Advancing Novel Immunotherapies: HOOKIPA ASCO Data Review

June 7, 2021

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2021 ASCO ANNUAL MEETING

First report of the safety/tolerability and preliminary antitumor activity of HB-201 and HB-202, an arenavirus-based cancer immunotherapy, in patients with HPV16+ cancers

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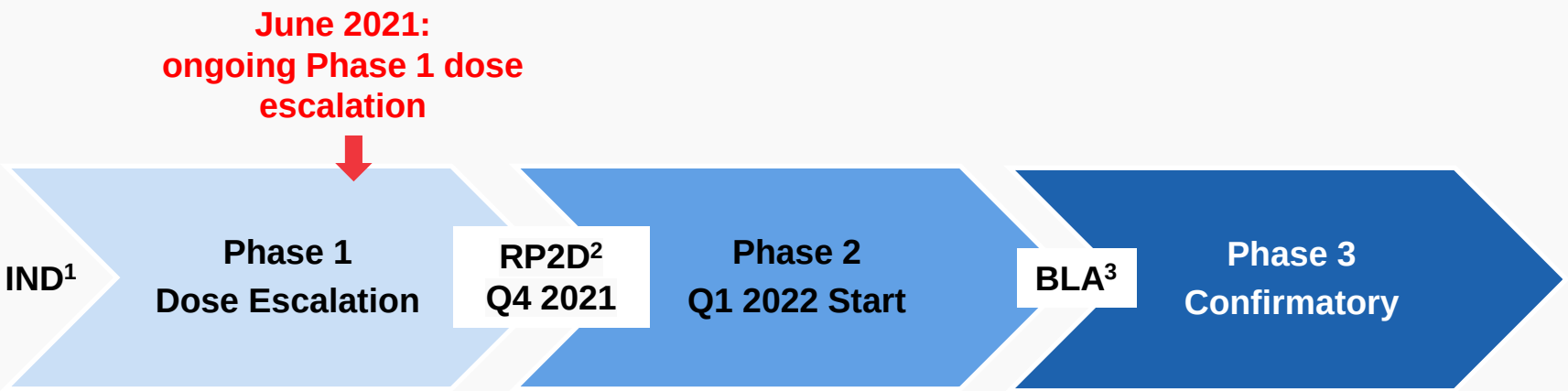
HB-200 in HPV16+ cancers: Ongoing monotherapy clinical trial in late-stage patients who progressed on multiple earlier line treatments



- **Clinical proof of mechanism** (CD8+ T cell induction)
- **HB-200 in monotherapy trial** (not in combination with any other agent) shows **preliminary efficacy in advanced, heavily pre-treated patients**

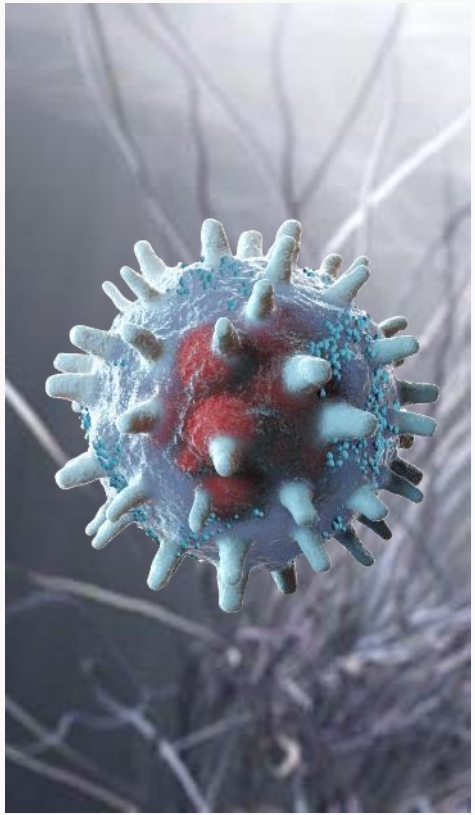
[1]IND, Investigational New Drug Application. [2]RP2D, Recommended Phase 2 Dose. [3]BLA, Biologics License Application.

In our ongoing HB-200 phase 1 trial we have demonstrated POC, defined by clinical proof of mechanism and early clinical efficacy

Up to **40%**	**18%**	**3.5 months**
Tumor Antigen-Specific Polyfunctional T cells	Response Rate In 3rd+ Line Head & Neck Cancer	Median Progression Free Survival

- HB-200 level of tumor antigen specific T cells is unprecedented
- Historically, objective response rates to active immunization in third+ line head and neck cancer patients have been zero.
  - HB-201 shows encouraging preliminary ORR of 18% and
  - Median Progression Free Survival of 3.5 months (longer than CPI's in 2nd line)

3 key messages: Unprecedented T cell responses, monotherapy efficacy, and broad platform potential in cancer



1 Hookipa's arenavirus platform generates unprecedented T cell responses, at times converting almost half of the $CD8^+$ T cell pool to be specific for the desired cancer target

2 HB-200 is the only systemic (IV) active immunization treatment with clinical efficacy as a monotherapy in cancer patients who progressed on standard-of-care, including checkpoint inhibitors

3 Hookipa's versatile arenavirus platform has broad potential applications across multiple cancers by inducing antigen-specific $CD8^+$ T cells, thereby solving a key hurdle of cancer immunotherapy

Agenda

1. **Immunogenicity Data: Perspectives and Interpretation (D Zamarin)**
2. Clinical Data Confirm the Arenavirus Mode of Action: Driving Unprecedented Tumor Antigen-Specific $CD8^+$ T Cell Levels (I Matushansky)
3. Clinical Efficacy as an IV Monotherapy in the Post-CPI Setting (I Matushansky)
4. Early Data Suggestive of a Relationship Between the Mode of Action and Biological Activity (I Matushansky)
5. The Bright Future for Hookipa's Arenavirus Platform in Oncology (J Aldag)
6. Q & A

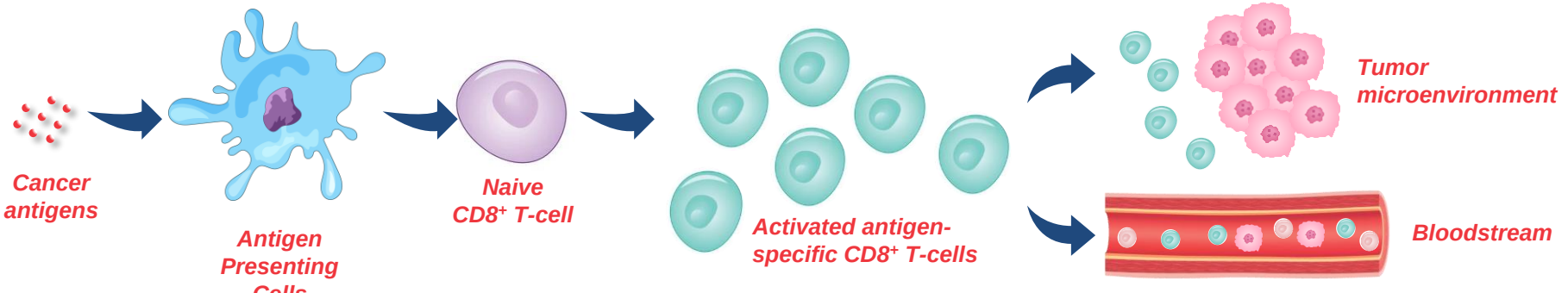
HB-200 Immunogenicity Data: Perspectives and Interpretation

Dmitriy Zamarin, M.D., Ph.D.

Translational Research Director in Gynecologic Medical Oncology at Memorial Sloan Kettering Cancer Center

& HB-200 Study Co-Investigator

CD8+ T cells are central and critical to an antitumor response because they kill cancer cells and cells infected with viruses



Cancer antigens released upon cell death are taken up and **presented by** immune cells called **antigen-presenting cells** (**APCs**) to inactive T cells (killer CD8+ and helper CD4+ T cells).

For **CD8+ T cells**, this interaction triggers **activation, replication**, entry into the **bloodstream**, and migration to the tumor microenvironment.

At the tumor site, these tumor infiltrating lymphocytes **kill cancer cells.**
In the circulation, activated CD8+ T cells and memory cells can detect and **kill circulating tumor cells**

Hiam-Galvez HJ, et al. Nat Rev Caner. 2021;1-15
Waldman AD, et al. Nat Rev Immunol. 2020; 20:651-668

Cancer can evade the immune system…

However, as cancer progresses, the immune response lessens due to chronic stimulation leading to T cell exhaustion and immunosuppressive factors at the site of T cell activation and in the tumor microenvironment.

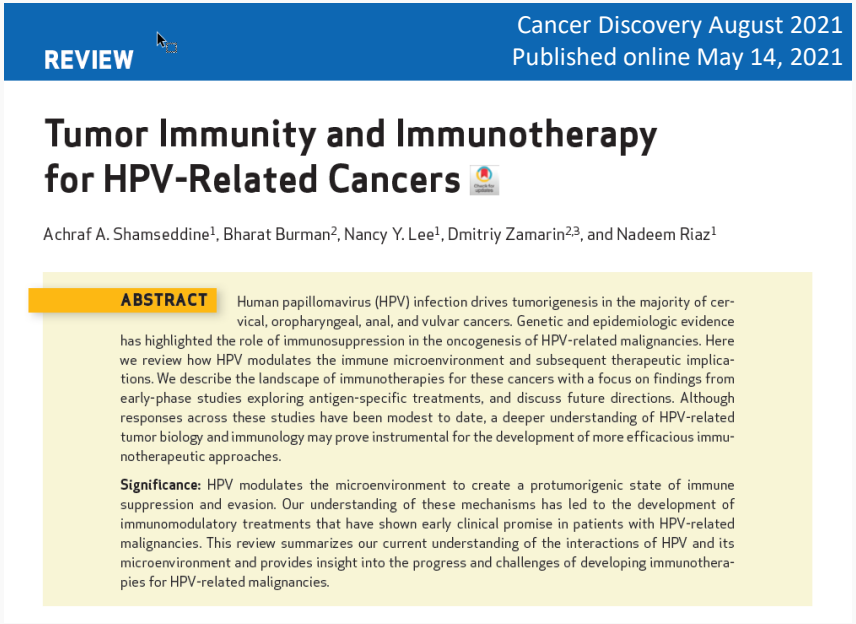
This allows the **cancer** to **evade the immune system, grow, and spread.**

…so active immunization therapies are designed to improve recognition of tumor antigens by the immune system

Active immunization therapies “wake up” the immune system by boosting pre-existing responses or by developing new responses.

