

- Start with a single, pure strain of non-pathogenic, Gram-negative bacteria
- Reduce LPS-endotoxin level by ~90%
- Kill the bacteria and stabilize so they remain intact after i.v. administration
- Product is a frozen suspension of killed, intact bacteria
- Chemical modification yields NCE
  Broad patent coverage: CoM + Methods
  4 issued US & 27 issued foreign patents
  Additional world-wide applications
  Nominal expiry – 2 families 2033/2039

Result and Predictions

- Decoy therapeutic is significantly less toxic *in vivo* than untreated bacteria and several live competitor products
- i.v. bacteria are passively targeted to liver, spleen and tumors, and cleared rapidly
- Predict "Goldilocks" effect:
  - Immune activation better than with i.t. dosing: Critical activation in spleen and can target primary liver cancer and liver metastasis from other tumors
  - Passive targeting and rapid clearance precludes continuous, systemic exposure common to small molecule, antibody and CAR therapies:

    Reduced chance of systemic toxicity

Indaptus Result – Effective, Safe and Patented

Decoy therapeutics exhibit many unique properties

- Single agent anti-tumor activity + tumor eradicating synergy with 5 different existing therapies
- Reduced toxicity and broad therapeutic index (no increase in toxicity with combinations)
- Safe induction of both innate and adaptive immune pathways (MoA) confirmed
- Innate and adaptive immunological memory leading to rejection of tumor re-challenge
- Efficacy in mouse syngeneic and human tumor xenograft models (CRC, HCC, Pancreatic, NHL)
- GMP batch of drug product produced (Decoy20) – stable for ≥6 months at -70°C, -20°C and 5°C
- IND-enabling toxicology with GMP drug product – no induction of cytokine release syndrome
- Significant single agent activity in pre-clinical models of HBV and HIV

Decoy Treatment Does Not Impair Anti-Tumor Cytokine/Chemokine Induction

Despite being less toxic, Decoy therapeutics induce similar amounts of anti-tumor cytokines and chemokines, uncoupling toxicity from anti-tumor activity

Secretion by Human PBMCs* *In Vitro*	Untreated Bacteria	Decoy Therapeutic (Decoy10)	Decoy Therapeutic (Decoy20)
Anti-Tumor Cytokine	pg/mL (mean of triplicate determinations ± %CV at same bacterial dose for each cytokine		
GM-CSF	1,094 ± 22	1,197 ± 2	1,695 ± 23
IFNγ	175,866 ± 7	47,488 ± 3	55,321 ± 10
IL-12p70	176 ± 14	528 ± 7	428 ± 37
TNFα	49,782 ± 11	77,919 ± 13	99,247 ± 16

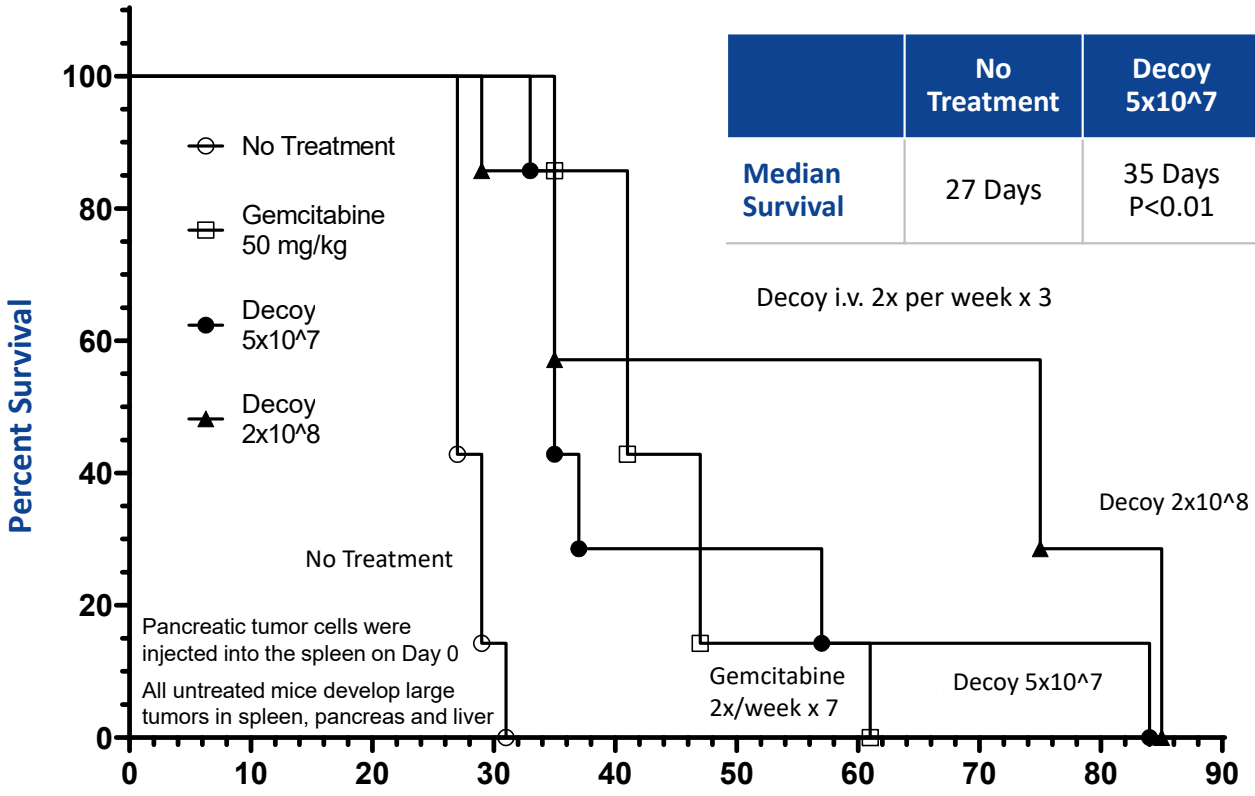
*Peripheral Blood Mononuclear Cells

Decoy therapeutics are more broadly active than mono-specific TLR agonists

Secretion by Human PBMCs *In Vitro*	CpG (TLR9)	Poly(I:C) (TLR3)	R848 (TLR7/8)	LPS (TLR4)	Decoy10* (TLR2,4,5,9)
Anti-Tumor Cytokine	pg/mL (triplicate full titration peak average from two exp)				
GM-CSF	0	2	136	27	1,246
IFNγ	7	248	61,914	33,293	171,284
IL-12p70	4	15	205	84	375
TNFα	65	334	36,663	24,944	73,069
MIP-1α**	0	272	17,866	19,278	29,942

*Decoy therapy tested at doses therapeutically relevant for *in vivo* models
**From one experiment

Single Agent Activity - Metastatic Mouse Pancreatic Carcinoma



	No Treatment	Decoy 5x10^7	Gem	Decoy 2x10^8
Median Survival	27 Days	35 Days P<0.01	41 Days P<0.01	75 Days P<0.01

Decoy i.v. 2x per week x 3

(Treatment started on Day 5 with 7 mice/group)

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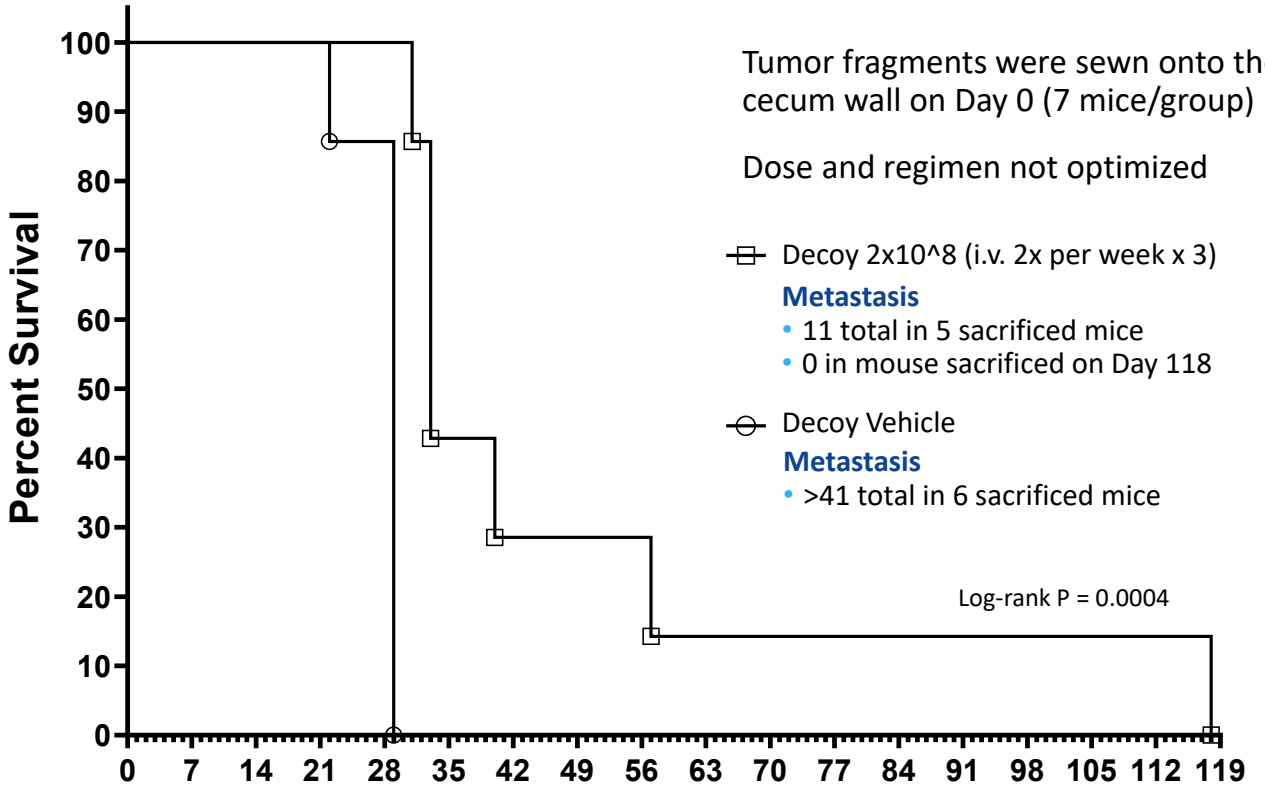
Single Agent Activity - Orthotopic Mouse Colorectal Carcinoma



Tumor fragments were sewn onto the cecum wall on Day 0 (7 mice/group)

