Next-Generation Cancer Therapeutics

April 2015
This presentation contains "forward-looking statements" as that term is defined under the Private Securities Litigation Reform Act of 1995 (PSLRA), including statements regarding expectations, beliefs or intentions regarding our business, technologies and products strategies or prospects. Such forward-looking statements are characterized by future or conditional verbs such as “may,” “will,” “expect,” “intend,” “anticipate,” “believe,” “estimate” and “continue” or similar verbs. Actual results may differ from those projected due to a number of risks and uncertainties, including, but not limited to, the possibility that some or all of the pending matters and transactions being considered by the Company may not proceed as contemplated, as well as risks inherent in additional financing, developing and obtaining regulatory approvals of new, commercially-viable and competitive products and product candidates, including timelines, the size of clinical trials, sufficiency of data from those trials and the requirements of the FDA for potential approval of Cynviloq™ and by all other matters described in the Company's filings with the Securities and Exchange Commission, including the risk factors set forth therein. These statements are made based upon current expectations that are subject to risk and uncertainty and information available to the Company as of the date of this presentation. The Company does not undertake to update forward-looking statements in this presentation to reflect actual results, changes in assumptions or changes in other factors affecting such forward-looking information. Assumptions and other information that could cause results to differ from those set forth in the forward-looking information can be found in the Company's filings with the Securities and Exchange Commission, including its most recent periodic report. We intend that all forward-looking statements be subject to the safe-harbor provisions of the PSLRA.
A Comprehensive Oncology Company

Deep and Complementary Pipeline Creates Significant Opportunities-
Novel breakthrough combination therapeutic regimens and modalities to attack cancer
Significant reduction in clinical development costs and timeline
Significant commercial edge in future drug pricing

Small Molecules

Cytotoxics
CYNVILOQ™

Targeted Therapy
MYC inhibitor
TRAIL modulator

Biologics

Immunotherapy
PD-1, PD-L1, CTLA-4
Bispecific Abs

Targeted Therapy
Anti-VEGFR2 ADC
Anti-c-MET ADC
Bispecific ADC

Cell Therapy

Adoptive Cellular Immunotherapy
Chimeric Antigen Receptor Tumor-attacking Neukoplast®
(Partnership with Conkwest)

Supportive Care

RTX
Intractable Cancer Pain

CYNVILOQ, CAR.TNK, CAR.TNK (Chimeric Antigen Receptor Tumor-attacking Neukoplast) are trademarks owned by Sorrento Therapeutics, Inc. Neukoplast is a trademark owned by Conkwest, Inc.
Patient enrollment in TRIBECA registration trial completed. Pilot PK suggests bioequivalence (BE) between Cynviloq and albumin-bound paclitaxel

Exclusive global partnership with Conkwest to develop next generation anti-cancer cellular immunotherapy with "Off-the-Shelf" CAR.TNK™ (Chimeric Antigen Receptor Tumor-attacking NeuKoplast)

First joint venture with NantWorks and Abraxis BioScience Inc. founder, Dr. Patrick Soon-Shiong, to develop next generation immunotherapies for the treatment of cancer and autoimmune disease.

NantCell global collaboration to discover and develop novel anti-cancer immunotherapies against neoepitopes of tumor-specific antigens discovered using NantWorks' proprietary pan-omics based, precision medicine approach

Licensing agreement to develop and commercialize anti-PD-L1 mAb with Lee’s Pharmaceutical for greater Chinese Market

Exclusive research and option agreement to generate and develop antibody-drug conjugates (ADCs) with Morphotek / Eisai
Deep and Complementary Pipeline Creates Significant Opportunities

