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): **February 28, 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

On February 28, 2018, Heat Biologics, Inc. (the"Company") issued a press release announcing interim results from its Phase 2 study investigating HS-110 in combination with the Bristol-Myers Squibb's anti-PD-1 checkpoint inhibitor, nivolumab (Opdivo®), in patients with non-small cell lung cancer (NSCLC) whose cancers have progressed after treatment with one or more lines of therapy.

Among the 35 patients in the Intent-to-Treat ("ITT") population, 6 patients (17%) achieved a partial response and 14 patients (40%) achieved disease control. Evaluable ITT patients (those who underwent at least one follow-up scan regardless of treatment duration) demonstrated overall response and disease control rates of 26% and 67%, respectively. Overall responses appeared durable and long lasting. The survival data are still maturing, and median overall survival has not yet been reached. The combination of HS-110 and nivolumab was well tolerated, with no additional toxicities compared to what has been observed with single agent checkpoint inhibitors.

As predefined in the clinical protocol, patient subgroups were evaluated for levels of tumor infiltrating lymphocytes ("TIL") and for PD-L1 checkpoint protein expression on tumor cells. Four of 9 "cold tumor" patients with low TIL levels (<10%) at baseline had partial responses. HS-110 also showed a durable effect in patients with low levels of PD-L1, with 3 of 9 patients responding. Both of these patient populations respond poorly to checkpoint inhibitors.

A copy of the press release is attached as Exhibit 99.1 to this Current Report on Form 8-K and is incorporated herein by reference.

The Company is also hosting an analyst and investor event on February 28, 2018 to present the interim Phase 2 results. In connection with this analyst and investor event, the Company intends to discuss the slide presentation attached as Exhibit 99.2 to this Current Report on Form 8-K, which is incorporated herein by reference.

The press release attached as Exhibit 99.1 and the slide presentation attached as Exhibit 99.2 to this Current Report on Form 8-K include "safe harbor" language pursuant to the Private Securities Litigation Reform Act of 1995, as amended, indicating that certain statements contained in the press release or the slide presentation are "forward-looking" rather than historical.

The Company undertakes no duty or obligation to update or revise information included in this Current Report on Form 8-K or the related Exhibits 99.1 or 99.2.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Press Release dated February 28, 2018
99.2	Heat Biologics, Inc. presentation dated February 28, 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: February 28, 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	Press Release dated February 28, 2018
99.2	Heat Biologics, Inc. presentation dated February 28, 2018

EXHIBIT 99.1

Heat Biologics Announces Positive Interim Data from its Phase 2 Clinical Trial of HS-110 and Nivolumab in Non-Small Cell Lung Cancer (NSCLC)

- Tumor shrinkage and disease control demonstrated in a majority of evaluable patients
- HS-110 + nivolumab combination showed durable responses in difficult-to-treat low TIL patients and low PD-L1 patients, who respond poorly to checkpoint inhibitors
- Data consistent with HS-110 mechanism of action
- Live Webcast at 8am Eastern Time today

DURHAM, NC – February 28, 2018 – Heat Biologics, Inc. (Nasdaq: HTBX), a biopharmaceutical company developing drugs designed to activate a patient's immune system against cancer, today announced interim results from its Phase 2 study investigating HS-110 in combination with Bristol-Myers Squibb's anti-PD-1 checkpoint inhibitor, nivolumab (Opdivo®), in patients with advanced non-small cell lung cancer (NSCLC) whose cancers have progressed after treatment with one or more lines of therapy.

Among the 35 patients in the Intent-to-Treat ("ITT") population, 6 patients (17%) achieved a partial response and 14 patients (40%) achieved disease control. Evaluable ITT patients (those who underwent at least one follow-up scan regardless of treatment duration) demonstrated overall response and disease control rates of 26% and 67%, respectively. Overall responses appeared durable and long lasting. The survival data are still maturing, and median overall survival has not yet been reached. The combination of HS-110 and nivolumab was well tolerated, with no additional toxicities compared to what has been observed with single agent checkpoint inhibitors.

As predefined in the clinical protocol, patient subgroups were evaluated for levels of tumor infiltrating lymphocytes ("TIL") and for PD-L1 checkpoint protein expression on tumor cells. Four of 9 "cold tumor" patients with low TIL levels (<10%) at baseline had partial responses. HS-110 also showed a durable effect in patients with low levels of PD-L1, with 3 of 9 patients responding. Both of these patient populations respond poorly to checkpoint inhibitors.

George Peoples, M.D., Chief Medical Officer, stated, "The results from this combination trial with HS-110 and nivolumab are very promising, demonstrating durable responses in those patients with low levels of TIL and PD-L1. These patients represent the most difficult-to-treat patient groups and comprise the majority of the NSCLC population."

“We look forward to continuing patient enrollment to better define the optimal NSCLC population and inform the design of a pivotal trial,” commented Jeff Wolf, Chairman and CEO. “These data are consistent with the mechanism of action of our T-cell Activation Platform that promotes a robust T-cell immune response, an important component of an effective immunotherapy combination against cancer.”

DURGA Trial Design

The ongoing DURGA trial is a single arm multicenter trial that was designed to evaluate the combination of HS-110 and nivolumab in patients with NSCLC. Patients with advanced and previously treated NSCLC were treated with weekly HS-110 for 18 weeks and nivolumab 3 mg/kg every 2 weeks until disease progression or death. The primary endpoints are 1) safety and tolerability, and 2) objective response rate as defined by RECIST 1.1 criteria. Secondary endpoints include disease control rate, duration of response, peripheral blood immune response, progression-free survival and overall survival.

Live Webcast with Slides

The interim data from the DURGA trial will be presented at an analyst event, taking place at 8am Eastern Time, today, Wednesday February 28th. To register and watch a live webcast of the event, visit http://lifesci.rampard.com/20180228.

About Heat Biologics, Inc.

Heat Biologics is a biopharmaceutical company developing immunotherapies designed to activate a patient’s immune system against cancer by inducing CD8+ “Killer” T-cells. Our T-cell Activation Platform (TCAP) produces therapies designed to turn “cold” tumors “hot,” and be administered in combination with checkpoint inhibitor therapies and other immuno-modulators to increase their effectiveness. We are currently enrolling patients in our Phase 2 clinical trial for non-small cell lung cancer, in combination with Bristol-Myers Squibb’s nivolumab (Opdivo®). Pelican Therapeutics, a subsidiary of Heat, is focused on the development of co-stimulatory monoclonal antibody and fusion protein-based therapies designed to activate the immune system. We also have numerous pre-clinical programs at various stages of development. For more information, please visit www.heatbio.com.

Forward Looking Statements

This press release includes forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995 on our current expectations and projections about future events. In some cases, forward-looking statements can be identified by terminology such as “may,” “should,” “potential,” “continue,” “expects,” “anticipates,” “intends,” “plans,” “believes,” “estimates,” and similar expressions. These statements are based upon current beliefs, expectations and assumptions and include statements regarding our continued enrollment of patients in this trial, the future pivotal trial and the potential benefits of our products. These statements are based on management’s expectations and assumptions as of the date of this press release and are subject to a number of risks and uncertainties, many of which are difficult to predict that could cause actual results to differ materially from current expectations and assumptions from those set forth or implied by any forward-looking statements, including the ability of Heat’s *ImPACT* therapy to perform as designed, to demonstrate safety and efficacy, as well as results that are consistent with results from the interim data and prior results, the ability to enroll patients and complete the clinical trials on time and achieve desired results and benefits, Heat’s ability to obtain regulatory approvals for commercialization of product candidates or to comply with ongoing regulatory requirements, regulatory limitations relating to Heat’s ability to promote or commercialize its product candidates for specific indications, acceptance of its product candidates in the marketplace and the successful development, marketing or sale of products, Heat’s ability to maintain its license agreements, the continued maintenance and growth of its patent estate, its ability to establish and maintain collaborations, its ability to obtain or maintain the capital or grants necessary to fund its research and development activities, and its ability to retain its key scientists or management personnel, its ability to successfully integrate Pelican, and the other factors described in Heat’s most recent annual report on Form 10-K and other filings with the SEC. The information in this release is provided only as of the date of this release and the company undertakes no obligation to update any forward-looking statements contained in this release based on new information, future events, or otherwise, except as required by law.

Contact

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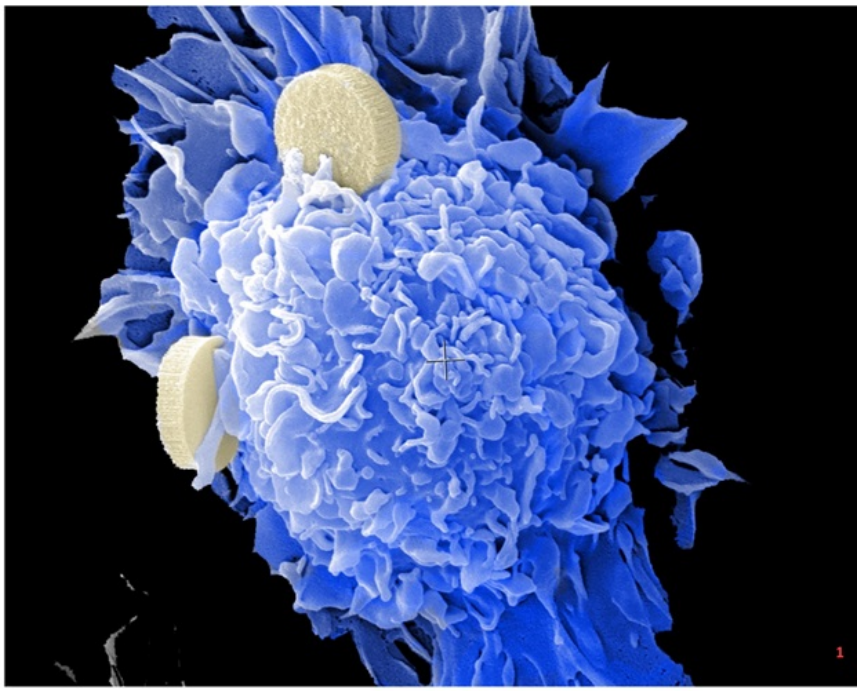
mconger@heatbio.com

Heat Biologics

Heat Biologics

Analyst and Investor Day
February 28, 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, future trial results being consistent with interim results, 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 most recent Annual Report on Form 10-K 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.

