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, 2019**

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 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 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 7.01. Regulation FD Disclosure.

On February 28, 2019, updated interim results from Heat Biologics, Inc.'s (the "Company's") ongoing 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) were presented in a poster that was presented at the ASCO-SITC Clinical Immuno-Oncology Symposium by Daniel Morgensztern, M.D., Associate Professor of Medicine and Director of Thoracic Oncology, Washington University School of Medicine, and Lead Investigator in the trial. A copy of the poster titled "VIAGENPUMATUCEL-L (HS-110) PLUS NIVOLUMAB IN PATIENTS WITH ADVANCED NON-SMALL LUNG CANCER (NSCLC)" is attached to this Current Report on Form 8-K as Exhibit 99.1.

The poster presentation attached as Exhibit 99.1 to this Current 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 Current Report or the Exhibit.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits.

The following exhibit is filed with this Current Report on Form 8-K:

Exhibit Number	Description
99.1	ASCO Poster Presentation titled "VIAGENPUMATUCEL-L (HS-110) PLUS NIVOLUMAB IN PATIENTS WITH ADVANCED NON-SMALL LUNG CANCER (NSCLC)"

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, 2019

HEAT BIOLOGICS, INC.

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

EXHIBIT INDEX

Exhibit Number	Description
99.1	ASCO Poster Presentation titled “VIAGENPUMATUCEL-L (HS-110) PLUS NIVOLUMAB IN PATIENTS WITH ADVANCED NON-SMALL LUNG CANCER (NSCLC)”

VIAGENPUMATUCEL-L (HS-110) PLUS NIVOLUMAB IN PATIENTS WITH ADVANCED NON-SMALL LUNG CANCER (NSCLC)

Daniel Morgensztern[1], Lyudmila Bazhenova[2], Saiama N Waqar[1] Lori McDermott[3], Jeff Hutchins[3], Wael Harb[4], Vamsidar Velcheti[5], Roger B. Cohen[6]

[1]Washington University School of Medicine, St. Louis, MO; [2]UC San Diego, Moores Cancer center, San Diego, CA; [3]Heat Biologics, Inc, Durham, NC; [4]Horizon Oncology Center, Lafayette, IN, [5]Taussig Cancer Institute, Cleveland Clinic, Cleveland, OH, [6]University of Pennsylvania Perelman School of Medicine, Philadelphia, PA

Background

Viagenpumatucel-L (HS-110) is an allogeneic cellular immunotherapy that incorporates a broad range of tumor antigens that are known to be shared amongst a high proportion of patients with non-small cell lung cancer (NSCLC). This cell system contains HLA-A1 (a human histocompatibility surface antigen) for purposes of drug identity and gp96-Ig (a transgene constructed from sequences encoding the human gp96 gene with the C-terminal KDEL sequence removed and replaced with the Fc portion of human IgG1). This construct is designed to enable the cell to express the heat shock protein/adjuvant gp96 in secreted form. The secreted gp96 acts as a chaperone and adjuvant to induce cellular immune responses to various tumor antigens expressed by the host cell. These characteristics make gp96 unique because it can both activate (MHC and T-cell costimulator up-regulation) and deliver chaperoned antigens to an APC for display via MHC I, in order to elicit CD8+ T-cell mediated immune responses [1,2].

The HS110-102 "Durga" Trial is an exploratory, multi-cohort master protocol evaluating HS-110 in combination with anti-PD1 mAbs in the treatment of advanced non-small lung cancer. Here we present interim data from the first two Cohorts, A and B, for all patients enrolled on or before the efficacy data cut-off. Cohort A (n=42) consists of previously treated patients who have never received an immune checkpoint inhibitor (CPI). Cohort B (n=20) is comprised of patients with progressive disease (PD) after receiving a minimum of 4 months of CPI therapy at any time prior to study entry. Trial ID: **NCT02439450**

Mechanism of Action

- Allogeneic cellular vaccine
- AD100 transfected with gp96-Ig

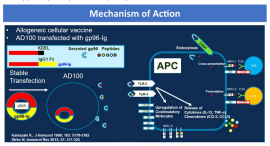


Figure 1: Viagenpumatucel-L (HS-110) Mechanism of Action
HS-110 is developed from the AD100 lung adenocarcinoma cell line transfected with gp96-Ig, where the gp96 KDEL ER retention sequence is replaced by IgG1 Fc. Gp96-Ig acts as a chaperone protein for tumor associated antigens that is recognized by CD91 on APCs, resulting in cross-presentation of antigen to MHC I for the selection of antigen-specific CD8 cells. At the same time, Gp96-Ig binding to TLRs 2 and 4 leads to upregulation of co-stimulatory molecules, MHC I and secretion of cytokines and chemokines.