<table>
<thead>
<tr>
<th>INDICATION</th>
<th>TARGET</th>
<th>PRECLINICAL</th>
<th>PHASE 1</th>
<th>PHASE 2</th>
<th>PHASE 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>CYNVILOQ™</td>
<td>Metastatic Breast Cancer</td>
<td>T R I B E C A*</td>
<td>Registration trial completed</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Non-Small Cell Lung Cancer</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>RTX</td>
<td>Intractable Cancer Pain</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>G-MAB Bi-Specific Ab</td>
<td>Immuno-oncology</td>
<td>PD-L1, PD1, CD47, CD137</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>PD-L1/c-MET; PD-L1/CTLA-4, PD-L1/EGFR</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ADC</td>
<td>VEGFR2, c-MET, CXCR5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MYC Inhibitor</td>
<td>Solid tumors and hematological malignancies</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* TRIBECA 505 (b)(2) Bioequivalence trial versus albumin-bound paclitaxel (Abraxane®) (paclitaxel albumin-bound particles for injectable suspension) (albumin-bound). Abraxane® is a registered trademark of and marketed by Celgene Corp. PDL1.TNK, CD123.TNK, ROR1.TNK, PSMA.TNK are trademarks owned by Sorrento Therapeutics, Inc.
Lead Oncology Product Opportunity

Cynviloq
(Paclitaxel polymeric micelle)

Registration Trial
**Cynviloq: Next Generation Paclitaxel Therapy**

**Generation**

1st
- **Taxol®**
- paclitaxel

2nd
- **Albumin-bound**
- paclitaxel

3rd
- **Cynviloq**
- paclitaxel polymeric micelle

**Formulation**

<table>
<thead>
<tr>
<th>Generation</th>
<th>Formulation</th>
<th>Maximum Tolerated Dose</th>
<th>Peak Product Sales</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st</td>
<td>Cremophor EL excipient: Polyoxyethylated castor oil</td>
<td>175 mg/m²</td>
<td>~ $1.6B (WW in 2000)</td>
</tr>
<tr>
<td>2nd</td>
<td>Biological polymer: Donor-derived human serum albumin (HSA)</td>
<td>260 mg/m²</td>
<td>$2.2 B* (2020) MBC, NSCLC, PC</td>
</tr>
<tr>
<td>3rd</td>
<td>Chemical polymer: Poly-lactide and polyethylene glycol diblock copolymer</td>
<td>&gt;300 mg/m² (up to 435 mg/m²)</td>
<td>Conversion of paclitaxel sales + new indications</td>
</tr>
</tbody>
</table>

*Celgene Presentation at JPM Healthcare Conference Jan 2015
Cynviloq Clinical Development Summary

<table>
<thead>
<tr>
<th>Phase 1:</th>
<th>Trials established MTD at &gt;300 mg/m² - Dana Farber Cancer Inst, Russia, &amp; S. Korea (total n=80) &gt;300 mg/m² (q3w) vs. 175 mg/m² (Taxol; weekly)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phase 2:</td>
<td>Completed trials in MBC, NSCLC, PC, OC, BC; in US - Yale Cancer Center, Russia, S. Korea (total n=259) Possible Phase 3 sNDA programs in these tumor types</td>
</tr>
<tr>
<td>Phase 2b*:</td>
<td>Chemo-naïve Stage IIIb/IV NSCLC vs Taxol in S. Korea (total n=276; Cynviloq n=140) 230 mg/m² + cis (q3w) vs. Taxol 175 mg/m² + cis; non-inferiority established</td>
</tr>
<tr>
<td>Phase 2*:</td>
<td>1st line treatment of OC vs Taxol in S. Korea (total n=100; Cynviloq n=50) 260 mg/m² + carbo (q3w) vs. Taxol 175 mg/m² + carbo; non-inferiority established</td>
</tr>
<tr>
<td>Phase 3:</td>
<td>MBC in S. Korea (total n=209; Cynviloq n=105 vs Taxol n=104) GPMBC301. An Open-label, Randomized, Parallel, Phase 3 Trial to Evaluate the Efficacy and Safety of Cynviloq compared to Genexo® (Paclitaxel with Cremophor EL) in Subjects with Recurrent or Metastatic Breast Cancer</td>
</tr>
<tr>
<td>PM-Safety:</td>
<td>Completed for MBC and NSCLC (total n=502) Efficacy and safety data supportive of 505(b)(2) submission</td>
</tr>
</tbody>
</table>

Total number of patients across all trials: 1,260

Data on file: * Investigator Initiated Study
## Comparative Phase 3 MBC Clinical Results