$CD8^+$ T cells are at the center of the action!

Results of active immunization therapies to-date: Systemic (IV) monotherapy has not resulted in objective responses in advanced/metastatic cancers



REVIEW

Cancer Discovery August 2021
Published online May 14, 2021

Tumor Immunity and Immunotherapy for HPV-Related Cancers

Achraf A. Shamseddine[1], Bharat Burman[2], Nancy Y. Lee[1], Dmitriy Zamarin[2,3], and Nadeem Riaz[1]

ABSTRACT Human papillomavirus (HPV) infection drives tumorigenesis in the majority of cervical, oropharyngeal, anal, and vulvar cancers. Genetic and epidemiologic evidence has highlighted the role of immunosuppression in the oncogenesis of HPV-related malignancies. Here we review how HPV modulates the immune microenvironment and subsequent therapeutic implications. We describe the landscape of immunotherapies for these cancers with a focus on findings from early-phase studies exploring antigen-specific treatments, and discuss future directions. Although responses across these studies have been modest to date, a deeper understanding of HPV-related tumor biology and immunology may prove instrumental for the development of more efficacious immunotherapeutic approaches.

Significance: HPV modulates the microenvironment to create a protumorigenic state of immune suppression and evasion. Our understanding of these mechanisms has led to the development of immunomodulatory treatments that have shown early clinical promise in patients with HPV-related malignancies. This review summarizes our current understanding of the interactions of HPV and its microenvironment and provides insight into the progress and challenges of developing immunotherapies for HPV-related malignancies.

CONCLUSION AND FUTURE DIRECTIONS

"Future work will need to identify the best way to harness improvements in antigen-specific immune response to improve oncologic outcomes in patients."

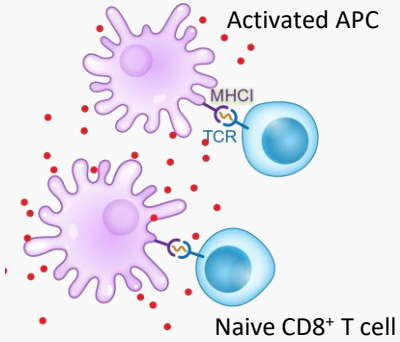
Summary of Active Immunization Therapies for HPV-Related Cancers

- Monotherapy successes only in pre-neoplastic/pre-invasive settings
- Positive data read-outs in combination with PD(L)1 inhibitors or other therapies
- Ongoing trials in advanced/metastatic cancers are testing combinations.

CD8⁺ T cells are the most critical and powerful soldiers in the anticancer immune response

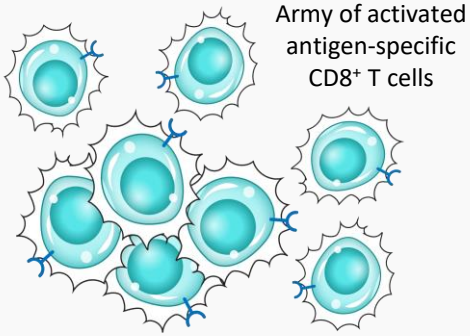
Steps to Create Highly-Specialized Tumor Killers

Activate



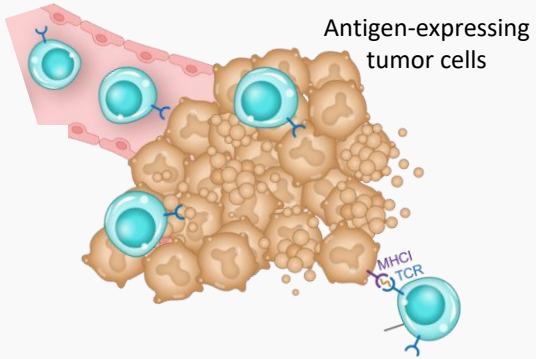
Antigen presenting cells **activate** naive T cells **specific** for the cancer antigen

Multiply



T cells are activated, **multiply**, and learn how to seek out tumors cells with the specific tumor antigen

Deploy



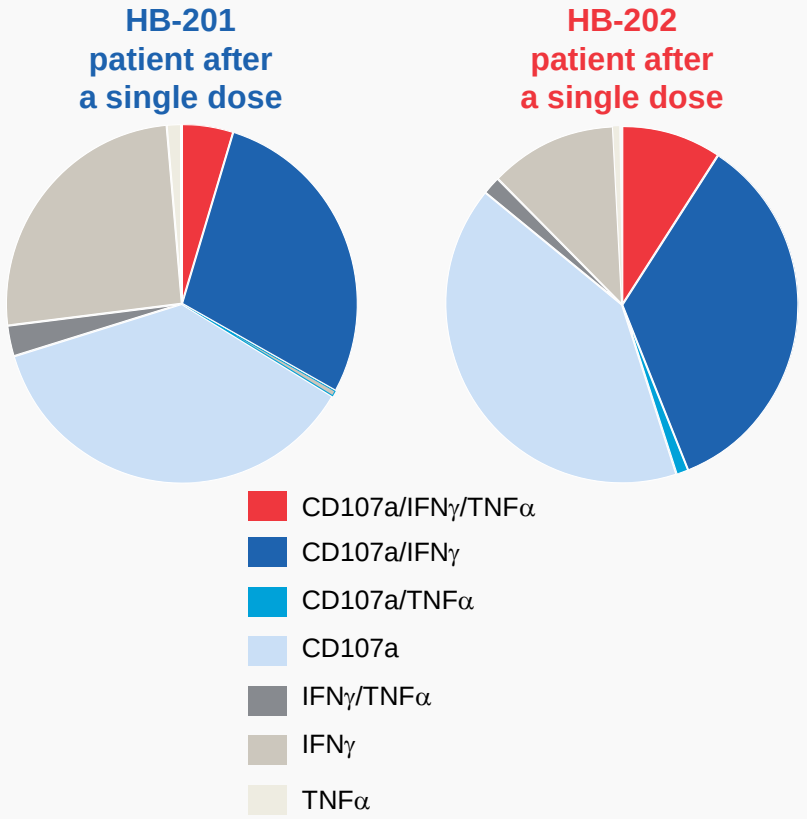
These specialized and armed soldiers **travel to the tumor** site and interact with and kill their enemy targets with minimal collateral damage

APC, antigen presenting cell.

Question #1: How are CD8+ T cells assessed for their killing potential?

How “fit” are the T cells for killing?

- Cytokine positivity is the main indicator of killing potential of T cells
- Key markers include:
  - Interferon gamma (IFNγ)
  - Tumor necrosis factor (TNFα)
  - Lysosomal-associated membrane protein-1 (CD107a)
- Polyfunctionality: The more markers (e.g., cytokines, cytotoxic markers) for which a T cell is positive, the more potent it is for killing

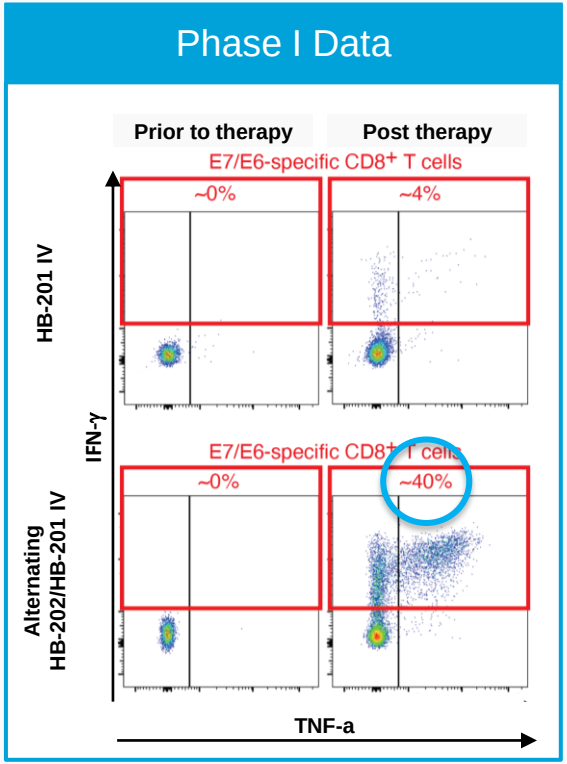


Data as of February 17, 2021. Katchar et al, AACR 2021 Late-Breaker.

Question #2: How many tumor-specific killer (CD8⁺) T cells must be recruited?

How big should the killer (CD8⁺) T cell army be?

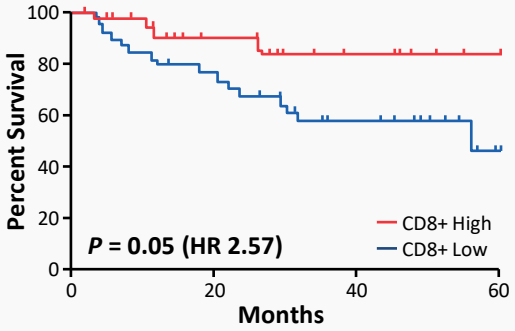
- The size is usually expressed as percentage and represents the portion of the CD8⁺ T Cell population which is specific for the cancer-antigen of interest
- While there is no established threshold, a level of 3-5% is a strong indicator of response
- The truest way to assess T Cell levels in blood is to measure directly, without prior *ex vivo* expansion



Please visit Hookipapharma.com to view an KOL roundtable video with additional thought leader perspectives on these questions.