Study Design and Endpoints

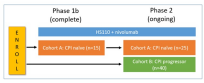


Figure 2: Study Design
This single-arm, open-label trial has a phase 1b portion of 15 previously treated patients that have never received a CPI with a phase 2 expansion, that added Cohort B: Patients with PD after treatment failure with CPI.

Primary Endpoints
Ph 1b: Safety
Ph 2: ORR (RECIST 1.1)

Secondary Endpoints
OS, PFS, DCR, DOR and iRECIST measurements

Exploratory Endpoints
Immune response, and correlation of clinical outcomes with Baseline: CD8+ TIL levels, and PD-L1 expression on tumor cells.

Study Schema

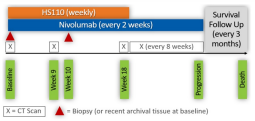


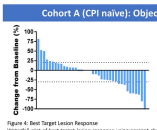
Figure 3: HS110-102 Study Schema
Patients receive weekly HS-110 (1 x 10^7 cells) intradermally for 18 weeks via 5 injections of 0.1ml each, and biweekly nivolumab 240 mg IV until disease progression or unacceptable toxicity.

Patient Characteristics

		Cohort A (N = 44)	Cohort B (N = 31)
Median age (range)		65 (37-87)	66 (50-84)
Female gender		24 (55%)	18 (58%)
White race		39 (89%)	25 (81%)
ECOG PS 1		27 (61%)	11 (35%)
EGFR or ALK positive		9 (20%)	3 (10%)
Histology	Adeno Squamous	41 (93%) 3 (7%)	25 (81%) 6 (19%)
Smoking status	Current/past Never	37 (84%) 7 (16%)	27 (87%) 4 (13%)
Prior lines of tx	1 2 or more Unavailable	27 (61%) 13 (30%) 4 (9%)	5 (16%) 18 (58%) 8 (26%)
PD-L1	< 1% ≥ 1% Unevaluable	17 (39%) 13 (29%) 14 (32%)	8 (26%) 14 (45%) 9 (29%)
CD8+ TIL	≤ 10% > 10% Unevaluable	12 (27%) 8 (18%) 24 (55%)	9 (29%) 10 (32%) 12 (39%)

Table 1: Patient Characteristics
Baseline patient demographics of the safety population (n=75). Note that only the patients enrolled prior to the data cut-off were included in the efficacy population (n=62).

Cohort A (CPI naïve): Objective Response Rate



RECIST 1.1 ORR = 21.4%
(95% CI, 10.3 - 36.8%)

PR	9 (21%)
SD	12 (29%)
Not evaluable	4 (10%)
DCR	**21 (50%)**

Figure 4: Best Target Lesion Response
Waterfall plot of best target lesion response using percent change from baseline of the SLD (sum of longest diameters) for all patients who received at least 1 post-baseline scan (n=38).

Cohort A (CPI naïve): Overall Survival & Tumor Burden

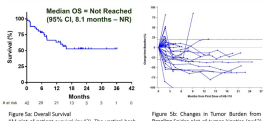


Figure 5a: Overall Survival
KM plot of patient survival (n=42). The vertical hash marks represents patient censoring. Median follow up of 14.4 months with 60% of patients still alive.

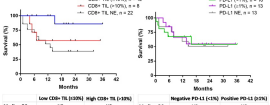


Figure 5b: Changes in Tumor Burden from Baseline Spider plot of tumor kinetics (n=42) demonstrating durability of decreased burden.

	Low CD8+ TIL (≤10%)	High CD8+ TIL (>10%)
Median OS	NR	NR
12 month OS	75%	50%
HR 0.89 (95% CI, 0.86, 2.91)		

	Negative PD-L1 (<1%)	Positive PD-L1 (≥1%)
Median OS	NR	NR
12 month OS	44%	54%
HR 0.85 (95% CI, 0.26, 2.79)		

Figure 6: Overall Survival – Subgroup Analysis by CD8+ TIL and PD-L1 Levels
KM plot of patient survival (n=42) based on protocol-defined subgroups of CD8+TIL (high and low) and PD-L1 status (positive and negative) at baseline. The vertical hash marks represents patient censoring.