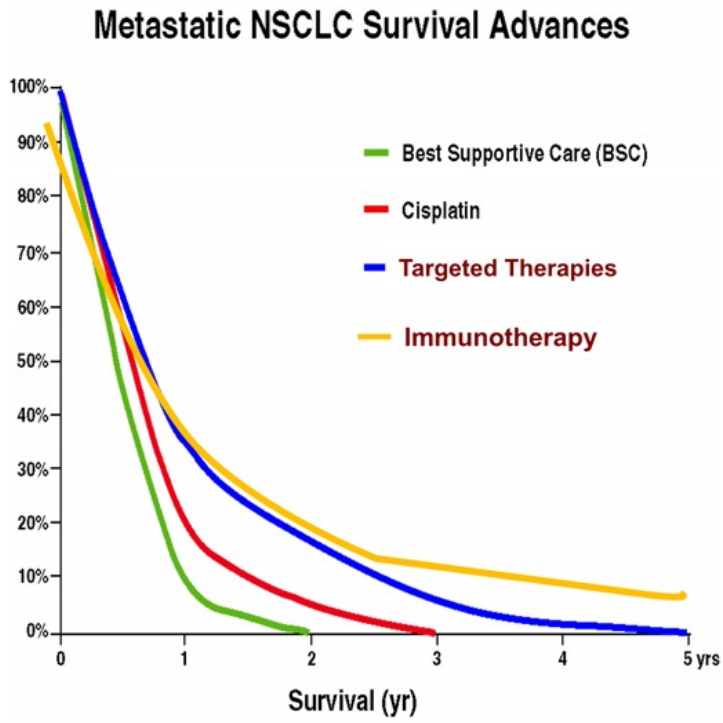
Penn Medicine

Roger B. Cohen

Professor of Medicine
University of Pennsylvania
Associate Director Clinical Research Abramson Cancer Center
Chief Clinical Research Officer Abramson Cancer Center
Co-Director, Head and Neck Cancer Research Center

the cure is within
ABRAMSON CANCER CENTER

Therapy for Advanced Lung Cancer

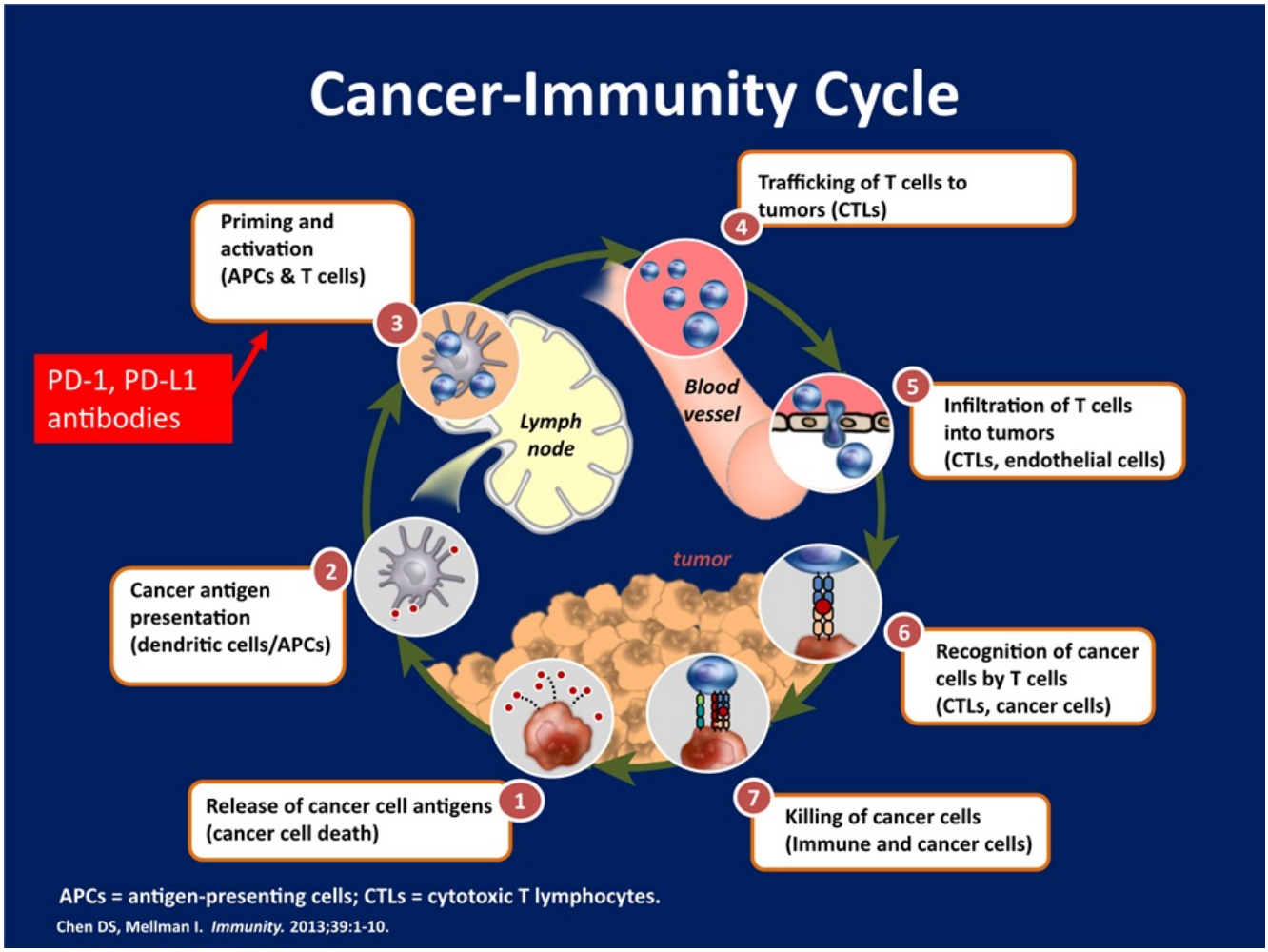


There have been some modest gains since 1980

Are we doing better with immunotherapy?

Penn Medicine

the cure is within
ABRAMSON CANCER CENTER


Cancer-Immunity Cycle
Priming and activation (APCs & T cells)
PD-1, PD-L1 antibodies
Trafficking of T cells to tumors (CTLs)
Blood vessel
Lymph node
Infiltration of T cells into tumors (CTLs, endothelial cells)
tumor
Cancer antigen presentation (dendritic cells/APCs)
Recognition of cancer cells by T cells (CTLs, cancer cells)
Release of cancer cell antigens (cancer cell death)
Killing of cancer cells (Immune and cancer cells)
1
2
3
4
5
6
7
APCs = antigen-presenting cells; CTLs = cytotoxic T lymphocytes.
Chen DS, Mellman I. Immunity. 2013;39:1-10.

Immune Checkpoints

Immune checkpoints are normal ‘brakes’ on the activity of the immune system; checkpoint proteins turn off activated T cells when they are no longer needed

Immune checkpoints have been ‘hijacked’ by the cancer to evade the immune system

Checkpoint Inhibitors remove the checkpoint and “take the brakes off the immune system”

Now the immune system can ‘see’ the tumor and kill it

1st Line NSCLC Treatment Landscape

Incurable NSCLC (adenocarcinoma) without activating EGFR, ALK, etc. mutations:

- If PD-L1 is ≥ 50%, consider Keytruda as monotherapy
- If PD-L1 <50%, consider Keytruda in combination with chemotherapy
- Platinum doublet chemotherapy

Incurable NSCLC (squamous cell):

- If PD-L1 is ≥ 50%, consider Keytruda as monotherapy
- If PD-L1 <50%, platinum doublet chemotherapy

Soon, many patients will get a checkpoint inhibitor in the first line of therapy

2nd Line NSCLC Treatment Landscape

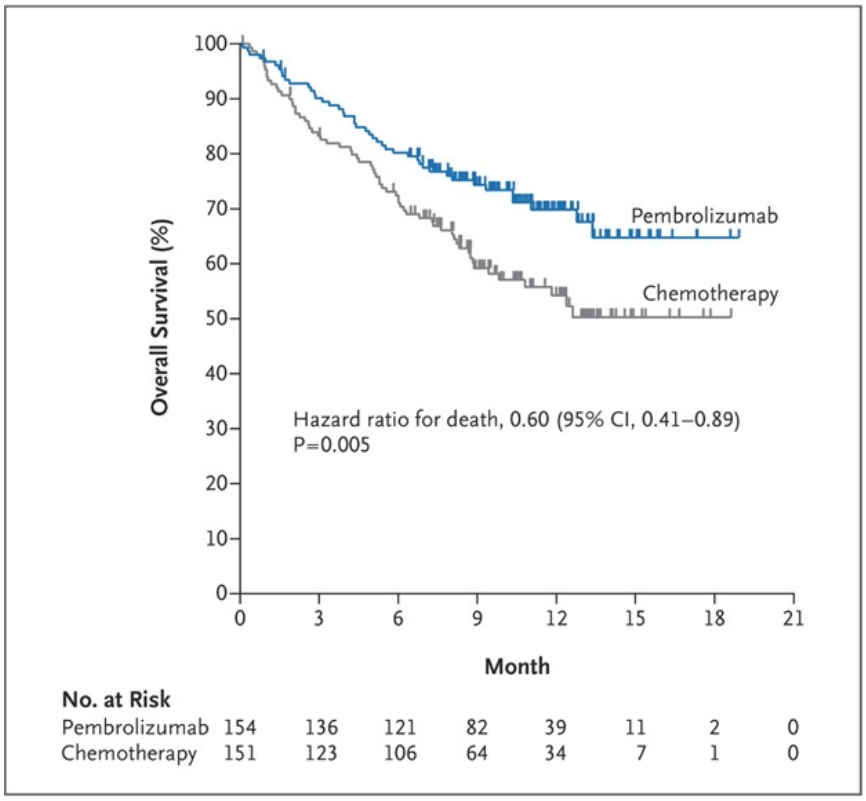
- Adenocarcinoma: chemotherapy (taxanes) or checkpoint inhibitor if not given previously
- Squamous Cell: chemotherapy (taxanes) or checkpoint inhibitor (Keytruda, Opdivo, Tecentriq) if not given previously

What if the patient already received a checkpoint inhibitor and wants more immunotherapy?