Dose and regimen not optimized

Decoy 2x10^8 (i.v. 2x per week x 3)

Metastasis

- 11 total in 5 sacrificed mice
- 0 in mouse sacrificed on Day 118

Decoy Vehicle

Metastasis

- >41 total in 6 sacrificed mice

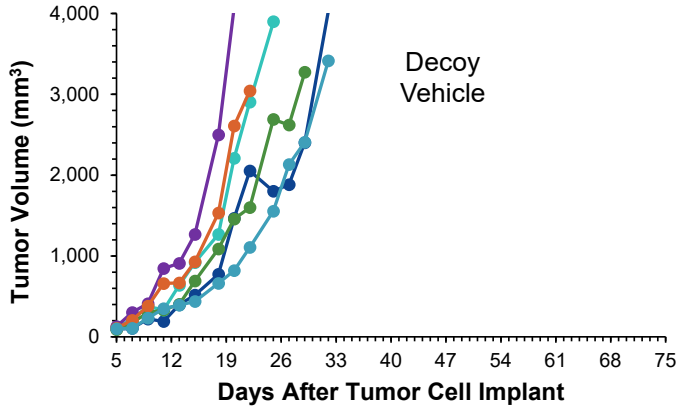
Log-rank P = 0.0004

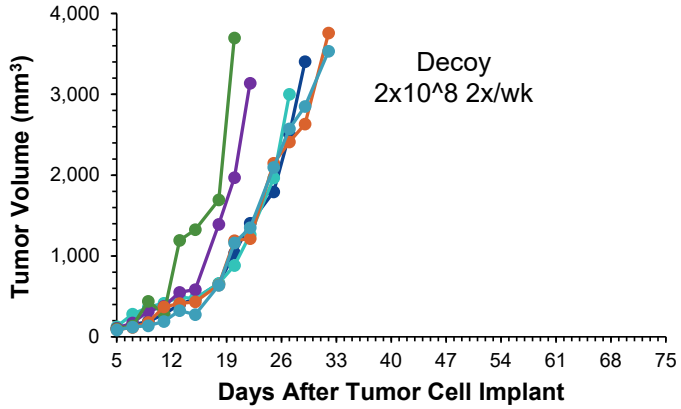
Also, combination-based eradications with s.c. tumors

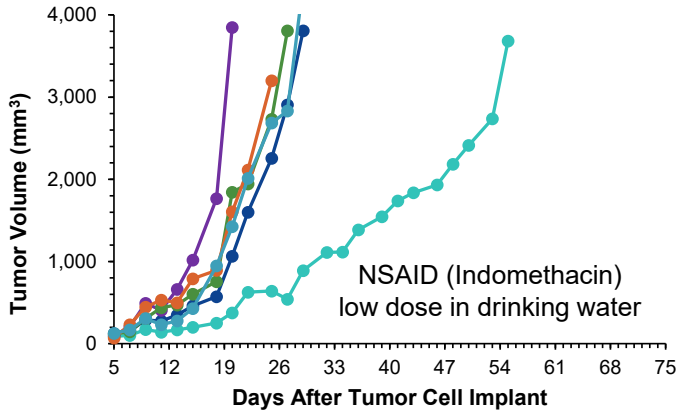
Decoy Synergizes With a Non-Steroidal Anti-Inflammatory Drug (NSAID) to Safely Eradicate Subcutaneous Mouse Hepatocellular Carcinomas (HCC)

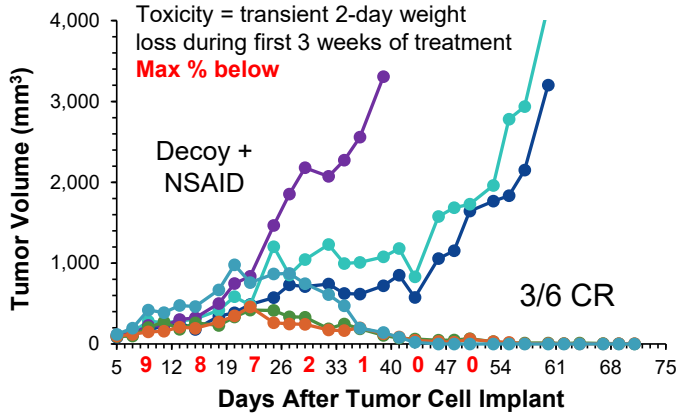
Treat 6 mice per group with Decoy 2x per week i.v. for 7 weeks / Start treatment at 103 mm^3

NSAIDS reduce myeloid-derived immune suppressive cells

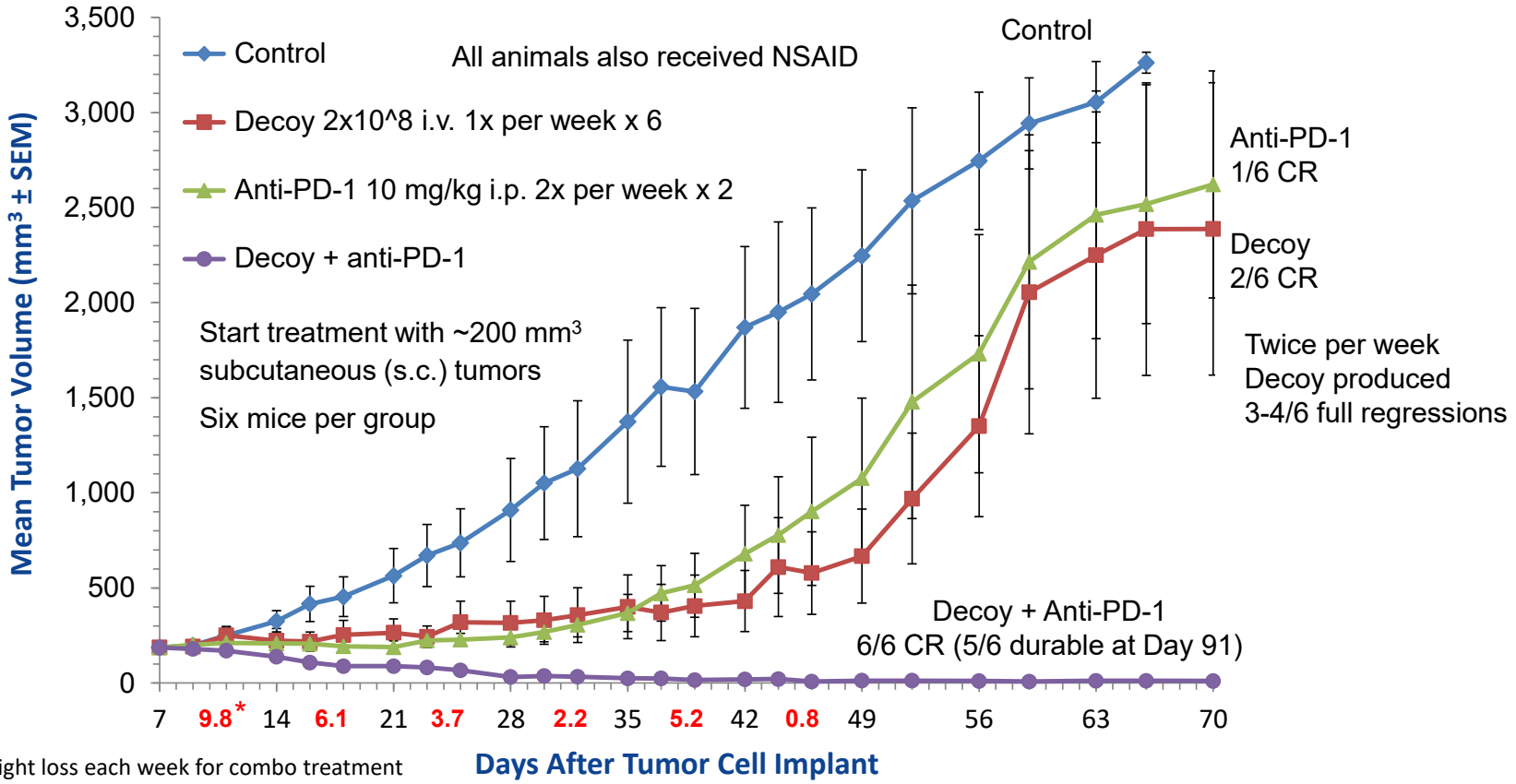








Combination With Anti-PD-1 Checkpoint Therapy Produces 100% Complete Responses With Hepatocellular Carcinoma



* Max % transient weight loss each week for combo treatment
No increase in toxicity with triple combo