<table>
<thead>
<tr>
<th>Group</th>
<th>Overall Response Rate (%)</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cynviloq, South Korea*</td>
<td>39.5%</td>
<td>105</td>
</tr>
<tr>
<td>Taxol, South Korea*</td>
<td>28.5%</td>
<td>104</td>
</tr>
<tr>
<td>Albumin-bound paclitaxel, US**</td>
<td>36%</td>
<td>209</td>
</tr>
<tr>
<td>Taxol, US**</td>
<td>27%</td>
<td>205</td>
</tr>
<tr>
<td>Albumin-bound paclitaxel, China***</td>
<td>40%</td>
<td>81</td>
</tr>
<tr>
<td>Taxol, China***</td>
<td>33%</td>
<td>82</td>
</tr>
</tbody>
</table>

*p = 0.03, **p = 0.001, ***p = 0.025

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Bioequivalence = Accelerated Pathway to Market

TRIBECATM

(TRial establishing BioEquivalence between Cynviloq™ and Albumin-bound paclitaxel)

- Patients with MBC

<table>
<thead>
<tr>
<th>Cycle 1</th>
<th>Cycle 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albumin-bound paclitaxel (n = 27)*</td>
<td>Cynviloq (n = 27)*</td>
</tr>
<tr>
<td>Albumin-bound paclitaxel</td>
<td>Cynviloq</td>
</tr>
</tbody>
</table>

Key Parameters:

- Dose: 260 mg/m²
- Infusion time: 30 min
- Duration: 3 weeks + crossover for 3 weeks
- Endpoints: AUC and Cmax (90% CI)

Note: Previous trial size estimate of 100 patients was based on PK simulation of albumin-bound paclitaxel and Cynviloq historical data with both drugs given at different doses and infusion rates. Based on the recent positive initial PK data and subject to FDA guidance, 53 patients may be sufficient to establish BE.

TRIBECATM (TRial establishing BioEquivalence between Cynviloq™ and Albumin-bound paclitaxel) is a trademark owned by Sorrento Therapeutics, Inc.
Pilot PK Data Analyses Suggest BE vs. Albumin-Bound Paclitaxel

BE Assessment and Sample Size Estimate

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Ratio of Cynviloq/Albumin-bound paclitaxel (%)</th>
<th>90% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ln(AUC$_{0 \text{ to } \infty}$)</td>
<td>109.1</td>
<td>93.98 – 126.58</td>
</tr>
<tr>
<td>Ln(C$_{\text{max}}$)</td>
<td>102.5</td>
<td>83.10 – 126.35</td>
</tr>
<tr>
<td>Point estimate</td>
<td>110 - Ln(AUC$_{0 \text{ to } \infty}$)</td>
<td>N = 53 with 90% power</td>
</tr>
</tbody>
</table>
111 patients recruited from Eastern Europe, USA and Asia
Initial reported AEs consistent with historical nab-paclitaxel toxicity profile
Estimated Timeline and Next Steps*

First patient dosed: **March 31, 2014**

Last patient in: **January 2015**

NDA filing: **Q3 2015**

Product launch (MBC and NSCLC): **2016**

*Estimates, subject to discussions with the FDA.*
Resiniferatoxin (RTX): A Novel, Non-opiate Analgesic
Intraganglionic: injection into or near the dorsal root ganglion

Intrathecal: injection into the cerebrospinal fluid space

Cross Sections of spinal cord*

Absence of TRPV1-positive cells after RTX treatment

* Adapted from Karai et al., 2004
Summary of Interim Data from the Phase 1/2 NIH Sponsored Trial

DESIGN OVERVIEW

6 advanced cancer patients with severe refractory pain received a single injection of RTX. Neuropathic pain, visceral and bone pain 2º to bone metastases (49-61 years; 4 M/ 2F, MBC, H&N, pancreatic, lymphoma, SCLC, endometrial cancer).

