
UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934

Date of Report (date of earliest event reported): **May 31, 2017**

Heat Biologics, Inc.

(Exact name of registrant as specified in charter)

Delaware

(State or other jurisdiction of incorporation)

001-35994

(Commission File Number)

26-2844103

(IRS Employer Identification No.)

**801 Capitola Drive
Durham, NC 27713**

(Address of principal executive offices and zip code)

(919) 240-7133

(Registrant's telephone number including area code)

N/A

(Former Name and Former Address)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of registrant under any of the following provisions:

- ☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)
- ☐ Soliciting material pursuant to Rule 14a-12(b) under the Exchange Act (17 CFR 240.14a-12)
- ☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))
- ☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Indicate by check mark whether the registrant is an emerging growth company as defined in in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by checkmark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☒

Item 8.01. Other Events

Heat Biologics, Inc. (the “Company”) will be making several investor presentations over the next several weeks. In connection with the presentations, the Company intends to discuss the slide presentation attached as Exhibit 99.1 hereto, which is incorporated herein by reference.

The slide presentation attached as Exhibit 99.1 to this Report includes “safe harbor” language pursuant to the Private Securities Litigation Reform Act of 1995, as amended, indicating that certain statements contained in the slide presentation or in the press release are “forward-looking” rather than historical.

The Company undertakes no duty or obligation to update or revise information included in this Report or any of the Exhibits.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Heat Biologics, Inc. investor presentation dated May 31, 2017



SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Dated: May 31, 2017

HEAT BIOLOGICS, INC.

By: /s/ Jeff Wolf
Name: Jeff Wolf
Title: Chairman, President and
Chief Executive Officer



EXHIBIT INDEX

Exhibit Number	Description
99.1	Heat Biologics, Inc. investor presentation, dated May 31, 2017



NASDAQ:HTBX

May 31, 2017

Forward Looking Statements

This presentation includes statements that are, or may be deemed, “forward-looking statements” within the meaning of the Private Securities Litigation Reform Act of 1995, as amended. In some cases, these forward-looking statements can be identified by the use of forward-looking terminology, including the terms “believes,” “estimates,” “anticipates,” “expects,” “plans,” “intends,” “may,” “could,” “might,” “will,” “should,” “approximately” or, in each case, their negative or other variations thereon or comparable terminology, although not all forward-looking statements contain these words. They appear in a number of places throughout this presentation and include statements regarding our intentions, beliefs, projections, outlook, analyses or current expectations concerning, among other things, our ongoing and planned discovery and development of drugs targeting cancer, the strength and breadth of our intellectual property, our ongoing and planned preclinical studies and clinical trials, the timing of and our ability to make regulatory filings and obtain and maintain regulatory approvals for our product candidates, our ability to partner our product development, the degree of clinical utility of our products, particularly in specific patient populations, expectations regarding clinical trial data, our results of operations, financial condition, liquidity, prospects, growth and strategies, the length of time that we will be able to continue to fund our operating expenses and capital expenditures, our expected financing needs and sources of financing, the industry in which we operate and the trends that may affect the industry or us.

By their nature, forward-looking statements involve risks and uncertainties because they relate to events, competitive dynamics, and healthcare, regulatory and scientific developments and depend on the economic circumstances that may or may not occur in the future or may occur on longer or shorter timelines than anticipated. Although we believe that we have a reasonable basis for each forward-looking statement contained in this presentation, we caution you that forward-looking statements are not guarantees of future performance and that our actual results of operations, financial condition and liquidity, and the development of the industry in which we operate may differ materially from the forward-looking statements contained in this presentation as a result of, among other factors, the factors referenced in the “Risk Factors” section of our Annual Report on Form 10-K for the year ended December 31, 2016 and our quarterly report on Form 10-Q for the subsequent quarters (collectively, our “SEC Filings”). In addition, even if our results of operations, financial condition and liquidity, and the development of the industry in which we operate are consistent with the forward-looking statements contained in this presentation, they may not be predictive of results or developments in future periods. Any forward-looking statements that we make in this presentation speak only as of the date of such statement, and we undertake no obligation to update such statements to reflect events or circumstances after the date of this presentation, except as required by law.

You should read carefully the factors described in the “Risk Factors” sections of our SEC Filings to better understand the risks and uncertainties inherent in our business.

Investment Opportunity

POTENTIAL BEST-
IN-CLASS

Activation of

Cancer
Killing CD8+ T Cells



Multiple
Platform
TECHNOLOGIES



ROBUST
Pipeline of
COMBINATION
Therapies



Favorable
Safety Profile



Signals in lung cancer
with

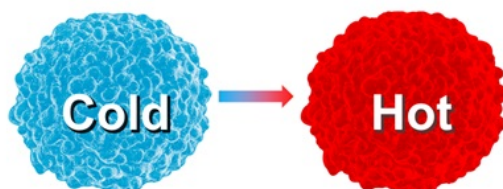
CHECKPOINT
Inhibitors



Turning **COLD** Tumors **HOT**



Our Mission is to Activate CD8+
"Killer" T cells to Turn "COLD" Tumors
"HOT"



Our goal is to combine with checkpoint inhibitors and other immunotherapies to dramatically increase their effectiveness

What are COLD and HOT tumors?

