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): January 13, 2020

UNUM THERAPEUTICS INC.
(Exact name of registrant as specified in its charter)

Delaware	**001-38443**	**46-5308248**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(I.R.S. Employer Identification No.)**

200 Cambridge Park Drive, Suite 3100
Cambridge, Massachusetts **02140**
(Address of principal executive offices) **(Zip Code)**

Registrant's telephone number, including area code (617) 945-5576

Not Applicable
(Former name or former address, if changed since last report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the 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))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common stock, $0.001 Par Value	**UMRX**	**The Nasdaq Global Select Market**

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

Emerging growth company ☒

If an emerging growth company, indicate by check mark 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.

Unum Therapeutics Inc. (the "Company") is furnishing a corporate presentation, attached as Exhibit 99.1 to this Current Report on Form 8-K, which the Company intends to use from time to time in meetings with investors and others beginning on January 13, 2020. The corporate presentation will also be available in the investor relations section of the Company's website at http://unumrx.com.

The information in this Item 7.01 and Exhibit 99.1 attached hereto shall not be deemed "filed" for purposes of Section 18 of the Securities and Exchange Act of 1934, as amended, or otherwise subject to the liabilities of that section, nor shall they be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, except as expressly set forth by specific reference in such filing.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Unum Therapeutics Inc. corporate presentation.

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.

Date: January 13, 2020

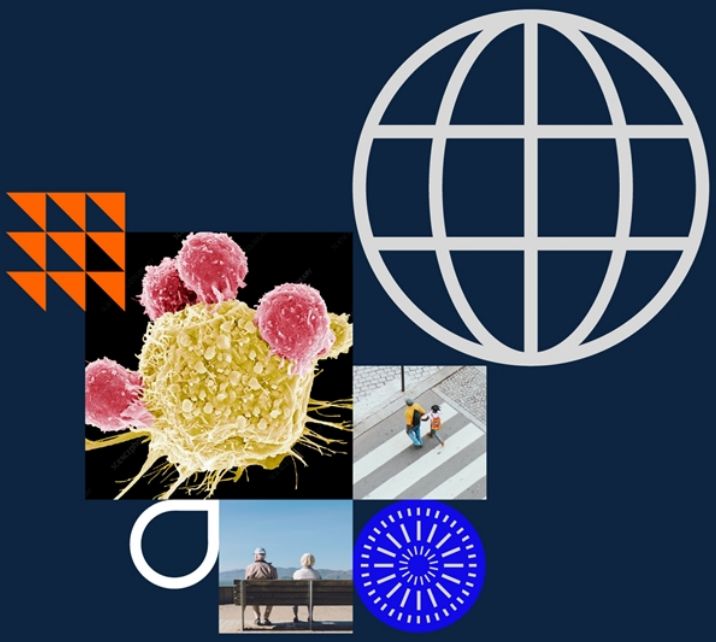
UNUM THERAPEUTICS INC.

By: /s/ Charles Wilson
Charles Wilson, Ph.D.
Chief Executive Officer and President

Exhibit 99.1

UNUM THERAPEUTICS

Delivering novel cell therapies to cure cancer



CORPORATE PRESENTATION
JANUARY 2020

Forward-Looking Statements and Risk Factors

This presentation and the accompanying oral commentary contain forward-looking statements that involve risks, uncertainties and assumptions. If the risks or uncertainties ever materialize or the assumptions prove incorrect, our results may differ materially from those expressed or implied by such forward looking statements. All statements other than statements of historical fact could be deemed forward-looking, including, but not limited to, any statements of the plans, strategies, and objectives of management for future operations, including our clinical development and commercialization plans; any projections of financial information; any statement about historical results that may suggest trends for our business; any statement of expectation or belief regarding future events; potential markets or market size, technology developments, our clinical product pipeline, clinical data or the implications thereof, enforceability of our intellectual property rights, competitive strengths or our position within the industry; any statements regarding the anticipated benefits of our collaborations or other strategic transactions; and any statements of assumptions underlying any of the items mentioned.