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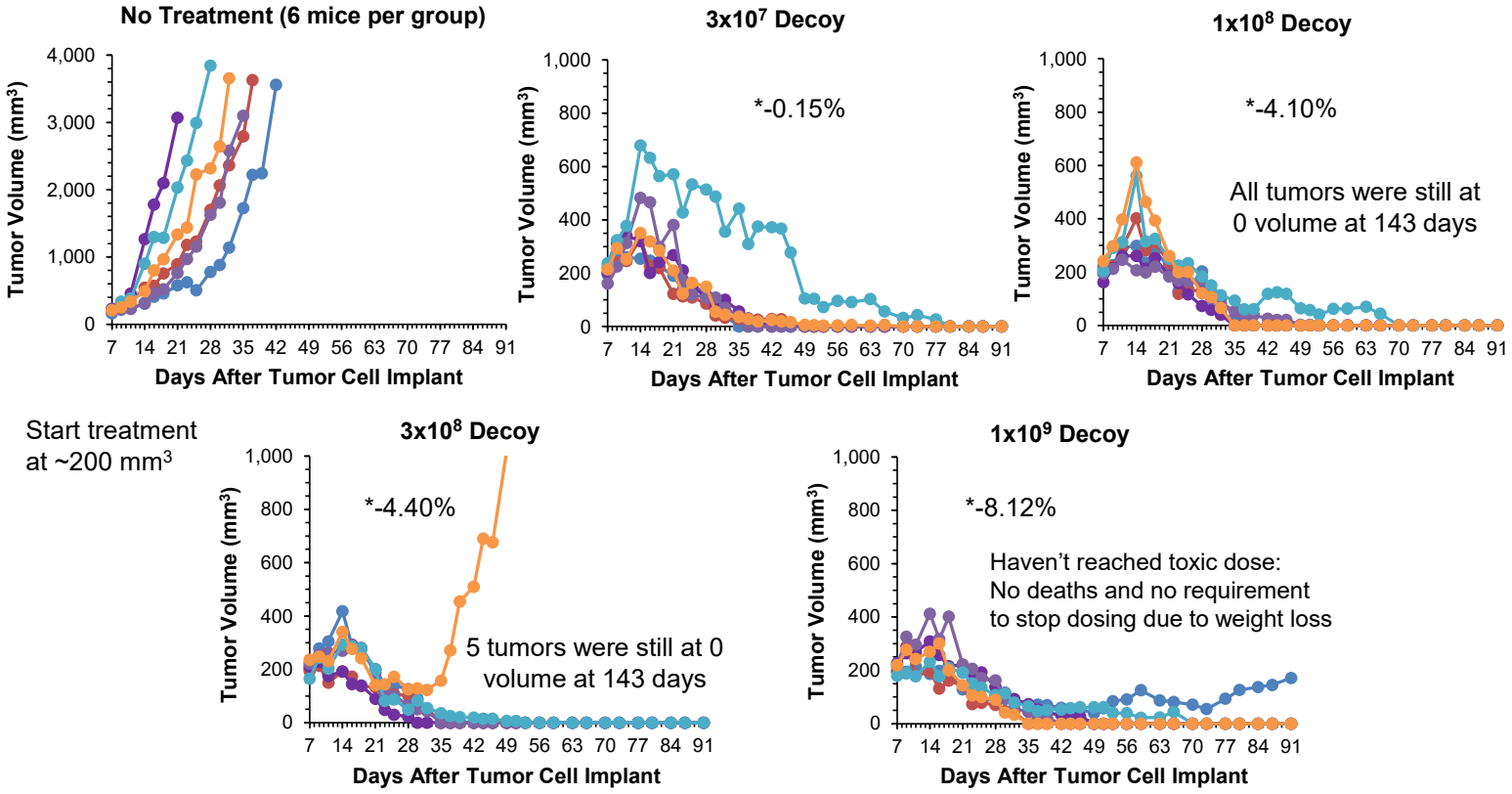
Synergistic Eradication of Murine HCC Exhibits a Very Wide Decoy Therapeutic Index (≥33-fold)

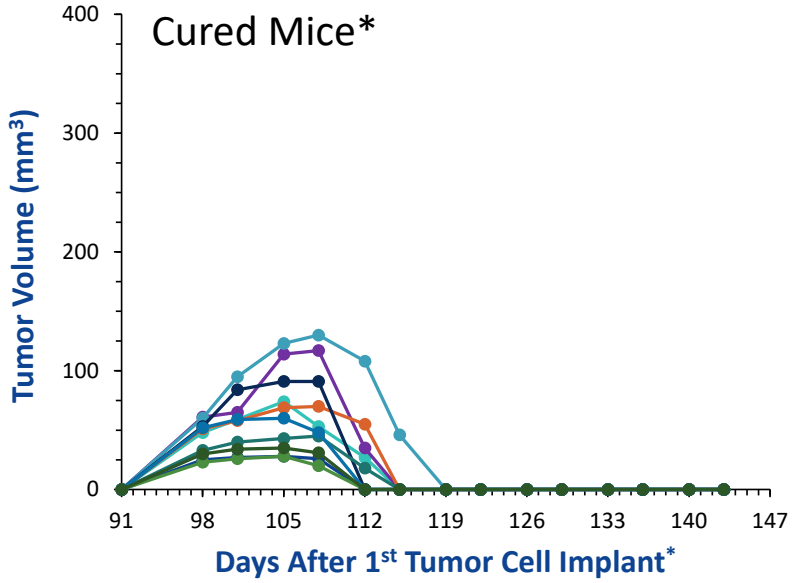
All animals also received a non-steroidal anti-inflammatory drug (NSAID)



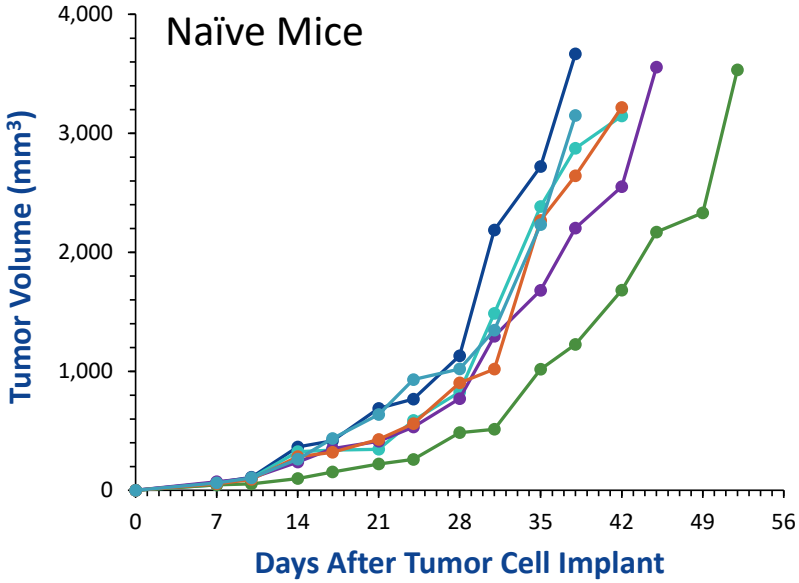
*Maximum transient body weight loss relative to start of treatment

Mice Cured by Decoy + NSAID + Anti-PD-1 and Re-Challenged with Fresh HCC Tumor Cells Reject the Tumors (Immunological Memory)

Eleven Cured Mice were Re-Challenged with Fresh HCC Tumor Cells on Day 91 on the Opposite Flank from the First Challenge



Six Naïve Mice were Challenged with the Same Tumor Cells as the Cured Mice on the Same Day



*All 1st challenge tumor sites remained tumor-free

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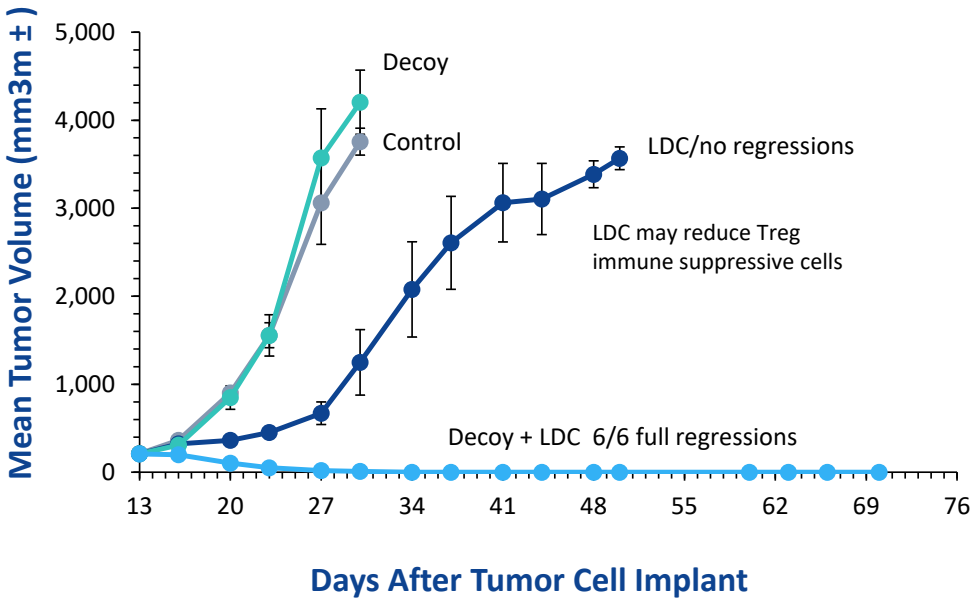
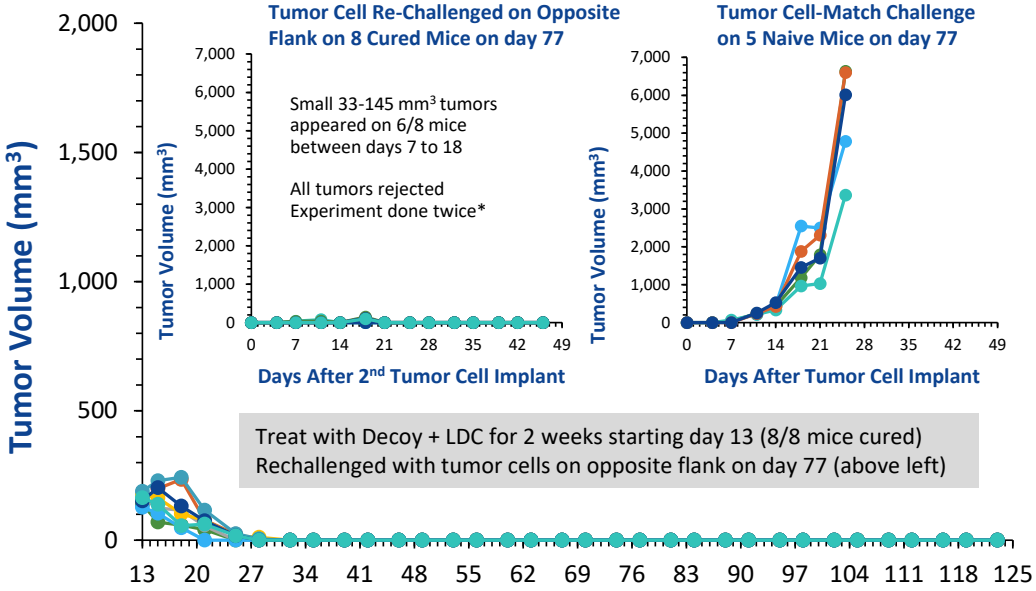
Decoy Produces Similar Results in Multiple Mouse Models

Decoy Therapeutic Synergizes with Low-Dose Chemotherapy (LDC) to Safely Induce Regression of s.c. Mouse Non-Hodgkin's-Lymphoma (NHL)