COLD Tumors

Tumors that have not been subject to robust CD8+ "Killer" T cell attack

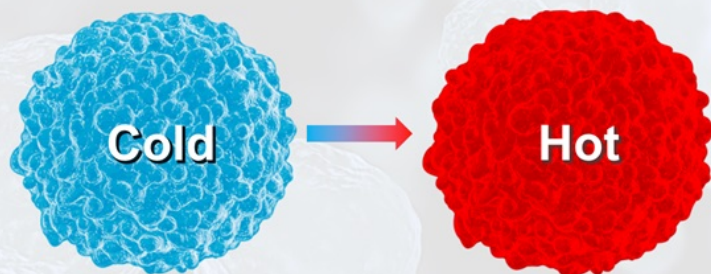
Biopsied tumors contain minimal CD8+ T cells

HOT Tumors

Tumors that have been subject to robust CD8+ "Killer" T cell attack

Biopsied tumors are loaded with CD8+ T-cells

HOT tumors are associated with clinical response



Turning **COLD** Tumors **HOT**

The Checkpoint Revolution

- Checkpoint inhibitors are dramatically changing the standard of care against a wide variety of cancers
 - 5 checkpoints approved to treat seven different cancers since 2014
 - Additional checkpoints against new tumors expected later this year
- Citibank, Goldman Sachs project checkpoints will be used to treat up to **60%** of cancers, generating **\$30B** – **\$40B** revenues per year

Approved Checkpoint Inhibitors

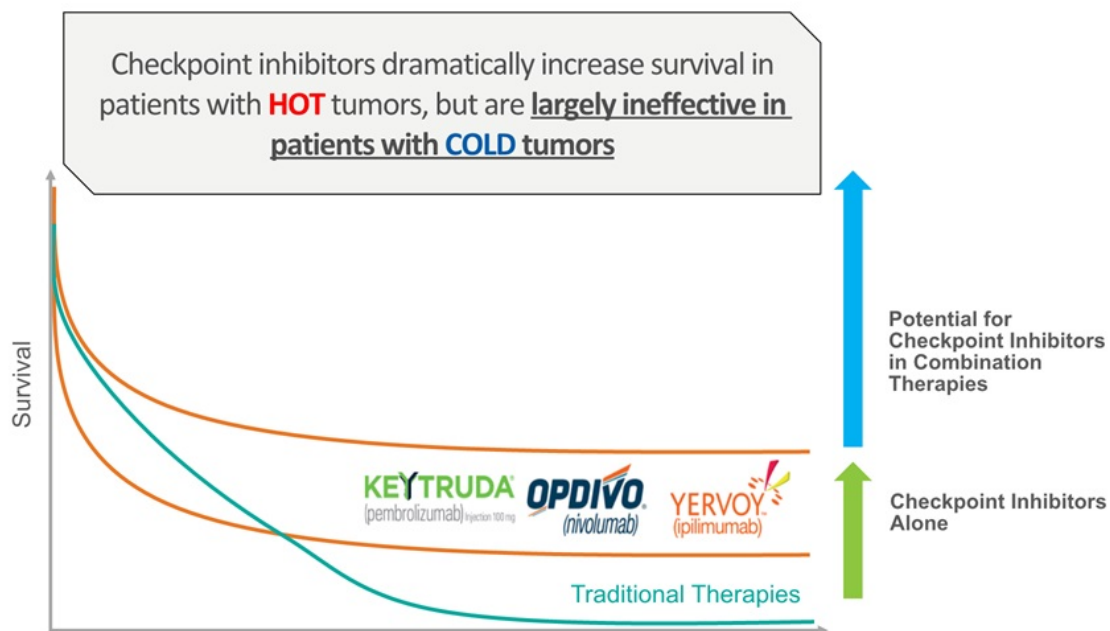
Targets	Checkpoint	Company	Approved Indications
CTLA-4	Ipilimumab	BMS	Metastatic melanoma
PD-1	Nivolumab	BMS	Metastatic melanoma NSCLC Bladder cancer Hodgkin lymphoma Renal cell carcinoma
PD-1	Pembrolizumab	Merck	Metastatic melanoma NSCLC Head and neck
PD-L1	Atezolizumab	Genentech	Bladder cancer
PD-L1	Avelumab	Pfizer	Merkel cell carcinoma

Additional checkpoint approvals expected later this year

But there is a problem...

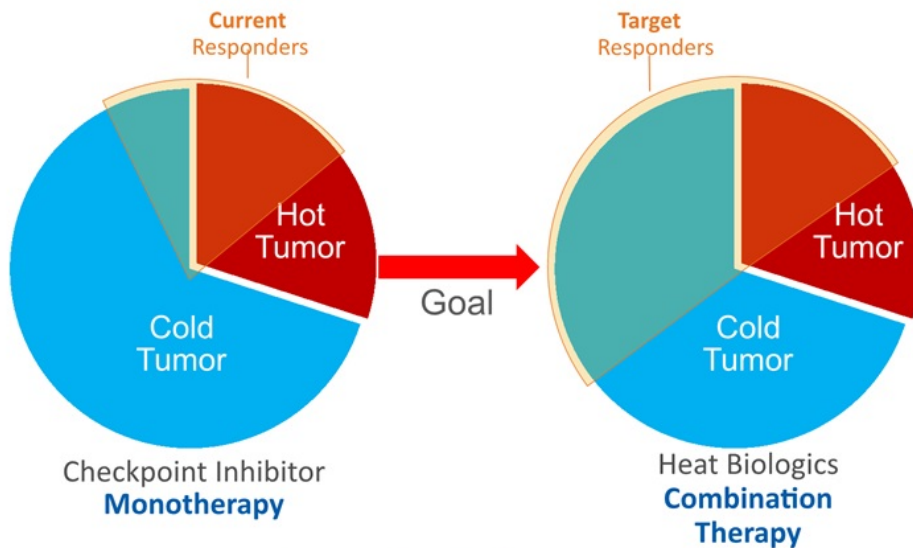


The Problem with Checkpoints



Turning COLD tumors **HOT**

Focused on turning **COLD** tumors **HOT** to enhance the effectiveness of checkpoint therapy for the majority of patients who don't respond to checkpoints alone

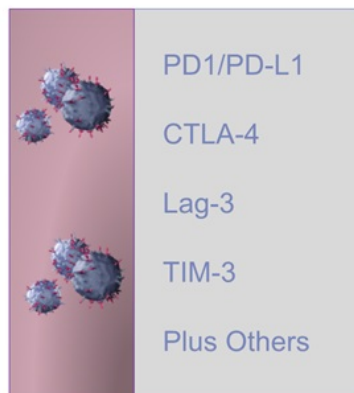


Our goal is to **dramatically increase** the number of patients responding to checkpoints

