A BOLD NEW PERSPECTIVE ON CANCER

Presentation • Fall 2019
ABOUT INTENSITY

PIONEERING A NOVEL DIRECT CANCER KILL AND ANTIGEN RECOGNITION TECHNOLOGY

RAISED ~$20 MILLION
STARTED IN 2012

PROPRIETARY DISCOVERY PLATFORM: DfuseRx℠
Creates novel anti-cancer formulations using unique tumor diffusing and cancer cell-penetration enhancer molecules

LEAD PRODUCT, INT230-6, IN PHASE 1/2 FOR SOLID TUMORS

Granted Fast Track Designation by FDA for triple negative breast cancer
- No dose limiting adverse events

ROBUST IP POSITION
- Issued US & foreign patents: all major countries; 100% company owned

PARTNERSHIPS
CRADA with the National Cancer Institute
Clinical collaboration with Merck
OUR EXPERIENCED TEAM

Lewis H. Bender, MIT ChE, MS, MA, MBA
CEO and Founder
• Prior CEO: Genomic testing company: Interleukin Genetics
• Prior CEO, CTO, VP, BD & Manufacturing: Emisphere Technologies
• Drug delivery expertise
• Preclinical through Phase III development experience
• BD agreement: e.g. Roche, Novartis, Lilly, Elan, Merck

Ian B. Walters, MD, MBA
CMO
• Clinical Development 30+ compounds: Sorrento, BMS, Millennium, PDL, Rockefeller University
• Translational Medicine: Rockefeller University, Sorrento, PDL
• At BMS 7+ years: Oversaw oncology protocol review, and IO clinical collaborations

Board of Directors
Mark A. Goldberg, MD, Former President & COO of PAREXEL
Declan Doogan, Ph.D., Former VP Development Pfizer
Emer Leahy, Ph.D., CEO Psychogenics
Lewis H. Bender, CEO Intensity
OUR CHALLENGE: Late Stage Solid Tumors

PROBLEM 1
TWO FORMS OF DISEASE: Regional & systemic

LARGE VISIBLE TUMORS invading organs and/or tissues

Indeterminate # of unseen METASTASES

Genetically, quite similar; phenotypically, very different.

PROBLEM 2
HYPOXIC TUMOR ENVIRONMENTS:
Impeded delivery of drugs or immune cells

HYPOXIC ZONE: dense, under high pressure, & containing fibrin

• Often resistant to conventional cancer therapies; tumor hypoxia correlates with advanced stages of malignancy

PROBLEM 3
IMMUNE RECOGNITION:
Immune cells have difficulty recognizing/distinguishing cancer from normal cells

OUR SOLUTION: Diffusion-based tech

DOSE LOCALLY ACT GLOBALLY

via DIRECT TUMOR KILLING and global attack by T-cells

OUR DRUGS:
- Intratumoral administration
- Penetrate hypoxic zone
- Attenuate tumors, enabling immune systems to recognize and eradicate all disease
- Potential to treat most solid tumors
LEAD PRODUCT

INT230-6

Proven anti-cancer agents Cisplatin & Vinblastine co-formulated in a fixed ratio with a DIFFUSION ENHANCER MOLECULE

Note: structure shown is not actual enhancer compound
THE CLEAR SOLUTION TO THE PROBLEMS: DIFFUSION

Intensity has developed a dispersion technology to kill cancer cells even in hypoxic tumor environments without cell membrane disruption.

Our drugs are clear water solutions that penetrate “leaky” cancer cell membranes.

When the drug is inside the cancer cell, damage to the nucleus & tubulin occurs.

Studies indicate that normal, healthy cells do not absorb INT230-6.