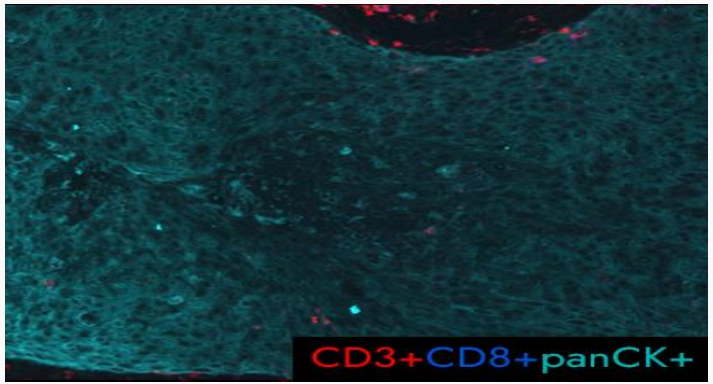
Question #3: How do we assess if T cells in the blood get into the tumor?

Presence of high levels of CD8$^+$ T cells in the tumor (tumor-infiltrating lymphocytes or TILs) predict longer survival in HPV$^+$ head and neck cancer

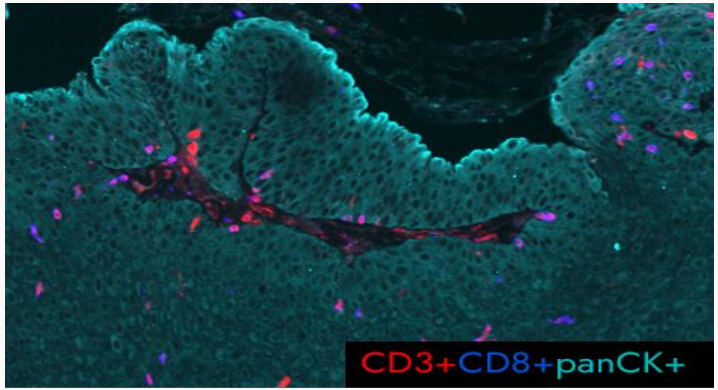


de Ruiter EJ, et al. Oncoimmunology. 2017;6(11):e1356148.

Pre-treatment



Post-treatment



PanCK$^+$ is the marker used to indicate the tumor tissue.
CD3$^+$ is the general T cell marker.

Putting Hookipa's arenaviral HB-200 into historical perspective of active immunization therapies

HB-200, as intravenous monotherapy, delivers unprecedented T cell immunogenicity and, for the first time, leads to clinical responses in advanced/metastatic cancers

- Significant and historically unprecedented immunogenicity
- Tumor shrinkage in advanced cancer from systemically administered monotherapy immunization has not been demonstrated previously
- Connection between immunogenicity and efficacy
  - Arenaviral non-lytic mechanism of action is that $CD8^+$ T cells will drive efficacy
  - Active immunization in advanced cancers could work *IF* sufficient levels of tumor antigen-specific $CD8^+$ T cells are generated
  - Hookipa's data, though early, support the causal relationship between immunogenicity and efficacy

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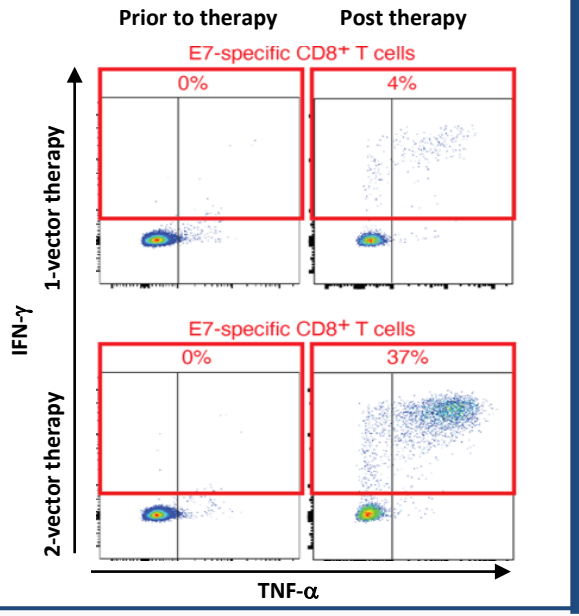
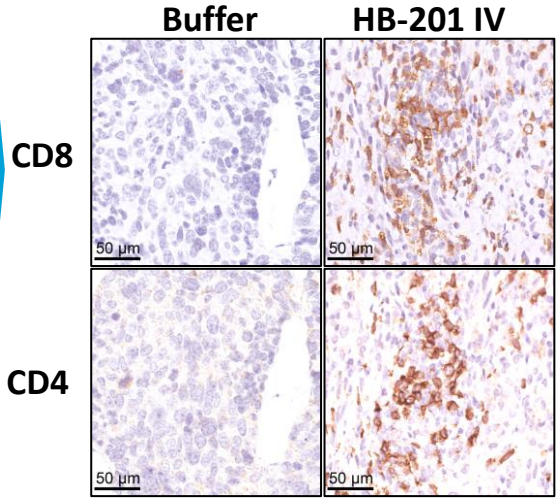
T cells induced by HB-200 enter the tumor and kill tumor cells in preclinical models

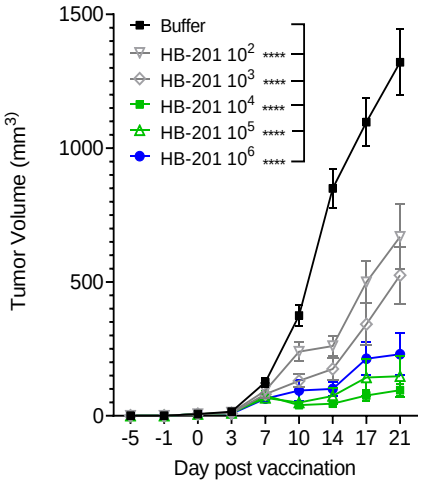
Very high levels of antigen-specific CD8+ T cells in blood



CD8+ T cells enter tumor[1]



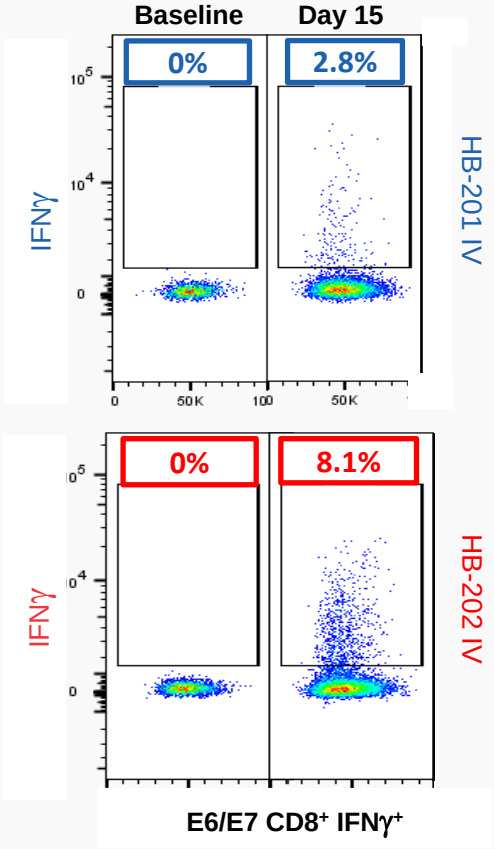
Tumor killing and improved survival[2]



[1]*Tumor infiltrating lymphocytes in TC-1 model (data on file).* [2]*Schmidt S, et al. Oncoimmunol 2020; 9:1809960.*

In patients, a single dose of HB-201 OR HB-202 drives robust T cell induction up to 8% of antigen specific IFNγ⁺ CD8⁺ T cells

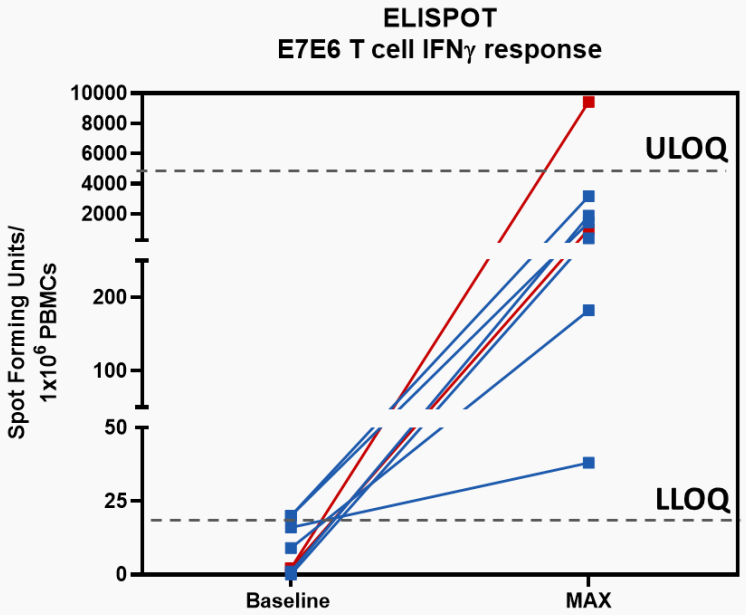
- Direct ELISpot measurements (no *ex vivo* expansion):
  - **250-fold** (multiple) increase in antigen-specific T cells
- Robust T cell levels[1]:
  - **3%** antigen-specific CD8⁺ T cells (HB-201)
  - **8%** antigen-specific CD8⁺ T cells (HB-202)



[1]*Intracellular cytokine staining (ICS) followed by flow cytometry.*
Data as of February 17, 2021. Katchar et al, AACR 2021 Late-Breaker.