Checkpoint Combination Advantages

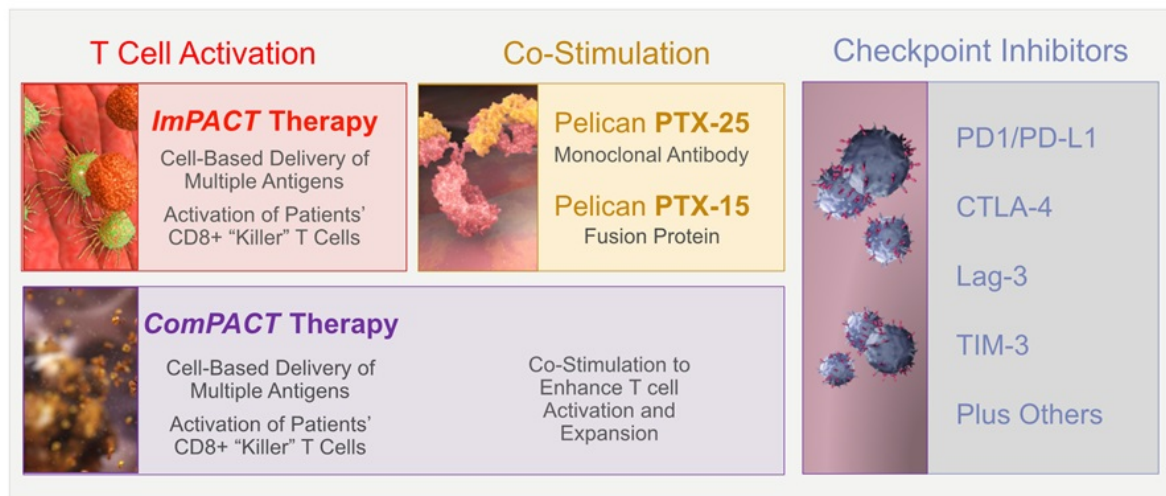
Heat's *ImPACT* Technology

- Activates CD8+ T cells, a critical component of effective anti-cancer immunity
- Activated T cells invade tumor site
- Simple once-a-week intradermal injection
- Well-tolerated with combinations
- Clinical and preclinical data supports activity with *ImPACT* and checkpoint inhibitors



Combination Therapies

Unlocking the Body's Natural Defenses with a Broad Range of Combination Therapies



ImPACT / ComPACT Therapies

“Off-the-shelf” Cellular Therapies to Activate “Killer” T Cells

T Cell Activation



ImPACT Therapy

Cell-Based Delivery of
Multiple Antigens
Activation of Patients'
CD8+ “Killer” T Cells



ComPACT Therapy

Cell-Based Delivery of
Multiple Antigens
Activation of Patients'
CD8+ “Killer” T Cells

Co-Stimulation to
Enhance T cell
Activation and
Expansion

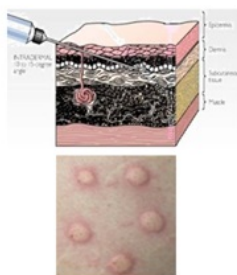
Robust, Multi-Antigen T Cell Activation



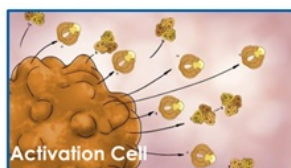
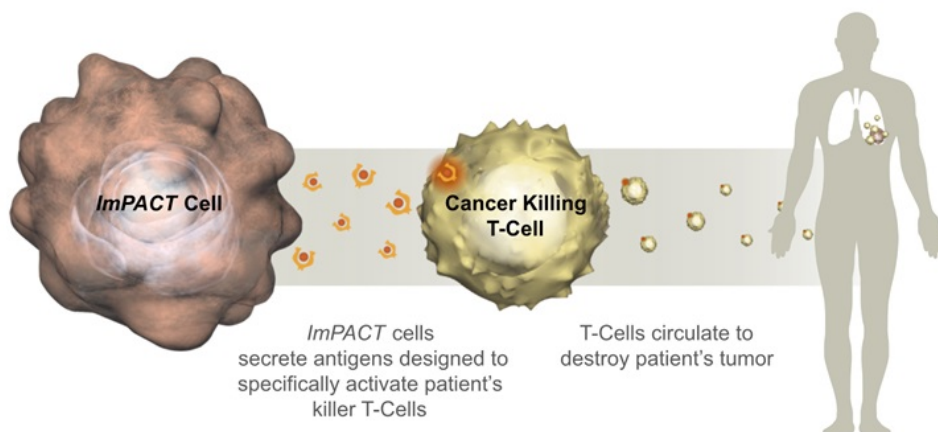
- Same process for *ComPACT* and *ImPACT*
- Frozen, fully-diluted single-dose vial
- Final drug product: 1 million or 10 million cells
- Easily scaled manufacturing

**Low COG, off-the-shelf
alternative to
autologous therapies**

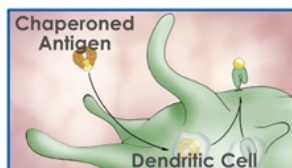
ImPACT: Immune Pan-Antigen Cytotoxic Therapy



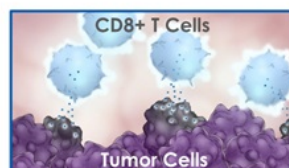
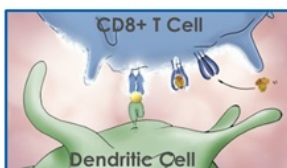
Cluster of five 0.1 mL intradermal injections



Activated cells **EXPRESS** chaperoned antigens



Chaperoned antigens **activate** dendritic cells which then **ACTIVATE & PROLIFERATE** CD8+ T cells



CD8+ T cells locate and **ELIMINATE** cancer cells

Heat Pipeline

Combination Therapies Designed to Activate the Body's Own CD8+ T Cells to Fight Cancer