- They will need a 'rescue' strategy: a checkpoint inhibitor "plus *something* to make the checkpoint inhibitor work or work again"
- *Something* =
  - Radiation ("RadVax")
  - Addition of a 2nd immune modulating drug: IDO inhibitor, IFNγ, or antibodies against CTLA-4, OX40, B7H3, CSFR1, LAG-3, TIGIT, TIM -3, etc.
  - **A vaccine (ex: HS-110) that induces CD8+ T cells to infiltrate the tumors**

The Big Challenge:

Most patients with NSCLC don't respond to checkpoint inhibition



Even the "ideal" pts (PD-L1 ≥ 50%) don't all respond to checkpoint inhibitors

Reck M et al. N Engl J Med 2016;375:1823-1833

Penn Medicine

The NEW ENGLAND JOURNAL of MEDICINE

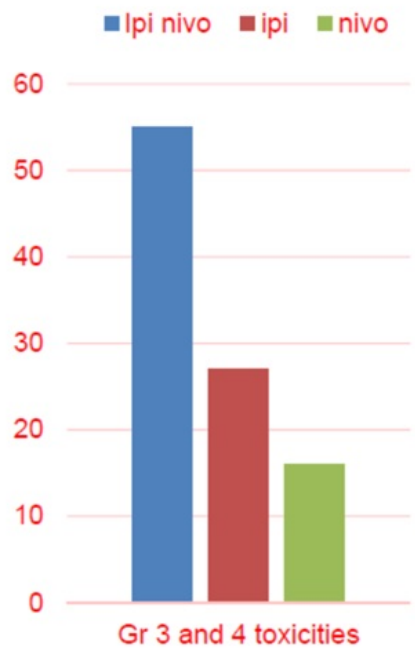
the cure is within
ABRAMSON CANCER CENTER

Considerable Unmet Need in All Lines of Therapy

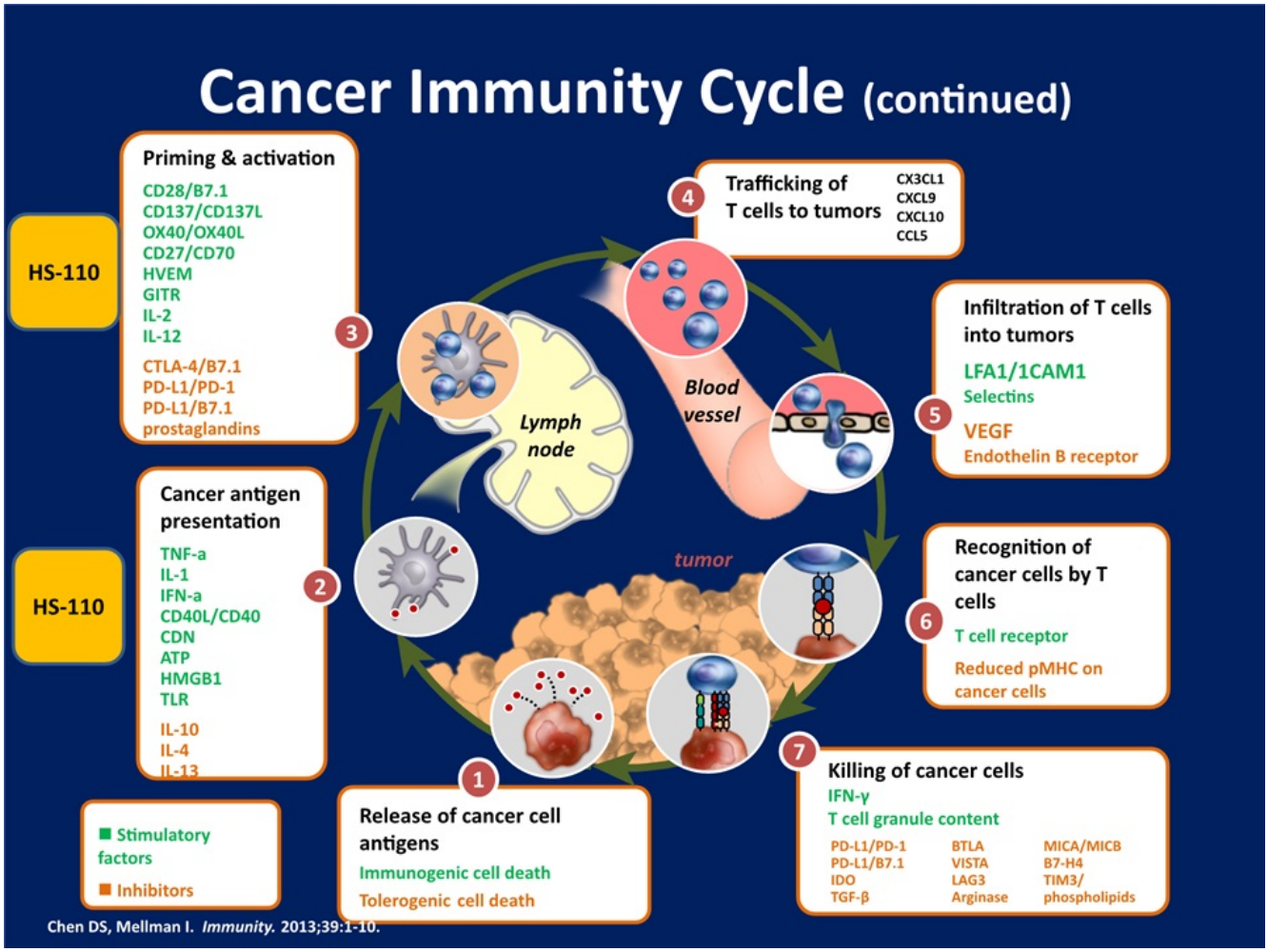
- In the PDL-1 intermediate (1-50%) patients the response rate is lower: ~20-25%
- In the PDL-1 negative (<1%) patients the response rates are < 10%
- And patients who do respond are not cured
  - They eventually get worse and die from NSCLC
- Reasons for failure of checkpoint inhibition likely include:
  - There are no T-cells in the tumor (the tumors are 'cold')
  - There are other white blood cells in the tumors that block the T-cells from doing their job
  - The cancer is using checkpoints other than PD-1/PD-L1
  - The cancer substitutes new checkpoints when we block PD-1/ PD-L1
  - Unknown mechanisms of immune evasion

Immunotherapy Combinations

- Improving response rates and response duration will require IO combinations
- Existing combinations, such as Opdivo-Yervoy are significantly more toxic than Opdivo monotherapy
- Immune-related toxicities include pneumonitis, colitis, rashes, hepatitis, nephritis, encephalitis and others
- Additive immune therapies that don't add significant toxicity are needed



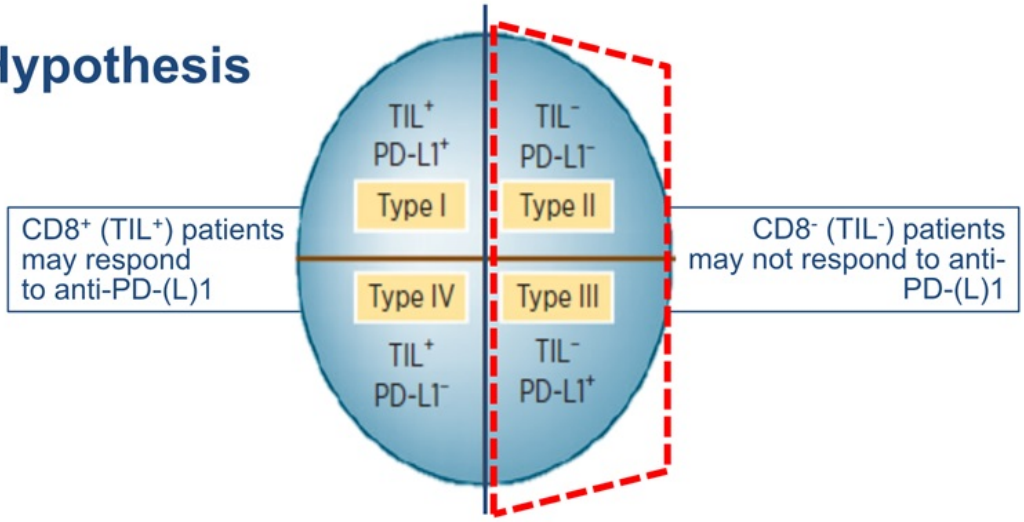
Larkin et al, N Engl J Med 2015; 373:23-34


Cancer Immunity Cycle (continued)
HS-110
Priming & activation
CD28/B7.1
CD137/CD137L
OX40/OX40L
CD27/CD70
HVEM
GITR
IL-2
IL-12
CTLA-4/B7.1
PD-L1/PD-1
PD-L1/B7.1
prostaglandins
3
4
Trafficking of T cells to tumors
CX3CL1
CXCL9
CXCL10
CCL5
Infiltration of T cells into tumors
LFA1/1CAM1
Selectins
VEGF
Endothelin B receptor
5
Lymph node
Blood vessel
tumor
HS-110
Cancer antigen presentation
TNF-a
IL-1
IFN-a
CD40L/CD40
CDN
ATP
HMGB1
TLR
IL-10
IL-4
IL-13
2
6
Recognition of cancer cells by T cells
T cell receptor
Reduced pMHC on cancer cells
1
Release of cancer cell antigens
Immunogenic cell death
Tolerogenic cell death
7
Killing of cancer cells
IFN-γ
T cell granule content
PD-L1/PD-1
PD-L1/B7.1
IDO
TGF-β
BTLA
VISTA
LAG3
Arginase
MICA/MICB
B7-H4
TIM3/ phospholipids
Stimulatory factors
Inhibitors
Chen DS, Mellman I. Immunity. 2013;39:1-10.

