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): **April 20, 2018**

Heat Biologics, Inc.

(Exact name of registrant as specified in charter)

Delaware
(State or other jurisdiction of incorporation)

001-35994	**26-2844103**
(Commission File Number)	*(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 corporate 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 April 20, 2018

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: April 20, 2018

HEAT BIOLOGICS, INC.

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

EXHIBIT INDEX

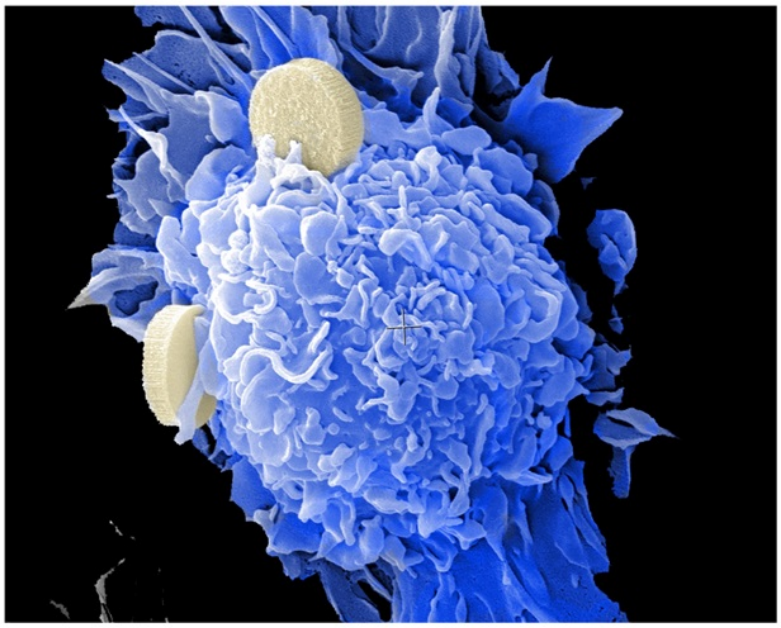
Exhibit No.	Description
99.1	Heat Biologics, Inc. investor presentation dated April 20, 2018

Heat Biologics

Heat Biologics

Corporate Presentation

April 20, 2018



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 complete clinical trials and 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, 2017 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.

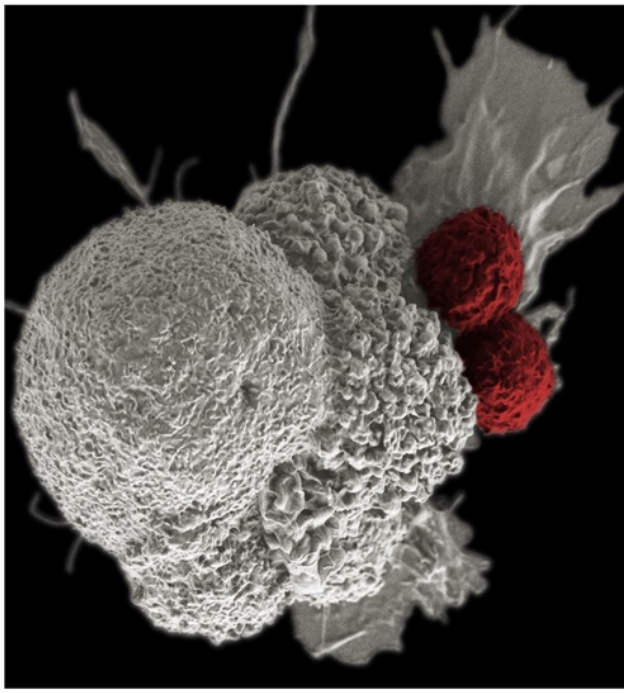
You should read carefully the factors described in the "Risk Factors" sections of our SEC Filings to better understand the risks and uncertainties inherent in our business.

Heat Biologics

Our Mission

*To activate CD8+ "Killer" T-cells to turn **"COLD"** tumors **"HOT"***

We seek to combine with checkpoint inhibitors and other immunotherapies to dramatically increase their effectiveness



Heat Biologics

- Tumors that **have not been** subject to robust CD8+ "Killer" T-cell attack
- Biopsied tumors contain minimal CD8+ T-cells

- Tumors that **have been** subject to robust CD8+ "Killer" T-cell attack
- Biopsied tumors are loaded with CD8+ T-cells

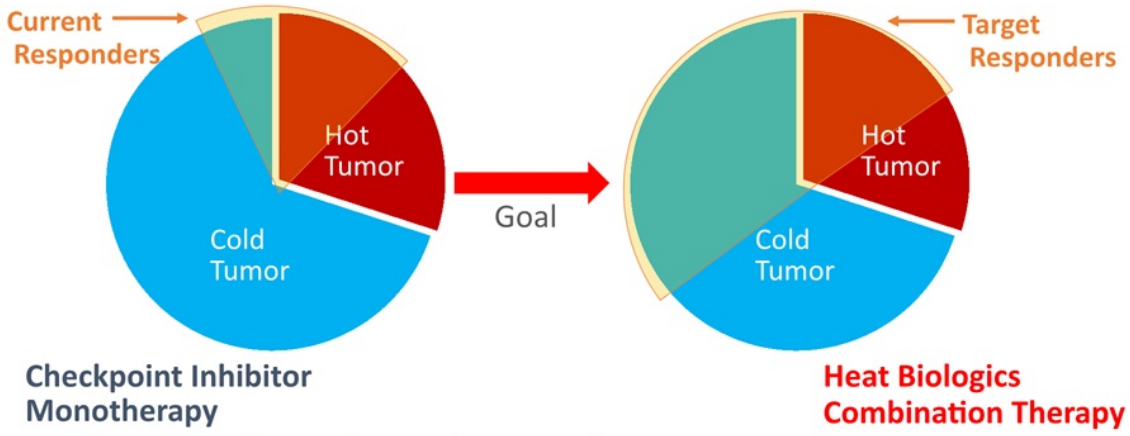
HOT tumors are associated with clinical response