THERAPEUTIC VACCINES			
HS-110 (viagenpumatucl-L)	NSCLC	Phase II	<i>ImPACT</i> activation technology in combination with nivolumab and other checkpoint inhibitors TBA
HS-120	NSCLC	Preclinical	<i>ComPACT</i> activation technology in combination with checkpoint inhibitors TBA
CO-STIMULATORS			
PTX-25	TBA	Preclinical	Humanized monoclonal antibody, functional agonist of human TNFRSF25
PTX-15	TBA	Preclinical	TL1A-Ig fusion protein, functional agonist of human TNFRSF25

Met Clinical Endpoints to Progress to Phase 2

HS-110 Phase 2 Lung Trial Design

Objective

- Evaluate objective response rate of HS-110 with a PD-1 checkpoint inhibitor
- Currently 2nd line therapy or greater

Patient Population

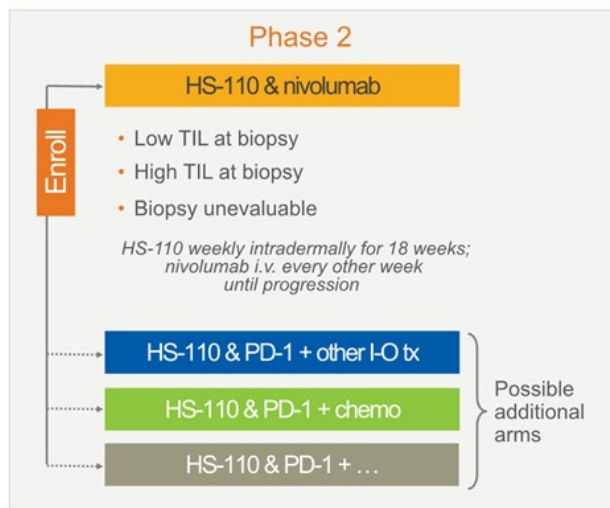
- Phase 1b expanded to Phase 2 in 1Q17 based upon efficacy

Secondary Endpoints

- Safety and tolerability, immune response, overall survival and progression-free survival

Enrollment

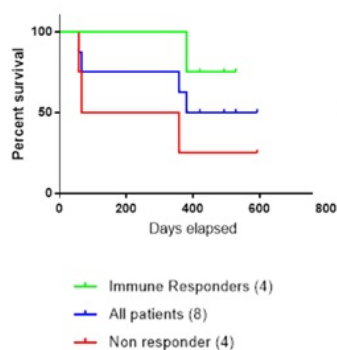
- 5 – 10 U.S. sites
- Up to 60 patients
- Partnership with Yale Cancer Center on TIL analysis



Flexible trial design permits additional combinations

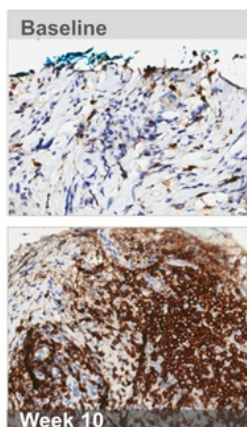
Activated Immune Response Correlates to Clinical Response

Overall Survival



Source: HS-110 Ph 1b NSCLC data (left); HS-110 Ph 1b NSCLC data reported at World Lung Dec. 2016 (right)

Response at Week 10



Key Findings

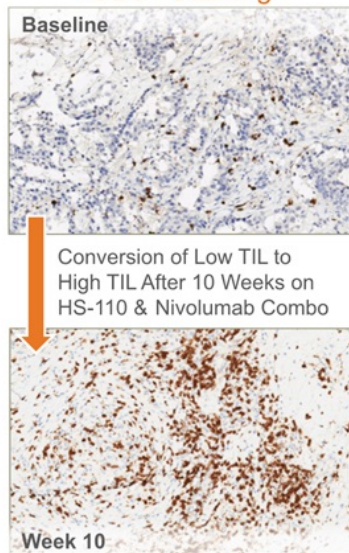
- Immune response to HS-110 observed in all 5 patients who had reduced tumors
- Patients with no immune response to HS-110 did not have reduced tumors
- Timing of immune responses to HS-110 corresponded to clinical responses
- Of 5 patients enrolled in the low TIL cold tumor cohort, 3 (60%) **exhibited substantial tumor reduction**
- This exceeds the 10% response observed in low TIL patients treated with nivolumab alone

Source: March, 2017 Heat Biologics Trial Update

T Cell Tumor Infiltration

T Cell Infiltration Observed Deep Inside Tumors

CD8+ Staining



Clinical evidence that HS-110 is turning COLD tumors HOT

- “Killer” CD8+ T cells driven deep into tumors
- “Cold” tumors with no previous activation made highly active (“HOT”)
- Expression of PD-L1 increased with CD8+ T cell infiltration in some tumors

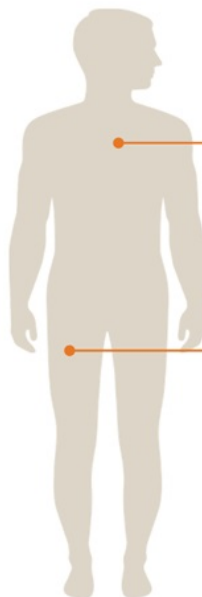
Source: Data extracted from a patient as reported in Heat's HS-110 Ph 1b NSCLC trial results, Dec 2016

Positive Safety Signals

1,000+ Doses – No Serious Adverse Reactions

Favorable Safety Profile To Date

- Over 1,000 doses administered to ~200 patients
- Only one patient ended treatment due to a non-serious adverse reaction*
- No systemic use of steroids required to treat reactions
- No serious adverse reactions
- No additive toxicities