These statement are based on estimates and information available to us at the time of this presentation and are not guarantees of future performance. Actual results could differ materially from our current expectations as a result of many risks and uncertainties, including but not limited to, risks associated with: the success, cost and timing of our product development activities and clinical trials; our ability to obtain regulatory approval for and to commercialize our product candidates; our ability to establish a commercially-viable manufacturing process and manufacturing infrastructure; regulatory requirements and regulatory developments; the effects of competition and technological advances; our dependence on third-party collaborators and other contractors in our research and development activities, including for the conduct of clinical trials and the manufacture of our product candidates; our ability to obtain, maintain, or protect intellectual property rights related to our product candidates; among others. For a further description of the risks and uncertainties that could cause actual results to differ from those expressed in these forward-looking statements, as well as risks relating to our business in general, see our periodic filings filed from time to time with the Securities and Exchange Commission. Unless as required by law, we assume no obligation and do not intend to update these forward-looking statements or to conform these statements to actual results or to changes in our expectations.

All of Unum Therapeutics ("Unum") product candidates are investigational product candidates and their safety and efficacy have not yet been established. Unum has not obtained marketing approval for any product, and there is no certainty that any marketing approvals will be obtained or as to the timelines on which they will be obtained.

Any data pertaining to Unum product candidates is interim data and may include investigator-reported interim data for which Unum has not yet independently reviewed the source data. The interim data may not be representative of the final results that may be obtained in the corresponding trials and results from earlier trials may not be representative of results obtained in later trial or pivotal trials.

Cancers are notorious for developing ways to suppress, evade and attack the body's natural defense systems, rendering many therapies ineffective.

Unum Therapeutics is focused on developing cures for patients with solid tumors through cell therapy approaches designed to improve the body's immune response to cancer.

Experienced Leadership Team

Unum's leadership possesses biotech and large pharma drug development experience and deep scientific expertise in cancer immunotherapy

CHUCK WILSON, Ph.D.
President and CEO

SETH ETTENBERG, Ph.D.
Chief Scientific Officer

GEOFFREY HODGE
Chief Technical Officer

MATTHEW OSBORNE
Chief Financial Officer

JESSICA SACHS, M.D.
Chief Medical Officer

MERT AKTAR
VP, Corporate &
Business Development

HEATHER HUET, Ph.D.
VP, Program Leadership &
Portfolio Management

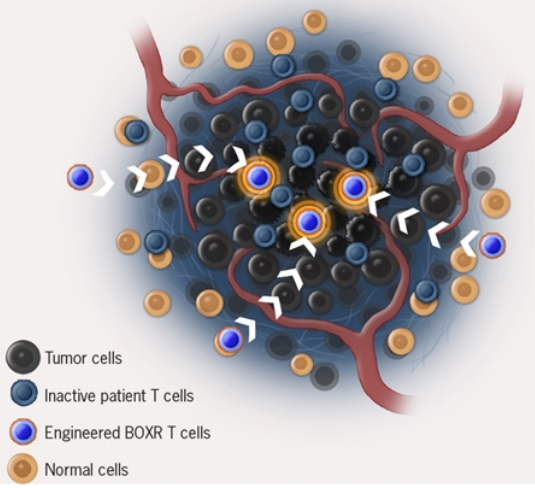
SARA SALTZMAN
VP, Regulatory Affairs

ERIN SCHELLHAMMER
VP, People

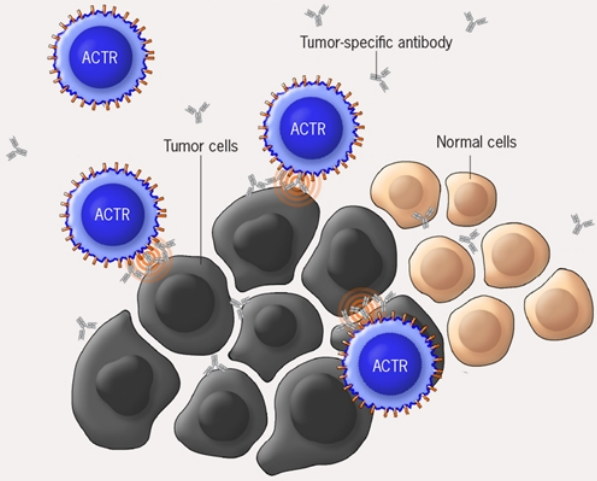
ALEXION
Biogen
Intellia THERAPEUTICS
genzyme
MILLENNIUM
NOVARTIS
Shire
Synageva
Takeda
Voyager THERAPEUTICS
Xcellerex