Combining Therapeutic Vaccines with Checkpoint Inhibitors

What might a therapeutic vaccine like HS-110 do?
Generate CD8+ cells (TILs) that penetrate tumors and make them "hot"

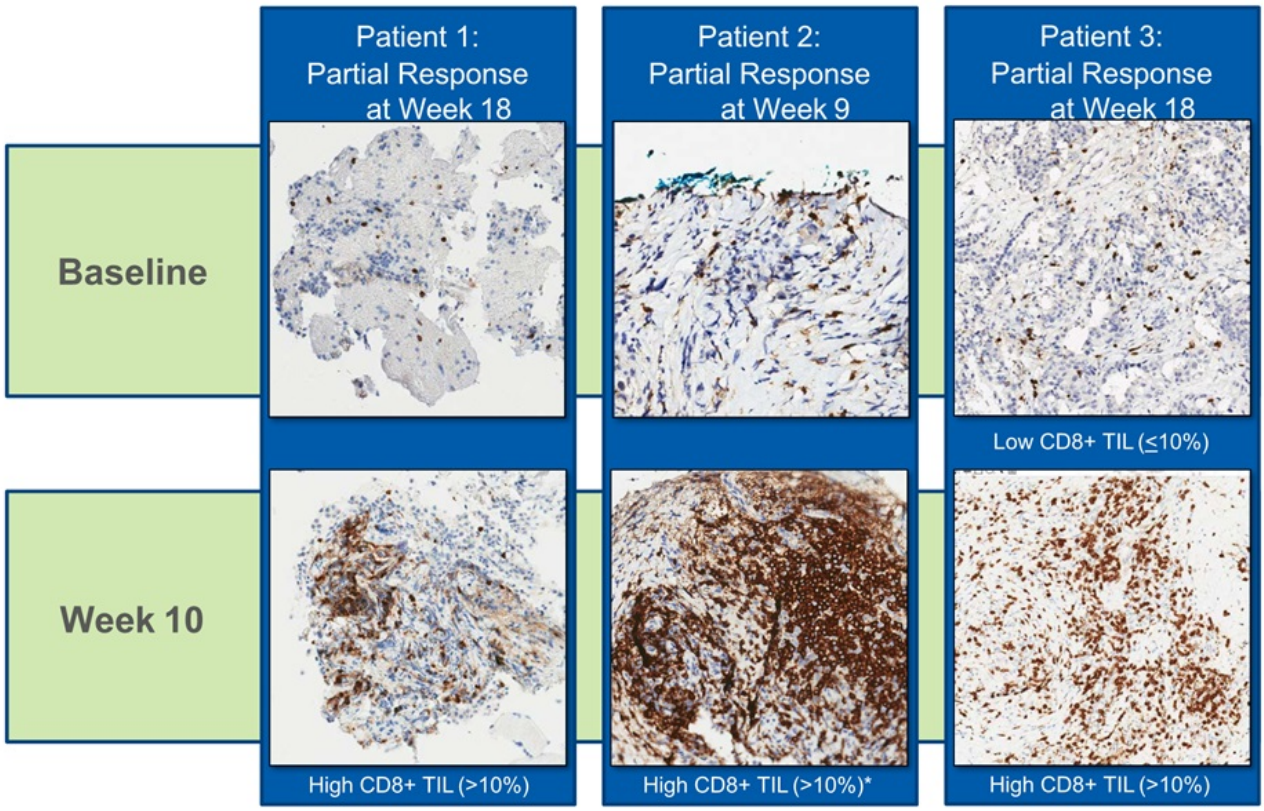
Hypothesis



Teng et al., 2015 Can Res
Gettinger et al., 2015 JCO

Convert TIL- tumors to TIL+

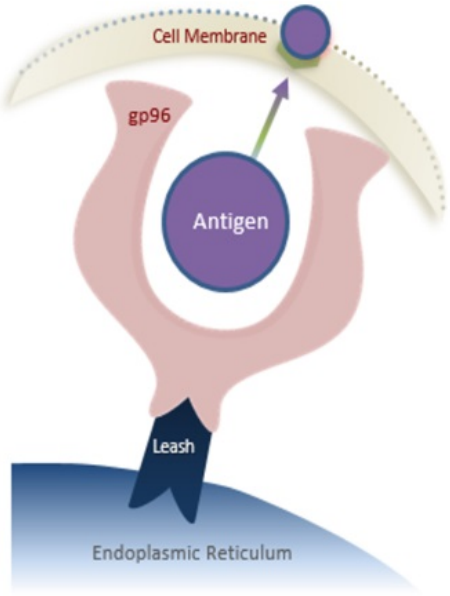
Biopsies from the DURGA Trial: TIL Infiltration Associated with Clinical Response



Previous lung cancer vaccines were not designed to elicit a robust CD8+ T-cell response

Introducing gp96- *The Immune System's "Swiss Army Knife"**

A Natural "Molecular Warning System"



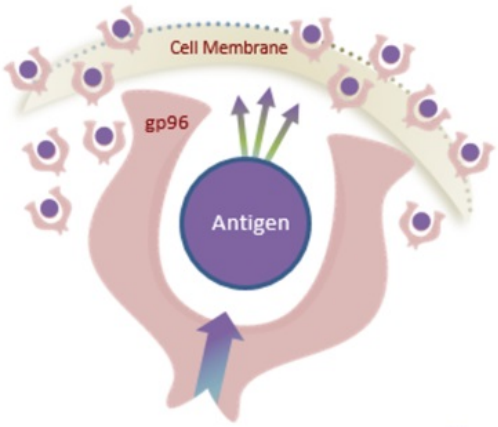
- Gp96 "chaperones" newly-created proteins to the cell membrane where they are released and embedded
- Gp96 + its ferried protein are naturally released only via necrosis
  - Exposure of gp96 outside the cell activates an immune response to the antigen it is carrying
  - Enables MHC I antigen cross-presentation specifically to CD8+ T-cells
  - Activates a cytotoxic T-cell response to the cargo antigen
- Gp96 among the most powerful immune adjuvants
- Gp96 is the only adjuvant that generates exclusively CD8+ ("killer") T-cells

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

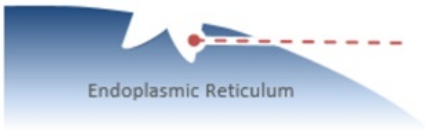
Heat's gp96 *ImPACT* Therapy

Heat Biologics *ImPACT*® technology reprograms cancer cells to continuously secrete their own antigens bound to heat shock protein gp96

Severing the Leash



- 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 the gp96 chaperone
  - This process mimics necrotic cell death
- Activates a powerful pan-antigen cytotoxic T-cell immune response

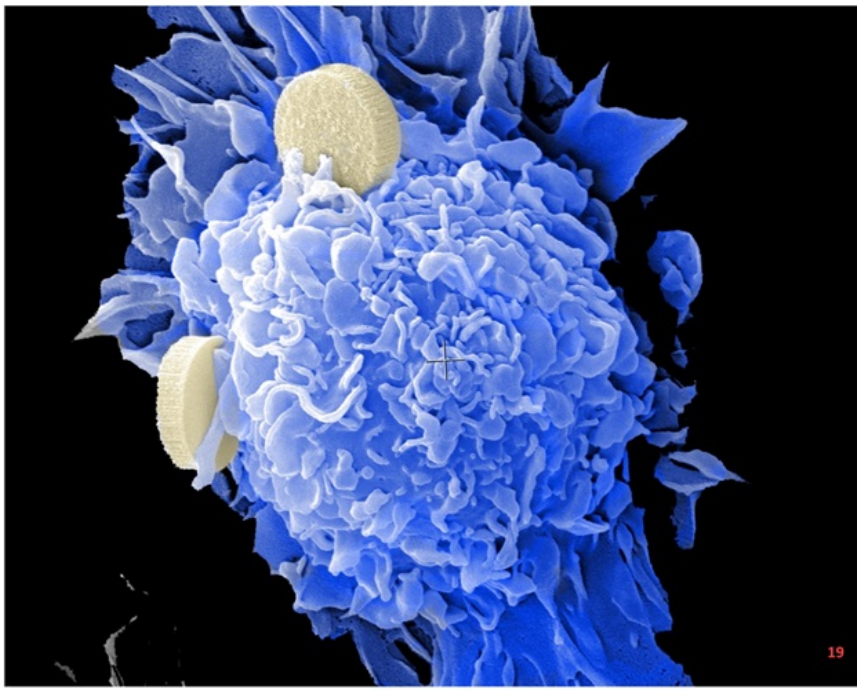


Heat Biologics *ImPACT* technology removes the leash that binds gp96 to the cell, replacing with a sequence that allows cells to continually secrete gp96 along with their "chaperoned" antigens

Heat Biologics

Heat Biologics

ImPACT Mechanism of Action
Jeff Hutchins PhD
Chief Scientific Officer
February 28, 2018



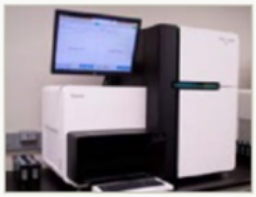
Heat Biologics

ImPACT/ComPACT Manufacturing

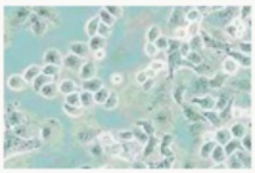
Cell-Based, Multi-Antigen T Cell Activation