Immune Reaction*

≤ Grade 3 toxicity



Injection Reactions

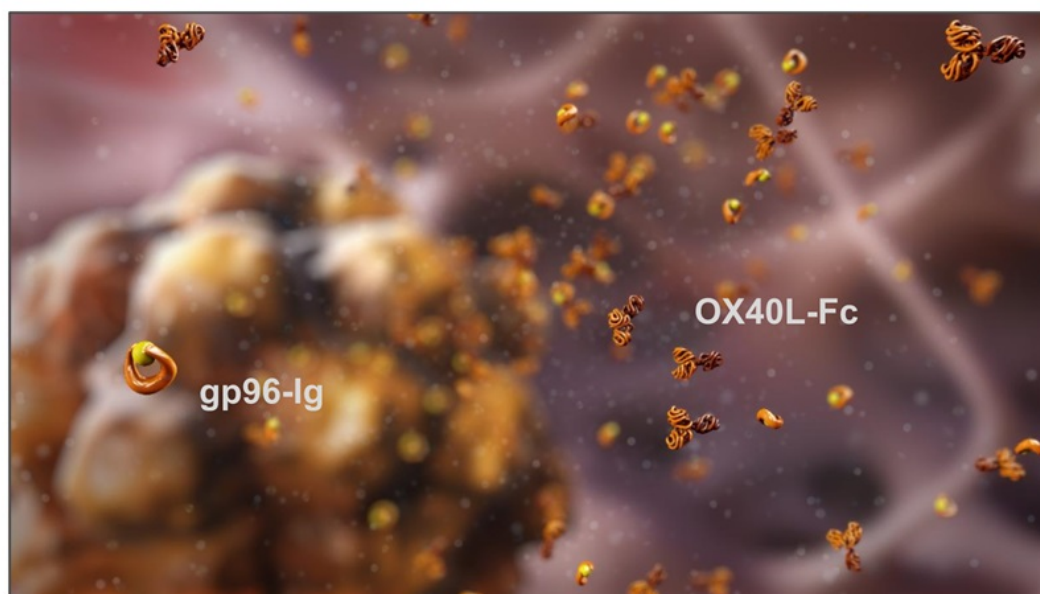
Week 1

Week 2



*Represents the only patient of ~200 patients dosed who discontinued treatment for a vaccine-related adverse event

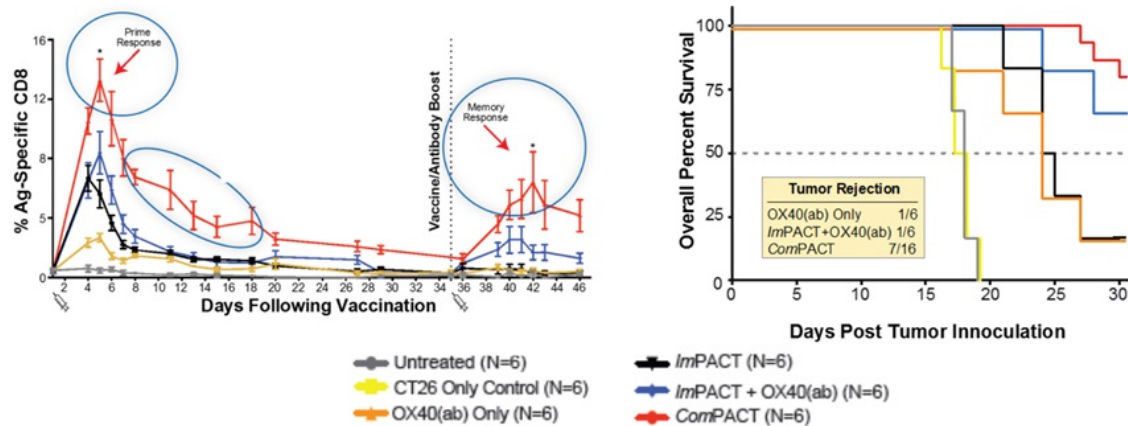
ComPACT Platform Technology



The first potential dual-acting immunotherapy designed to deliver T cell activation and costimulation in a single product – combination therapy without additive costs



ComPACT Outperforms OX40 Monoclonal Antibodies in Preclinical Models

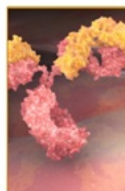


- ComPACT leads to ~50% complete tumor rejection as compared to ~16% with OX40 agonist antibody combinations

T Cell Co-Stimulation

T Cell Co-stimulation to Enhance Immune Response Against Cancer

Co-Stimulation



Pelican PTX-25
Monoclonal Antibody

Pelican PTX-15
Fusion Protein

CO-STIMULATORS

PTX-25	TBA	Preclinical	Humanized monoclonal antibody, functional agonist of human TNFRSF25
PTX-15	TBA	Preclinical	TL1A-Ig fusion protein, functional agonist of human TNFRSF25

Heat Biologics Acquires Pelican Therapeutics

- Heat has **acquired 80% controlling interest** in Pelican
- Pelican will operate as a subsidiary in Texas
- PTX-25 - potential best-in-class co-stimulator
- Pre-clinical synergy with Heat's *ImPACT* and checkpoint therapy
- \$15.2M CPRIT Grant to fund clinical development



Emerging Target in T Cell Co-Stimulation

Many companies are pursuing co-stimulators with less specificity for CD8+ "memory" activation

Target	Lead mAb	Clinical Stage	Companies	Comments
4-1BB/4-1BBL	Urelumab, PF-05082566	Phase 1/2	BMS Pfizer	Original phase II halted, now enrolling at lower doses
CD27	Varlilumab	Phase 2	BMS Celldex	mAb works by ADCC, no clinical evidence of agonism
OX40/OX40L	MEDI0562, MEDI6383	Phase 1	Genentech GSK Medimmune	Enrolling
GITR	TRX518	Phase 1	Merck	Enrolling
CD40/CD40L	CP-870893	Phase 1	Pfizer	Enrolling
HVEM/BTLA		Preclinical	BMS	IND Enabling
HVEM/LIGHT		Preclinical	BMS	IND Enabling
TNFRSF25/TL1A	PTX-25, PTX-15	Preclinical	HEAT/PELICAN	IND Enabling