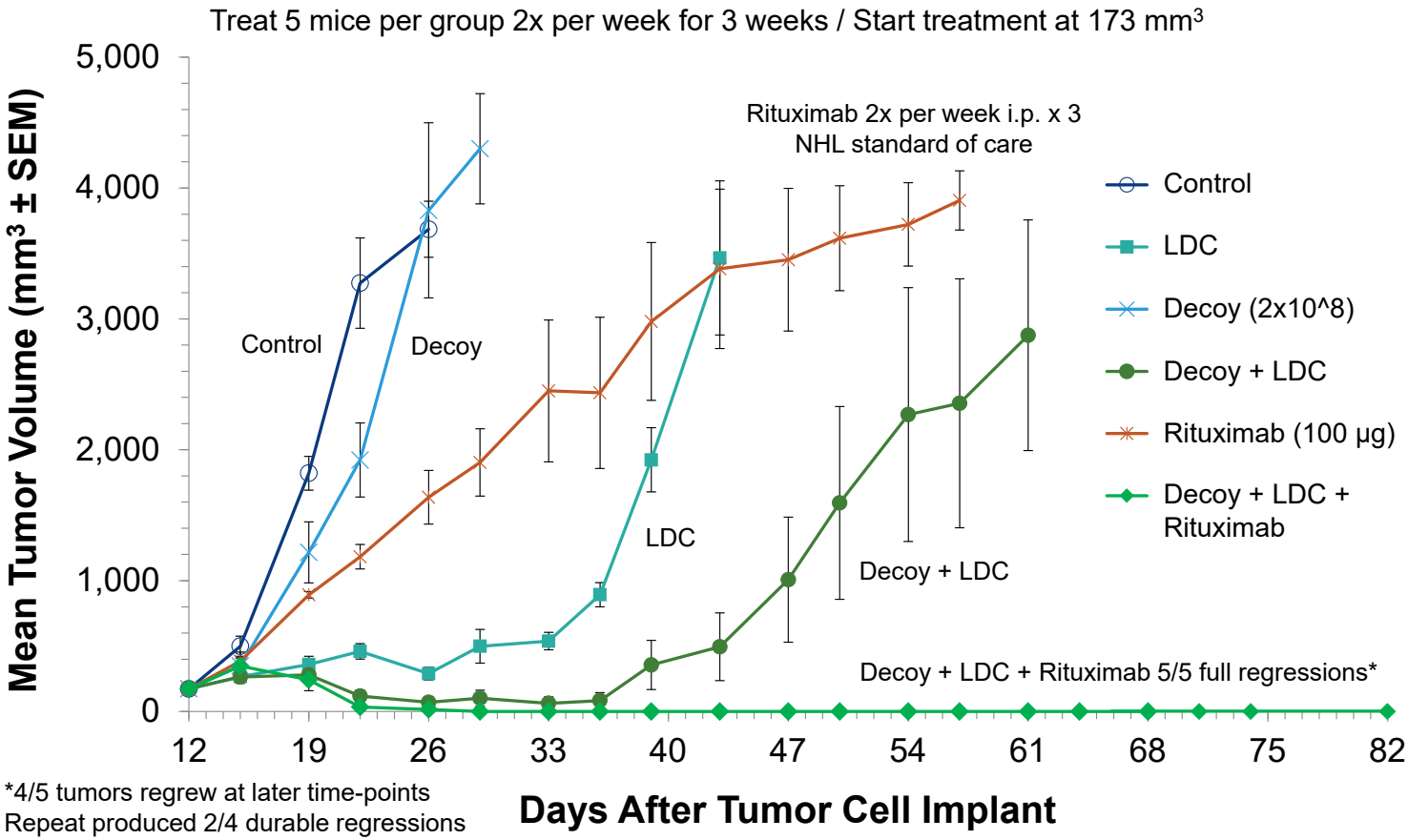


Mice Cured by Decoy + LDC and Re-Challenged with Fresh NHL Tumor Cells Reject the Tumors (Immunological Memory)



*Immunological memory also seen with innate-only human tumor xenograft model

Decoy Technology Also Regresses Human Tumor Xenografts



*4/5 tumors regrew at later time-points
Repeat produced 2/4 durable regressions

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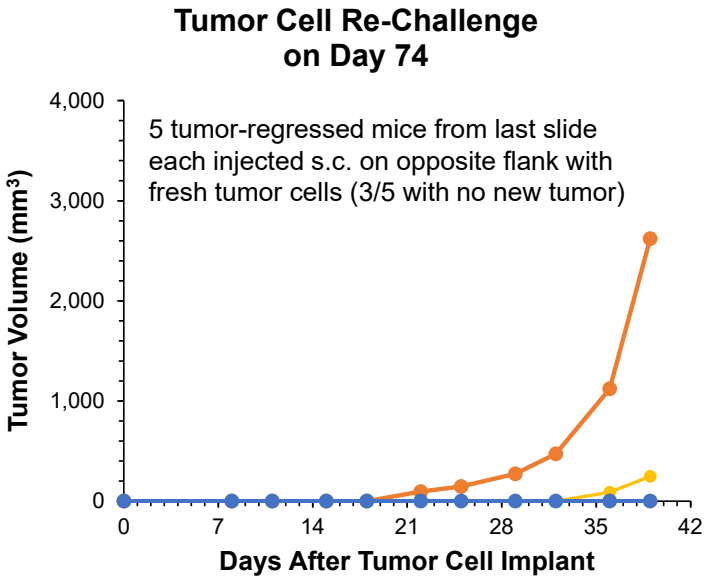
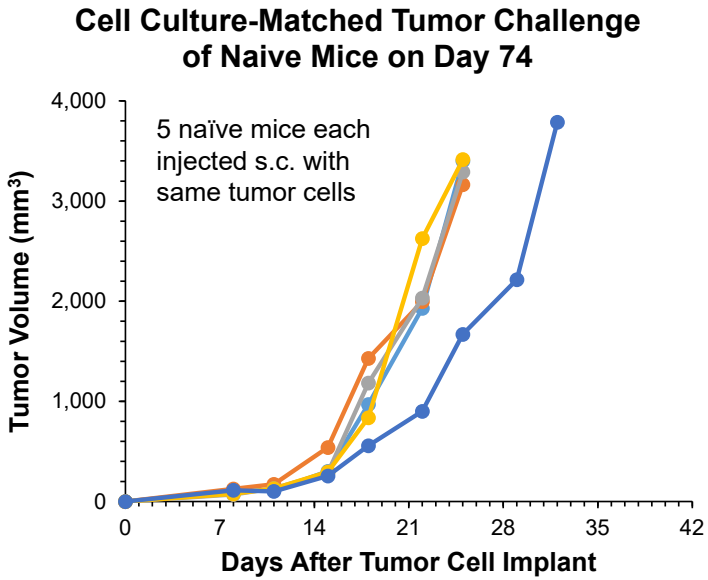
Decoy Technology can Induce Immunological Memory Via the Innate Immune System





- Tumor regression with immunological memory via the innate immune system alone is very rare in preclinical models, but consistent with a multiple danger signal mechanism
- Results suggest that Decoy technology may synergize with other marketed ADCC mechanism-based, targeted antibody therapeutics (~12 on market)

Decoy Technology Platform

Potential utility as anti-viral therapy - Hepatitis B Virus (HBV), HIV and Others

- HBV is a chronic liver infection affecting 257 million people world-wide
  - Only 2% treated with current therapies / Major cause of cirrhosis and HCC / 887,000 deaths per year
- Cytokines have strong anti-viral activity, but single, oral TLR agonists have failed in the clinic
- Multi-TLR agonist Decoy therapy is passively targeted to liver and safely induce cytokines
- Standard pre-clinical AAV-HBV mouse model of chronic HBV carried out twice:

Decoy Therapeutic Produces Broader Anti-HBV Activity Than Standard of Care Reverse Transcriptase Inhibitor Entecavir

	Inhibition (including for up to 6 months after cessation of treatment)					
	HBV Replication		HBe Antigen		HBs Antigen	cccDNA-Like Molecule
	Plasma	Liver	Plasma	Liver	Plasma*	Liver
Entecavir	✓					
Decoy Therapeutic	✓	✓	✓	✓	✓	✓

*Mild reduction by Decoy also in liver

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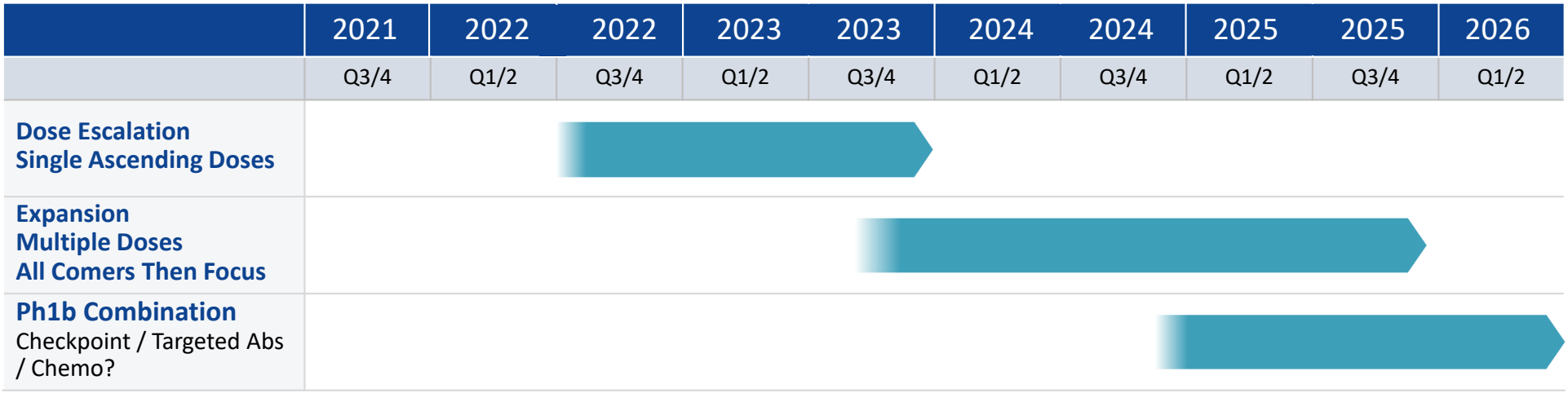
Indaptus Clinical Development Plan



Target Indications Include 6 of the World's 12 Deadliest Cancers

12 Deadliest Cancers World-Wide (Decoy Targets)			
		% of Yearly Deaths	% of Yearly Cases
1	Lung	18.4	11.6
2	**Colorectal**	**9.0**	**10.0**
3	Stomach	8.2	5.7
4	**Liver**	**8.2**	**4.7**
5	Breast	6.6	11.6
6	Esophagus	5.3	3.2
7	**Pancreas**	**4.5**	**2.5**
8	Prostate	3.8	7.1
9	**Cervical**	**3.3**	**3.2**
10	Leukemia	3.2	2.4
11	**N-H Lymphoma**	**2.6**	**2.8**
12	**Bladder**	**2.1**	**3.0**
Decoy Indications % of Total		**29.7%**	**26.2%**