Screen → **Select** → **Scale-Up**

Compare CTA Expression on Tissue/Cell Lines

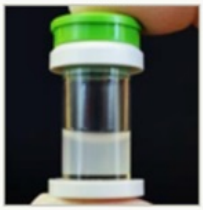


ID Cell Line with Most Antigen Overlap



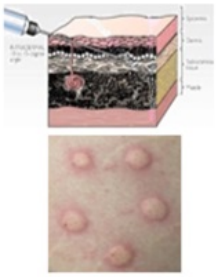
Transfect with Activation Technology

Scale-Up for Manufacturing by CMO

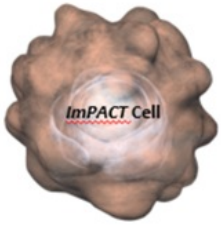


Low COG, off-the-shelf alternative to autologous therapies

ImPACT: Immune Pan-antigen Cytotoxic Therapy

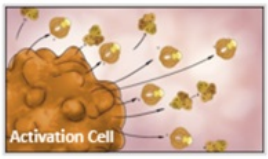


Cluster of five 0.1 mL intradermal injections

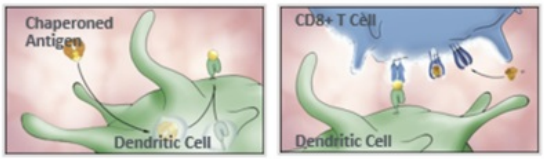


ImPACT cells secrete antigens designed to specifically activate patient's killer T-cells

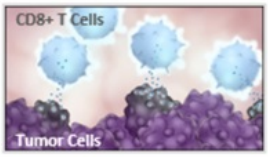
T-cells circulate to destroy patient's tumor



Activated cells **EXPRESS** chaperoned antigens

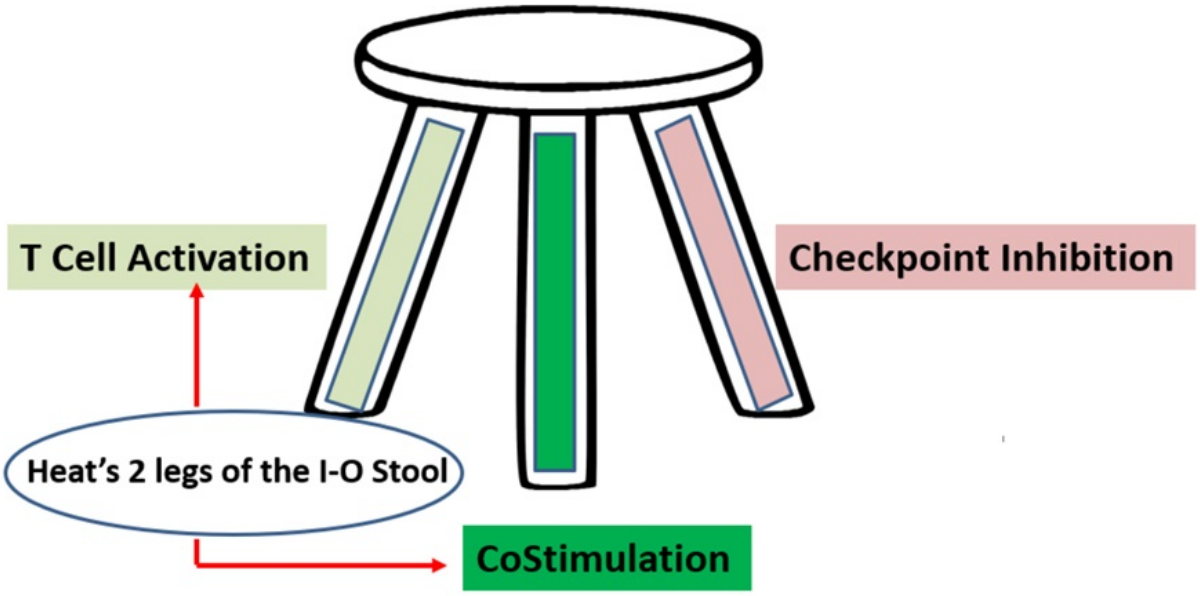


Chaperoned antigens activate dendritic cells, which then **ACTIVATE** & **PROLIFERATE** CD8+ T-cells



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

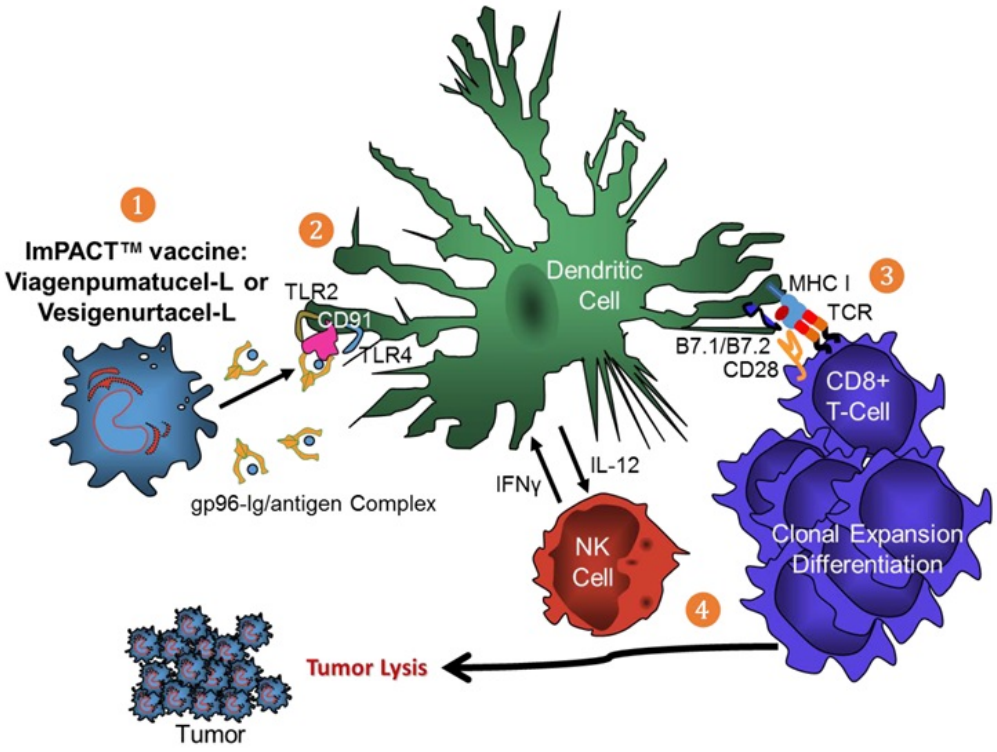
Successful Immuno-oncology: A 3 Legged Stool



Heat Biologics

ImPact Generates an Adaptive Immune Response

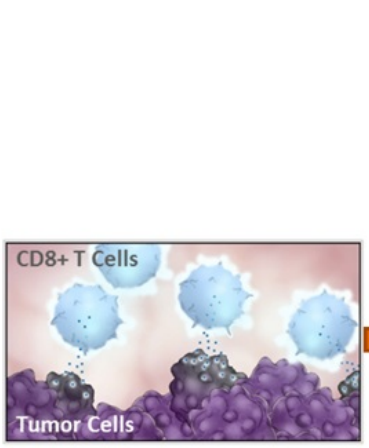
1. **Secretion of gp96 -Ig** carrying tumor specific proteins represented on the patients tumor.
2. **Activation of APCs** (TLR2/4) and cross-presentation of antigens (CD91).
3. **Specific T-cell receptor** engagement.
4. **Clonal Expansion** of Tumor Antigen Specific T cells.



Heat Biologics

Clinical Proof of Mechanism in NCSLC

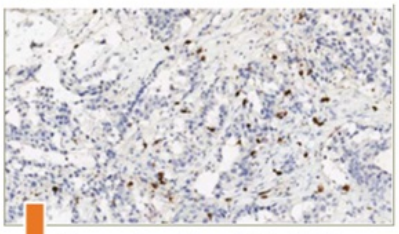
Histopathological evidence that HS-110 is turning COLD tumors HOT



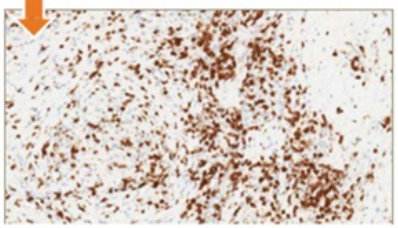
CD8+ T-cells locate and **ELIMINATE** cancer cells