None of these co-stimulators have advanced beyond phase 2 trials

Only Pelican is targeting TNFRSF25, an emerging target in immuno-oncology



Potential Best-in-Class Co-Stimulator

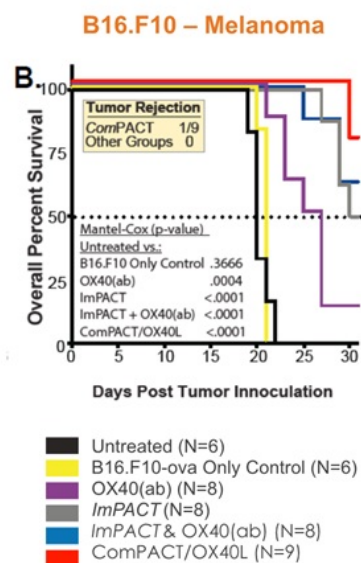
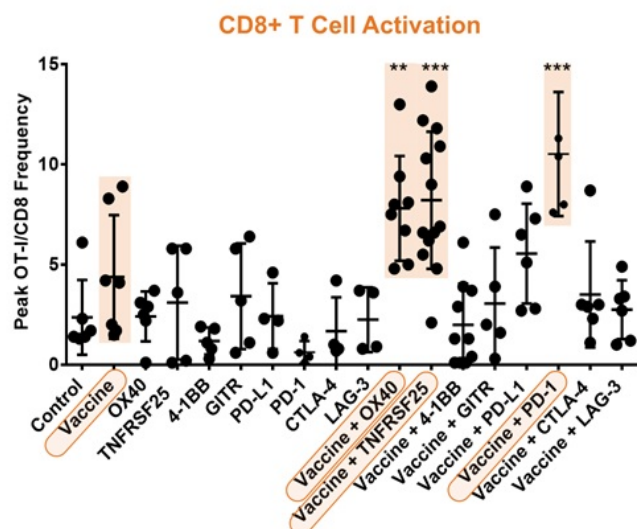
- PTX-25 is a potential best-in-class T cell co-stimulator specific to “killer” CD8+ “memory” T cells
- Pre-clinical studies show advantages over competing T cell co-stimulator programs based on CD8+ T cell specificity
- \$15.2 million New Company Product Development Award from the Cancer Prevention and Research Institute of Texas (CPRIT)
 - Propels PTX25 through a ~70-patient first-in-man clinical program
 - Advance multiple products through preclinical development, and at least one through Phase I trials

Pre-clinical studies show advantages over competing T cell co-stimulator programs based on CD8+ T cell specificity

- Antigen-specific T cell proliferation
- Increased effector cytokine production
- *Increased effector immune function*
- *Increased survival in preclinical models*

Pre-Clinical Data

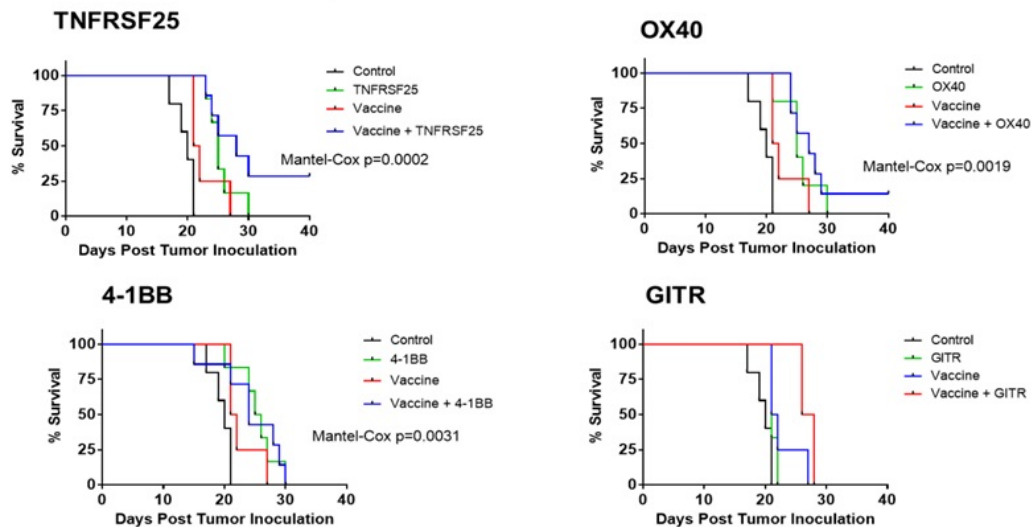
Strong supporting pre-clinical data combining co-stimulators with Heat's *ImPACT/ComPACT* therapies



Source: Fromm et al. Society for Immunotherapy of Cancer Annual Meeting, 2016

PTX-25 Comparative Pre-Clinical Anti-Tumor Activity

TNFRSF25 agonism with murine mAb shows increased survival compared to other co-stimulators



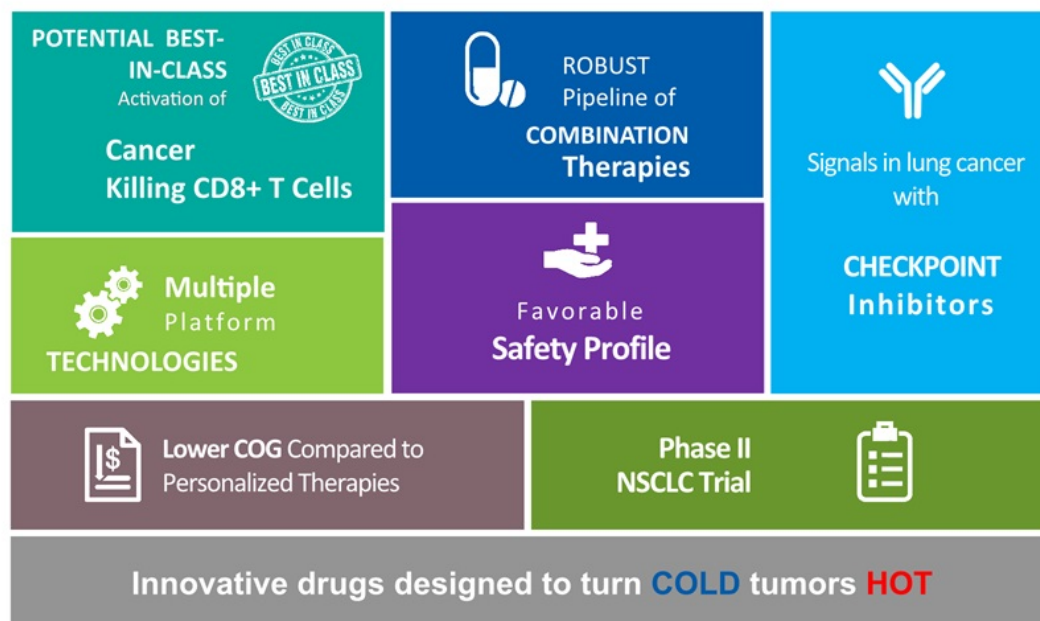
Comparative activity of OX40, GITR, 4-1BB and TNFRSF25 agonist mAbs in a long-established (9-day) B16-F10 melanoma model