Source: CA CANCER J CLIN 2018;68:394-424

High Unmet Medical Need

Percent five-year survival
for patients with metastatic disease

3% - 17%

Source: American Cancer Society

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Experienced Management and Board of Directors

Leadership experience in new modalities and early development

Roger J. Pomerantz, M.D.	Chairman	ContraFect, SERES THERAPEUTICS, MERCK INVENTING FOR LIFE, Johnson & Johnson
Michael J. Newman, Ph.D.	Founder, CSO, Director	Roche, SANDOZ, NOVARTIS, Decoy Biosystems
Jeffrey Meckler	CEO, Director	CRYSTAL, Cypress BIOSCIENCE, INC., Pfizer
Anthony J. Maddaluna	Director	amri Complex Science. Expert Solutions., Pfizer
W. Brad Hayes	Director	LabCorp, Builders FirstSource, Patheon A HEALTHIER WORLD. DELIVERED.
Brian O'Callaghan	Director	Obseva nature meets nurture, PETRA PHARMA, SONRGY, ACUCELA, Sangart
Hila Karah	Director	florafotonica, DARIO HEALTH, PERCEPTIVE ADVISORS
Hoonmo Lee	Director	KDDF, Lilly

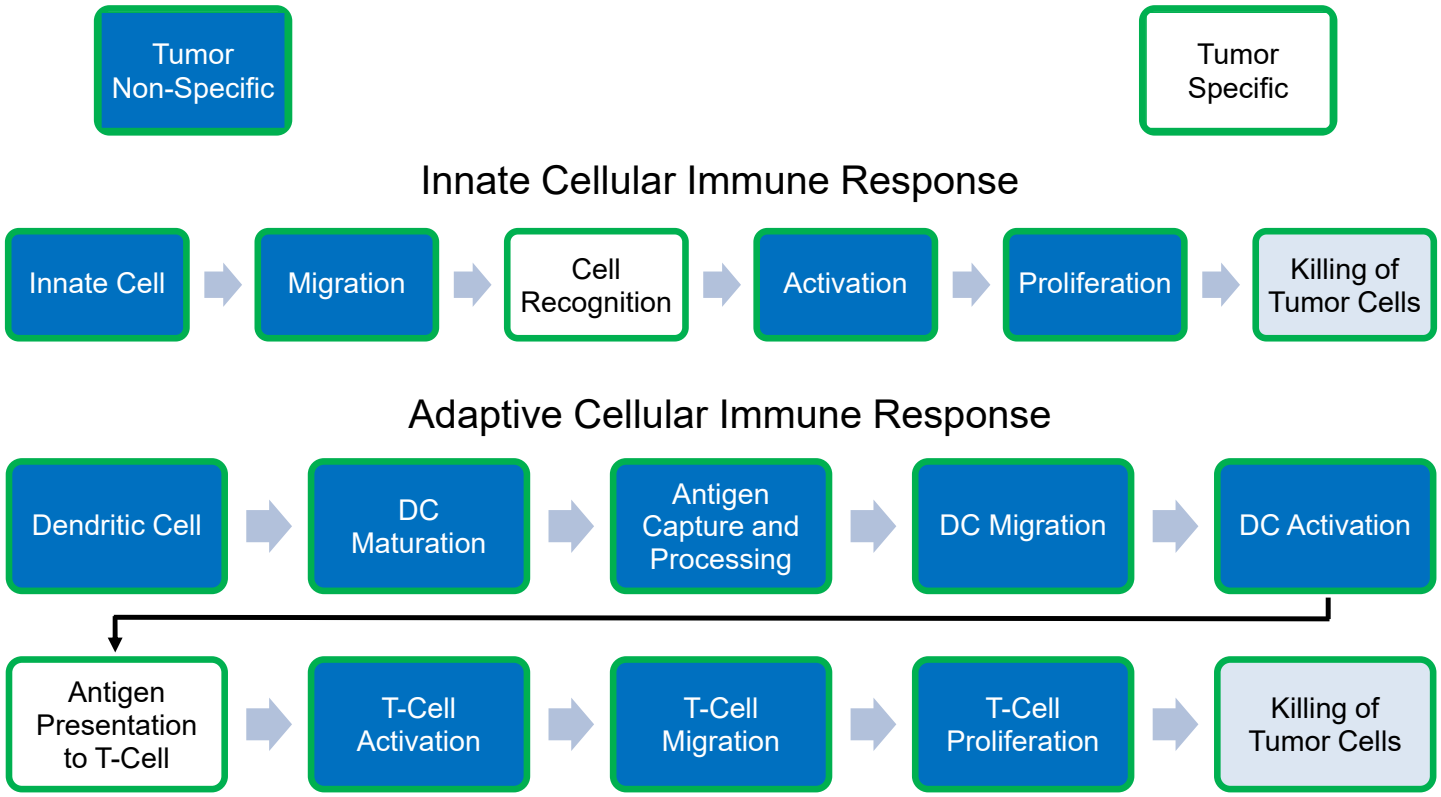
Thank you.

Appendix

There are No Intrinsically "Bad" Cytokines/Chemokines
Good or Bad Depends on Time, Place, Amount and How Long

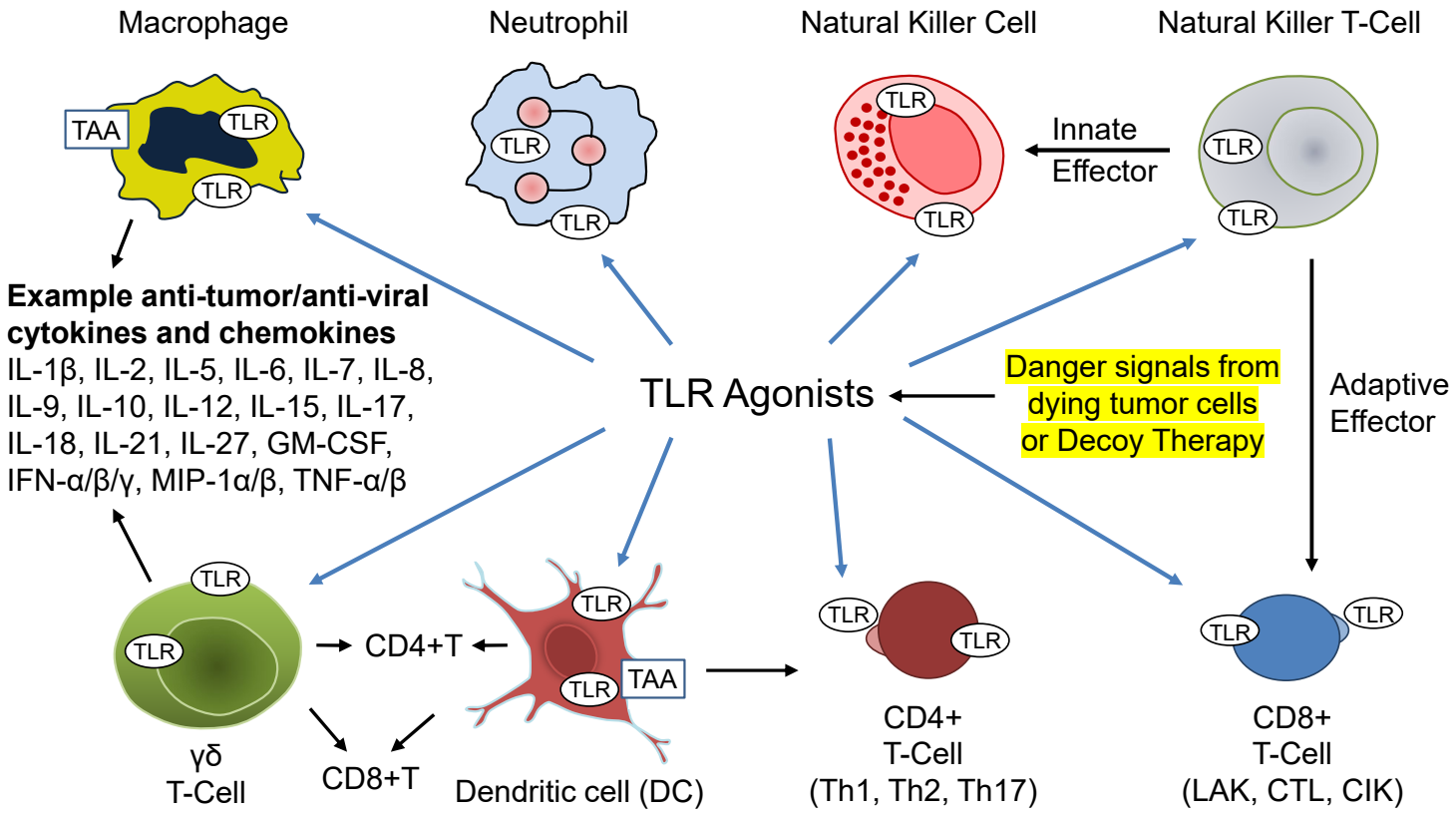
Cytokines and Chemokines Inducing Migration, Activation, Maturation and/or Proliferation	Responsive Immune Cell Type: All Participate in Anti-Tumor Immune Responses
GM-CSF, IL-1β, IL-4, IL-12, IL-15, IFN-γ	Dendritic Cells
IL-2, IL-12, IL-18, TNF-α	Gamma-Delta (γδ) T-Cells
IL-1β, IL-8, IFN-γ, MIP-1α, TNF-α	M1 Macrophage
IL-2, IL-10, IL-12, IL-15, IL-18, IL-21, IFN-γ	NK Cells
IL-12, IL-18, IL-21, IFN-γ	NKT Cells
GM-CSF, IFN-α, IL-4, IL-8, MIP-1α, TNF-α	Neutrophils
GM-CSF, IL-1β, IL-2, IL-5, IL-6, IL-7, IL-8, IL-9, IL-10, IL-12, IL-15, IL-17, IL-18, IL-21, IFN-γ, MIP-1α, TNF-α, TNF-β	T-Cells (Th1, Th17 or Th2 CD4+ or CD8+) Including CIK, CTL, LAK

How Can Bacterial Danger Signals Activate Anti-Tumor Immunity?
Most Steps Required for Innate and Adaptive Cellular Immune Responses are Non-Specific



All non-specific steps are induced or promoted by immune system “danger signal” molecules, which also enhance specific (tumor antigen recognition) steps / also many steps don’t occur in the tumor