Multiple dose treatment with HB-202/HB-201 monotherapy is highly immunogenic

Robust E7E6-Specific T cell IFNγ Response



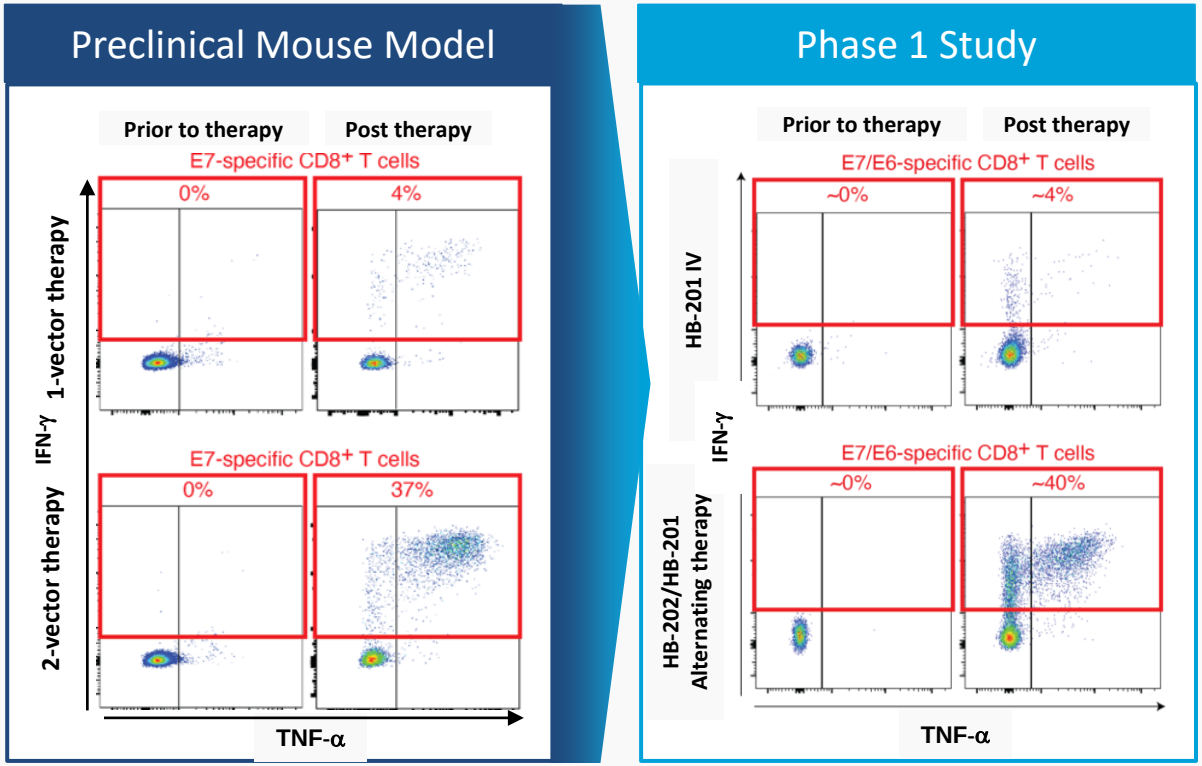
ULOQ: Upper Limit Of Quantification
LLOQ: Lower Limit of Quantification
ELISpot: Enzyme-linked immune absorbent spot

Key Points:

- All patients show increased $CD8^+$ T cell levels
- Unprecedented levels of circulating $HPV16^+$ E7/E6-specific $CD8^+$ T cells, 6% average and up to 40%
- Fast response: High levels of $CD8^+$ T cells achieved within 2 weeks of initial dose

Expansion of E7/E6-specific CD8⁺ T cells in patients mirrors that observed in mouse models

- Induction of a substantial T cell response, with up to **40% of E7/E6-specific circulating CD8⁺ T cells**
- These activated cells are producing TNFα and/or IFNγ



Adapted from Bonilla et al Cell Rep Med 2021

Early clinical data confirm the arenavirus mode-of-action: Driving unprecedented antigen-specific CD8$^+$ T cell levels

Key Points:

- ✓ All patients show increased CD8$^+$ T cell levels
- ✓ Unprecedented levels of circulating HPV16$^+$ E7/E6-specific CD8$^+$ T cells (up to 40%)
- ✓ Patient T cell data mirrors mouse model data which translated into broad tumor control

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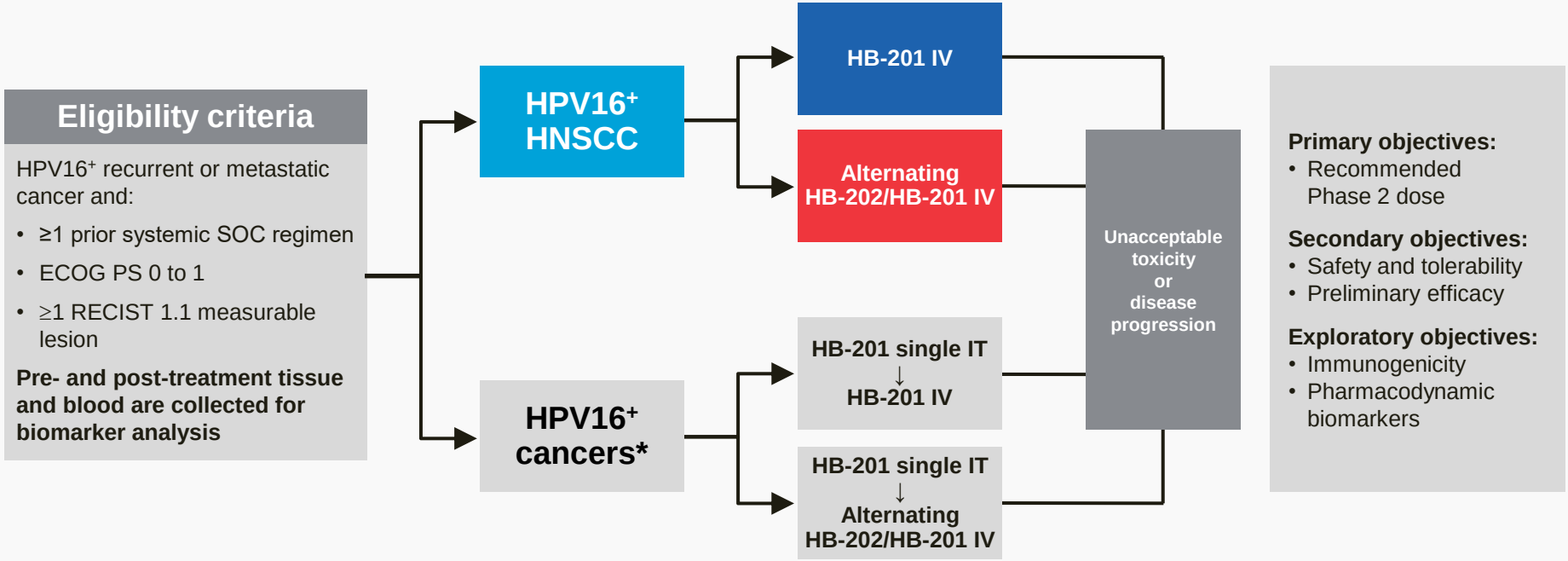
Ongoing Phase 1 study (NCT04180215) in patients with HPV16⁺ cancers is evaluating multiple HB-201 and HB-202/HB-201 doses and regimens



3+3 dose escalation with additional biomarker and schedule finding cohorts. Dosing schedules assessed include: Q3w–Q6w and Q2w.
Dose levels explored to-date: HB-201 IV: Dose level one: 5x10^5 and Dose level two: 5x10^6 RCV FFU.
Alternating HB-202/HB201 IV: Dose level one: HB-202=1x10^6 and HB201=5x10^6 RCV FFU. Dose level two: HB202=1x10^7 and HB201=5x10^6 RCV FFU.
**HPV16⁺ cancers with accessible lesion amenable for biopsy and IT administration.*
Tumor tissue and blood samples (including serum and plasma) were collected during the study unless agreed otherwise between the Sponsor and the Investigator.
ECOG, Eastern Cooperative Oncology Group; HNSCC, head and neck squamous cell carcinoma; HPV, human papillomavirus; IT, intratumoral; IV, intravenous; PS, performance status; RECIST, Response Evaluation Criteria in Solid Tumors; SOC, standard of care.

ASCO data set includes 38 patients dosed, with 18 patients still on treatment

As of Mar 31, 2021 data cut-off:

38 patients dosed

18 patients on treatment

20 discontinuations:
- 17 progressions
- 2 consent withdrawals
- 1 death (progression)

HB-201 IV	Alternating HB-202/HB-201 IV	Other regimens
Every 3 weeks	*Every 3 weeks*	*1. Initial Intra-tumoral dose 2. Every 2 weeks*
14 patients treated	**8 patients treated**	**16 patients treated**
11 patients with ≥1 post-dose efficacy scan	**4 patients with ≥1 post-dose efficacy scan**	

Most patients had head and neck cancers and were heavily pretreated with a checkpoint inhibitor and/or platinum chemotherapy

Cohorts	Total	HNSCC	Non-HNSCC
Number of HPV16+ patients	**38**	32	6
Primary Site Oropharynx Other, n details	**29 (76%)** **9 (24%)**	29 (91%) 1 Nasal 1 Nasopharynx 1 Unknown	0 (0%) 3 Cervical 1 Vaginal 1 Anal 1 Penile
Age, years, median (range)	**62 (30-86)**	64 (30-86)	54 (49-66)
Gender, male	**30 (79%)**	29 (91%)	1 (17%)
Race, White	**34 (90%)**	30 (94%)	4 (80%)
ECOG PS 1	**23 (61%)**	19 (59%)	4 (67%)
Prior lines of therapy, median (range)	**3 (1-10)**	3 (1-10)	3 (2-3)
Prior CPI use	**31 (82%)**	28 (88%)	3 (50%)
Prior platinum use	**34 (90%)**	29 (91%)	5 (83%)
Prior cetuximab use	**18 (47%)**	18 (56%)	0 (0%)
Distant metastasis at baseline	**30 (79%)**	24 (75%)	6 (100%)

Patients were generally heavily pretreated

82% previously received a CPI

79% had baseline distant metastasis

Data cut-off: Mar 31, 2021

Data shown as n (%) unless otherwise indicated. All patients who did not have a baseline ECOG of 1, had an ECOG of 0.
CPI, checkpoint inhibitor; ECOG, Eastern Cooperative Oncology Group; HNSCC, head and neck squamous cell carcinoma; HPV, human papillomavirus.