Novel Technologies Designed to Improve T Cell Functionality and Targeting

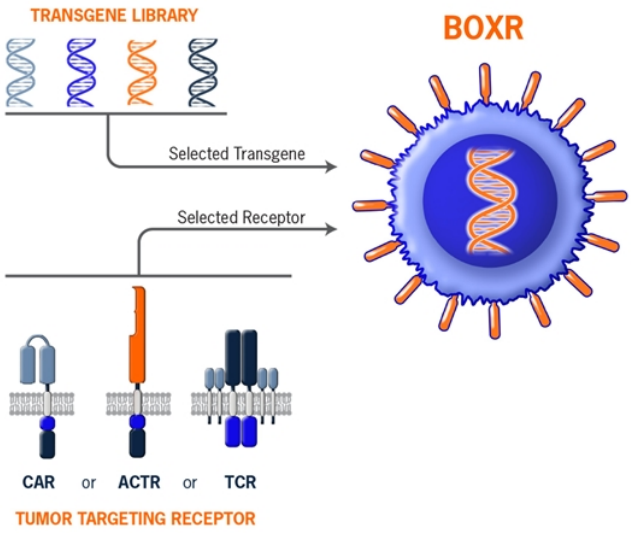
Bolt-On Chimeric Receptor (BOXR):
Overcoming Tumor Immunosuppression