Toll-Like Receptor (TLR) Agonists from Bacteria Directly Activate Immune Cells and Indirectly Activate by Inducing Secretion of Cytokines and Chemokines



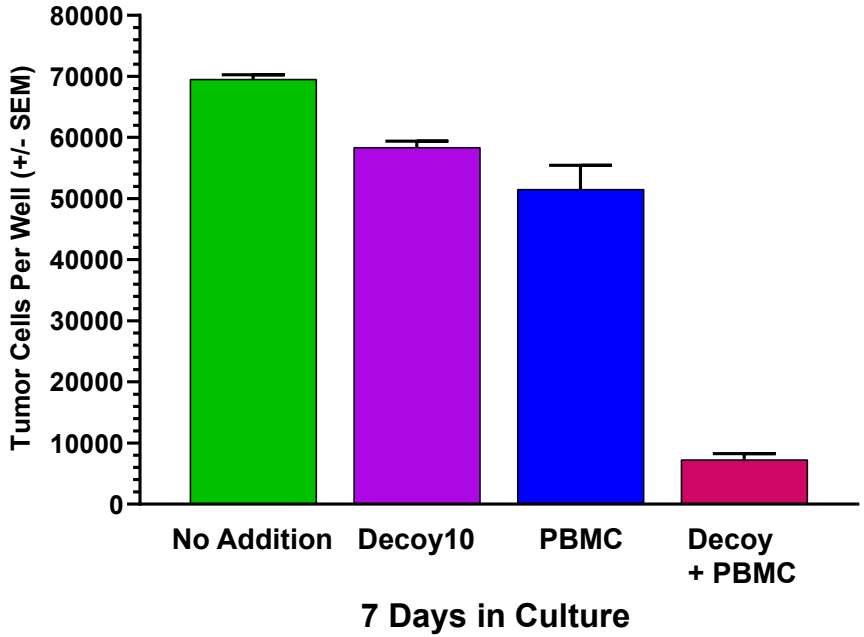
Immune cells can kill tumor or virus-infected cells or inhibit viral infection via cytokine secretion, cytotoxic granules, apoptosis, antibody-dependent cellular cytotoxicity (ADCC) and reactive oxygen/nitrogen species (RO/NS)

Patented Decoy Treatment Kills Bacteria and Significantly Reduces LPS-Endotoxin Activity and *In Vivo* Pyrogenicity

Treatment	Live Bacteria	LPS Endotoxin Activity (LAL Assay)	Pyrogenicity Threshold (Rabbit Assay)
No Treatment	100%	44.7 Units / 10^6 Bacteria	$3x10^4$ Bacteria
Decoy	0	3.6 Units / 10^6 Bacteria	$9x10^5$ Bacteria
Change induced by treatment	Killed all bacteria	92% reduction	97% reduction (requires more bacteria to increase rabbit temperature)

Decoy therapeutics are also 100 to 2,500-fold less toxic in mice (LD_{50}) than live, attenuated bacterial products

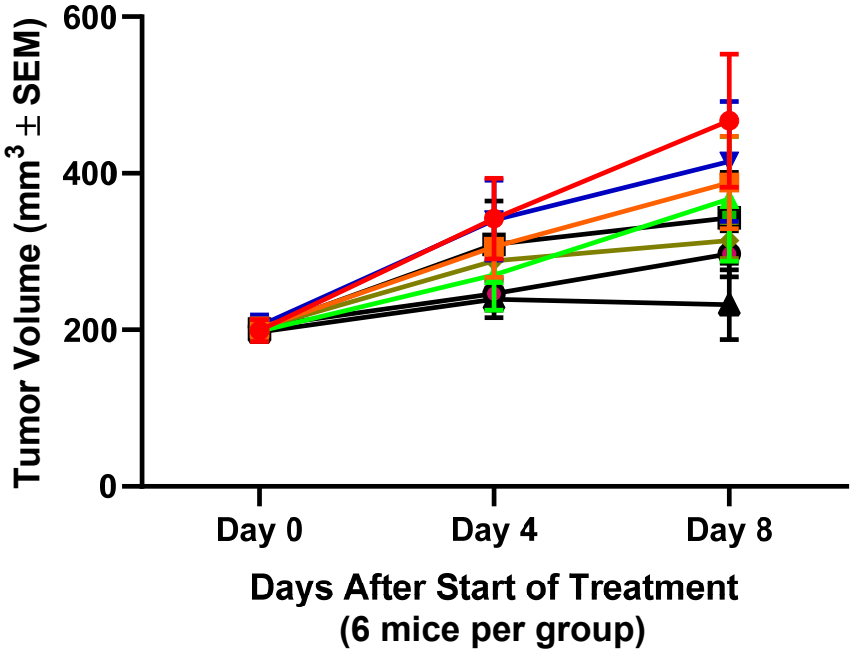
Decoy Therapeutic Synergizes with Human PBMCs to Kill Human Breast Carcinoma Cells *In Vitro*



Killed, non-pathogenic Decoy therapeutic is not intrinsically toxic to tumor cells (broad dose-response not shown), but can activate immune cells to kill tumor cells

Tumor-Eradicating Combinations Transform “Cold” HCC Tumors to “Hot”

Treatment Effects on Tumor Volume
(RNA prepared on Day 8 for NanoString Analysis)



Fold increase in
Tumor Inflammation Signature Score (TIS)
“Cold” vs. “Hot” Tumor

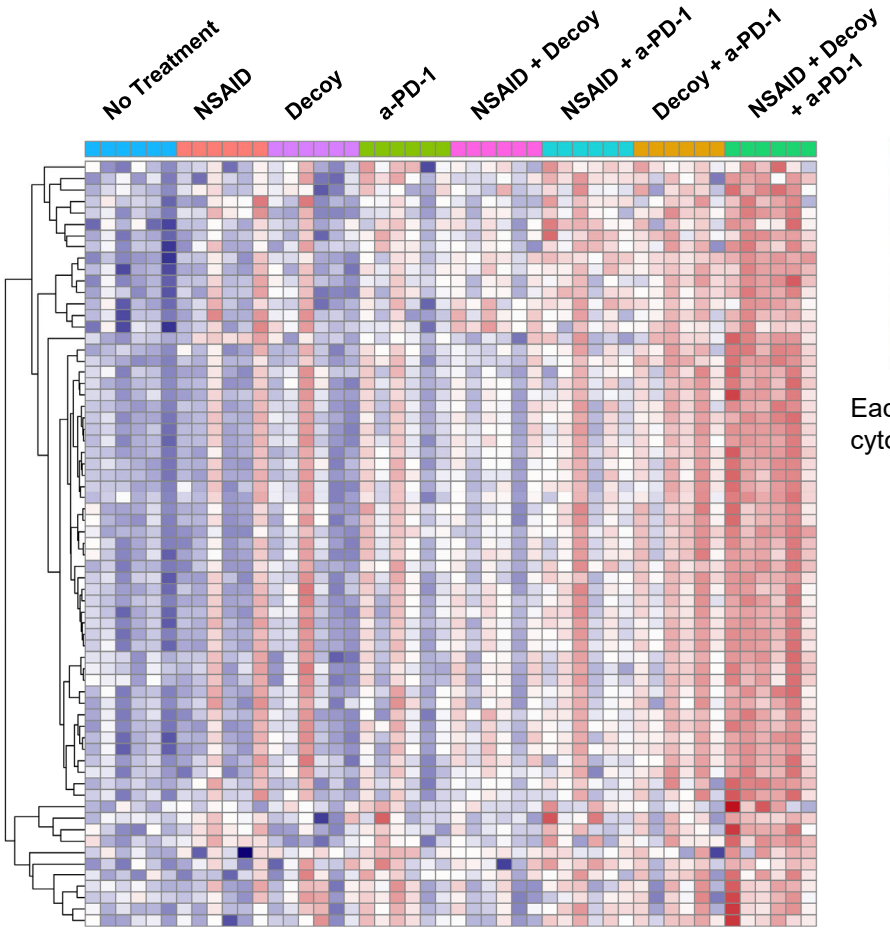
NT		0 / 6
NSAID	1.68	0 / 6
Decoy	1.61	0 / 6
a-PD-1	2.30	0 / 6
N + D	2.21	2 / 6
N + P	2.92	1 to 2 / 6
D + P	3.60	2 / 6
N + D + P	5.89	5 to 6 / 6

Number of long-term regressions per group

Systemic Administration of Decoy Therapy, NSAID and Anti-PD-1 Induces Cytokine Immune Pathways in HCC Tumors

NanoString 770 gene expression analysis: Cytokines and Receptors in tumor

Mice with 200 mm^3 tumors were treated for 1 week before tumor removal and RNA isolation/analysis

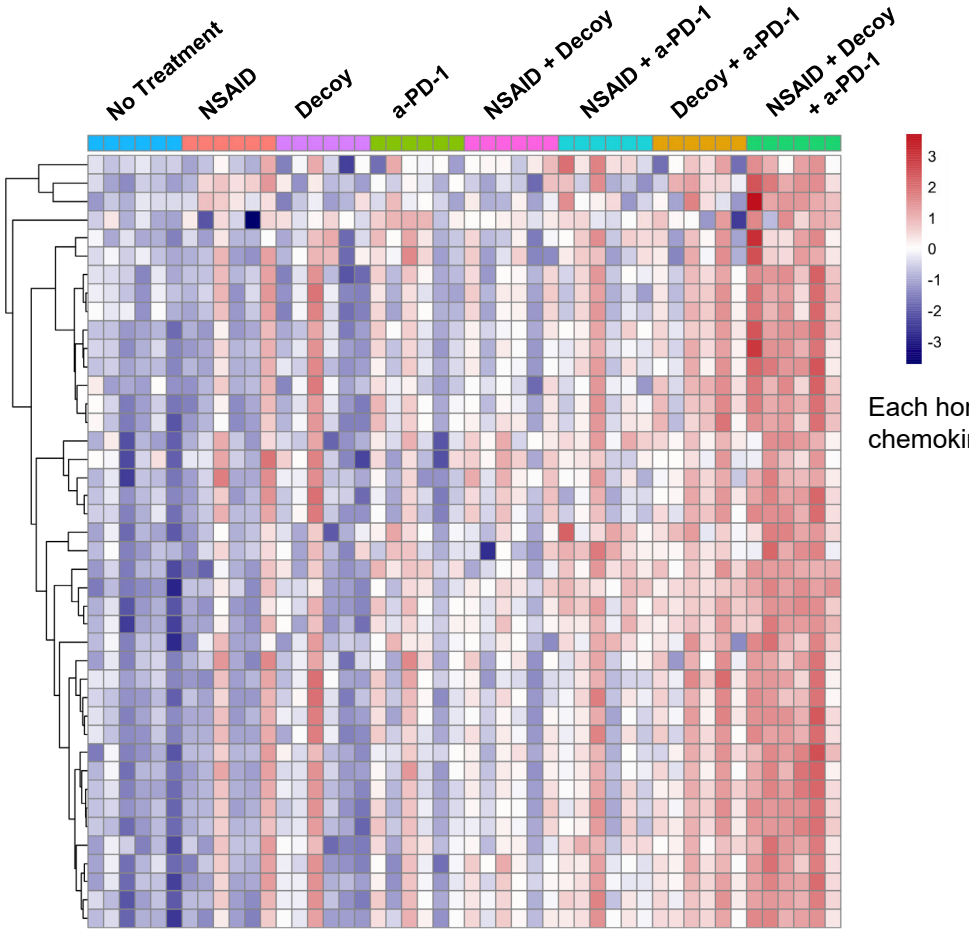


Each horizontal row represents a different cytokine or cytokine receptor gene

Systemic Administration of Decoy Therapy, NSAID and Anti-PD-1 Induces Chemokine Immune Pathways in HCC Tumors