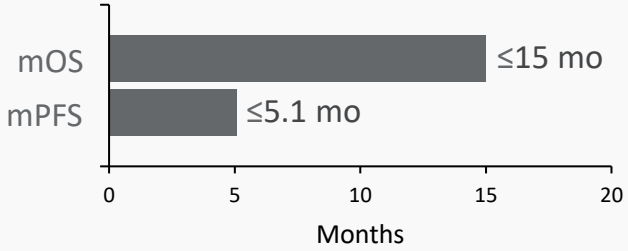
As cancer progresses, treatment outcomes decline with each line of therapy, so 2nd line outcomes are worse than 1st line and 3rd line are worse than 2nd line

Outcomes for 1st and 2nd Line Standard of Care Treatments[1,2]

Increasing disease burden and decreasing QOL

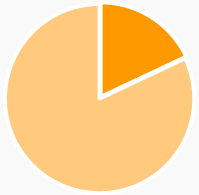
1st Line[3,4]

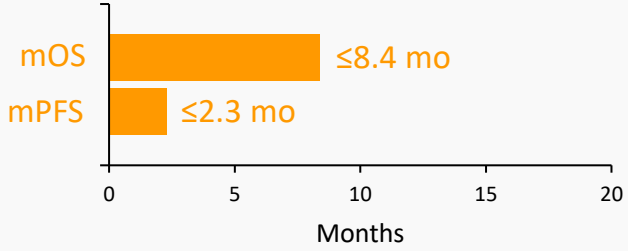
Response Rate ≤36%



2nd Line[4,5]

Response Rate ≤18%



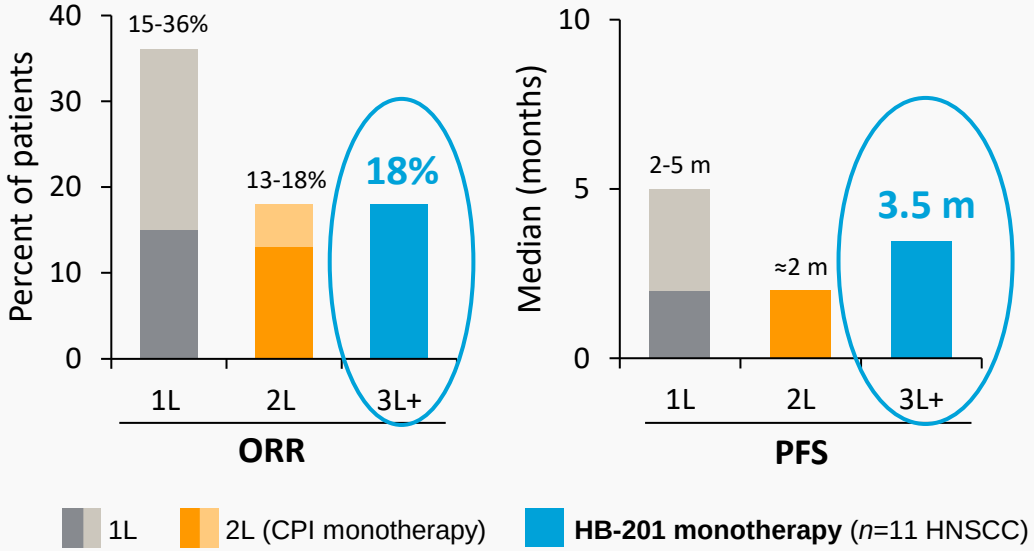


As **metastatic disease progresses** through lines of therapy

- Expectations of **treatment outcomes are reduced** as resistance to existing therapies develops
- **Patients are sicker** and may be unable to tolerate significant toxicities common with traditional cancer therapies

[1]Keytruda (pembrolizumab). Prescribing Information. [2]Pai S, et al. J ImmunoTherapy Cancer. 2019;7:96. [3]CT + CPI or cetuximab; CPI alone in PD-L1+ tumors. [4]Data from phase 3 trials (KEYNOTE-048, KEYNOTE-040, and CheckMate 141). [5]PD-1i or CT monotherapy.
QOL, quality of life; L, line; mOS, median overall survival; mPFS, median progression-free survival; ORR, overall response rate; PD-1i, programmed death 1 inhibitor; PD-L1, programmed death ligand 1; SOC, standard of care.

Single agent HB-201 data in L3+ head & neck cancer patients looks better than L2 progression-free survival and similar to L2 response rate

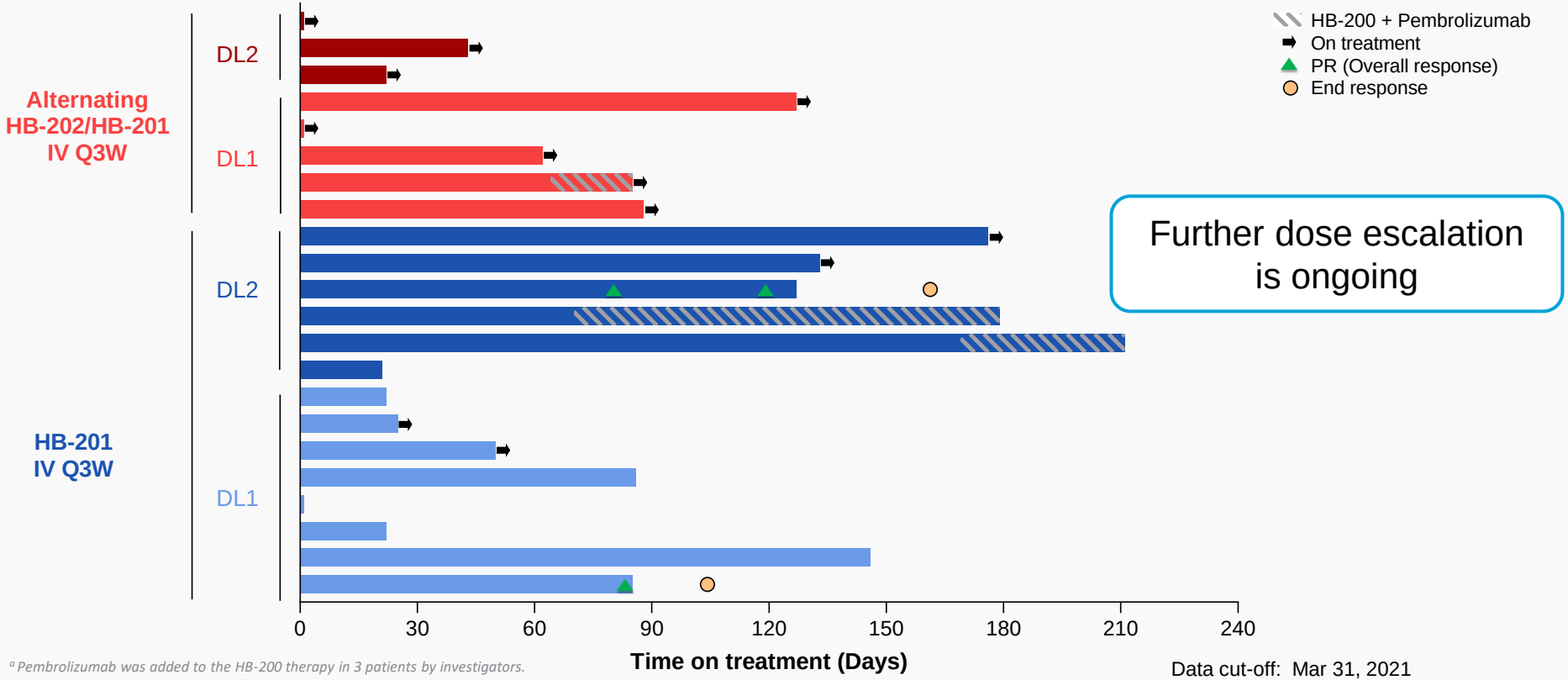


HB-200 L3+ Monotherapy data:

- **Progression Free Survival**
  - BETTER than 2L standard of care
- **Overall response rate**
  - Comparable to 2L PD1 inhibitor data
- **Disease control rates**
  - 73% for HB 201 IV Q3W
  - 100% for HB202/HB201 IV Q3W

CPI, checkpoint inhibitor; CT, chemotherapy; L, line of therapy; ORR, overall response rate; PFS, progression-free survival; SOC, standard of care.
https://www.merck.com/product/usa/pi_circulars/k/keytruda/keytruda_pi.pdf
https://packageinserts.bms.com/pi/pi_opdivo.pdf