Heat Biologics

Current NSCLC checkpoint inhibitor treatment

Unmet medical need in 2nd line NSCLC

17% - 20% response rate in a PD-L1 unselected population*



Checkpoint Inhibitor Monotherapy

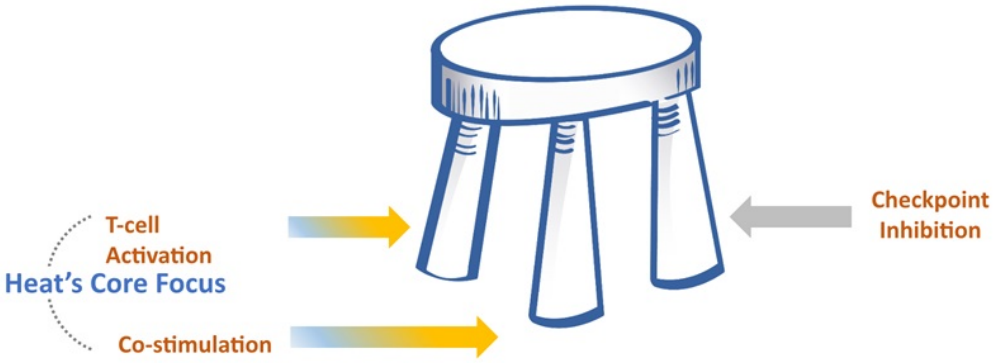
Heat Biologics Combination Therapy

Combination therapies can improve patient outcomes

**Borghaei H, et al. NEJM 2015;373:1627-39*
**Nishio M, et al. JCO 2015; 33:15_suppl, 8027-8027*

Immuno-Oncology Combination Therapy

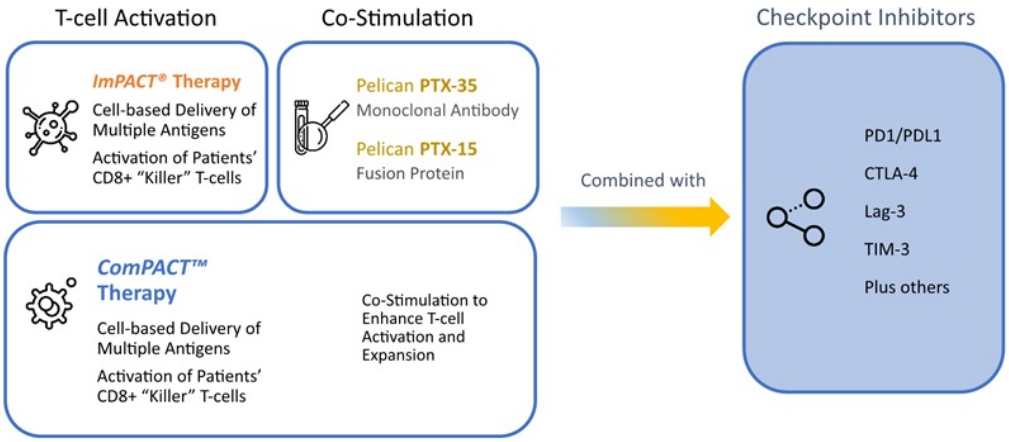
The three legs of an Immuno-Oncology Stool



Heat's goal is to dramatically increase the number of patients responding to checkpoint inhibitors

Combination Therapies

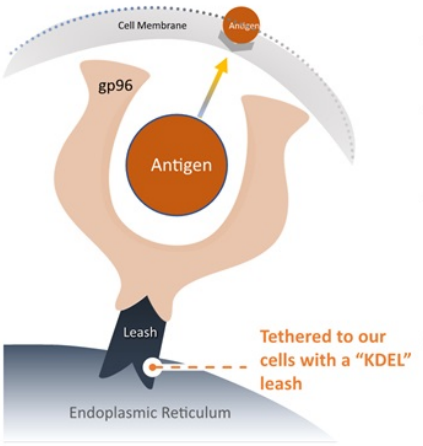
Unlocking the Body's Natural Defenses with a Broad Range of Combination Therapies



Heat Biologics

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

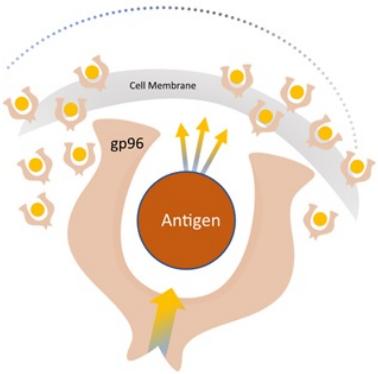
Schild, H. & Rammensee, H.* ***Gp-96 – The Immune System's Swiss Army Knife**. Nature Immunology 2, 100-101 (2000)*

Heat Biologics

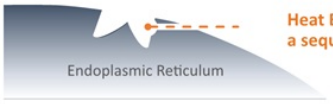
ImPACT Platform

"Severing the Leash"

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 tumors



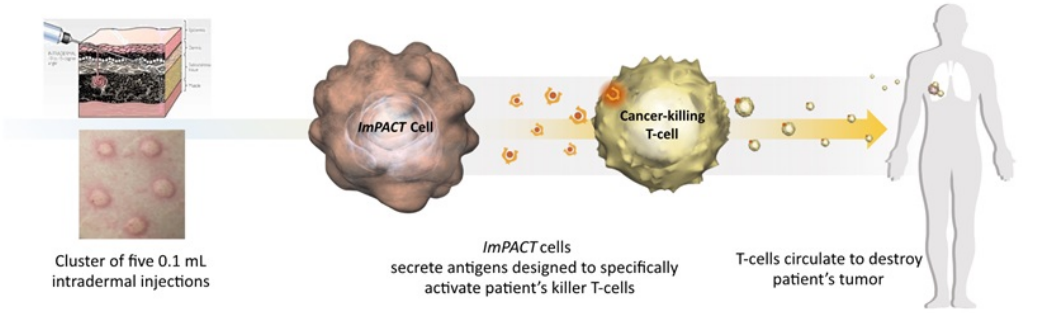
- Genetically modify tumor cells by "severing the leash" that binds the gp96 to the endoplasmic reticulum of the cell and replacing it with a sequence that pumps gp96 out of the cell
- Enables living cancer cells to "pump-out" their own surface antigens along with their gp96 chaperone
  - Mimics necrotic 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

Heat Biologics

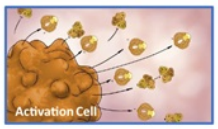
ImPACT®: Immune Pan-antigen Cytotoxic Therapy



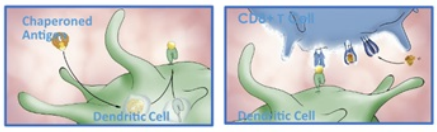
Cluster of five 0.1 mL intradermal injections

ImPACT cells secrete antigens designed to specifically activate patient's killer T-cells

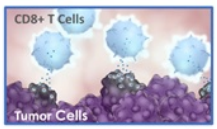
T-cells circulate to destroy patient's tumor



Activated cells **EXPRESS** chaperoned antigens



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

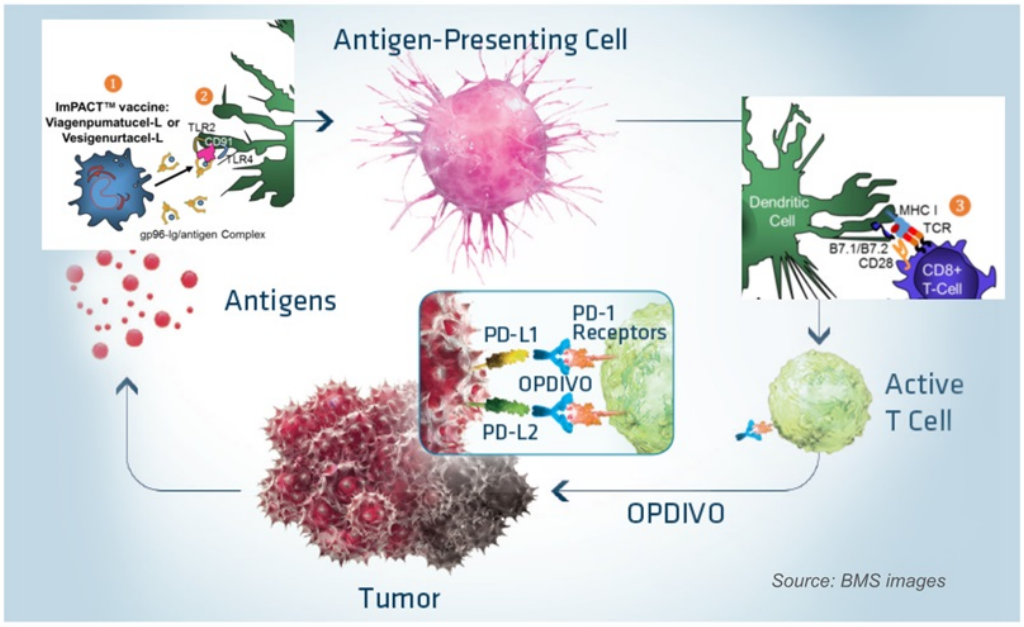


CD8+ T-cells locate and **ELIMINATE** cancer cells

Heat's unique cell-secreted gp96 drives activation of dendritic cells via TLR signaling and CD8+ T cells via antigen cross presentation