"LEAKY" CANCER CELL MEMBRANE

HEALTHY CELLS UNHARMED

Membrane Fluidity in Cancer Cell Membranes as a Therapeutic Target...
www.cell.com/biophysj/abstract/S0006-3495(14)02570-3
INT230-6: Absorbed by tumors when dosed intratumorally

**CYTOTOXIC AGENTS ALONE + dye**

- Drug NOT absorbed, significant leakage
- Drug leaks into interstitial space
- Little to no drug dispersion in all tumors tested

**INT230-6 + dye**

- Injections made to center of tumor over 90 sec
- INT230-6 is ABSORBED
- No leakage
- Significant dispersion in all tumors tested

Results of intratumoral tumor absorption by DfuserRx technology confirmed at 3 different labs

Mouse pancreatic cancer model
INT230-6: Formulation’s efficacy is superior to cytotoxic agents alone (IV or IT)

Growth Inhibition

<table>
<thead>
<tr>
<th>Tumor Volume (mm³)</th>
<th>Days</th>
</tr>
</thead>
<tbody>
<tr>
<td>No treatment</td>
<td></td>
</tr>
<tr>
<td>Cis/Vbl IV</td>
<td></td>
</tr>
<tr>
<td>Cis/Vbl IT</td>
<td></td>
</tr>
<tr>
<td>INT230-6 IT</td>
<td></td>
</tr>
</tbody>
</table>

Survival

<table>
<thead>
<tr>
<th>% Remaining</th>
<th>Days</th>
</tr>
</thead>
<tbody>
<tr>
<td>No treatment</td>
<td></td>
</tr>
<tr>
<td>Cis/Vbl IT</td>
<td></td>
</tr>
<tr>
<td>INT230-6 IT</td>
<td></td>
</tr>
</tbody>
</table>

Research grade formulation
INT230-6: Clinical grade formulation is more potent

- Large tumor volume ($\geq 225 \text{ mm}^3$)
- Low dose (~4mg/kg)
- 80% complete responders
- 100% of tumors regress from baseline
- One cycle of 3 or 5 daily doses

Data generated by the National Cancer Institute under CRADA
Immune Activation Mechanism Data
INT230-6: Causes increase in tumor microenvironment APCs

Tumor xenografts were stained with 1 μg/mL of anti-CD11c antibodies at Day 10 (40× shown)

~10 days after INT230-6 dosing, there is an increase in dendritic cells in the tumor microenvironment

- Cisplatin induces immunologic cell death by release of calreticulin to the cell surface.
- Cisplatin also produces high mobility group box 1 (HMGB1) protein, stimulating dendritic cell processing through interaction TLR-4.

Results using INT230-6 generated by NCI published 2019 jointly with Intensity Therapeutics in OncoImmunology 7/2019

Intratumorally delivered formulation, INT230-6, containing potent anticancer agents induces protective T cell immunity and memory

Anja C. Bloom, Lewis H. Bender, Shweta Tiwary, Lise Pasquert, Katharine Clark, Tianbo Jiang, Zheng Xia, Alzea Morales-Kastresana, Jennifer C. Jones, Ian Walters, Masaki Terabe, and Jay A. Berzofsky

Vaccine Branch, Center for Cancer Research, National Cancer Institute, Bethesda, MD, USA; Intensity Therapeutics, Westport, CT, USA