Antibody-Coupled T Cell Receptor (ACTR):
Selective Tumor Targeting

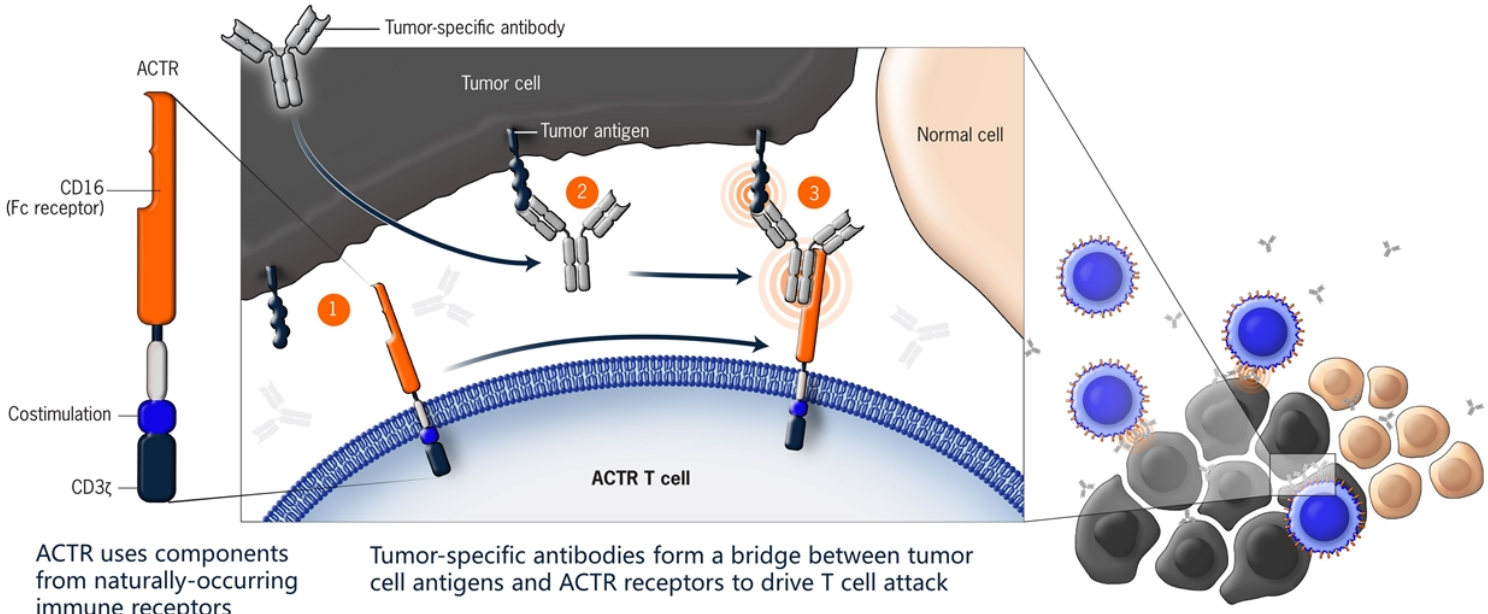


BOXR Technology: Overcoming the Solid Tumor Microenvironment (TME)



- BOXR program candidates are designed to overcome the suppressive effects of the TME, including:
  - Competition for metabolites
  - Exhaustion due to chronic stimulation
  - Resistance to immunosuppressive cell types
- Novel bolt-on transgenes coupled with different targeting receptors to potentially yield multiple product candidates

ACTR Technology: Next Generation Cell Therapy



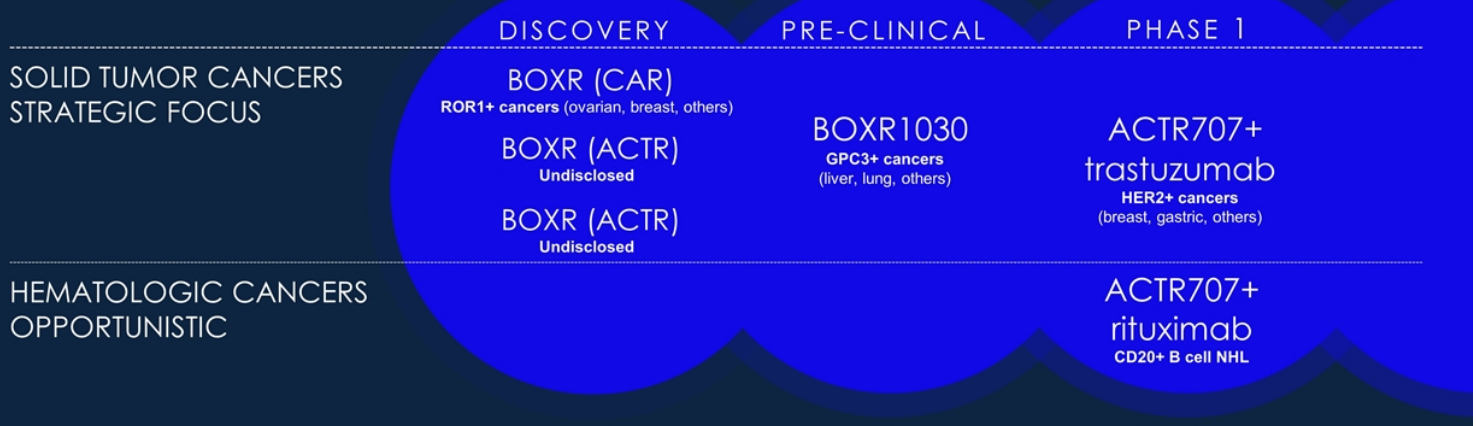
ACTR uses components from naturally-occurring immune receptors

Tumor-specific antibodies form a bridge between tumor cell antigens and ACTR receptors to drive T cell attack

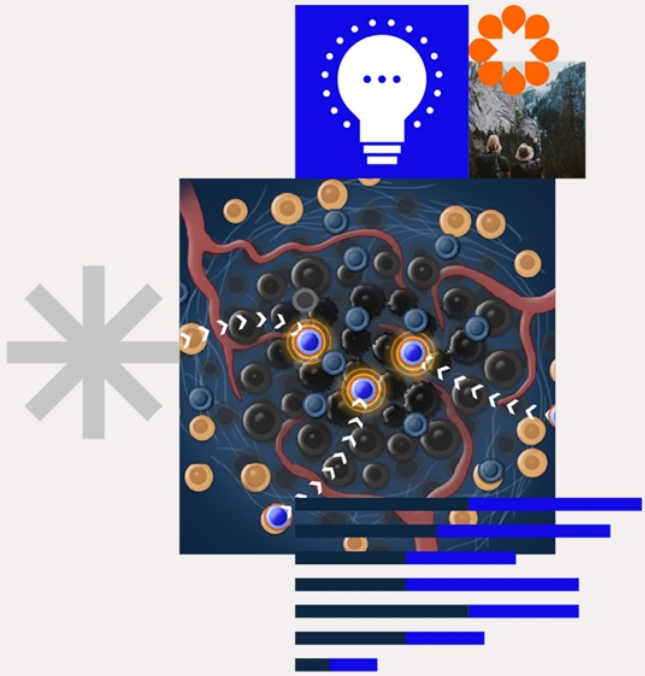
ACTR T cells drive selective tumor cell attack while sparing normal tissue