CD8+ Staining

Baseline



Conversion of Low TIL to High TIL After 10 Weeks on HS-110 & Nivolumab Combo



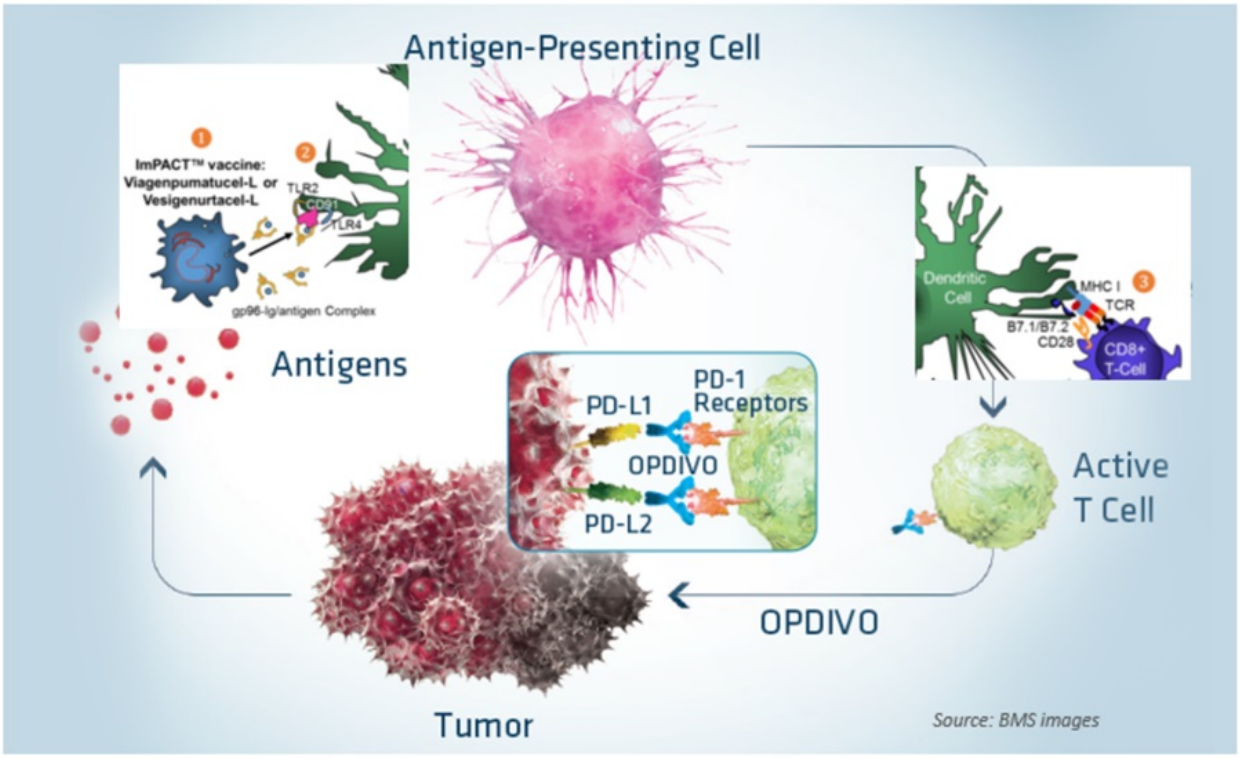
Week 10

- Increased levels of CD8+ T cells deep into the tumor
- Tumors with no previous immune activation made highly active
- Association with radiographic clinical response

Heat Biologics

ImPACT + Opdivo Combination Therapy

The potential to improve clinical responses and survival, without additional toxicity

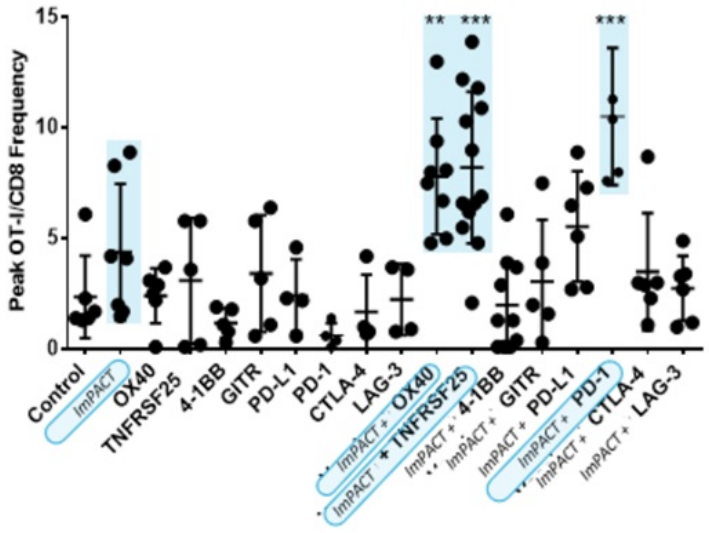


Heat Biologics

Pre-clinical Data of T-cell Expansion

✓ Strong support for our clinical approaches

CD8+ T-cell Expansion



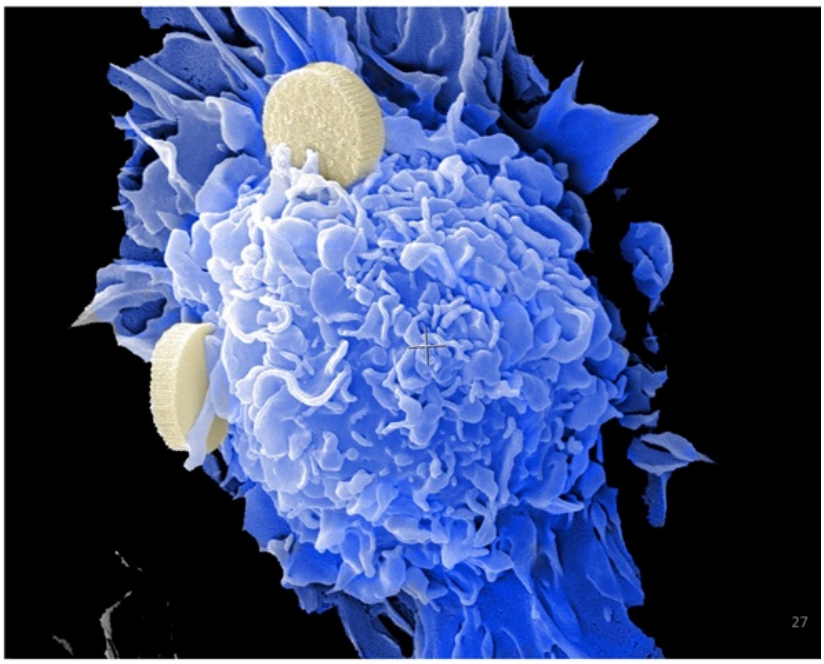
- Higher T-cell responses observed in mice treated with *ImPACT* alone
- *ImPACT* boosted CD8+ T-cells to even higher levels when combined with co-stimulator agonist antibodies: OX40, TNFRSF25, PD-1
- Findings suggest synergies when combining *ImPACT* with Pelican's TNFRSF25 antibody

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

Heat Biologics

Heat Biologics

DURGA Interim Data Review
George Peoples MD FACS
Chief Medical Officer
February 28, 2018



Heat Biologics

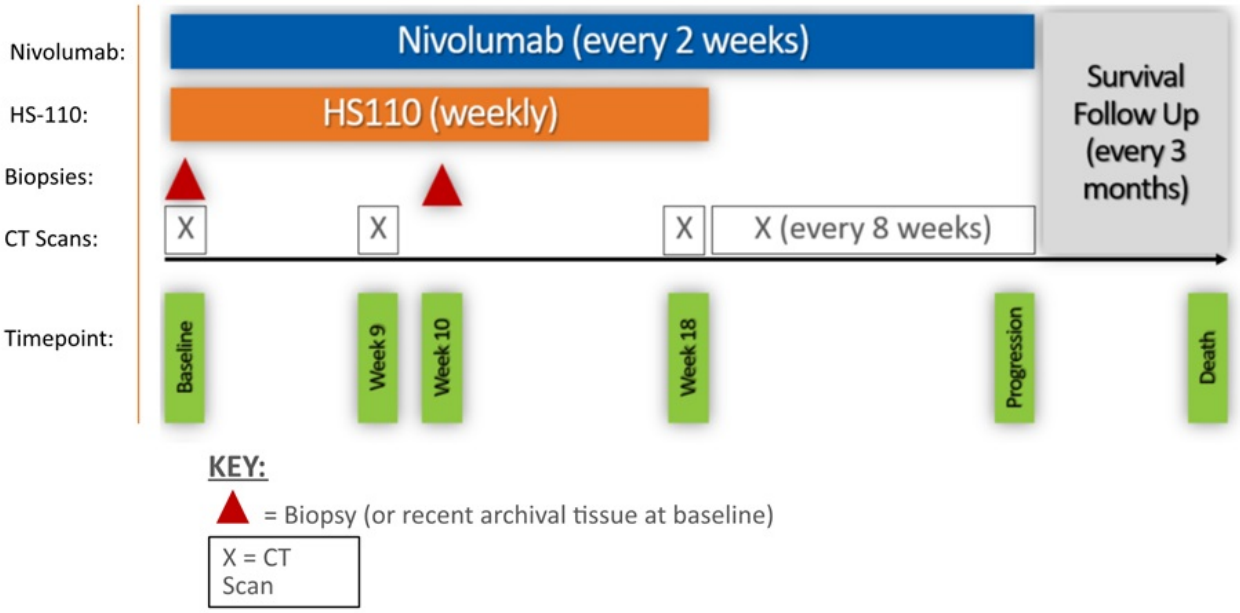
HS-110-102 DURGA Trial

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 with a PD-1 checkpoint inhibitor (nivolumab)
Design	• Single arm multicenter trial of up to 120 patients • Cohort analysis based on histology, prior checkpoint inhibitor therapy, TIL levels and PD-L1 expression
Endpoints	• Objective Response Rate (RECIST 1.1) • Duration of Response • Progression-free Survival • Overall Survival • Immune Response • Safety & Tolerability
Population	Previously treated, advanced NSCLC Current Analysis: • Adenocarcinoma • Checkpoint inhibitor naïve New Populations for enrollment: • Squamous cell carcinoma • Checkpoint inhibitor relapsed

Heat Biologics

DURGA Schema

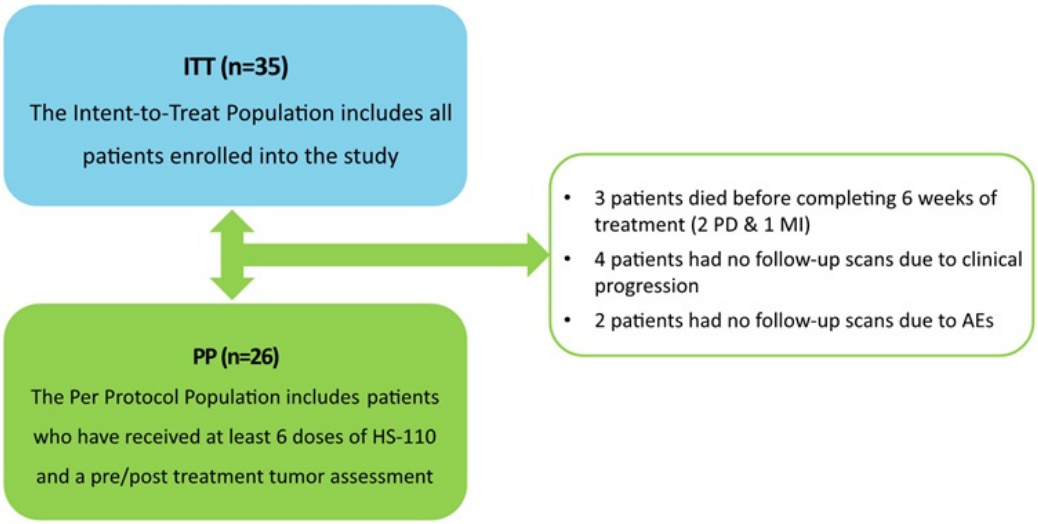


KEY:

▲ = Biopsy (or recent archival tissue at baseline)

X = CT Scan

Pre-Specified Patient Populations Analyzed

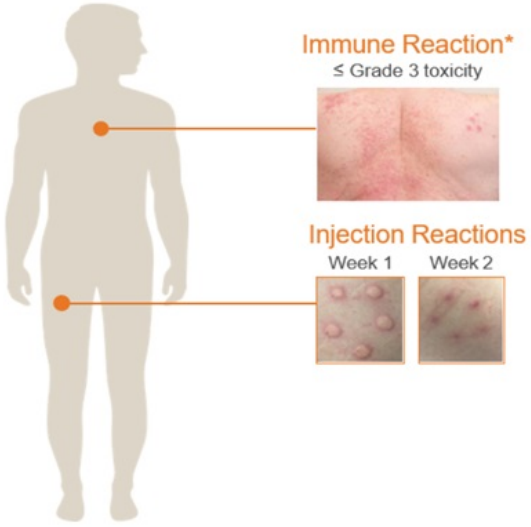


ImPACT (HS-110) Safety Profile to Date

~1,000 Doses – No Serious Adverse Reactions

Favorable Safety Profile To Date

- Almost 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 SOC
- No additive toxicities with SOC



*Represents the only patient of ~100 patients dosed who discontinued treatment for a vaccine-related adverse event

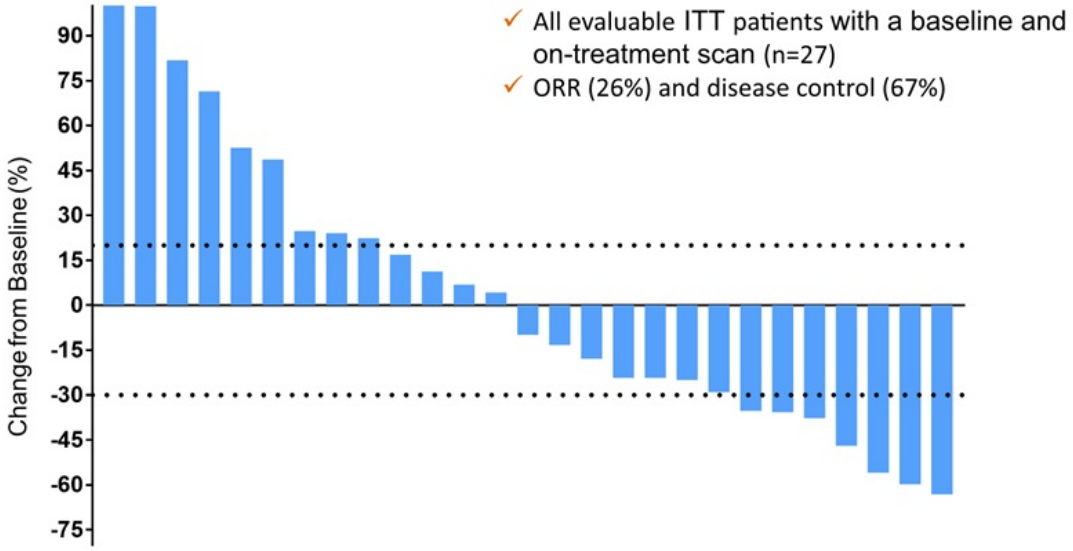
Primary Efficacy Analysis

Population	Objective Response Rate (RECIST 1.1)	Disease Control Rate (RECIST 1.1)
ITT (n=35)	17%	40%
PP (n=26)	23%	50%

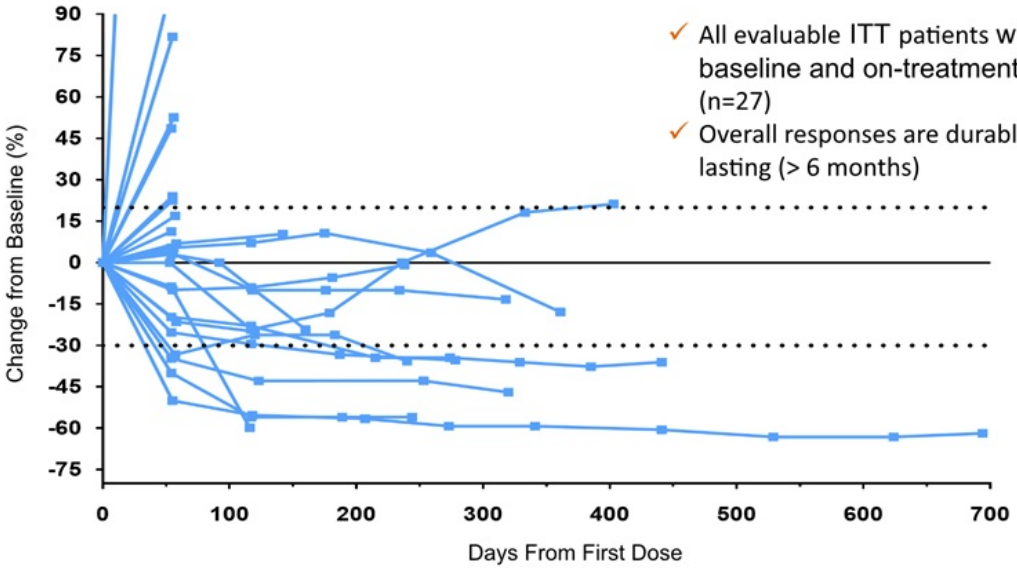
ORR: Objective Response Rate is defined as the % of patients who have reached Partial Response (PR) per RECIST 1.1 which requires a 30% reduction in the sum of the longest diameters of all target lesions from baseline.

DCR: Disease Control Rate is defined as the % of patients who have reached Partial Response (PR) or Stable Disease (SD) per RECIST 1.1 which requires that the sum of the longest diameters of all target lesions does not increase more than 20% from baseline.

Best Target Lesion Response



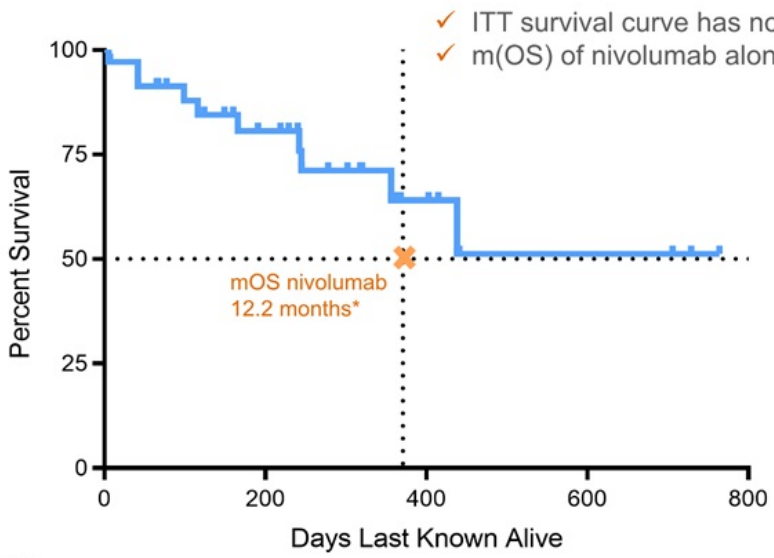
Durable Target Lesion Response



- ✓ All evaluable ITT patients with a baseline and on-treatment scan (n=27)
- ✓ Overall responses are durable and long lasting (> 6 months)

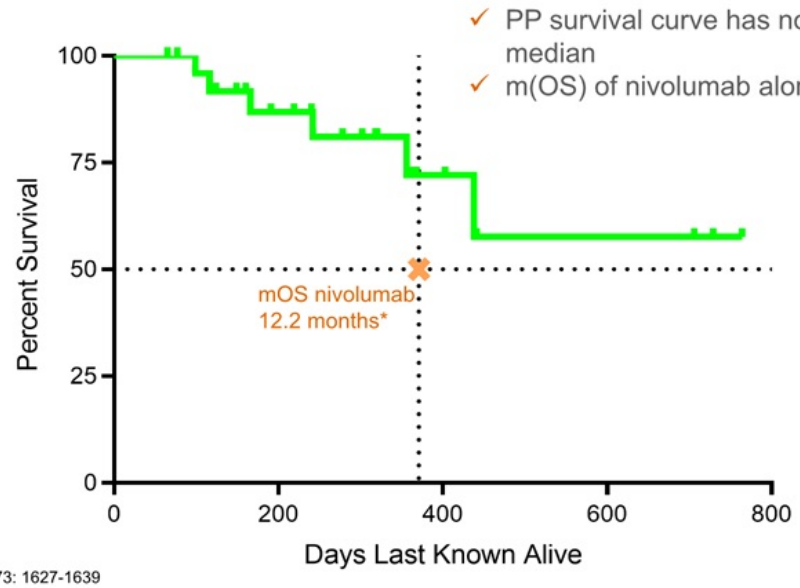
All enrolled patients (ITT) with a baseline and on-treatment scan

ITT Overall Survival: Encouraging and Still Maturing



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

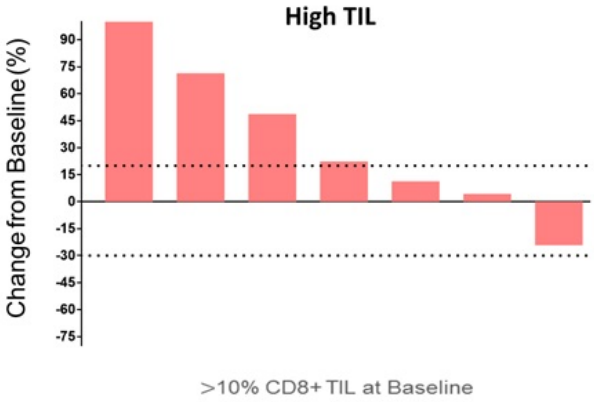
PP Overall Survival: Encouraging and Still Maturing