NanoString 770 gene expression analysis: Chemokines and Receptors in tumor

Mice with 200 mm^3 tumors were treated for 1 week before tumor removal and RNA isolation/analysis

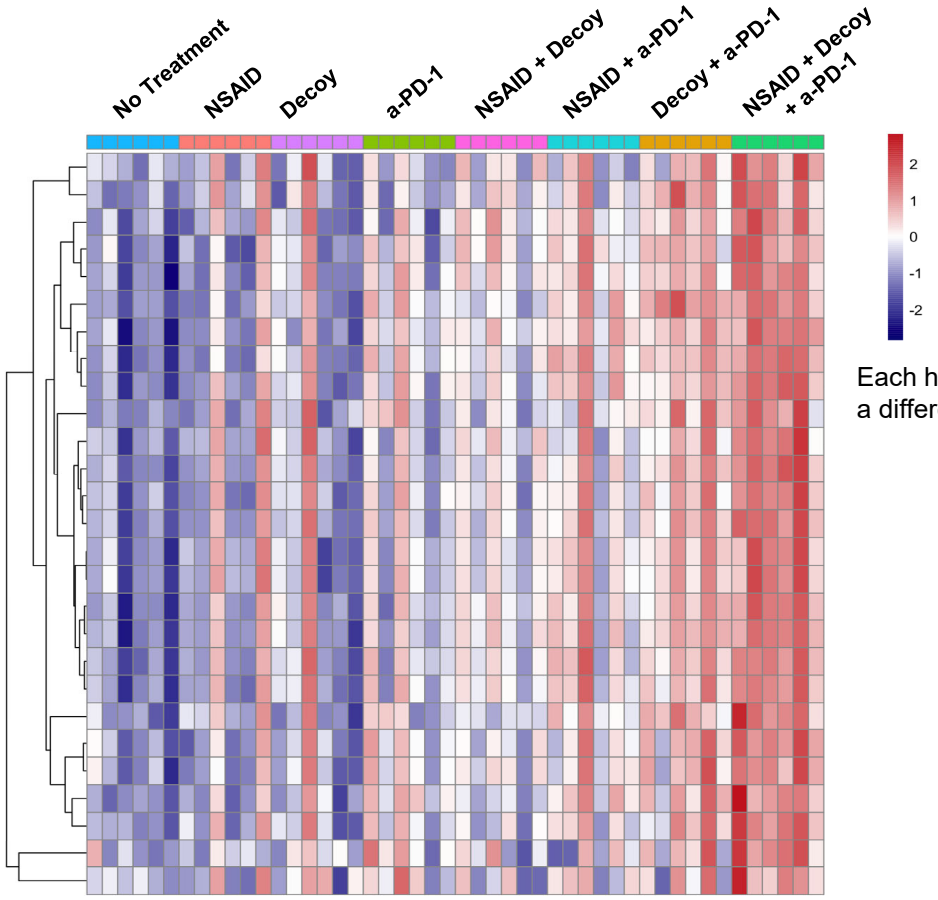


Each horizontal row represents a different chemokine or chemokine receptor gene

Systemic Administration of Decoy Therapy, NSAID and Anti-PD-1 Induces Innate Immune Pathways in HCC Tumors

NanoString 770 gene expression analysis: Innate Immune response in tumor

Mice with 200 mm^3 tumors were treated for 1 week before tumor removal and RNA isolation/analysis

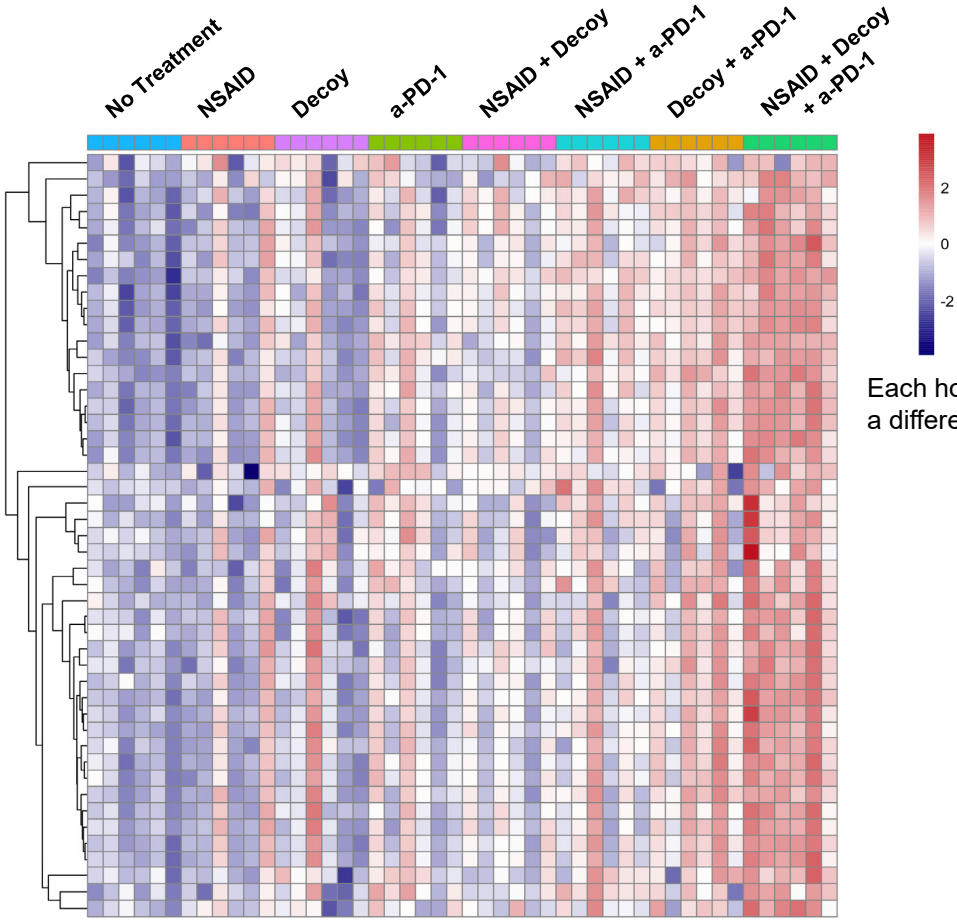


Each horizontal row represents a different innate pathway gene

Systemic Administration of Decoy Therapy, NSAID and Anti-PD-1 Induces Adaptive Immune Pathways in HCC Tumors

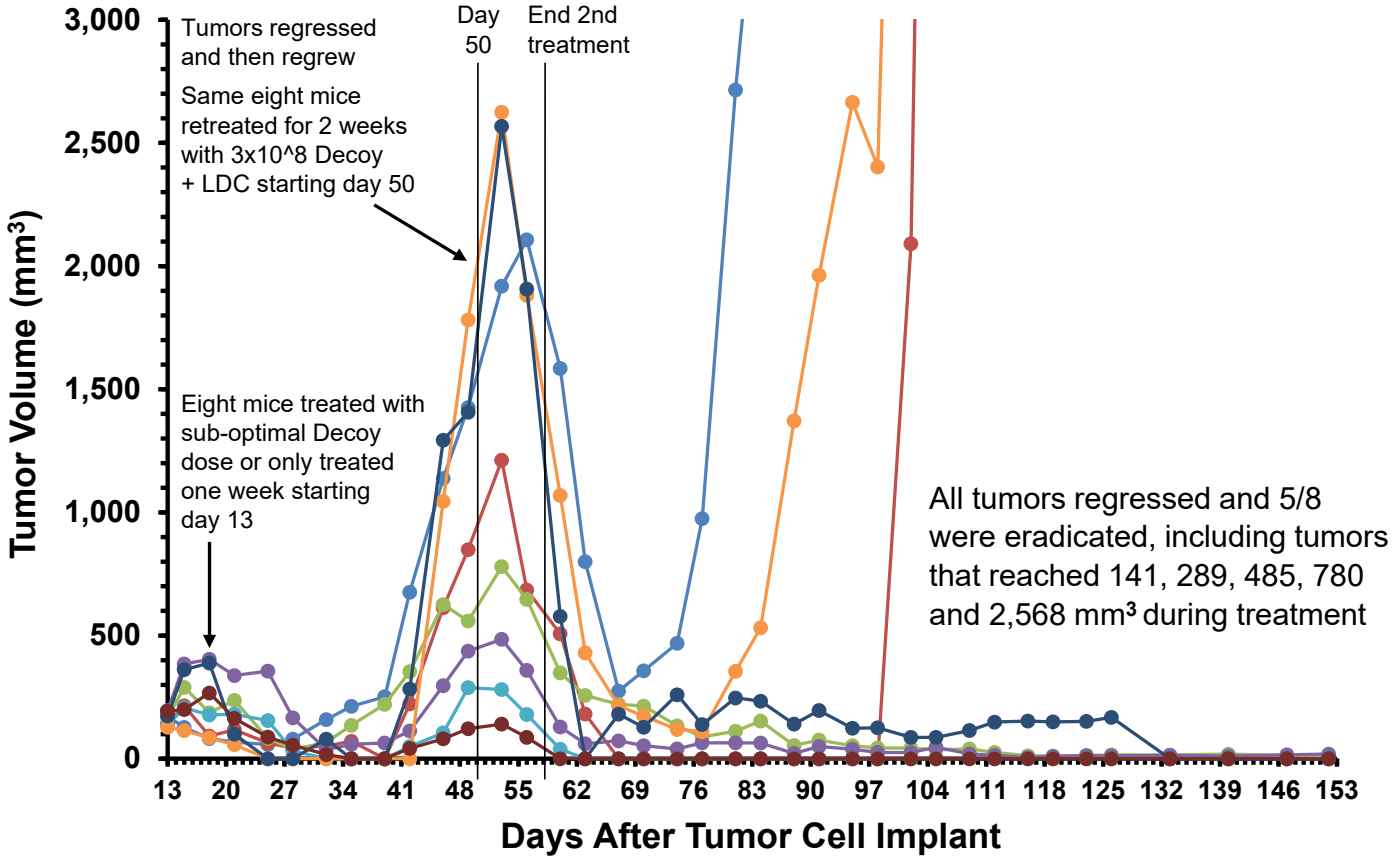
NanoString 770 gene expression analysis: Adaptive Immune response in tumor