UNUM THERAPEUTICS

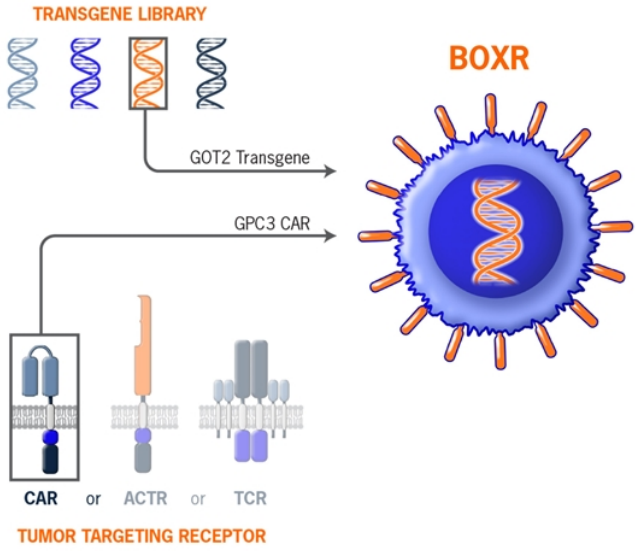
A Focused Plan to Treat More Cancers



BOXR



BOXR1030: GOT2 Transgene + GPC3 CAR



- Glutamic-oxaloacetic transaminase 2 (GOT2) is a critical mitochondrial enzyme for cell survival, proliferation and differentiation
- Glypican-3 (GPC3) is highly expressed in several solid tumor types; not in normal tissues
- BOXR1030 T cells have improved metabolism and function, yielding superior activity in xenograft animal models compared to parental CAR T[1]

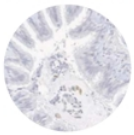
[1] Whiteman, KR et al. Co-expression of the Metabolic Enzyme GOT2 with a GPC3-Targeted CAR-T Overcomes Challenges of the Solid Tumor Microenvironment, Substantially Improving Therapeutic Efficacy in Solid Tumor Xenografts, SITC 2019

BOXR1030: Targeting GPC3+ Cancers

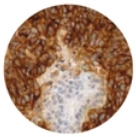
GPC3 a validated target; potential to improve upon current clinical programs

- GPC3 is highly expressed in several solid tumor types, not in normal tissues

LUNG (NORMAL)



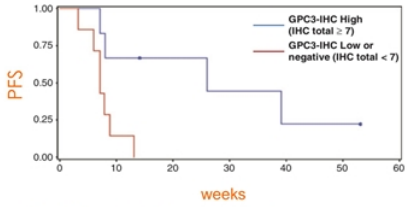
LUNG (SQUAMOUS)



INDICATION	%GPC3+
HCC	75
Lung SqCC	55
Lung SCLC	20
Lung Adeno	12
Cervical	20
Gastric	20
Ovarian	12

Aviel-Ronen S, et al. Modern Pathology. 2008.