Promising duration of treatment, with many patients still ongoing



Further dose escalation is ongoing

Data cut-off: Mar 31, 2021

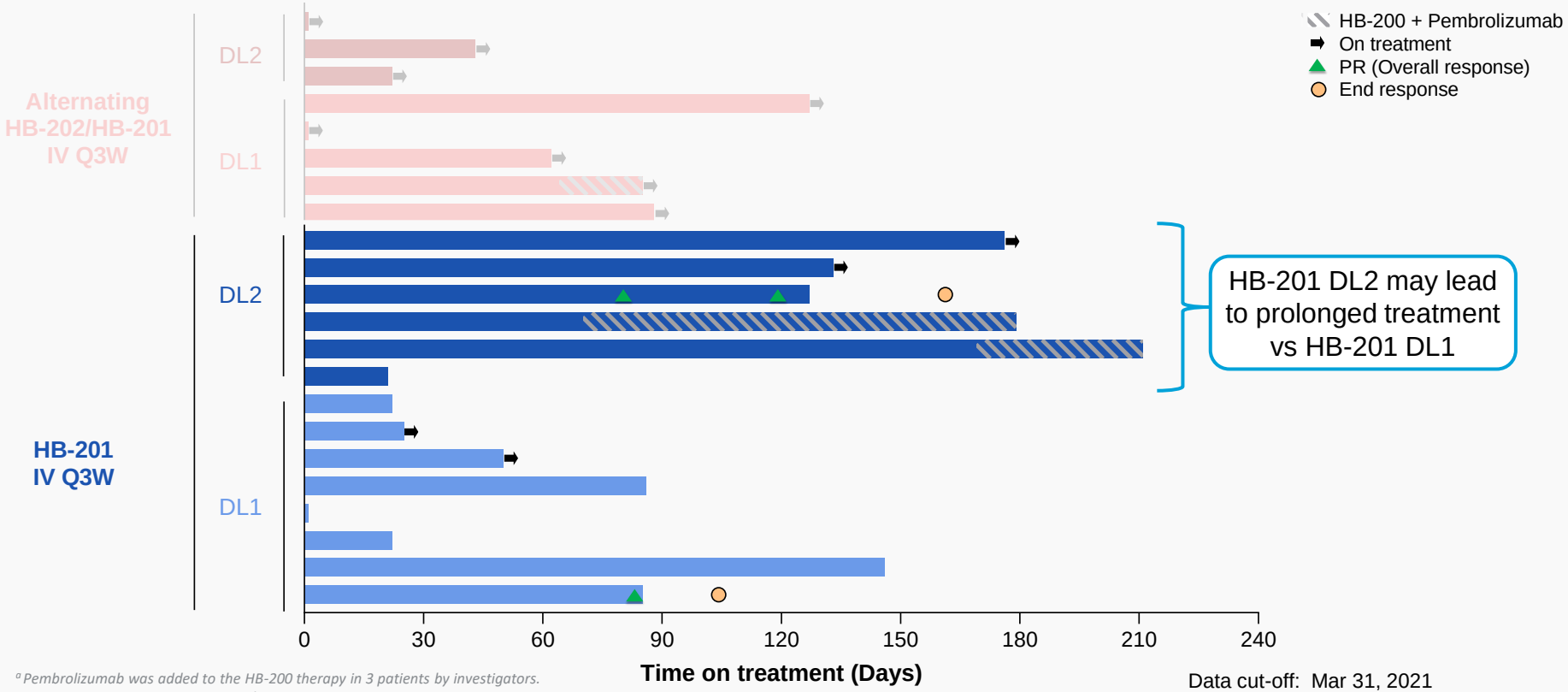
[a] *Pembrolizumab was added to the HB-200 therapy in 3 patients by investigators.*

Time on treatment = Last treatment/death date – first dose date + 1.

EDC data was used for some patients due to missing/incorrect data entry on TLF as of the data transfer date. Data shown is of patients receiving IV therapy only, every 3 weeks.

DL, dose level; EDC, electronic data capture; HNSCC, head and neck squamous cell carcinoma; IT, intratumoral; IV, intravenous; PR, partial response.

Dose level response trend emerging for HB-201



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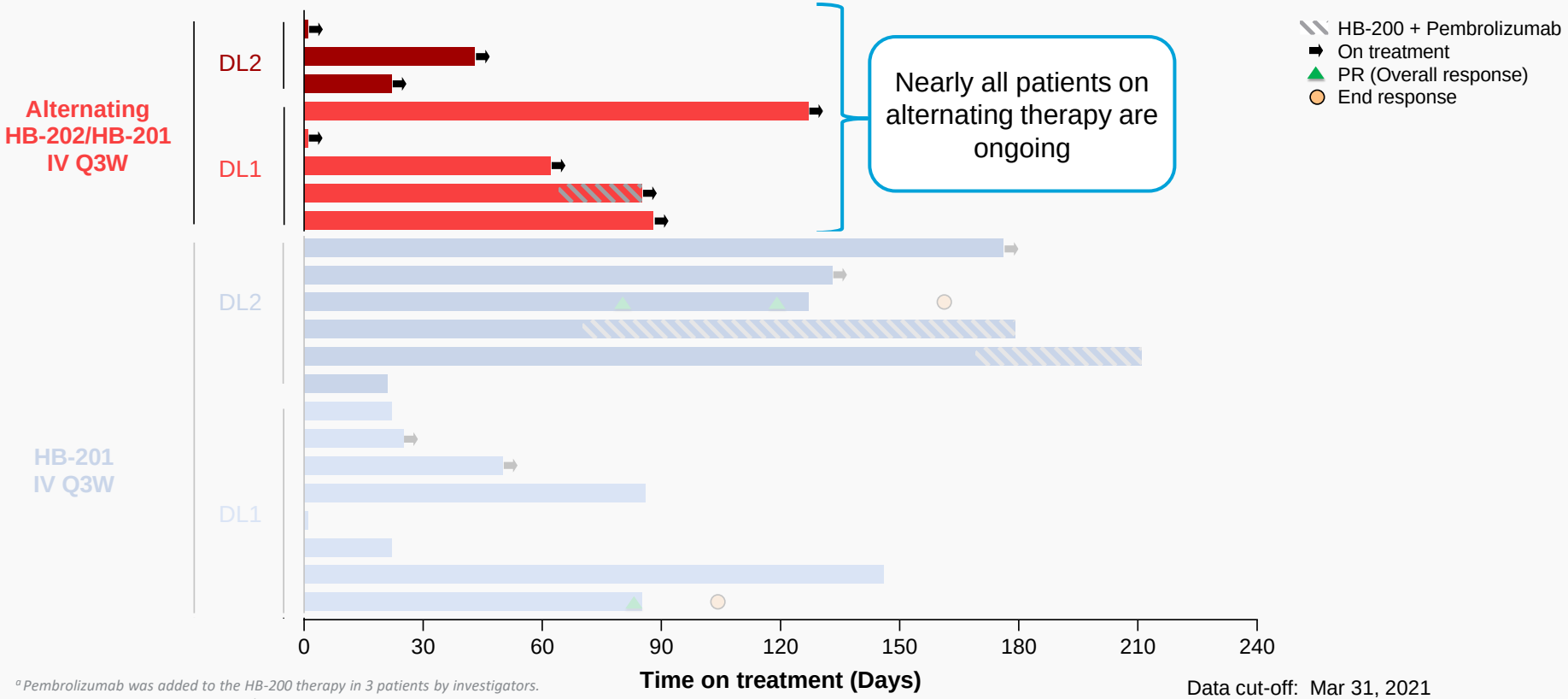
Nearly all patients on the alternating HB-202/HB-201 therapy are ongoing



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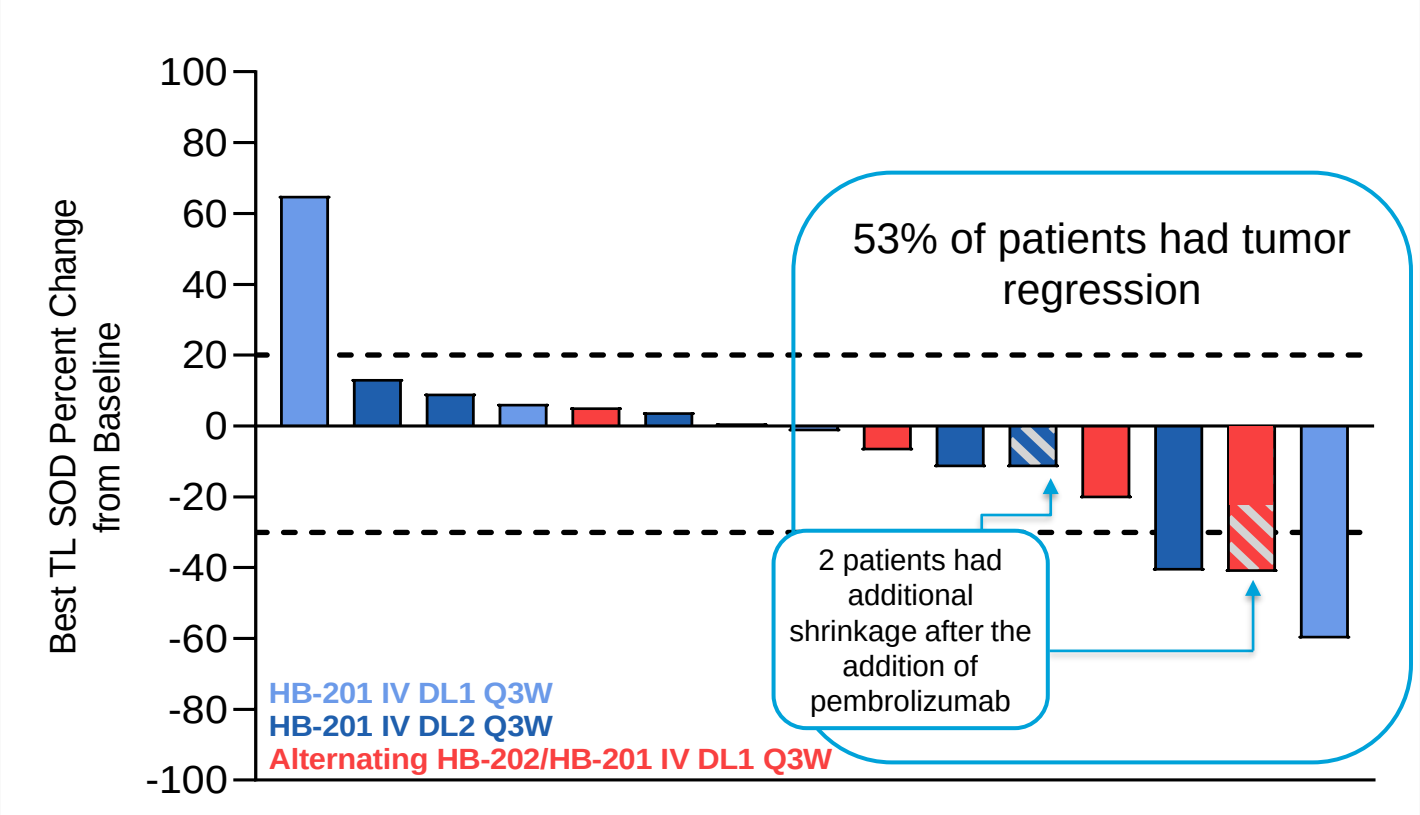
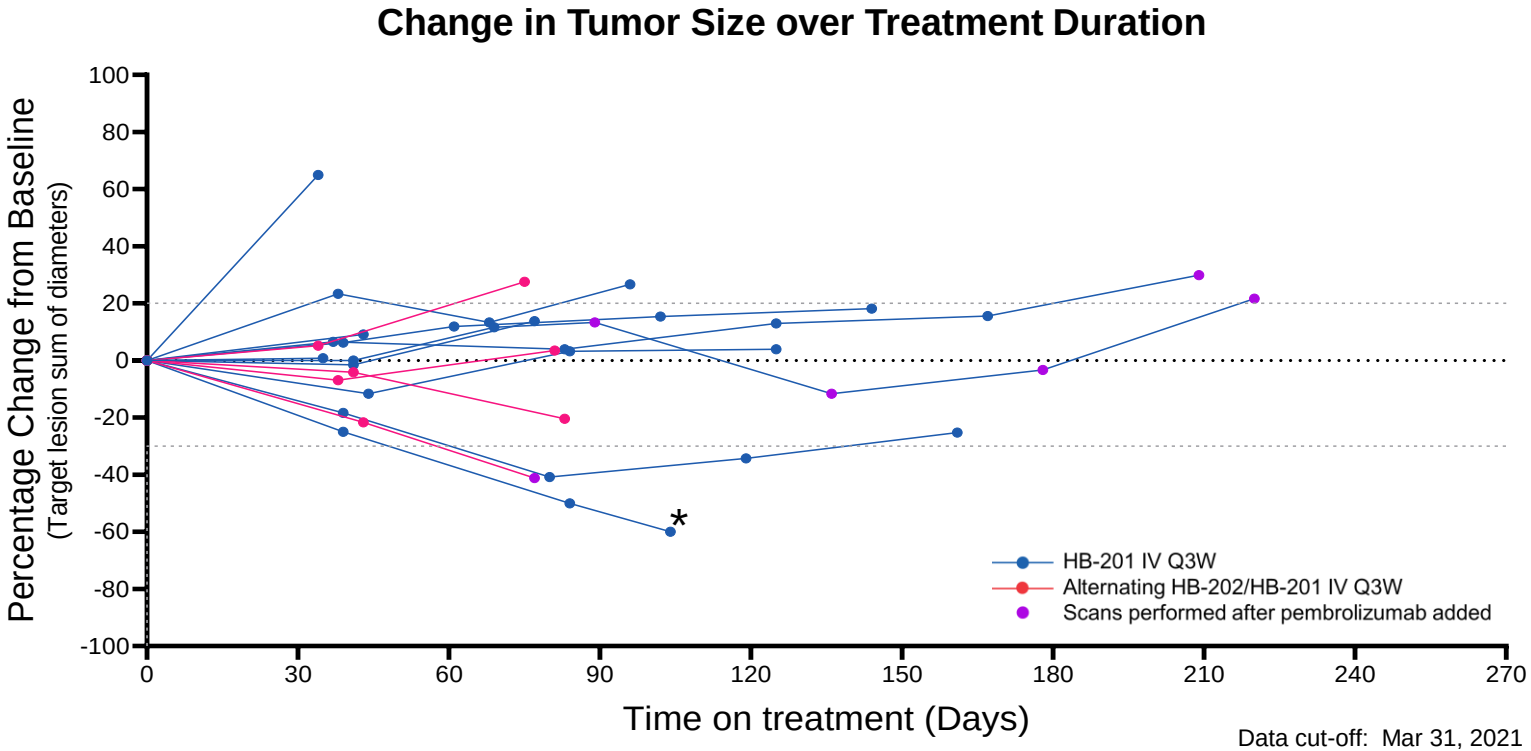
Encouraging monotherapy data in extensively pre-treated patients