<table>
<thead>
<tr>
<th>No unexpected toxicities</th>
<th>All 6 patients had near complete relief post-injection</th>
<th>100% of non-ambulatory patients could walk post injection (n=2)</th>
<th>MTD not reached, additional dose optimization being explored</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinically meaningful improvement in QOL</td>
<td></td>
<td>Improved pain scores with increased activity</td>
<td></td>
</tr>
</tbody>
</table>

RESULTS
Next Steps for RTX Development

Complete intractable cancer pain clinical Phase 1/2 trial (\textit{intrathecal injection}) under Sorrento IND; n=45-60 patients; optimization of dosing study

End of Phase 2 meeting with FDA (for \textit{intrathecal injection})

Initiate Phase 3 (\textit{intrathecal injection})

Phase 1/2 trial(s) (\textit{intraganglionic injection})

End of Phase 2 meeting with FDA (for \textit{intraganglionic injection})

~3 years for clinical development
Immunotherapy Programs

G-MAB + Neukoplast + Proprietary Toxins & Conjugation Chemistries
G-MAB: Library of Therapeutic Antibodies

Proprietary technology:
- RNA amplification used for library generation
- Freedom-To-Operate

No stacking royalties

Very high library diversity:
- $2.1 \times 10^{16}$ distinct antibodies

Fully human antibodies

High successful screening hit rate
- (over 70 targets screened)

Ideal for CAR-Generation

Difficult Targets:
- Small Peptides

High Value Oncology Targets:
- Immune modulation: PD-1, PD-L1, CD47
- Antibody Drug Conjugates: VEGFR2, c-Met

Most Difficult Targets:
- G Protein-Coupled Receptors (GPCRs)

Size of Target Antigen
Anti-PD-L1 mAb Exhibits Potent Activity

**Immune Modulation***

- T Cell Activation (%)
- IFN-γ (pg/mL)
- IL-2 (pg/mL)

**Tumor Mouse Model***

- Tumor Growth Inhibition (%)

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* mAbs @ 0.05 mg/mL
** xenograft model using H1975 human NSCLC cells; % inhibition relative to control mAb treatment
*** p<0.05, mean tumor volumes are significantly reduced in STI-A1010 group versus control groups as determined by Mann-Whitney u-test
Anti-PD1 mAb Exhibits Excellent Activity

Immune Modulation*

- T Cell Activation (%)
- IFN-γ (pg/mL)
- IL-2 (pg/mL)

Target Specificity

- Control
- Sorrento mAb

*Immune Modulation:
-Competitor mAb
-Sorrento mAb

- Human PD1
-Cyno PD1
-Human CTLA-4
-Human CD28
-Human ICOS
-PBS control
K-Lock

Current industry standard

Proprietary K-Lock chemistry

Sorrento’s homogenous ADC
No need for:
- non-natural amino acids
- genetic re-engineering
- enzymatic posttranslational modification

C-Lock

Maleimide conjugation
Destabilizes antibody structure
Drug-antibody linkage not stable
Altered PK profile
Off-target drug effects

C-Lock conjugation
Enhances ADC stability
Prolongs PK profile
Reduces off-target effects
## Proprietary High Potency Duostatin Toxins

<table>
<thead>
<tr>
<th>EC&lt;sub&gt;50&lt;/sub&gt; (pM)</th>
<th>Cancer</th>
<th>Her-2</th>
<th>DM1</th>
<th>MMAE</th>
<th>Duostatin 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBKR3</td>
<td>Breast</td>
<td>+++</td>
<td>95</td>
<td>72</td>
<td>30</td>
</tr>
<tr>
<td>HCC1954</td>
<td>Breast</td>
<td>+++</td>
<td>124</td>
<td>78</td>
<td>68</td>
</tr>
<tr>
<td>BT474</td>
<td>Breast</td>
<td>+++</td>
<td>818</td>
<td>126</td>
<td>214</td>
</tr>
<tr>
<td>MDA-MB-361</td>
<td>Breast</td>
<td>+++</td>
<td>218</td>
<td>151</td>
<td>35</td>
</tr>
<tr>
<td>ZR75</td>
<td>Breast</td>
<td>+++</td>
<td>215</td>
<td>298</td>
<td>264</td>
</tr>
<tr>
<td>HCC1419</td>
<td>Breast</td>
<td>+++</td>
<td>391</td>
<td>271</td>
<td>332</td>
</tr>
<tr>
<td>MDA-MB-453</td>
<td>Breast</td>
<td>++</td>
<td>1,877</td>
<td>&gt;100,000</td>
<td>452</td>
</tr>
<tr>
<td>MDA-MB-175</td>
<td>Breast</td>
<td>+</td>
<td>&gt;100,000</td>
<td>1,348</td>
<td>425</td>
</tr>
<tr>
<td>N87</td>
<td>Gastric</td>
<td>+++</td>
<td>368</td>
<td>139</td>
<td>260</td>
</tr>
<tr>
<td>OE-19</td>
<td>Gastric</td>
<td>+++</td>
<td>176</td>
<td>164</td>
<td>130</td>
</tr>
<tr>
<td>SKOV-3</td>
<td>Ovarian</td>
<td>+++</td>
<td>150</td>
<td>251</td>
<td>144</td>
</tr>
</tbody>
</table>