- In clinical studies to date, GPC3-targeted therapies were well-tolerated but demonstrated limited activity
- Codrituzumab (Chugai/Roche anti-GPC3 antibody)[1,2]
  - Phase 1: improved time to progression (TTP) in GPC3-high patients
  - Phase 2: (unselected patients) did not meet efficacy endpoints



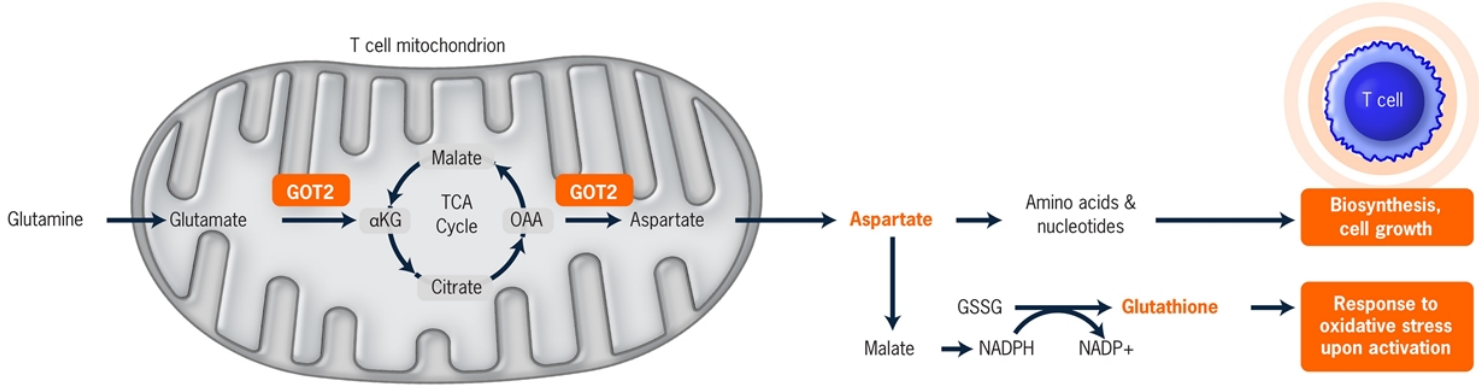
1. Zhu AX, et al. Clin. Cancer Res. 2013
2. Abou-Alfa GK, et al. J. Hepatology. 2016.

UNUM THERAPEUTICS

GOT2: A Regulator of Cellular Metabolism

GOT2 BOLT-ON | IMPROVED METABOLISM | BETTER T CELL FUNCTION | POTENT ANTI-TUMOR ACTIVITY

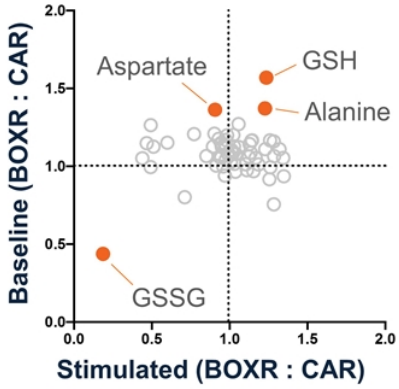
GOT2 is a critical enzyme for cell survival, proliferation and differentiation



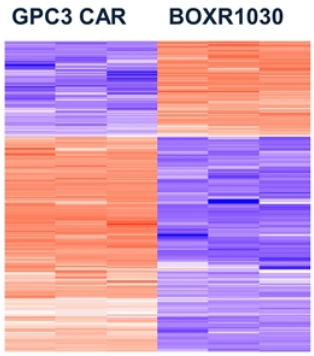
BOXR1030: Improving T Cell Metabolism in the TME

GOT2 BOLT-ON | **IMPROVED METABOLISM** | BETTER T CELL FUNCTION | POTENT ANTI-TUMOR ACTIVITY

Metabolic profiling reveals enriched metabolites in BOXR1030 consistent with GOT2 enzymatic activity



BOXR1030's unique transcriptional profile indicates improved metabolism and better stress response



Pathways with differential expression

Metabolism
- Fatty acid metabolism
- Adipogenesis
- Oxidative phosphorylation
- MTOR signaling
- Glycolysis

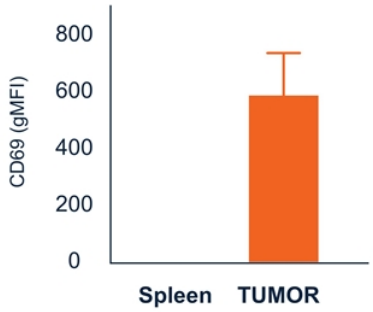
Response to Stress
- NFKB signaling
- Unfolded protein response
- G2M checkpoint