Mice with 200 mm^3 tumors were treated for 1 week before tumor removal and RNA isolation/analysis



Each horizontal row represents a different adaptive pathway gene

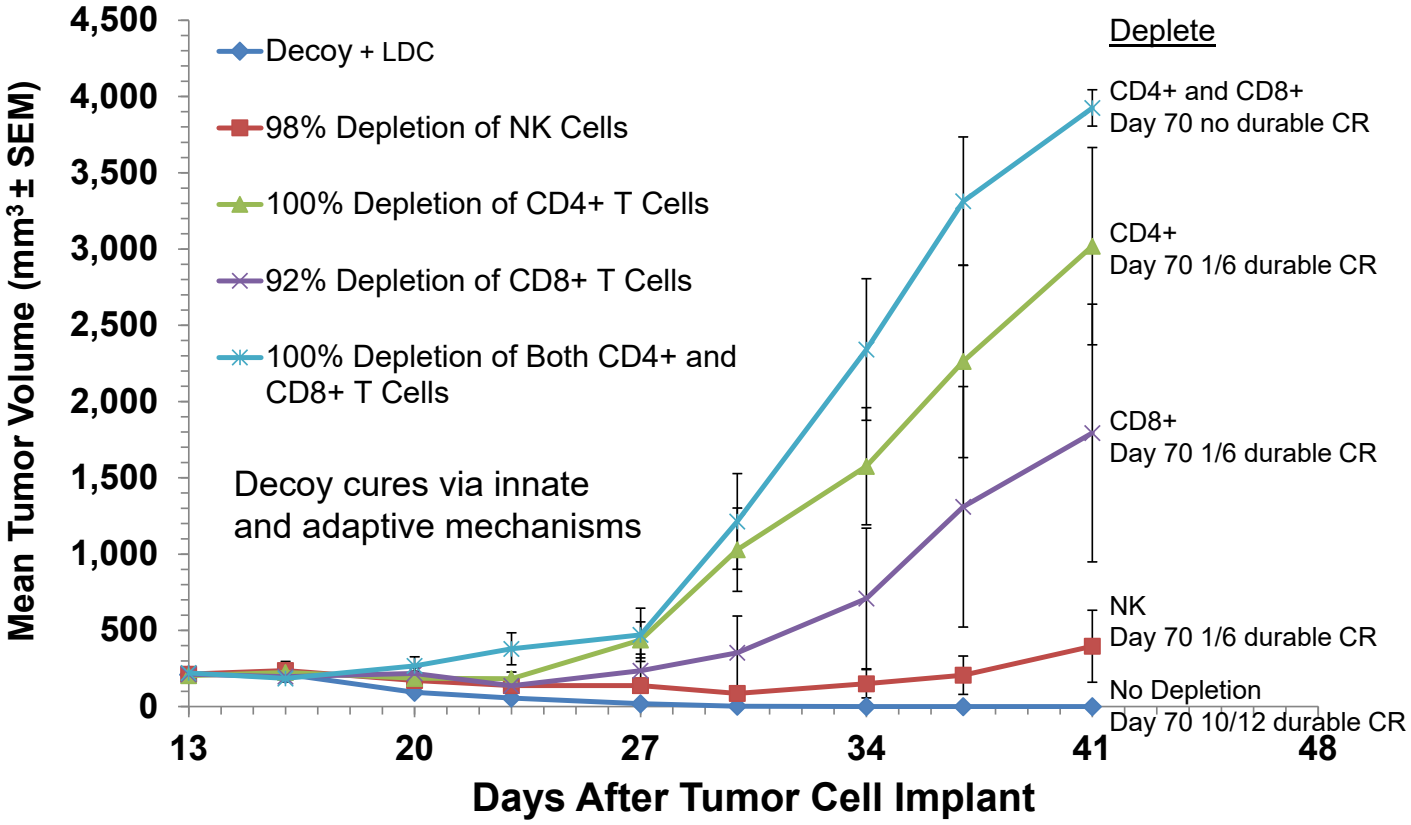
NHL Tumors That Regrow After 1 or 2 Weeks of Sub-Optimal Decoy + LDC Treatment are Sensitive to Optimal Retreatment



Suggests no or low neutralizing antibody resistance / Very large tumors can be eradicated!

High Percentage Eradication of s.c. NHL by Decoy + LDC Requires NK Cells and CD4+ and CD8+ T Cells

Treat all groups (6 mice per group) with i.v. Decoy + LDC for 2 weeks / Start treatment at ~200 mm^3



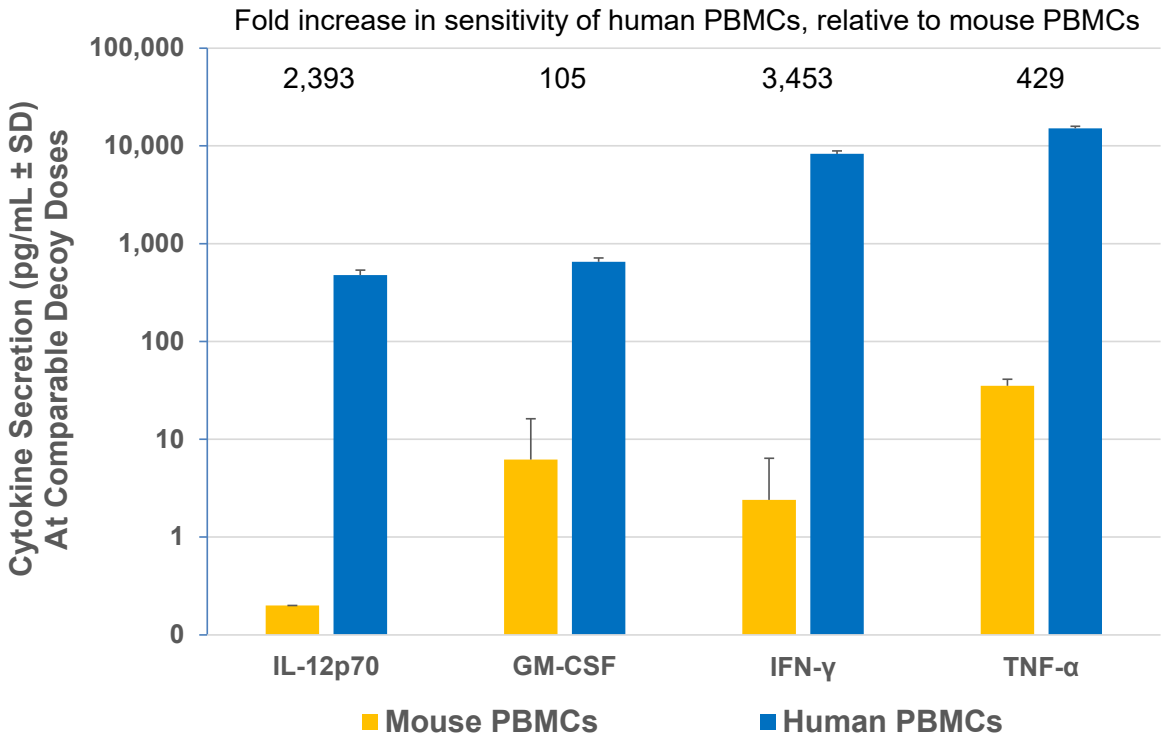
Frequently Asked Question

Q) **Humans are more sensitive than mice to the toxic effects of TLR agonists such as LPS. Does this mean that the Decoy product will be too toxic in humans?**

A) Humans are also more sensitive than mice to anti-tumor cytokine induction by TLR agonists, so if there is a therapeutic index in mice, there may also be one in humans (see next slide).

Decoy Therapy Induces Human PBMCs to Secrete ~100 to 3,500 Times Higher Levels of Anti-Tumor Cytokines Compared to Mouse PBMCs

Based on 48 hour *in vitro* (full) dose responses carried out in triplicate



This is probably why humans are more "sensitive" than mice to TLR agonists and why we will require correspondingly lower doses of Decoy therapy in humans for anti-tumor activity, preserving or possibly increasing the therapeutic index
Different sensitivity to TLRs by mouse and human PBMCs has been reported (Warren J. Infect. Dis. 201 223 2010)

Bacterial "Danger Signals" Approved to Prevent or Treat Early Stage Cancer

- **Bacillus Calmette-Guérin (BCG) (Sanofi/Merck)**
  - Tuberculosis vaccine (1921) made from live, attenuated *Mycobacterium bovis*
  - Approved for superficial bladder cancer via local (intravesical) administration
- **Picibanil™ (OK-432) (Chugai)**
  - Locally-administered, killed, Gram$^+$ component of Coley's Toxins - approved in Japan/Taiwan
  - Used mainly to treat lymphangiomas and vascular malformations
- **Mifamurtide (Mepact®) (Millennium/Takeda)**
  - Synthetic derivative of *Mycobacterium* cell wall muramyl dipeptide (NOD2/TLR4 agonist)
  - Approved in EU for non-metastatic osteosarcoma
- **Imiquimod (Aldara®) (Taro Pharmaceutical Industries/3M)**
  - Topical TLR7 agonist approved for superficial basal cell carcinoma
- **Monophosphoryl lipid A (MPL) (GSK)**
  - LPS analogue approved as adjuvant in HPV and Shingles vaccines (i.m. injection)

Limitations - All but one administered locally and none approved for advanced cancers