ABSTRACT
The benefits of anti-cancer agents extend beyond direct tumor killing. One aspect of cell death is the potential to release antigens that initiate adaptive immune responses. Here, a diffusion enhanced formulation, INT230-6, containing potent anti-cancer cytotoxic agents, was administered intratumorally into large (approx. 300 mm³) subcutaneous murine Colon26 tumors. Treatment resulted in regression from baseline in 100% of the tumors and complete response in up to 90%: CD8⁺ or CD8⁺/CD4⁺ T cell double-depletion at treatment onset prevented complete responses, indicating a critical role of T cells in promoting complete tumor regression. Mice with complete response were protected from subcutaneous and intravenous re-challenge of Colon26 cells in a CD4⁺/CD8⁺ dependent manner. Thus, immunological T cell memory was induced by INT230-6. Colon26 tumors express the endogenous retroviral protein gp70 containing the CD8⁺ T cell AH-1 epitope. AH-1-specific CD8⁺ T cells were detected in peripheral blood of tumor-bearing mice and their frequency increased 14 days after treatment onset. AH-1-specific CD8⁺ T cells were also significantly enriched in tumors of untreated mice. These cells had an activated phenotype and highly expressed Programmed cell-death protein-1 (PD-1) but did not lead to tumor regression. CD8⁺ T cell tumor infiltrate also increased 11 days after treatment: INT230-6 synergized with checkpoint blockade, inducing a complete remission of the primary tumors and shrinking of untreated contralateral tumors, which demonstrates not only a local but also systemic immunological effect of the combined therapy. Similar T-cell dependent inhibition of tumor growth was also found in an orthotopic 4T1 breast cancer model.
Selective depletion study:

- **IN3T20-6:** Mechanism of action includes anti-tumor attack by activated immune cells *(Clinical grade formulation)*

- **Baseline**
  - No CR

- **CR:**
  - IgG SHAM DEPLETION: CR: 8/10
  - CD4 DEPLETION: CR: 7/10
  - CD8 DEPLETION: No CR
  - CD4/CD8 DEPLETION: No CR

- **Tumor volume (mm³)**

- **% survival**

- **Log-rank test:** both p<0.0007

- **Final destruction of the tumor is immune cell dependent**

- **Both CD8 and CD4 T-cell responses are present**

*Data generated by the National Cancer Institute under CRADA*

Oncolimmunology: On-line July 16, 2019: https://doi.org/10.1080/2162402X.2019.1625687
INT230-6: Highly synergistic with anti-PD-1 checkpoint blockade (Data from the NCI)

Dual tumor model plus anti-PD-1 (colon 26):

Overall survival:

- Vehicle
- Anti-PD-1
- INT230-6
- Anti-PD-1 + INT230-6

PD1 has minimal effect on large primary tumors but regresses small mets.

Vehicle injected to primary has mild abscopal effect.

Untreated contralateral flank:

Treated contralateral flank:

Published in OncoImmunology: On-line July 16, 2019: https://doi.org/10.1080/2162402X.2019.1625687
INT230-6 plus anti-CTLA-4 shows synergy in dual tumor model, distal tumor regression (Data from the NCI)

Dual tumor model plus anti-CTLA-4 (colon 26):

-14 -3 0 5 10 study day

C26 primary  C26 anti-CTLA4

Overall survival:

Percent survival

Days post initial dose of drug

Injected tumors

Tumor volume (mm³)

Days post initial dose

INT230-6 plus anti-CTLA-4 shows synergy in dual tumor model, distal tumor regression (Data from the NCI)

Bystander tumors

Tumor volume (mm³)

Days post initial dose

OncoImmunology: On-line July 16, 2019: https://doi.org/10.1080/2162402X.2019.1625687
## INT230-6: Remarkable pre-clinical results

### MULTIPLE TUMOR TYPES

In vivo **efficacy in multiple tumor types:** breast, melanoma, colon, and pancreatic cancer

Large tumors, low doses

### DURABLE COMPLETE RESPONSES

Up to 80% of animals have a complete responses; **life long protection against the cancer**

### PREVENTION

**Prevention** of metastases

### REGRESSION

**Regression** of untreated, established **distal** tumors

### SYNERGY

Strong **synergy with immune checkpoint** inhibitors

### CROSS-PRIMING

Immunity to colon tumors leads to resistance against breast cancer

### NO LOCAL TOXICITY

No local toxicity following injection into 3 tissue sites (dogs) & multiple in humans

### T-CELL ACTIVATION

Mechanism is direct cell kill along with T-cell activation (CD8/CD4)
CLINICAL STUDY RESULTS
Engaged with major academic medical centers