Whiteman, KR et al. Co-expression of the Metabolic Enzyme GOT2 with a GPC3-Targeted CAR-T Overcomes Challenges of the Solid Tumor Microenvironment, Substantially Improving Therapeutic Efficacy in Solid Tumor Xenografts, SITC 2019

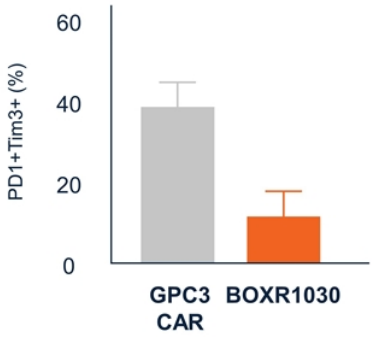
BOXR1030: Overcomes T Cell Exhaustion and Improves Function in Preclinical Studies

GOT2 BOLT-ON | IMPROVED METABOLISM | **BETTER T CELL FUNCTION** | POTENT ANTI-TUMOR ACTIVITY

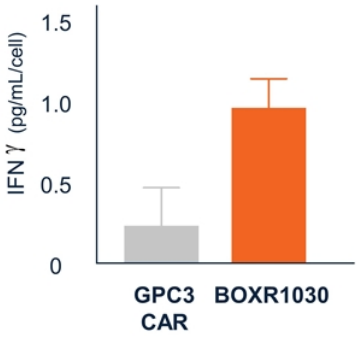
BOXR1030 activation only occurs at tumor site



BOXR T cells in the tumor display less exhaustion



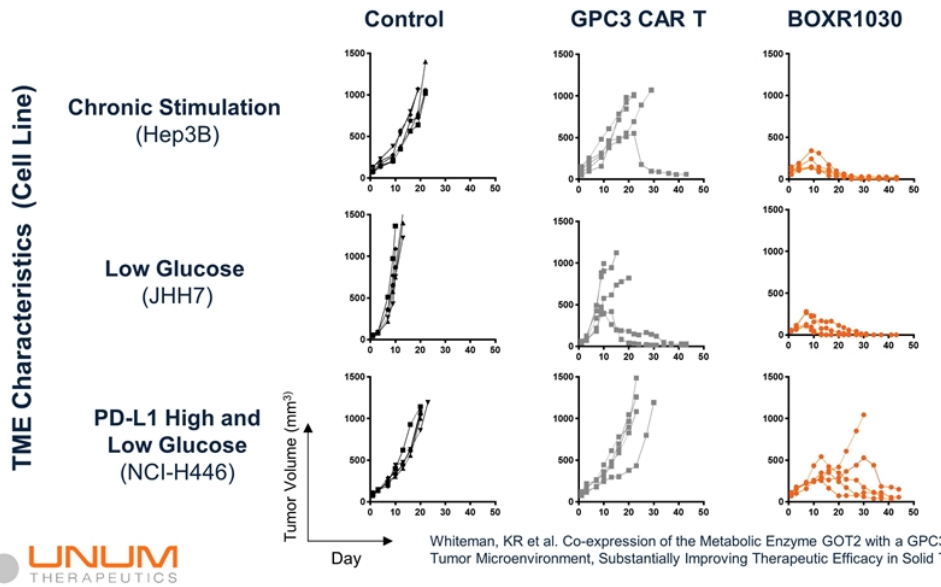
BOXR T cells in the tumor retain function



Whiteman, KR et al. Co-expression of the Metabolic Enzyme GOT2 with a GPC3-Targeted CAR-T Overcomes Challenges of the Solid Tumor Microenvironment, Substantially Improving Therapeutic Efficacy in Solid Tumor Xenografts, SITC 2019