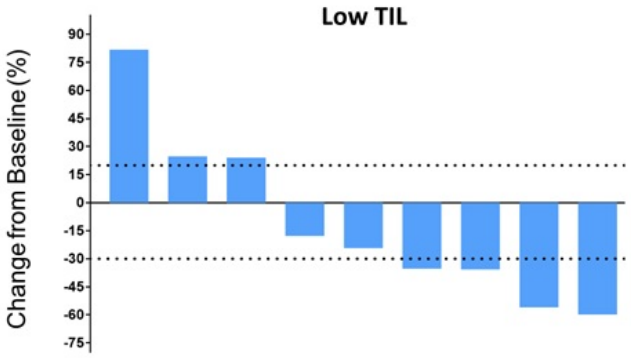
*N Engl J Med 2015; 373: 1627-1639

Target Lesion Response Based on Initial TIL Status

- ✓ HS-110 shows effect in low TIL "cold tumor" patients who typically do not respond well to PD-1 inhibitors
- ✓ 4 of 9 achieved a partial clinical response



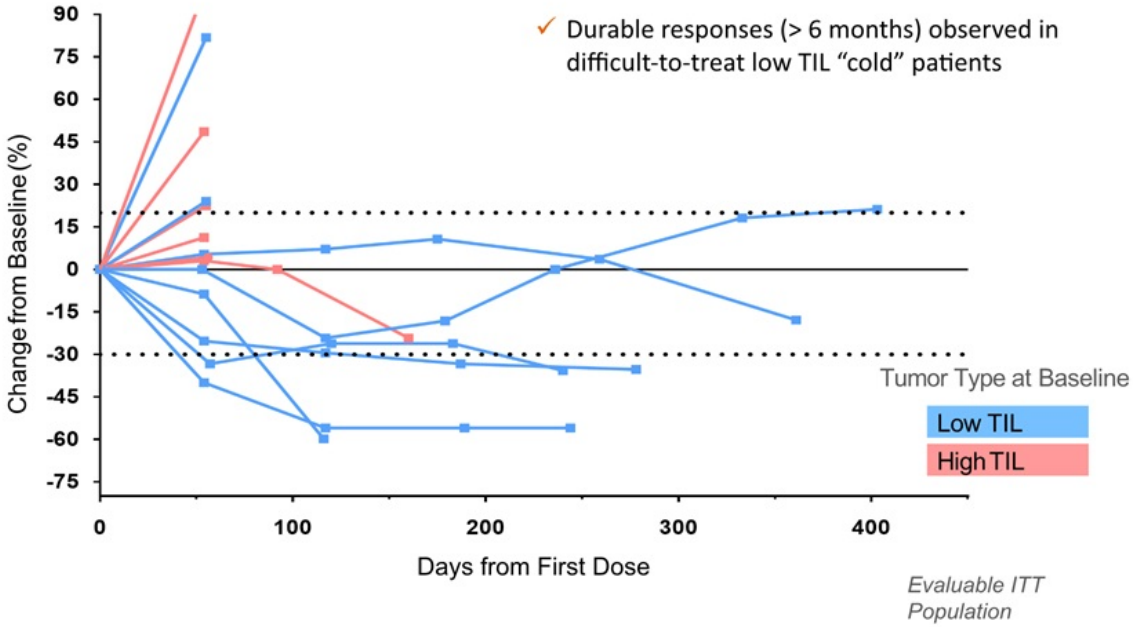
>10% CD8+ TIL at Baseline



≤10% CD8+ TIL at Baseline

Evaluable ITT Population

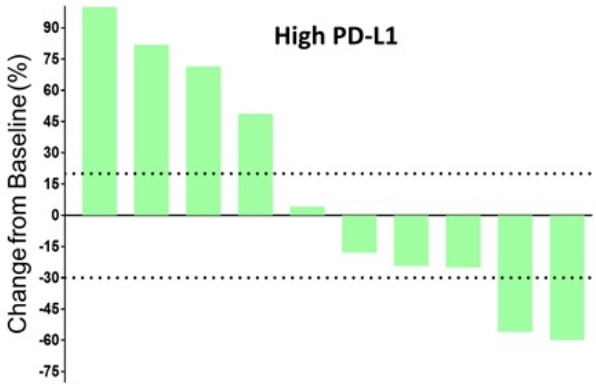
Durable Target Lesion Responses Based on Initial TIL Status



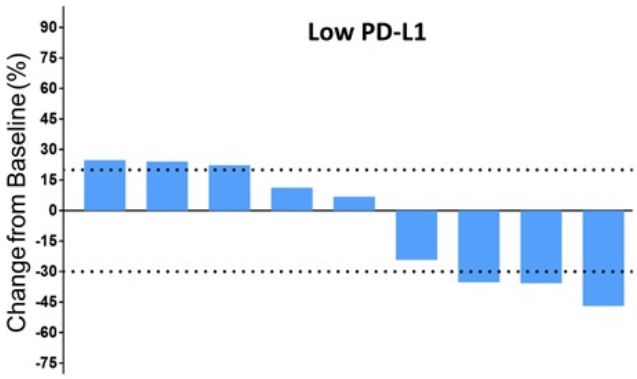
Evaluable ITT Population

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

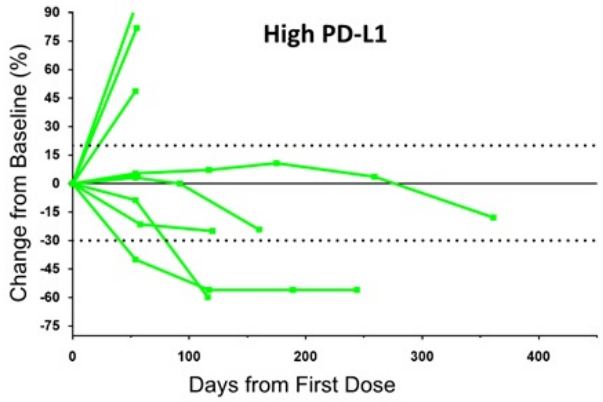


<1% PD-L1 Tumor Type at Baseline

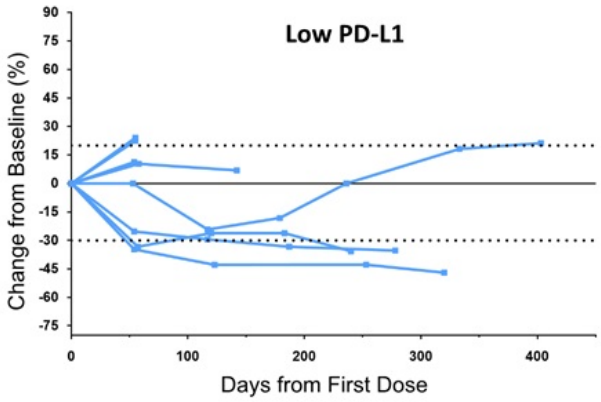
Evaluable ITT Population

Durable Target Lesion Responses Based on Initial PD-L1 Status

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



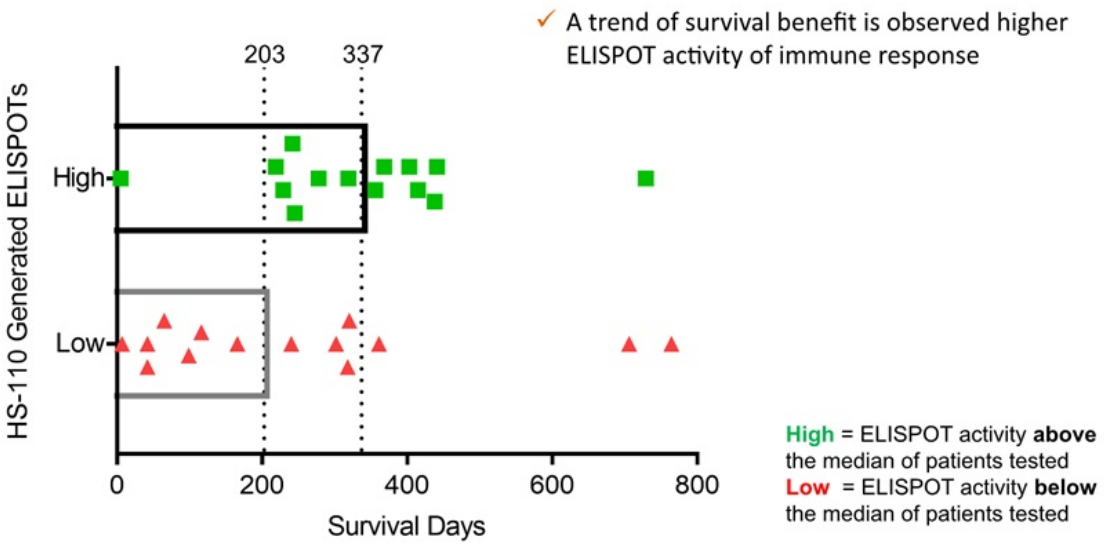
≥1% PD-L1 Tumor Type at Baseline



<1% PD-L1 Tumor Type at Baseline

Evaluable ITT Population

ELISPOT Activity and Survival



Summary of Interim Data

- ✓Tumor shrinkage and disease control demonstrated in a majority of evaluable patients
  - ✓ Overall responses are durable and long lasting
  - ✓ While survival data is still maturing, the median overall survival has not yet been reached
- ✓HS-110 shows durable responses in difficult-to-treat low TIL "cold tumor" patients
- ✓HS110 shows durable responses in low PD-L1 patients, who typically do not respond to checkpoint inhibitors
- ✓A trend of survival benefit is observed with higher ELISPOT activity reflective of tumor antigen-specific immune response

This data is consistent with HS-110 mechanism of action as well as data previously reported in our phase 1 trial