Corporate Highlights

Nasdaq HTBX	Shares Outstanding 35.6M	Shares Price \$0.70¹
Market Cap \$25M¹	Cash & Equiv. \$11.1M²	Grant Funds \$15.2M

1. As of May 26, 2017;
2. As reported as of March 31, 2017

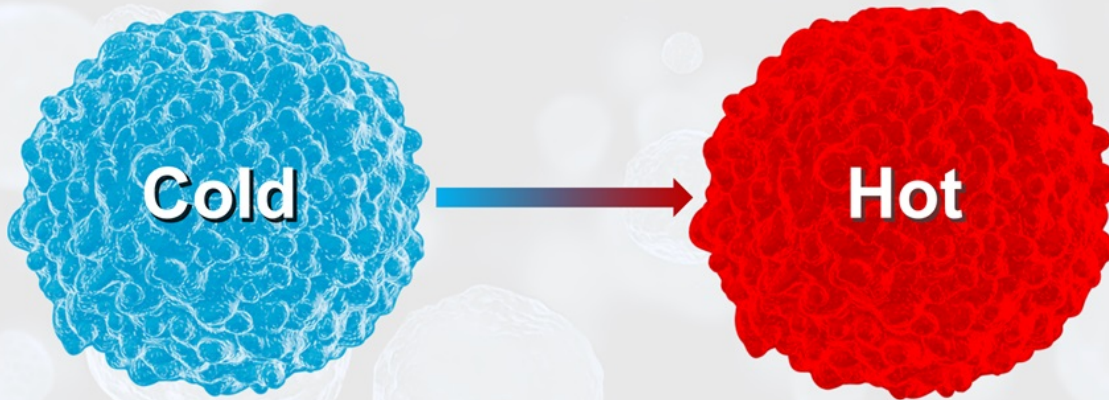
Heat Biologics Highlights





Heat Biologics

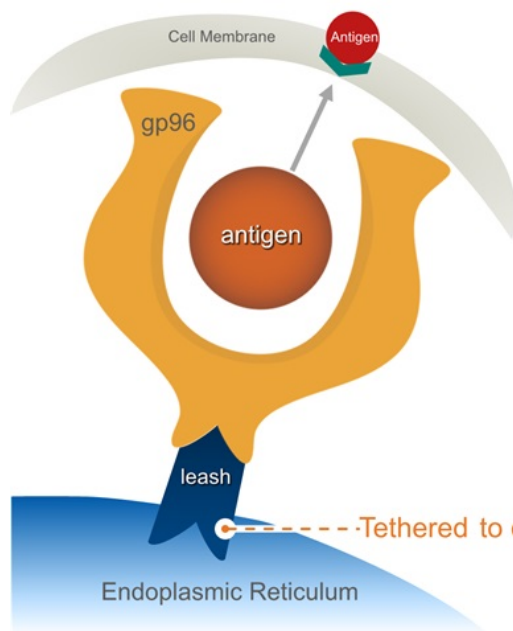
Turning **COLD** Tumors **HOT**



Appendix

Introducing gp96

The Immune System's "Swiss Army Knife"*

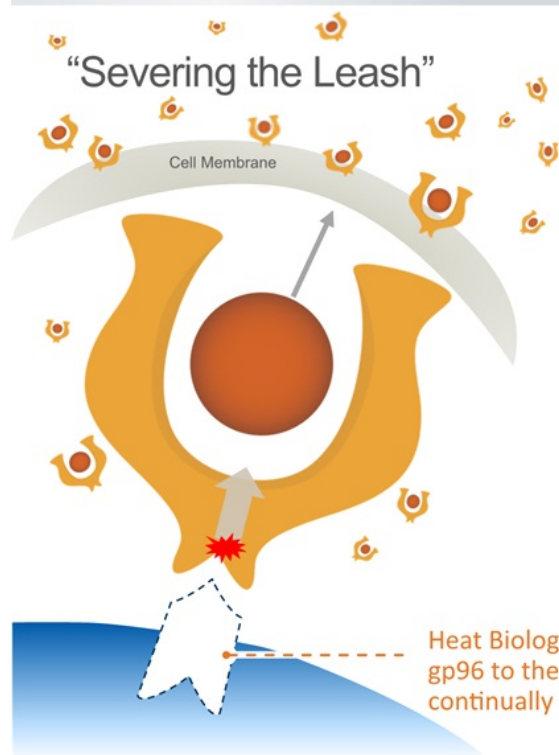


"Molecular Warning System"

- Natural biological process to deliver proteins (antigens) & gp96 adjuvant to our immune system
- Gp96 "chaperones" newly-created proteins to the cell membrane where they are released and embedded
- Activates a cytotoxic T-cell response to the antigen it is carrying when cells die through necrosis
 - Enables MHC I antigen cross-presentation to CD8+ T-cells
- Gp96 + protein are only naturally released via necrosis
 - Exposure of gp96 outside the cell activates an immune response to the antigen it is carrying
 - Enables MHC I antigen cross-presentation specifically to CD8+ T-cells
- Among the most powerful adjuvants and the only adjuvant to show exclusive specificity to CD8+ ("killer") T-cells
 - Provides long-term immunity against the infectious agent