ImPACT + Opdivo Combination Therapy

Potential to improve clinical responses and survival, without additional toxicity



Product Pipeline

Combination Therapies Designed to Activate CD8+ T-cells to Fight Cancer

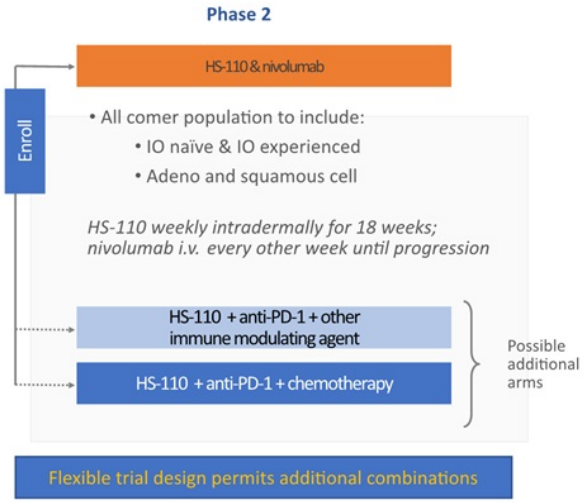
Combination Therapies	Indication	Pre-clinical	Phase 1	Phase 2	Phase 3	Approved	Comments
HS-110 (viagenpumatucel-L)	NSCLC						ImPACT® activation technology in combination with nivolumab and other checkpoint inhibitors TBA
HS-130	NSCLC						ComPACT™ activation technology in combination with checkpoint inhibitors TBA
Co-stimulators							
PTX-35	TBA						Humanized monoclonal antibody, functional agonist of human TNFRSF25 ($15.2M CPRIT grant)
PTX-15	TBA						TL1A-Ig fusion protein, functional agonist of human TNFRSF25

- Clinical proof of mechanism activating an immune response
- Activated T-cell immune response seen at 10 weeks
- T-cell infiltration seen deep inside tumors
- Positive safety profile, to-date

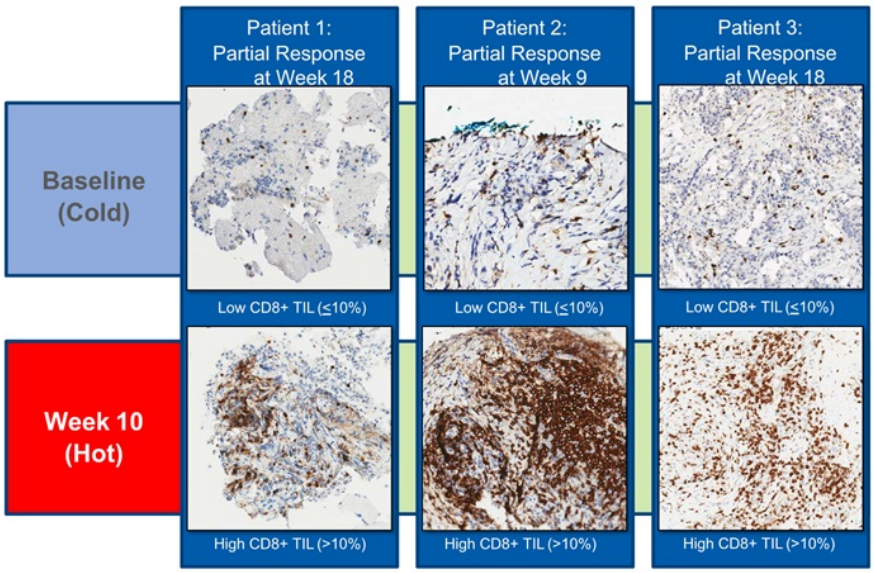
HS-110 (DURGA) Phase 2 Master Protocol: Design

A Phase 1b/2 study of Viagenpumatucel-L (HS-110) in Combination with Multiple Treatment Regimens in Patients with Non-Small Cell Lung Cancer (The "DURGA" Trial)

Objective	● Evaluate objective response rate of HS-110 and anti-PD-1 immune checkpoint inhibitor and other agents
Patient Population	● NSCLC ● Adenocarcinoma and squamous cell carcinoma ● 2nd line therapy or greater
Endpoints	● Safety and tolerability, immune response, objective response rate, duration of response, overall survival and progression -free survival
Enrollment	● Up to 25 U.S. sites ● Up to 120 patients TBD by iDMC ● Partnership with Yale Cancer Center on TIL analysis



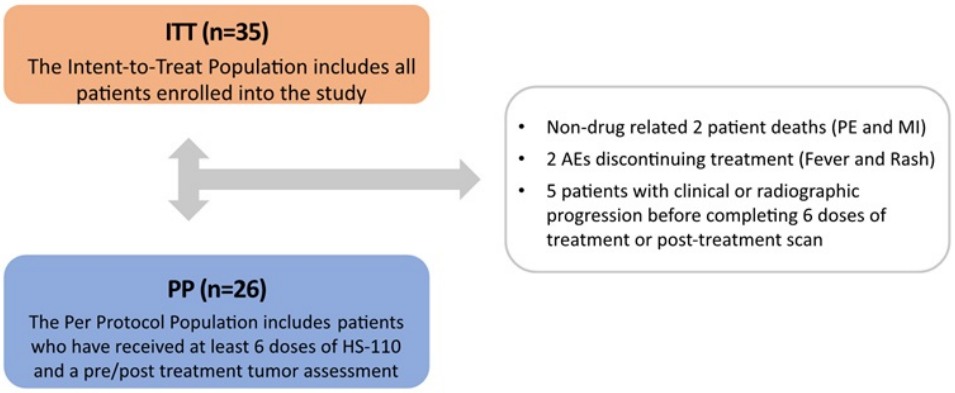
Clinical Evidence that HS-110 is Turning COLD Tumors HOT



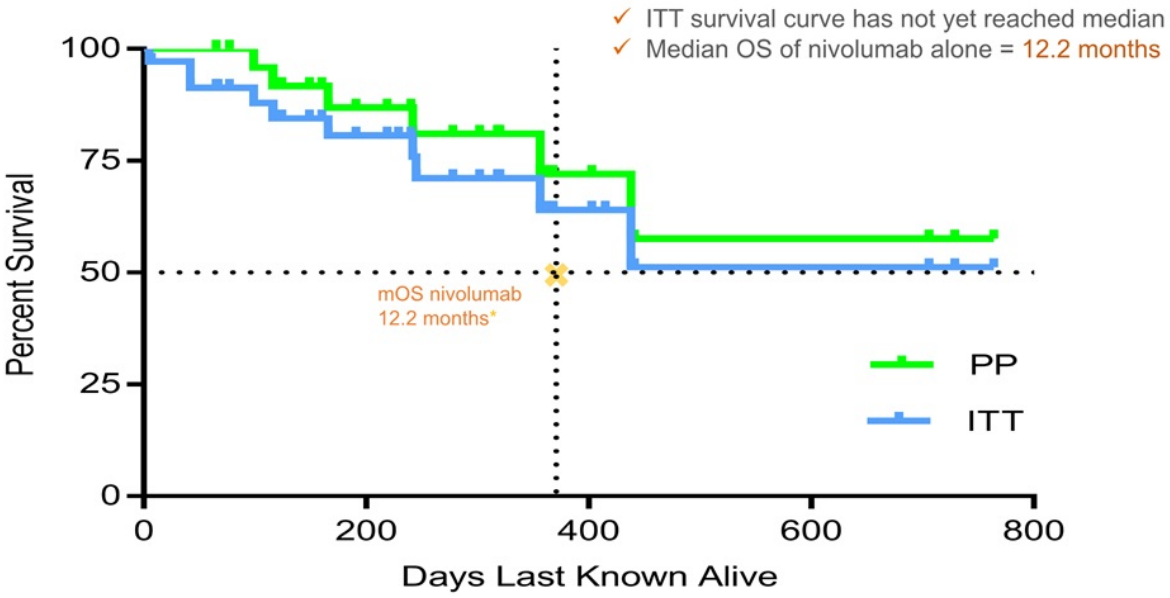
- "Killer" CD8+ T-cells driven deep into tumors
- "Cold" tumors with no previous activation made highly active ("HOT")
- Expression of PD-L1 increased with CD8+ T-cell infiltration in some tumors