Data cut-off: Mar 31, 2021

TL SOD: Target lesion sum of diameters.
Striped areas indicate decrease in target lesion change after pembrolizumab was added to therapy. IV, intravenous.

Two partial responses in HB-200 monotherapy group and third partial response after addition of pembrolizumab



*60% decrease was comprised of a lymph node <1 cm and, therefore an unconfirmed complete response of the target lesion

Emerging Data in Head & Neck Patients Progressed on Standard of Care, Including Checkpoint Inhibitors, Better than Earlier Line Patients

	HB-201 IV DL1&DL2 Q3W	HB-201/HB-202 IV DL1 Q3W
N, evaluable (≥1 scan)	11	4
Median time on treatment (days)	127	87
ORR, n (%)	2 (18.2)	0 (0.0)
PR, n (%)*	2 (18.2)	0 (0.0)
SD, n (%)	6 (54.5)	4 (100.0)
SD ≥16 wks	4 (36.4)	0 (0.0)
PD, n (%)	3 (27.3)	0 (0.0)
DCR, n (%)	8 (72.7)	4 (100.0)
PFS, median (mo)	3.45	3.58

*PR include 1 confirmed PR and 1 unconfirmed PR.

Data cut-off: Mar 31, 2021

EDC data was used for some patients due to missing/incorrect data entry on TLF as of the data transfer date.
DCR, disease control rate; DL, dose level; EDC, electronic data capture; HNSCC, head and neck squamous cell carcinoma; ORR, objective response rate; PFS, progression-free survival; NE, non-evaluable; PR, partial response; TL, target lesion; Q3W, every 3 weeks; Q6W; every 6 weeks; RECIST, Response Evaluation Criteria in Solid Tumors; SOD, sum of diameters; SD, stable disease; uCR, unconfirmed CR.

Benign safety profile, easy to combine with other therapies

All groups all cohorts (N = 38)	Treatment related	Treatment emergent
Any event	**20 (53%)**	28 (74%)
Grade ≥ 3	**0**	12 (32%)
Serious	**0**	7 (18%)
Leading to dose reduction	**0**	0
Leading to dose interruption	**0**	1 (3%)[a]
Leading to discontinuation	**0**	0
Death	**0**	1 (3%)[b]

The most common TEAEs (≥15%) were fatigue (32%), pyrexia (26%), nausea (18%), and hypertension (16%)

Key Take-aways:

- **Favorable safety** especially in pre-treated patients
- **Lack of overlap** with prototypical PD(L)1 inhibitor side effect profile
- **De-risked combinations** with checkpoint inhibitors and other relevant therapeutics

Data cut-off: Mar 31, 2021

[a]Treatment was interrupted in one patient due to bronchopulmonary hemorrhage (which resolved) and lung infection.
[b]One patient succumbed to hemorrhagic shock; post pulmonary hemorrhage attributed to progression of disease.
Median duration of treatment was 1.6 months (0–6.9 months) as defined as the lesser value of: (date of last dose or death – first date of first dose of treatment +1)/30.4375.
AE, adverse event; DLT, dose limiting toxicities; TEAE, treatment-emergent AE

Early signs that HB-200 is effective in controlling cancer as a monotherapy in the post checkpoint inhibitor setting

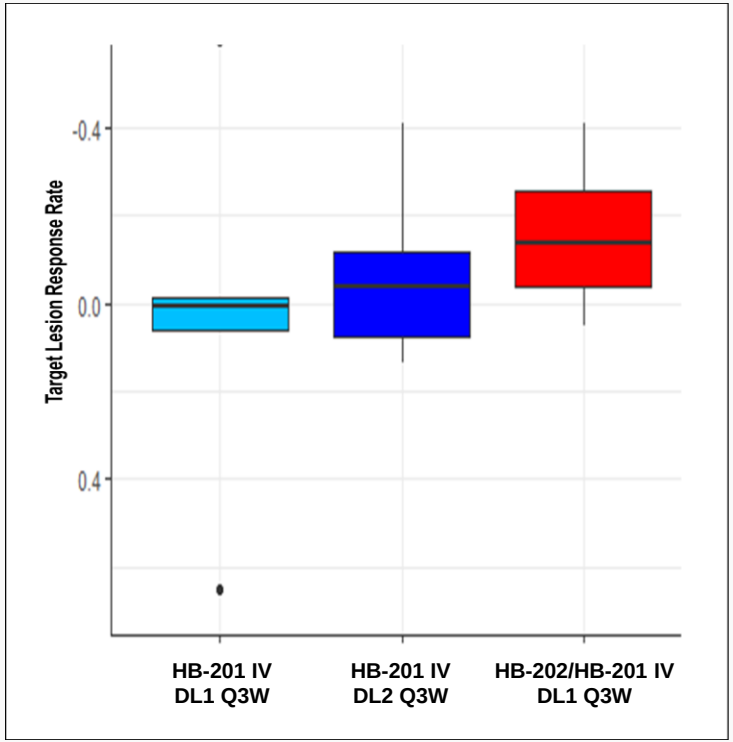
Key Points:

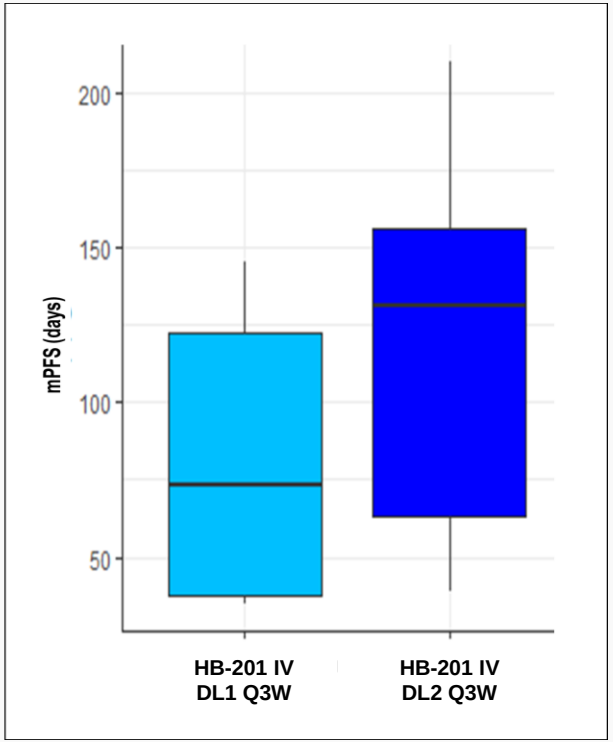
- ✓ Monotherapy HB-200, unlike other monotherapy active immunization therapies, provides clinical responses in 3rd+ line post CPI patients
- ✓ Two Objective Responses and a Disease Control Rate of 80% (12 of 15) in advanced patients
- ✓ Benign safety profile, supportive of combinations with many different therapies