Trastuzumab was used as targeting mAb
In Vivo Proof-of-Concept of Sorrento ADCs

**VEGFR2-ADC STI-D0168**

**c-MET-ADC STI-D0602**

A431 squamous-cell carcinoma cells; ^indicates dosing

U87 xenograft; dosing twice weekly; maytansinoid drug conjugates
Independent company focused on advancing next generation immunotherapies against cancer and autoimmune diseases.

Both companies will contribute to its pipeline of clinical and preclinical assets of novel and proprietary immunotherapies, ADCs, and bispecific antibodies.

Nantworks will contribute phase 3 antibody.

Nantibody will draw from NantWorks’ proteomic and genomic capabilities and Sorrento’s industry-leading, highly diverse G-MAB library.
AN EXCLUSIVE JOINT PARTNERSHIP
Advancing Cellular Immunotherapy Beyond CAR-T Cell Therapies

Neukoplast® NK cell line ("off-the-shelf")
- Broad anti-cancer activity in solid and liquid tumors
- No clinical DLTs/SAEs in over 40 patients treated

Vast diversity human antibody library
- High successful screening rate (over 70 targets screened)
- Proprietary technologies with FTO

Advanced proteomics platform
- Proprietary gene insertion (without use of lentiviruses)
- ‘GMP in a Box’ production technology
## CAR.TNK vs CAR-T: Key Differentiators

<table>
<thead>
<tr>
<th>Category</th>
<th>CAR.TNK</th>
<th>CAR-T</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cell Production</strong></td>
<td>Simple: Off-the-shelf universal product CAR-modified Neukoplast cells</td>
<td>Invasive: Autologous (patient-derived) invasive procedure/leukapheresis</td>
</tr>
<tr>
<td><strong>Transduction characteristics</strong></td>
<td>100%: Master cell bank with 100% of cells expressing CAR</td>
<td>Variable %: Variable CAR transfection &amp; expression</td>
</tr>
<tr>
<td><strong>MOA</strong></td>
<td>Broad: Multiple MOAs, targeting and killing through CAR-dependent and innate mechanisms (&quot;off-target / on-tumor&quot;)</td>
<td>Limited: Requires co-stimulators (CD80, CD86) not present in many solid tumors</td>
</tr>
<tr>
<td><strong>Safety</strong></td>
<td>Good: On-target / off-tumor effects limited due to short half life and lack of IL-6 production</td>
<td>Poor: Cytokine release syndrome, ICU; Prolonged bone marrow suppression; Cardiotoxicity; Reported cases of encephalitis; Death</td>
</tr>
<tr>
<td><strong>COGS</strong></td>
<td>Low: large scale bioreactor manufacturing for many patients</td>
<td>High: requires individual patient processing</td>
</tr>
</tbody>
</table>
Unmodified Neukoplast Clinically Validated In Several Phase 1 Studies

More than 40 patients treated
  Advanced metastatic disease refractory to chemo, biologics, cytokines, radiation, and surgery

Many patients received multiple dosing regimens (up to 6 months)