TIL Infiltration Associated with Clinical Response

Pre-Specified Patient Populations Analyzed



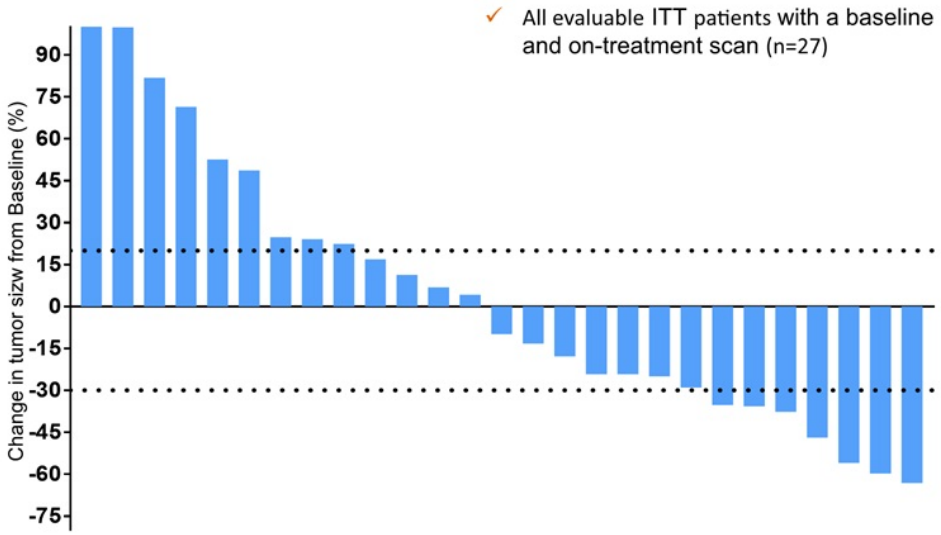
HS-110 Overall Survival



*N Engl J Med 2015; 373: 1627-1639

PP (n=26) is a subset of the ITT population (n=35)

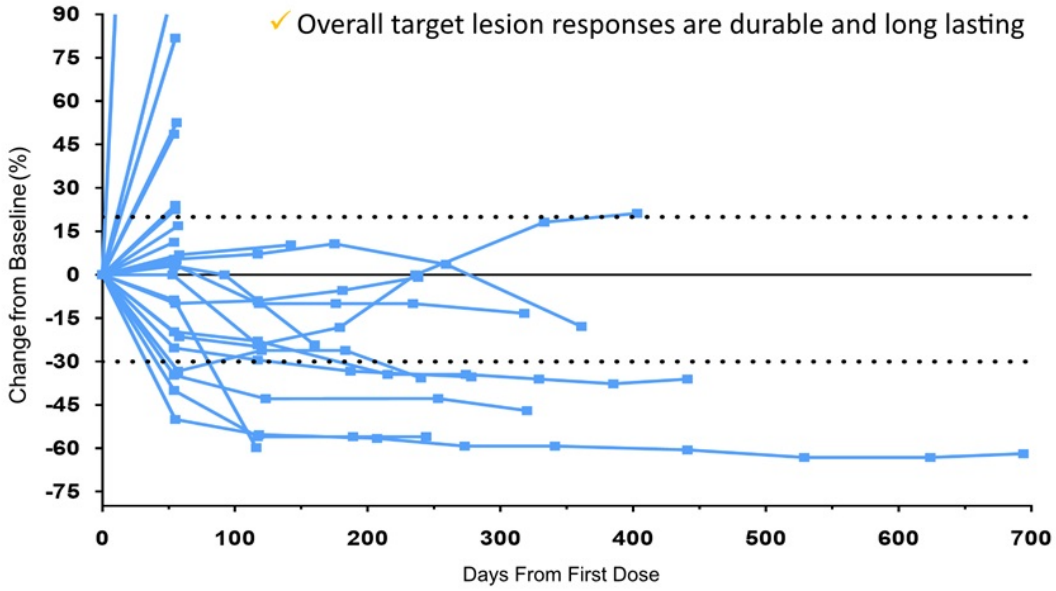
Best Target Lesion Response



All enrolled patients (ITT) with a baseline and on-treatment scan

Heat Biologics

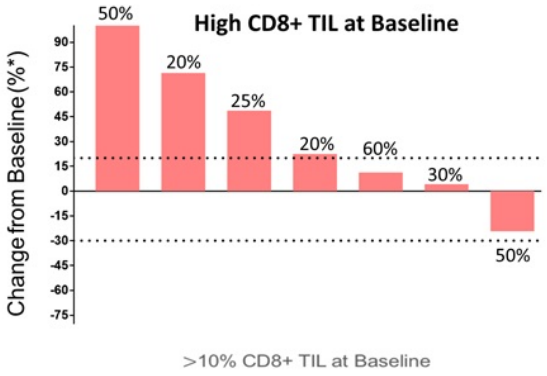
Durability of Target Lesion Response



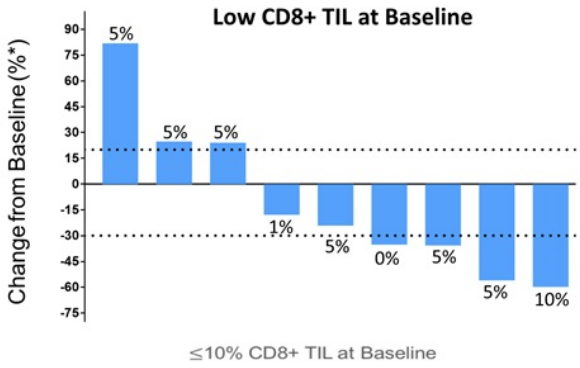
All enrolled patients (ITT) with a baseline and on-treatment scan

Target Lesion Response Based on Initial CD8+ TIL Status

HS-110 shows effect in “cold tumor” patients who typically do not respond well to PD-1 inhibitors