INT230-6 PHASE I/II CLINICAL TRIAL SITES
NEW SITES COMING

UNIVERSITY OF SOUTHERN CA
Norris Comprehensive Cancer Center, HOAG Cancer Center

UNIVERSITY HEALTH NETWORK TORONTO
Princess Margaret Cancer Center

COLUMBIA UNIVERSITY
The New York and Presbyterian Hospital

UNIVERSITY OF MA
UMASS Memorial Medical Center

TEMPLE UNIVERSITY
Fox Chase Cancer Center

JOHNS HOPKINS UNIVERSITY
Sydney Kimmel Center
INT230-6 Phase I/II adaptive clinical trial design
SAFETY IS PRIMARY ENDPOINT IN PHASE 1

Patients with metastatic disease who failed all therapies
Intra-patient dose escalation over 5 doses
Multiple deep tumors can be injected at 1 session

First Cohort Completed
• Superficial tumors (1:4)
• 1 dose per month
• Start at 5 mL

Second Cohort Completed
• Superficial tumors (1:4)
• Escalation to 30 mL
• Every 2 weeks

Third Cohort Completed
• All tumor types (1:4)
• Up to 30 mL
• 1 per month

Fourth Cohort Completed
• All tumor types (1:2)
• Escalation to 120 mL
• Dosed every 2 weeks

Current Cohort
• All tumor types (1:3)
• Escalation 220 mL
• Every 2 weeks

Next Cohorts
• Combination with Merck’s Keytruda
  • Safety lead in, then expansion cohorts in PC, Bile duct, SCC and CRC
• Combination with CTLA-4 (in discussions)

Escalation by:
- increasing dose amount
- increasing dose frequency
- increasing dose load per tumor
45 patients treated to date (15 different cancer types)

- Dose per tumor is set by tumor volume: multiple tumors can be injected at a single session
- > 150 deep tumor injections to date; including lung and liver with 4 injections in liver at 1 session
- As much as 180 mL dosed in a single session – no reported drug leakage
- No dose-limiting toxicities
- Excellent safety
Clinical Safety Data
Cisplatin and Vinblastine Pharmacokinetics as of May 31

- Dose proportional Pk
- Majority of drug stays in tumor
- Minimal systemic levels consistent across many tumor types
### INT230-6 Phase I safety summary (n=42)