*Schild, H. & Rammensee, H. *Gp-96 – The Immune System's Swiss Army Knife*.
Nature Immunology 2, 100-101 (2000)

ImPACT Therapy



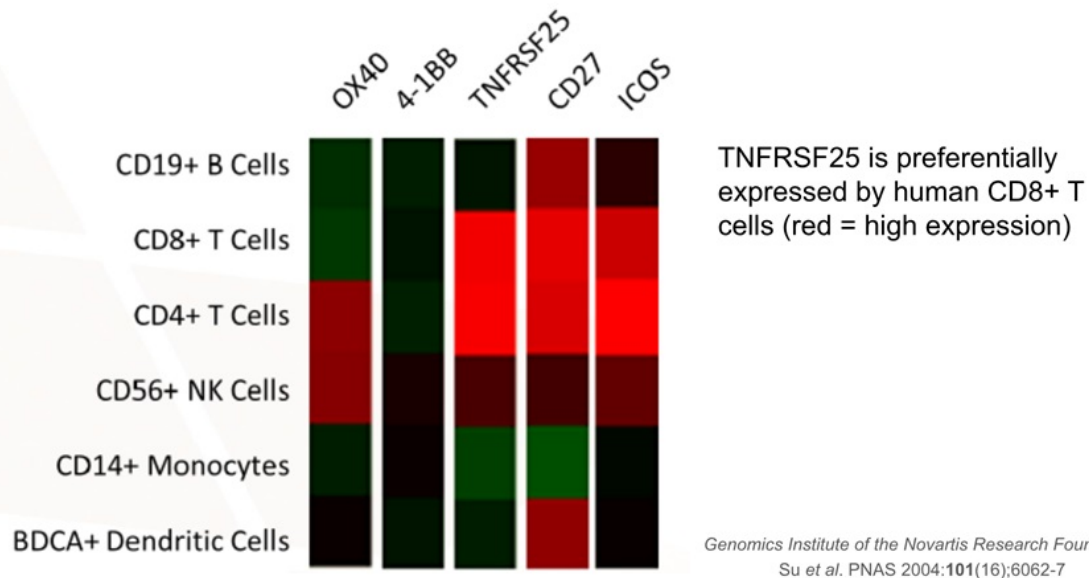
Heat Biologics *ImPACT* technology reprograms cancer cells to continuously secrete their own antigens bound to heat shock protein gp96 to seek out and destroy a variety of tumours

- Genetically modify tumor cells by “severing the leash” that binds the gp96 to the endoplasmic reticulum of the cell and replacing it with a sequence that pumps gp96 out of the cell
- Enables living cancer cells to “pump-out” their own surface antigens along with their gp96 chaperone
 - Mimics necrotic cell death
- Activates a powerful pan-antigen cytotoxic T-cell immune response

Heat Biologics *ImPACT* technology removes the leash that binds gp96 to the cell, replacing with a sequence that allows cells to continually secrete gp96 along with their “chaperoned” antigen

Expression of T cell Costimulators

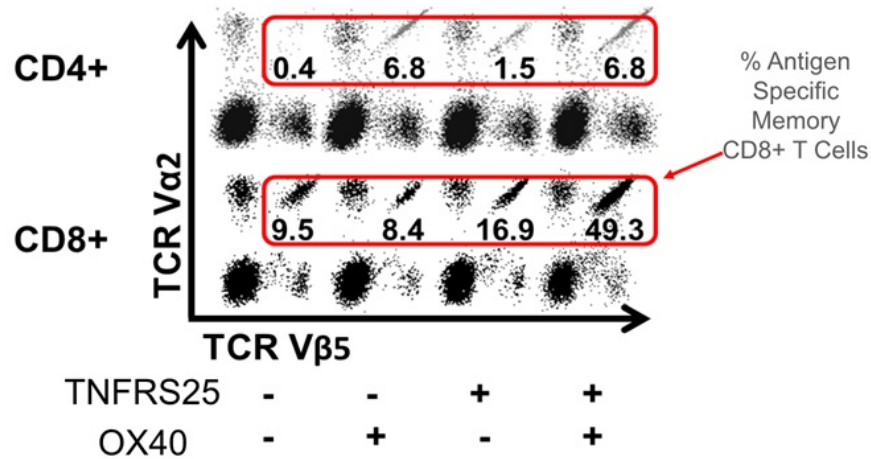
Compared to other T cell costimulators, TNFRSF25 is preferentially expressed on CD8+ T cells



Preferential CD8+ T cell Induction with TNFRSF25

Pre-clinical studies with murine agonist antibody shows preferential CD8+ T cell Induction; differentiation from other T cell costimulators

- The frequency of antigen-specific memory CD4+ or memory CD8+ T cells were examined following treatment of mice with a vaccine alone, or in combination with OX40 or TNFRSF25 antibodies



- TNFRSF25 preferentially 'boosts' CD8+ T cell immunity, whereas OX40 is preferential to CD4+ T cells

Highlights from Pelican Pre-clinical Studies

- mAb to TNFRSF25, drives the development of **antigen-specific CD8+ T cells** (this effect mimics TLIA, the natural monogamous ligand of TNFRSF25)
- mAb to TNFRSF25 results in costimulation and expansion of antigen-experienced memory T cells, both CD4+ and CD8+
 - notable is a significantly enhanced effect on **memory CD8+ T cells**
- Costimulation occurs only in the context of **TCR recognition of antigen**
- TNFRSF25 appears to have **superior activity** in stimulating memory CD8+ cells relative to OX40, 4-1BB and GITR
- Agonism with TNFRSF25 mAb leads to increases in **effector cytokine** and **effector immune function**, and **increases survival in mouse models**
- In mouse melanoma models, TNFRSF25 mAb results in increased survival **compared to agonism of OX40, GITR, 4-1BB** with respective agonist mAbs