>10% CD8+ TIL at Baseline



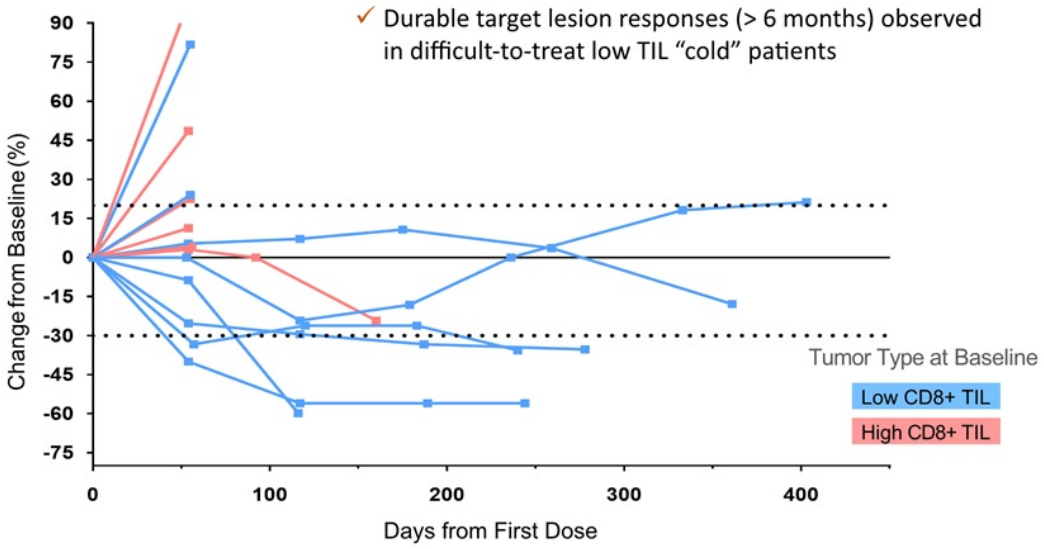
≤10% CD8+ TIL at Baseline

4 of 9 low CD8+ TIL achieved a partial clinical response

* % of CD8+ T-cells in tumor tissue at baseline

CD8+ TIL Evaluable ITT Population

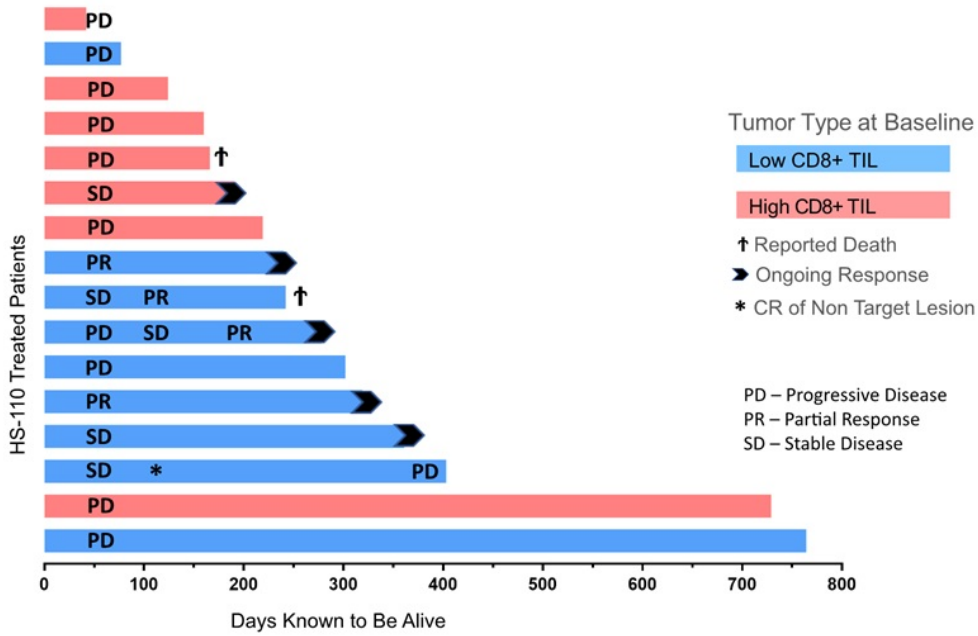
Durable Target Lesion Responses Based on Initial CD8+ TIL Status



CD8+ TIL Evaluable ITT Population

Heat Biologics

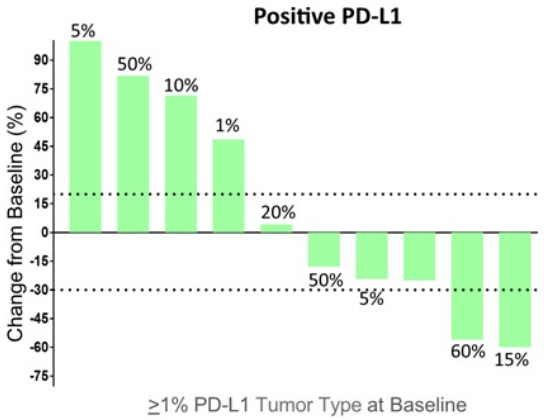
Encouraging Overall Survival Based on CD8+ TIL Status



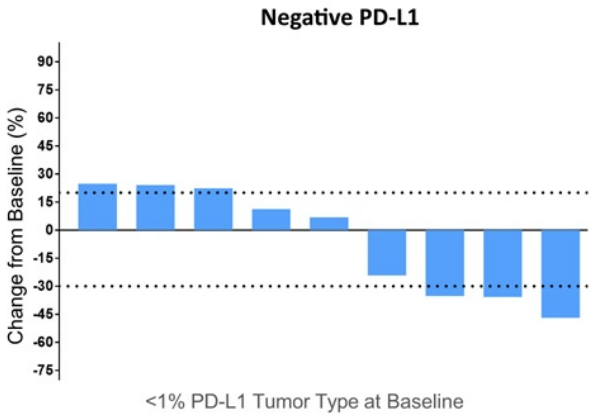
TIL Evaluable ITT Population

Target Lesion Response Based on Initial PD-L1 Status

HS-110 shows effect in low PD-L1 patients who typically do not respond to checkpoint inhibitors



≥1% PD-L1 Tumor Type at Baseline

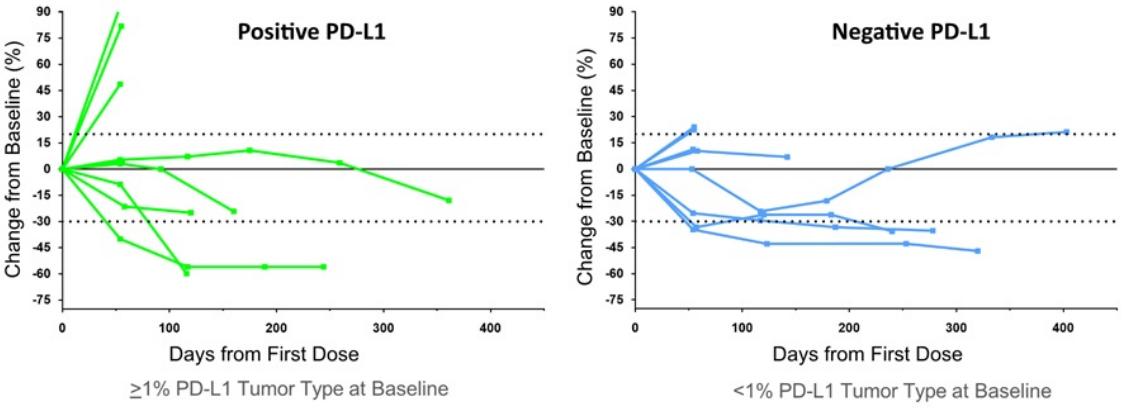


<1% PD-L1 Tumor Type at Baseline

PD-L1 Evaluable ITT Population

Durable Target Lesion Responses Based on Initial PD-L1 Status

Durable target lesion responses observed in difficult-to-treat low PD-L1 patients



≥1% PD-L1 Tumor Type at Baseline

<1% PD-L1 Tumor Type at Baseline

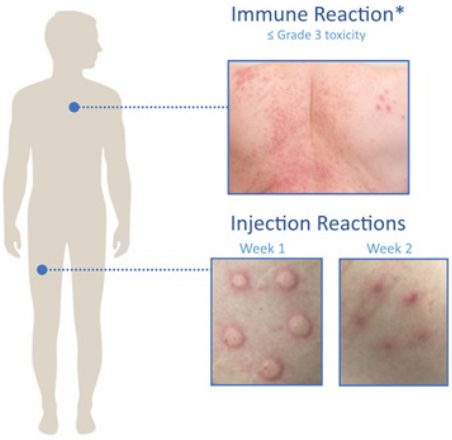
PD-L1 Evaluable ITT Population

HS-110 Safety Profile to Date

~1,000 *HS-110* Doses – No Serious Adverse Reactions

Favorable Safety Profile To Date

- ~1,000 doses administered to ~100 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 beyond those seen with standard of care
- No additive toxicities to standard of care