**LOW INCIDENCE OF TREATMENT RELATED ADVERSE EVENTS (SEPTEMBER 30 DATA)**

<table>
<thead>
<tr>
<th>PT</th>
<th>Grade 1</th>
<th>Grade 2</th>
<th>Grade 3</th>
<th>Grade 4</th>
<th>Grade 5</th>
<th>Total</th>
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</thead>
<tbody>
<tr>
<td>Any</td>
<td>9 (20.9%)</td>
<td>20 (46.5%)</td>
<td>7 (16.3%)</td>
<td>0</td>
<td>0</td>
<td>36 (83.7%)</td>
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<tr>
<td>Localized tumor-related pain</td>
<td>11 (25.6%)</td>
<td>8 (18.6%)</td>
<td>1 (2.3%)</td>
<td>0</td>
<td>0</td>
<td>20 (46.5%)</td>
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<tr>
<td>Fatigue</td>
<td>4 (9.3%)</td>
<td>10 (23.3%)</td>
<td>1 (2.3%)</td>
<td>0</td>
<td>0</td>
<td>15 (34.9%)</td>
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<tr>
<td>Nausea</td>
<td>11 (25.6%)</td>
<td>3 (7.0%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>14 (32.6%)</td>
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<tr>
<td>Decreased appetite</td>
<td>4 (9.3%)</td>
<td>7 (16.3%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>11 (25.6%)</td>
</tr>
<tr>
<td>Vomiting</td>
<td>9 (20.9%)</td>
<td>2 (4.7%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>11 (25.6%)</td>
</tr>
<tr>
<td>Anaemia</td>
<td>2 (4.7%)</td>
<td>4 (9.3%)</td>
<td>2 (4.7%)</td>
<td>0</td>
<td>0</td>
<td>8 (18.6%)</td>
</tr>
<tr>
<td>Back pain</td>
<td>3 (7.0%)</td>
<td>1 (2.3%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>4 (9.3%)</td>
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<tr>
<td>Chills</td>
<td>4 (9.3%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>4 (9.3%)</td>
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<tr>
<td>Dizziness</td>
<td>4 (9.3%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>4 (9.3%)</td>
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<tr>
<td>Abdominal pain</td>
<td>1 (2.3%)</td>
<td>1 (2.3%)</td>
<td>1 (2.3%)</td>
<td>0</td>
<td>0</td>
<td>3 (7.0%)</td>
</tr>
<tr>
<td>Blood creatinine increased</td>
<td>2 (4.7%)</td>
<td>1 (2.3%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3 (7.0%)</td>
</tr>
<tr>
<td>Dry mouth</td>
<td>2 (4.7%)</td>
<td>1 (2.3%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3 (7.0%)</td>
</tr>
<tr>
<td>Dysgeusia</td>
<td>2 (4.7%)</td>
<td>1 (2.3%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3 (7.0%)</td>
</tr>
<tr>
<td>Myalgia</td>
<td>2 (4.7%)</td>
<td>1 (2.3%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3 (7.0%)</td>
</tr>
<tr>
<td>Groin pain</td>
<td>1 (2.3%)</td>
<td>1 (2.3%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2 (4.7%)</td>
</tr>
<tr>
<td>Haematuria</td>
<td>2 (4.7%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2 (4.7%)</td>
</tr>
<tr>
<td>Headache</td>
<td>1 (2.3%)</td>
<td>1 (2.3%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2 (4.7%)</td>
</tr>
</tbody>
</table>

Related AE’s by max severity in more than 1 patient

No maximum tolerated dose yet found

Intensity Confidential Information
Good safety at doses administered and is well-tolerated

- Well-tolerated
- No maximum tolerated dose yet found; up to 180 mL injected at one time
- Primarily side effect: temporary discomfort at injection site, fatigue
- PK data show >75% to 90% of the two drugs remain in the tumor

Ongoing Phase I/II clinical trial: Safety, tolerability, & efficacy overview
Evidence of Anti-Cancer Activity
Prolonged benefit in several patients with highly refractory cancers who have failed all approved, appropriate therapies for their disease.

The top 17 patients had 4x the drug to tumor volume dose as the lower 17; this led to much better outcomes.
INT230-6 Phase I/II clinical trial:
Safety, tolerability, & efficacy overview

Early signs of efficacy

• Injected tumor response; INT230-6 appears to cause tumor necrosis and/or visual regression in injected lesions
• Increase in circulating CD8+/CD4+ T-cells
• Abscopal effect observed for untreated tumors in patients
• Shows activity as monotherapy

>30% increases in CD8+ and CD4+ T-cells compared to baseline (n=25) at 2 weeks

Ongoing Phase I/2 clinical study; safety data reporting will continue in 2019
Pre and Post Clinical Dosing of INT230-6: Down regulation of proliferating cells with upregulation of immune cell infiltrates.

Example of a breast cancer subject with a pre- and one month post-first dose biopsy.
* 75% reduction in Ki 67+ cells (magenta)
* 73% reduction in FoxP3 (orange)
* Significant increases into tumor of CD4 and CD8 T-cells (whole section image analysis).
INT230-6 Phase I/II trial: Case study highlights

DEEP SQUAMOUS CELL CARCINOMA PATIENT

PRIOR HISTORY: Failed 2 surgeries, radiation, chemotherapy
• Two large deep tumor nodules in muscle of left arm
• Prior to study (Jan ’18), recommendation for arm and shoulder amputation
• Failure on all prior therapy