Agenda

1. Immunogenicity Data: Perspectives and Interpretation (D Zamarin)
2. Clinical Data Confirm the Arenavirus Mode of Action: Driving Unprecedented Tumor Antigen-Specific $CD8^+$ T Cell Levels (I Matushansky)
3. Clinical Efficacy as an IV Monotherapy in the Post-CPI Setting (I Matushansky)
4. **Early Data Suggestive of a Relationship Between the Mode of Action and Biological Activity (I Matushansky)**
5. The Bright Future for Hookipa's Arenavirus Platform in Oncology (J Aldag)
6. Q & A

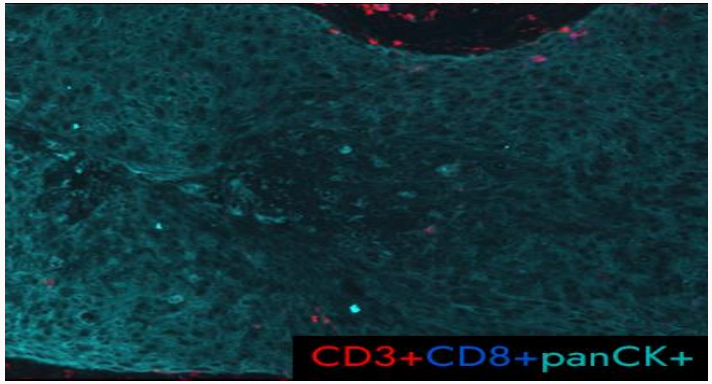
Response rates and progression free survival favor higher doses over lower doses and favor dual-alternating over single-vector therapy



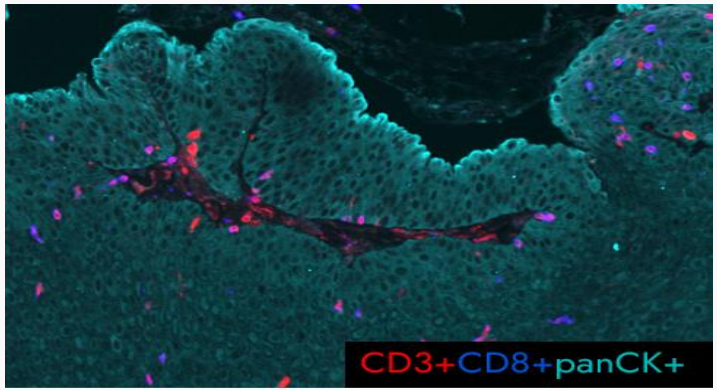


Biopsy data confirm HB-200 increases CD8⁺ T cells in tumor

Pre-treatment



Post-treatment



panCK⁺ is the marker used to indicate the tumor tissue.
CD3⁺ is the general T cell marker.

After HB-200 Therapy:

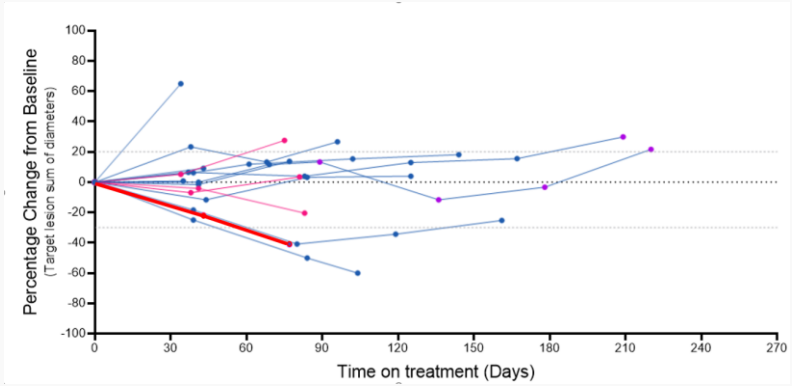
- CD8⁺ T cells penetrate tumor
- Tumors have increased levels of CD8⁺ T cells, consistent with the changes seen in the blood

Patient who received 2 doses of HB-201/HB-202: 40% tumor antigen-specific CD8⁺ T cell induction and tumor regression in soft tissue

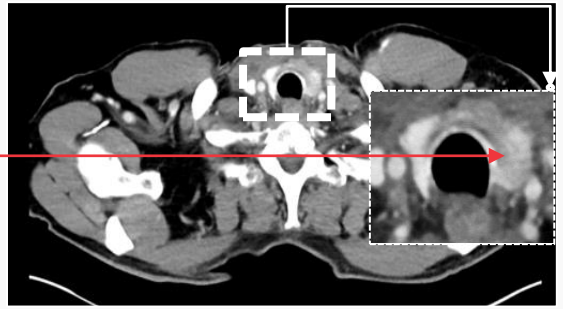
Prior treatments: 3 prior lines of therapy
Radiation therapy ► Cisplatin ► Monalizumab/durvalumab/cetuximab

Results: Progression in peri-thyroid soft-tissue metastases

Status: Started HB202/HB201 – with 40% shrinkage of target lesion

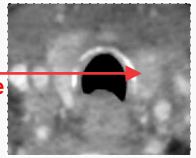


Baseline



Scan 1: 6 weeks
Post 1 cycle:
HB-201/HB-202 monotherapy
-20% shrinkage

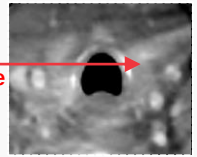
-20% shrinkage

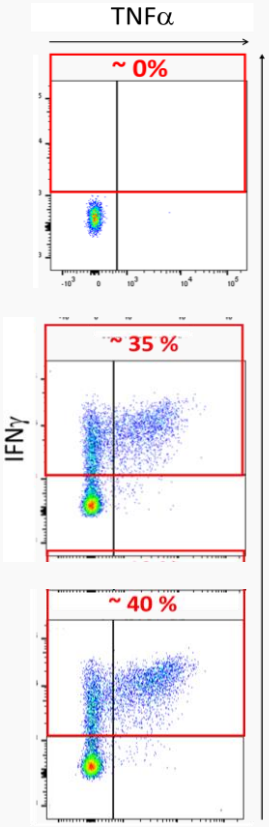


Pembrolizumab added

Scan 2: 12 weeks
Post 2 cycles:
HB-201/HB-202 + 1 cycle of Pembro
-40% shrinkage

-40% shrinkage





Early data suggest a relationship between T cells and clinical efficacy

Key Points:

- ✓ Efficacy measures improve with higher dose and with dual-alternating single vector therapy
- ✓ HB-200 therapy causes similar increases of $CD8^+$ T cells in blood and tissue biopsies
- ✓ In tissue, early evidence that HB-200 decreases immune suppression in tumor microenvironment

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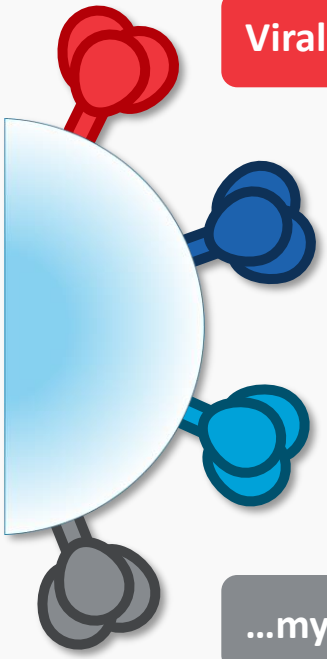
HB-200 clinical development program in HPV16+ cancers to initiate Phase 2 studies in early 2022 (potentially registration-enabling)

3 avenues to obtain accelerated approvals in 3 HPV16+ indications		
1st line advanced/metastatic head & neck cancer:	**2nd line advanced/metastatic head & neck cancer:**	**2nd line advanced/metastatic anal cancer:**
Randomized Phase 2 in combination with a PD1 inhibitor	Phase 2 expansion cohort of ongoing study with HB200 monotherapy	Phase 2 expansion cohort of ongoing study in combination with a PD1 inhibitor

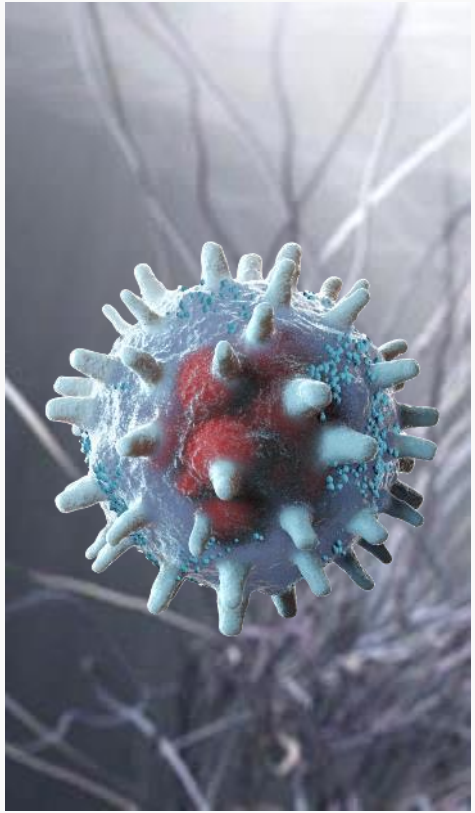
“Plug & Play” arenavirus technology: Engineered to drive robust, targeted and durable T cell responses against a broad range of cancers



Viral antigens (e.g., E7/E6)		HPV16⁺ tumors
Self antigens	PSA, PAP, PSMA	Prostate cancer
	AFP	Hepatocellular carcinoma
Mutated oncogenes (e.g., KRAS)		Colorectal cancer Lung cancer Pancreatic cancer, among others
…myriad options for the future		

AFP, Alpha-fetoprotein; PAP, prostatic acid phosphatase; PSA, prostate specific antigen; PSMA, prostate-specific membrane antigen.

Hookipa's expanding oncology pipeline: Value creating milestones ahead



1. Next comprehensive data update no later than 4Q 2021
2. RP2D defined in 4Q 2021
3. Start of Phase 2 HB-200 2nd Line Expansion Cohorts: 1Q 2022
4. Start of checkpoint inhibitor (CPI) combination study in 1st Line HNSCC: 1H 2022
5. HB-300 Prostate cancer IND: 3Q 2022
6. At least one additional IND *per annum* starting 2023

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