*Represents the only patient of ~100 patients dosed with HS-110 who discontinued treatment for a study related adverse event

Summary of Phase 2 Interim Data

- Tumor shrinkage and disease control demonstrated in a majority of evaluable patients
  - Overall responses are durable and long lasting
  - Median overall survival has not yet been reached
- HS-110 shows effect in difficult-to-treat low TIL and low PD-L1 patients who typically do not respond to checkpoint inhibitors
  - HS-110 shows durable responses in low TIL “cold tumor” patients
  - HS-110 shows durable responses in low PD-L1 patients
- A trend of survival benefit is observed with higher ELISPOT activity reflective of tumor antigen-specific immune response

Heat Biologics

Trial Learnings to Enhance Current Design

ImPACT provides a nurturing tumor antigen-specific T cell environment to support more responsive IO-driven therapy

- Enroll patients with low CD8+ TIL
- Enroll earlier in the treatment course or as front-line maintenance therapy
  - Clinical settings that provide maximal time to respond to HS-110
- Explore potential benefits to anti-PD-1 pre-conditioned treatment environment
- Modify current trial to identify and characterize patient population to carry forward to registrational trial

Trial modifications in progress

Heat Biologics Acquires Pelican Therapeutics

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

- Heat **acquired 80% controlling interest** in Pelican
- Pre-clinical synergy with Heat's *ImPACT®* and checkpoint therapy
- **$15.2M grant award** from the Cancer Prevention Research Institute of Texas (CPRIT) propels PTX-35 through a **~70-patient, first-in-human clinical program**
- PTX-35 is a potential **best-in-class, T-cell co-stimulator** specific to "killer" CD8+ "memory" T-cells

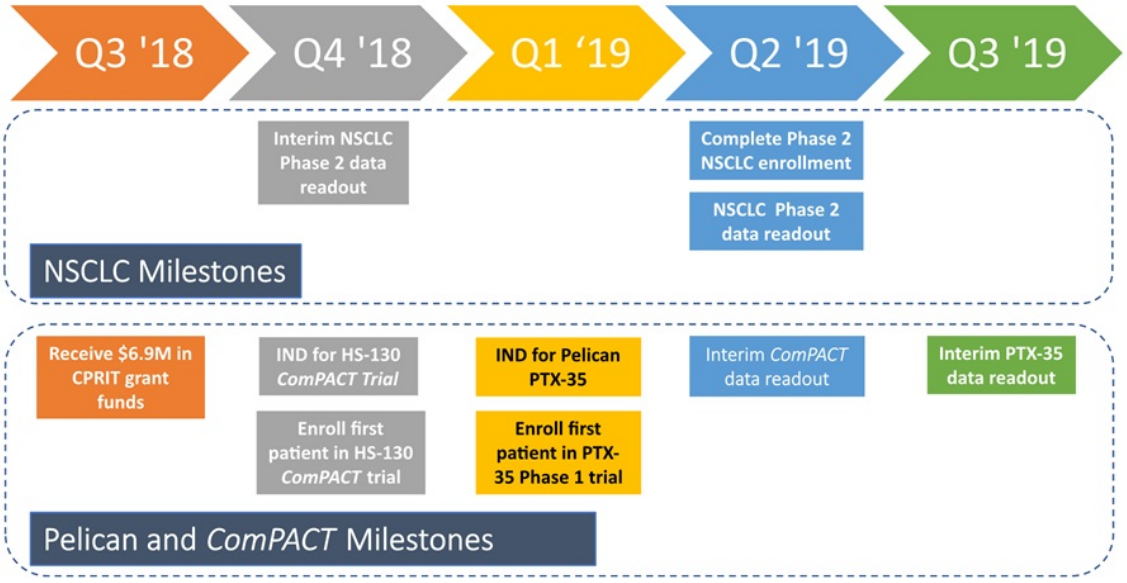
Pre-clinical studies highlight advantages over competing T-cell co-stimulator programs based on CD8+ T-cell specificity

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

Highlights from Pelican Pre-clinical Studies

- TNFRSF25 is preferentially expressed on CD8+ T-cells compared to other T-cell co-stimulators
  - Co-stimulation occurs only in the context of **TCR recognition of antigen**
  - Drives the development of **antigen-specific CD8+ T-cells** (mimics TL1A, the specific ligand of TNFRSF25)
- Superior activity is seen with TNFRSF25 in stimulating memory CD8+ cells relative to OX40, 4-1BB and GITR
- Agonism with TNFRSF25 mAb increases:
  - Effector cytokine function
  - Effector immune function
  - Survival in mouse models
- TNFRSF25 mAb results in increased survival compared to agonism of OX40, GITR, 4 -1BB with respective agonist mAbs in mouse melanoma models,

Corporate Milestones



Heat Biologics

Corporate Highlights

Nasdaq	Shares Outstanding	Share Price	Market Cap	Cash & Equiv.	Grant Funds
HTBX	5.66M	$1.22[1]	$7.0M[1]	$9.8M[2]	$15.2M

Capitalization table (as of 3.31.2018)	Shares	WAEP	% of Fully Diluted
Common shares outstanding	5,663,919		87.6%
Warrants	310,397	$14.60	4.8%
Outstanding stock options	426,393	$12.71	6.6%
Unvested restricted stock	63,287	$0.00	1.0%
Fully Diluted Shares Outstanding	**6,463,996**		100%

1. *Closing price as of April 18, 2018*
2. *Reported as of December 31, 2017*

Heat Biologics

Investment Highlights

Potential Best in Class Oncology Treatment - T-cell activating platform (TCAP) produces allogeneic, off-the-shelf therapies designed to activate the immune system to turn immunologically "cold" tumors "hot"

Combination Effect - Our therapies are designed to be administered with checkpoint inhibitors and other immuno-modulators to enhance immune response

Off-the-shelf Therapies - We can administer drug immediately without extracting patient material, simplifying treatment and substantially lowering cost of goods sold

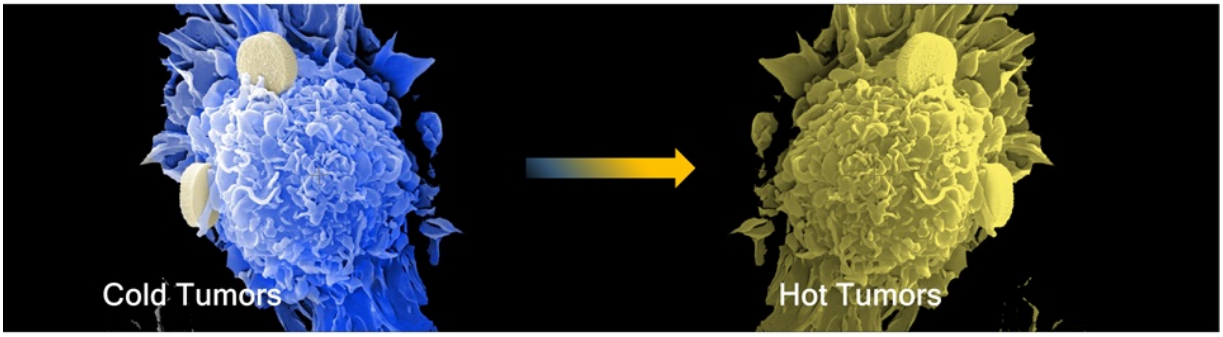
Clinical Data with Checkpoint Inhibitors - Positive interim data from ongoing Phase 2 trial of HS-110 + checkpoint inhibitor in non-small cell lung cancer (NSCLC)

Diverse Technology Platforms - Multiple complementary platform technologies

Strong Management Team - Senior team with broad experience in biotech, pharma, clinical development and research

Heat Biologics

Turning COLD Tumors HOT



Appendix

Heat Biologics

Management and Advisors

Management


Jeff Wolf
Founder & CEO


Jeff Hutchins, Ph.D.
CSO/COO


George Peoples, MD
Chief Medical Advisor


Ann Rosar
VP of Finance


Janice McCourt
VP of Business Devt.