AFTER INT230-6:
• Increase in necrosis and inflammation of tumor
• Increase in volume
• No RECIST progression
• Blood shows increase in:
  • T-cells (CD4+ and CD8+)
  • Macrophages
  • TNF-α

METASTATIC SQUAMOUS CELL CARCINOMA PATIENT

PRIOR HISTORY:
• Inoperable ulcerated tumor; no approved appropriate therapy

AFTER INT230-6:
• Decrease in tumor size >50% after 4 months dosing
• Blood shows increase in T-cells (CD4+ and CD8+) and decrease in macrophages

Results observed despite minimal dosing and non-optimal drug-to-tumor ratios
Case study spotlight: Chordoma patient

CHORDOMA PATIENT
a rare type of spinal or skull tumor – NO APPROVED DRUG THERAPY

There is no approved drug therapy;
90% of patients succumb to the disease

PRIOR HISTORY AND CLINICAL PRESENTATION:
• Failed surgery, radiation, 5 different lines of chemo, targeted and immunotherapy
• Tumors (245cc, 39cc, 37cc) protruding from spine/buttocks

ON STUDY TREATMENT:
• Received all 5 doses (20 or 30 mL per session)
• Main tumor was under-dosed

Patient presented with 3 large inoperable tumors in the sacrococcygeal area with metastases in lungs

Potential for early registration study in chordoma
Case study spotlight: Chordoma patient

**SPINE TUMOR**
Treated with low dose INT-230-6 to gluteal mass

Patient experienced **58% reduction** in primary spine tumor + near complete regression in untreated metastatic lung tumor

**LUNG TUMOR**
Untreated left lingula tumor (abscopal effect)

- Treated tumors became necrotic, cystic and regressed
- Abscopal effect seen in untreated lung tumor
- Patient requested re-treatment and is back on study

Pre-dose:
March 21, 2018
45.4mm x 26.9 mm

4 months into treatment:
July 27, 2018
26.7mm x 19.1 mm
PATIENT WITH CHORDOMA
Prolonged benefit in several refractory cancers receiving a proper dose to tumor volume into multiple tumors

- **Chordoma**
  - Progressed for 4 years on several therapies – had tumor PRs, stable 18 mon.; INT230-6 only – abscopal

- **Squamous cell carcinoma (2)**
  - Patient 1, after INT230-6 patient received anti-PD-1; had a complete response (CR)
  - Patient 2, had a partial response (PR) on INT230-6. 2 yrs. Stable. (9 mon. 1 dose of T-Vec no change)

- **Liposarcoma**
  - Failed multiple surgeries; failed Doxil plus Larturvo® (Olaratumab); then dosed INT230-5 has a PR, 35% regression of all tumors at 8 months (uninjected tumors also declined – abscopal effect)

- **Breast cancer patients (4)**
  - Failed 8 therapies – TNB subject received two sessions of INT230-6 now stable 13 months
  - Failed 7 therapies – Completed INT230-6; stable 11 months, regression of liver tumors, abscopal effect
  - Failed 8 therapies – TNB subject 1st dose 8/2019 2 large tumors –necrotic and sloughing off 10/2019
  - Failed 11 therapies - (pseudo?) progression at 4 months after INT230-6, dosed Ixabepilone*

*"Mediastinal and hilar lymphadenopathy has resolved, hepatic masses have decreased in size and activity significantly, aortocaval lymph node resolved, and bony lesions SUV down significantly. The patient is doing very well clinically and is traveling and active. Ixabepilone has ~ 19% ORR in refractory breast patients and PFS ~ 3 months."