Promising activity against different cancer types, including acute myelogenous leukemia (AML), lymphoma (NHL, HL), melanoma, renal cell cancer (RCC), and lung cancers (SCLC, NSCLC)

No DLTs; only 1 “grade 4 SAE” (hypoglycemia likely related to tumor lysis)
Lymphocytes from 2 healthy donors co-cultured with each other → vigorous proliferation

Co-cultured with Neukoplast (7 days) → no proliferation
CAR.TNK: CAR-modified Neukoplast

Clonal cell lines expressing one or more CARs to establish a range of distinct products

Multiple killing mechanisms - CAR-targeted as well as broad intrinsic anti-cancer activity of Neukoplast (“off-target / on-tumor”)

Engages the adaptive immune system through cytokine secretion and immune cell recruitment

Titratable: repeat dosing option; controllable dose exposure to manage safety risk
Serial Killing of Her2+ Cells by Her2.TNK Cells

Homing to Her2 expressing tumors

Inhibition of Her2+ RCC metastasis

Growth inhibition and killing correlate with Her2 expression levels

“Serial killing” of Her2+ target cells even after gamma radiation with 10 Gy

Selective cytotoxicity (spares normal cells)
Her2.TNK Demonstrate Tumor Homing and Potent Anti-Glioma Activity in Mice

Tumor homing of CAR.TNKs

Intracranial LN-319 glioblastoma xenografts in NSG mice

Schoenfeld et al. Mol Therapy, in press
Prospective CAR.TNKs for Development (Initial List)

<table>
<thead>
<tr>
<th>Target</th>
<th>Potential Indication(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>EGFRviii.TNK</td>
<td>Glioma</td>
</tr>
<tr>
<td>EphA3.TNK</td>
<td>Glioma, AML</td>
</tr>
<tr>
<td>L1CAM.TNK</td>
<td>Gastric, pancreatic, NSCLC</td>
</tr>
<tr>
<td>CSPG4.TNK</td>
<td>H&amp;N, breast, mesothelioma</td>
</tr>
<tr>
<td>BCMA.TNK</td>
<td>Myeloma</td>
</tr>
<tr>
<td>ROR1.TNK</td>
<td>CLL, ALL, MCL, breast, lung, pancreas</td>
</tr>
<tr>
<td>PSMA or PSCA.TNK</td>
<td></td>
</tr>
<tr>
<td>PDL1.TNK</td>
<td>Myeloma, RCC, NSCLC, TNBC</td>
</tr>
<tr>
<td>CS1.TNK</td>
<td>Myeloma</td>
</tr>
<tr>
<td>CD123.TNK</td>
<td>AML</td>
</tr>
<tr>
<td>CD19.TNK</td>
<td>CLL, ALL</td>
</tr>
<tr>
<td>CD22.TNK</td>
<td>CLL, ALL</td>
</tr>
</tbody>
</table>

CAR targets jointly selected by the Steering Committee

Lead company will be responsible for all pre-clinical and clinical development, regulatory filings, and commercialization

Profit sharing on all CAR.TNKs revenues proportional to contribution
Next Steps for CAR.TNK Development

H1 2015  Generation of CARs

H2 2015  Generation and evaluation of stable CAR.TNK cell lines

2016     IND-enabling studies, IND submission, and initiation of Phase 1 studies
A Comprehensive Oncology Company

Deep and Complementary Pipeline Creates Significant Opportunities-
Novel breakthrough combination therapeutic regimens and modalities to attack cancer
Significant reduction in clinical development costs and timeline
Significant commercial edge in future drug pricing

Small Molecules
Cytotoxics
CYNVILOQ™
Targeted Therapy
MYC inhibitor
TRAIL modulator

Biologics
Immunotherapy
PD1, PD-L1, CTLA-4
Bispecific Abs
Targeted Therapy
Anti-VEGFR2 ADC
Anti-CMET ADC
Bispecific ADC

Cell Therapy
Adoptive Cellular Immunotherapy
Chimeric Antigen Receptor
Tumor-attacking Neukoplast®
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Supportive Care
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Next-Generation Cancer Therapeutics

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