Lori McDermott
VP of Clinical Dev.


Gary Vinson
VP of Manufacturing

Scientific Advisors

Robert Levy, Ph.D. University of Miami	Roger Cohen, MD University of Pennsylvania
Robert Negrin, MD Stanford University	Llew Keltner, MD, Ph.D. Epistat
Anthony Tolcher, MD Next Oncology	Gary Acton, MD Advisor

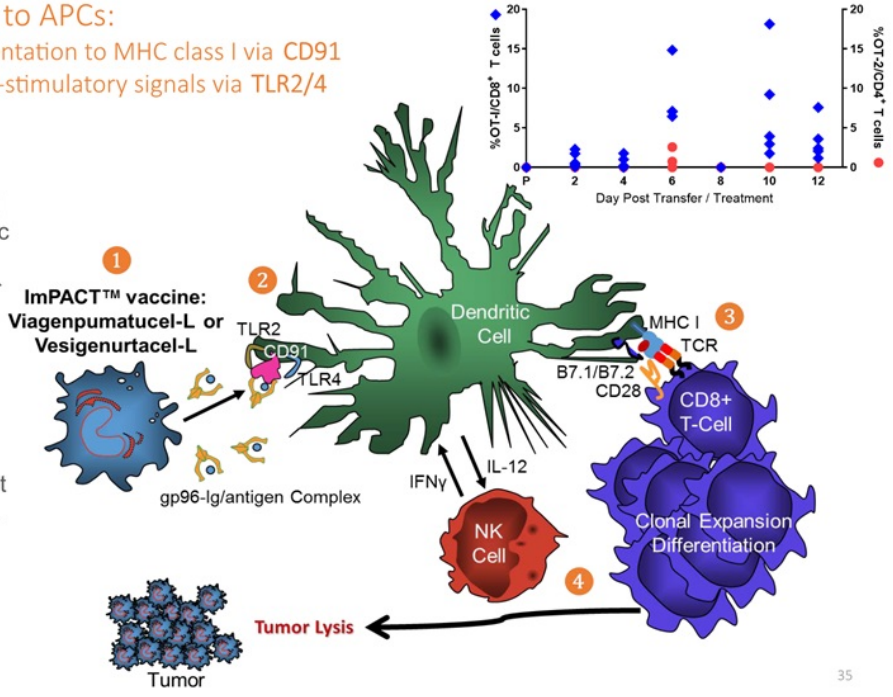
Heat Biologics

ImPact Generates an Adaptive Immune Response

2 signals Delivered to APCs:

- ✓ Antigen cross presentation to MHC class I via CD91
- ✓ Up regulation of co-stimulatory signals via TLR2/4

1. **Secretion of gp96-Ig** carrying tumor specific proteins represented on the patients tumor
2. **Activation of APCs** (TLR2/4) and cross-presentation of antigens (CD91)
3. **Specific T-cell receptor** engagement
4. **Clonal Expansion** of Tumor Antigen Specific T cells.



ImPACT® Manufacturing

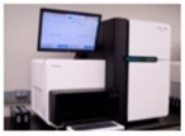
Robust, Multi-antigen T-cell Activation

Sample

Collect Cancer Cell Line & Patient Tissue Samples

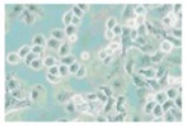
Screen

Compare Using Our Proprietary Technology



Select

ID Cell Line with Most Antigen Overlap



Transfect with Activation Technology

Scale

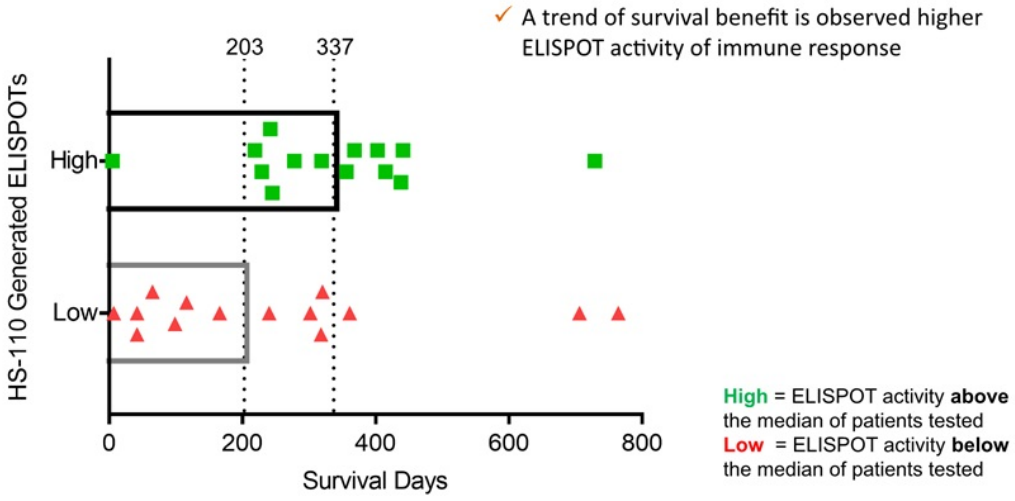
Scale-up for Manufacturing by CMO

- 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

Heat Biologics

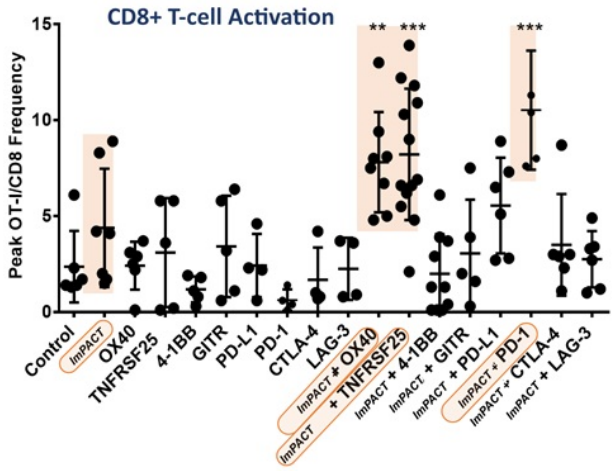
ELISPOT Activity and Survival



ELISPOT Evaluable ITT Population

Pre-clinical Data

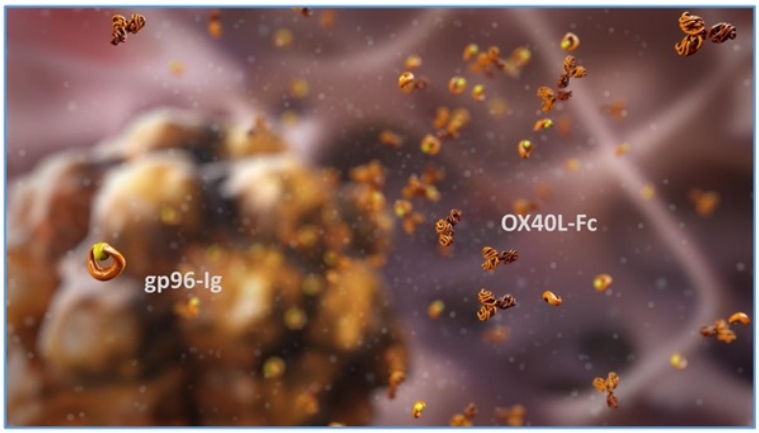
Strong supporting pre-clinical data



- Higher T-cell responses observed in mice treated with *ImPACT* alone
- *ImPACT*® boosted CD+ T-cells to even higher levels when combined with co-stimulator agonist antibodies: OX40, TNFRSF25, PD-1
- Findings suggest synergies when combining *ImPACT* with Pelican's TNFRSF25 antibody