His treating breast oncologist is doubtful that this response is from the Ixabepilone alone
Clinical Development
INT230-6 PLUS KEYTRUDA COHORT

• Safety lead in in superficial tumors

• Dosing INT230-6: Q2W (2 months) and Keytruda Q3W (2 yrs)
  • Start both drugs on Day 1

• Cohorts of ~10 to 15 patients per tumor type
  • Colorectal carcinoma (non-MSI high) *
  • Pancreatic *
  • Bile duct *
  • Squamous cell (any type)

* Non-immunogenic cancer: Can INT230-6 turn cold tumors hot?
### OUR PIPELINE

<table>
<thead>
<tr>
<th>Compound</th>
<th>Phase II Studies Start</th>
<th>Indications</th>
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<tbody>
<tr>
<td>INT230-6</td>
<td>2019</td>
<td>Monotherapy, Sarcoma, Int230-6 + Keytruda, INT3XXX + Keytruda, INT4XXX + Keytruda</td>
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<tr>
<td>INT230-6 + Keytruda</td>
<td>2019</td>
<td>Safety, then Phase 2</td>
</tr>
<tr>
<td>INT230-6 + CTLA-4</td>
<td>Expected 2020</td>
<td>Safety, then Phase 2</td>
</tr>
</tbody>
</table>

**Phase II Studies Start:** Planned in 1H 2020; After 6 patient lead in safety cohort

**Indication Agnostic:**
- INT230-6 + Keytruda
- INT230-6 + CTLA-4
- INT3XXX (Undisclosed Target)
- INT4XXX (Undisclosed Target)

**Preclinical Animal Pharmacology Testing:**
- Breast Cancer (Triple Neg.)
- Sarcoma
- Pancreatic Cancer
- Squamous Cell, CRC, Bile

**GlP Toxicology:**
- Per Fast Track: Monotherapy

**Phase I Dose Escalation:**
- INT230-6

**Phase II**
- INT230-6 + Keytruda
- INT230-6 + CTLA-4
- INT3XXX (Undisclosed Target)
- INT4XXX (Undisclosed Target)

**Phase III**
- INT230-6 + Keytruda
- INT230-6 + CTLA-4
- INT3XXX (Undisclosed Target)
- INT4XXX (Undisclosed Target)
INT230-6: Optimal for combinations with checkpoint inhibitors

- **PD-1 + stimulatory IO**
  - Extra stimulation
  - Not much benefit

- **PD-1 or CTLA4 + systemic chemotherapy**
  - Approved in lung cancer
  - Approved in breast cancer
  - Antigen release, but reduced immunogenicity from IV chemo agents
    - (with anti-PD-1)

- **OPTIMAL ANTIGEN RELEASE + IMMUNE PRIMING**
  - Immune system intact
  - Minimal toxicity
  - Direct tumor kill (bulk destruction)
  - Broad, quality antigen presentation

- **PD-1 or CTLA-4 + systemic chemotherapy**
Use of proceeds from next financing

• Phase 1/2 safety component of INT230-6 & Keytruda study to start 2019;
  • Start expansion phase 2 cohorts t 1H 2020 – possible efficacy read-out early 2021
  • Increase clinical staff
  • Increase number of enrolling centers
• Initiate phase 2 expansion (monotherapy) in TNBC and/or liposarcoma in Q1 2020 data read-out early 2021
• Initiate development of next generation DFuseRx drug product in 2020
• GMP production: On boarding of second supplier for GMP INT230-6 drug product manufacture & scale-up (2020)
• Initiate CTLA-4 combination study in 1H 2020 with data readout in 1H 2021
Summary of Intensity Therapeutics

**Corporate:**
- Partnerships with Merck & the National Cancer Institute
- Fast track designation from FDA
- Issued US and foreign patents
- Cash efficient:
  - Low burn - capital until Q4 2020

**Science & Technology:**
- Delivery platform (proprietary products)
- Product INT230-6: comprised of proven anti-cancer agents
- Clinical evidence of single therapy activity against multiple cancers with excellent safety
- Next generation products in preclinical testing
A BOLD NEW PERSPECTIVE ON CANCER

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