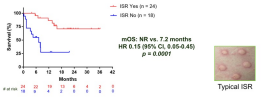


Figure 7: Overall Survival – Injection Site Reactions (ISR)
KM plot of patient survival (n=42) with ISR subgroups (yes or no) demonstrates a statistically significant survival benefit in patients experiencing at least one injection site reaction to HS-110 during study treatment. The vertical hash marks represents patient censoring.

Adverse Events

Adverse events (AEs) occurring in ≥10% of patients in the safety population (n=75) are: fatigue (31%), cough (21%), diarrhea (15%), anemia (13%), dyspnea (13%), nausea (13%), pruritis (13%), arthralgia (12%), hypoalbuminemia (12%), decreased appetite (11%), hyponatremia (11%), dizziness (11%) and constipation (11%). There were two grade 5 AEs, pulmonary embolism and acute myocardial infarction, neither of which were deemed related to treatment.

Cohort B (CPI progressors): Objective Response Rate

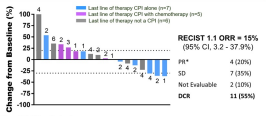


Figure 8: Best Target Lesion Response
Waterfall plot of best target lesion response using percent change from baseline of the SLD (sum of longest diameters) for all patients who received at least 1 post-baseline scan (n=18). The bar colors indicate the type of therapy received immediately preceding study entry. The numerical values presented with each bar indicate the number of lines of prior treatment. *per Investigator Assessment

Cohort B (CPI Progressors): Duration of Treatment

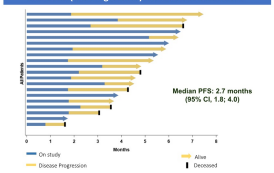


Figure 9: Duration of Treatment
Swimmer plot showing time on study and time of disease progression (n=20).

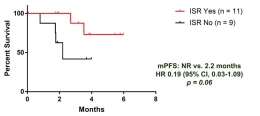


Figure 10: Progression Free Survival – Injection Site Reactions (ISR)
KM plot of patient progression free survival (n=20) with ISR subgroups (yes or no) shows progression-free survival benefit in patients experiencing at least one injection site reaction to HS-110 during study treatment.

Immune Activity and Response

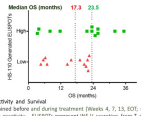


Figure 11: Immune Activity and Survival
Peripheral blood obtained before and during treatment (Weeks 4, 7, 13, EOT; n=24) to measure HS-110 derived immune reactivity. ELISPOTs represent INF-γ secretion from T cells in culture after stimulation with HS-110 lysate. High = Patients with absolute ELISPOT increases above the group median; Low = Patients with absolute ELISPOT increases below the group median.

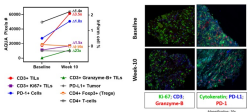


Figure 12: Changes in the Tumor Microenvironment
Using AQUA (CD3, Ki67, Granzyme B, PD-1, PD-L1) and inForm (CD4, Foxp3) immunohistochemistry-based analysis, cell phenotypes were quantitated in the tumor microenvironment (TME) of a patient at baseline and after 10 weeks of combination treatment.

Conclusions

HS-110 in combination with nivolumab is well tolerated.

Preliminary results demonstrate the ability of HS-110 plus nivolumab to induce CD8+ T-cell tumor infiltration in previously "cold" tumors.

In Cohort A, the occurrence of injection site reactions and increased INF-γ ELISPOTs may be associated with improved overall survival.

In Cohort B, early data suggest that the addition of HS-110 to nivolumab may restore responsiveness after tumor progression on prior CPI.

References

1. Strbo N, Garcia-Soto A, Schreiber TH, Podack ER. Secreted heat shock protein gp96-Ig: next-generation vaccines for cancer and infectious diseases. Immunologic research 2013;57:311-25.
2. Strbo N, Vaccari M, Pahwa S, et al. Novel vaccination modality provides significant protection against mucosal infection by highly pathogenic SIV. Journal of immunology (Baltimore, Md : 1950) 2013;190:2495-9.

Acknowledgements

The authors are grateful for the investigators, study staff, patients and their families for the commitment to this trial to help advance the treatment of non-small cell lung cancer.

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