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

ComPACT™ Platform Technology

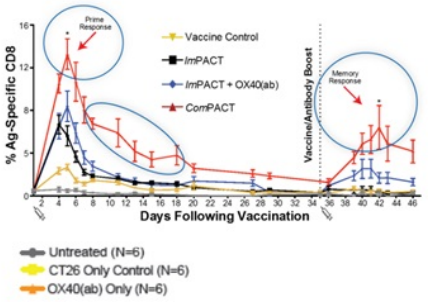


The first potential dual-acting immunotherapy designed to deliver T-cell activation and co-stimulation in a single product – combination therapy without additive costs

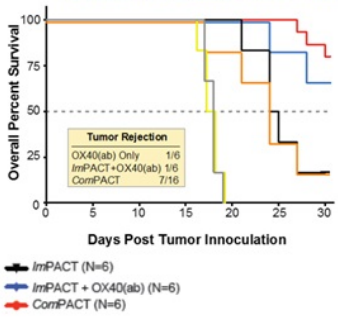
ComPACT™ Outperforms OX40

Monoclonal Antibodies in Pre-clinical Models

Superior primary and memory T-cell response in mouse model



Translates into increased overall survival and tumor reduction in a mouse tumor model



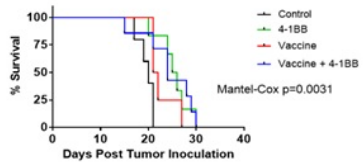
***ComPACT* generates ~50% complete tumor rejection compared to ~16% with OX40 agonist antibody combinations**

PTX-35 Comparative Pre-clinical Anti-tumor Activity

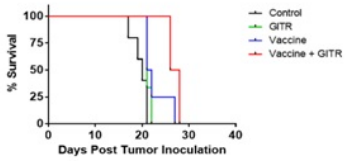
Activity of agonists TNFRSF25, 4-1BB, OX40 and GITR during nine-day B16-F10 melanoma model

4-1BB and GITR agonists have a moderate impact on survival

4-1BB

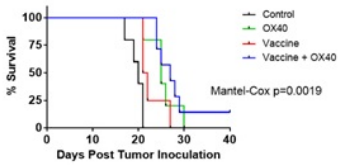


GITR

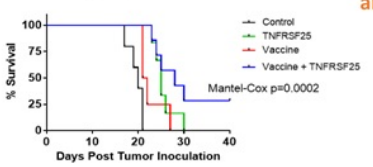


TNFRSF25 agonist leads to markedly increased survival compared to other co-stimulatory antibodies

OX40



TNFRSF25



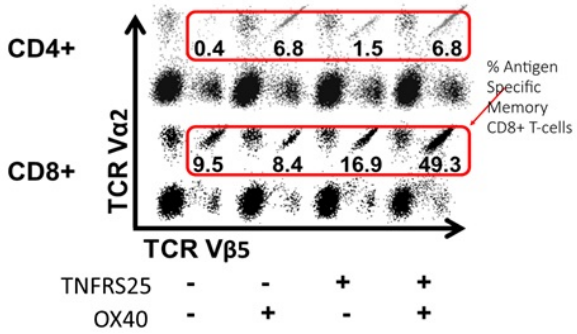
TNFRSF25 agonists leads to increased survival compared to other co-stimulatory antibodies

Schreiber T. et al. SITC 2014

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 co-stimulators

The frequency of antigen-specific memory CD4+ or memory CD8+ T-cells following treatment of mice with *ImPACT™* alone, or in combination with OX40 or TNFRSF25 antibodies



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

Schreiber et al. J Immunol 2012:189(7);3311-8

Emerging Target in T-cell Co-stimulation

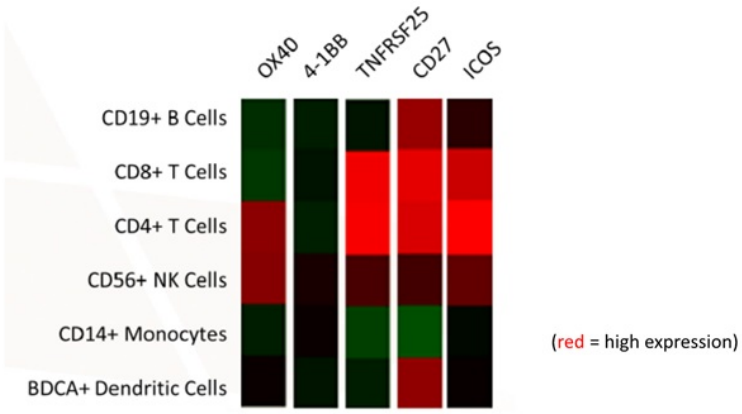
Many companies are pursuing co-stimulators with less specificity for CD8+ “memory” activation

Target	Agonist to Target	Clinical Stage	Company	Comments
4-1BB	PF-05082566 (utomilumab)	Phase 1/2 in solid tumors; Phase 3 with quadruple combination B-cell lymphoma	Pfizer	Phase 3 combination 4-1BB+epigenetic modulator+CD20 mAb and/or chemotherapy in refractory B-cell lymphoma
	BMS-663513 (urelumab)	Phase 1/2 in refractory metastatic melanoma	BMS	Combinations with PD-1 inhibitor
CD27	CDX-1127	Phase 1/2 combination with checkpoint	Celldex/BMS	Advanced refractory solid tumors; kidney cancer (sunitinab)
	Anti-CD27	Pre-clinical	Merck/Aduro	Shows synergy with checkpoint inhibitors
OX40	MEDI0562 (tavolimab)	Phase 1/2 in advanced solid tumors	MedImmune/AZ	Combination with PD-L1 (durvalumab); CTLA-4 (tremelimumab)
	GSK3174998	Phase 1/2 in advanced solid tumors	GSK	Combination with PD-1 (Keytruda®) in advanced tumors
	INCAGN01949	Phase 1/2 in advanced solid tumors	Incyte	Double/triple combinations: PD-1 and CTLA4; IgG1
	MOXR0916	Phase 1/2 in advanced solid tumors	Genentech	Combination with PD-L1 inhibition
	PF-04518600	Phase 1/2 in metastatic kidney cancer	Pfizer	Combination with Inlyta® (tyrosine kinase inhibitor); NCI and USC collaboration
	ABBV-368	Phase 1/2 in advanced solid tumors	AbbVie	Phase 2 planned in head/neck + nivolumab
GITR	GWN323	Phase 1	Novartis	Advanced tumors/lymphomas: planned combination studies, including PD-1
	INCAGN01876	Phase 1 advanced tumors	Incyte/Agenus	MoA involves Treg ADCC + co-stimulation
	MEDI1873	Phase 1 advanced tumors	MedImmune/AZ	Hexameric GITR ligand
	TRX-518	Phase 1 advanced tumors	Leap Therapeutics	Fc disabled. Modulates Tregs and T-effector cell ratio
ICOS	JTX-2011	Phase 1/2	Celgene/Jounce	Advanced solid tumors
TNFRSF25	PTX-35	IND filing Q4 2018	Heat-Pelican	Advanced solid tumors

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

Expression of T-cell Co-stimulators

TNFRSF25 is preferentially expressed on CD8+ T-cells compared to other T-cell co-stimulators



Genomics Institute of the Novartis Research Foundation Su *et al*. PNAS 2004:**101**(16);6062-7