BOXR1030: Reduced Tumor Burden in Mouse Xenografts

GOT2 BOLT-ON › IMPROVED METABOLISM › BETTER T CELL FUNCTION › POTENT ANTI-TUMOR ACTIVITY

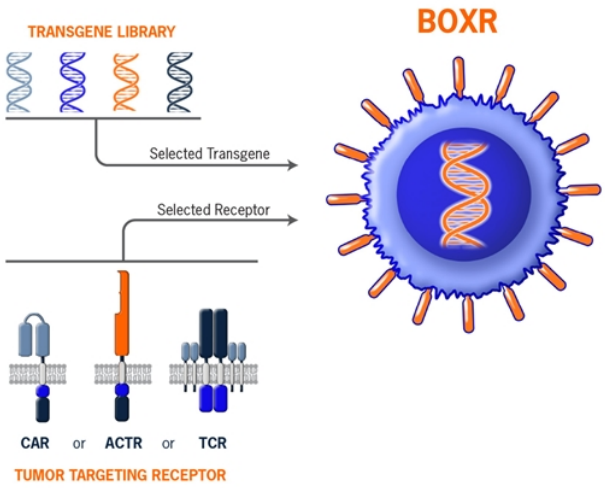


Addition of the GOT2 transgene resulted in complete tumor regressions with GPC3-targeted T cells

Whiteman, KR et al. Co-expression of the Metabolic Enzyme GOT2 with a GPC3-Targeted CAR-T Overcomes Challenges of the Solid Tumor Microenvironment, Substantially Improving Therapeutic Efficacy in Solid Tumor Xenografts, SITC 2019

BOXR Platform: Potential for Broad Pipeline Expansion

Bolt-On genes designed to improve T cell functionality in solid tumors can be readily incorporated into different cell therapy products



Unum's Bolt-On genes can yield multiple novel candidates from:

- Different Targeting Receptors
  - CAR
  - ACTR
  - TCR
- Different Immune Cells
  - αβ T cells
  - γδ T cells
  - NK cells
- Different Cell Sources
  - Circulating patient cells (autologous)
  - Donor-derived cells (allogeneic)
  - Tumor infiltrates (e.g., TILs)

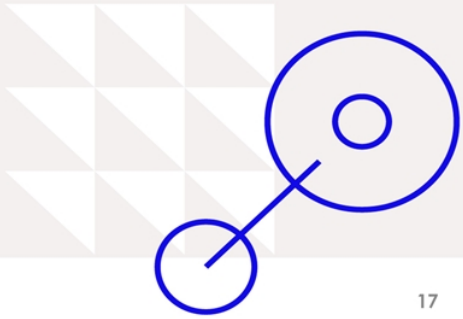
BOXR 1030/Platform Next Steps

BOXR1030

- GMP viral and T cell process development in progress
- Preclinical characterization of activity and preliminary safety studies conducted
- Clinical plan/protocol in preparation
- On track for IND filing in late 2020

BOXR PLATFORM

- Targeting ROR1 to address unmet need in triple negative breast cancer, ovarian and other indications
- Targeting multiple undisclosed solid tumor indications using proprietary antibodies



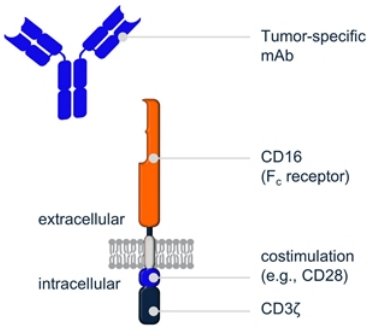
ACTR

ACTR: Antibody-Coupled T Cell Receptor

Autologous engineered T cell therapy designed to combine the cell-killing ability of T cells and the tumor-targeting ability of antibodies to exert potent antitumor activity

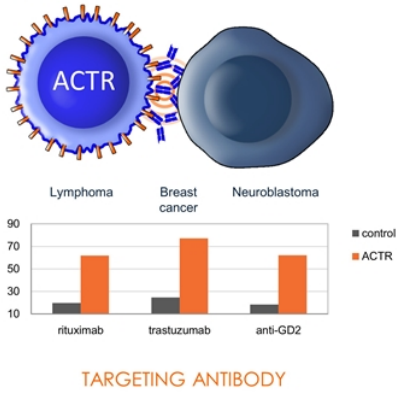
Structure

NK Fc receptor ectodomain linked to standard CAR signaling components



UNUM THERAPEUTICS

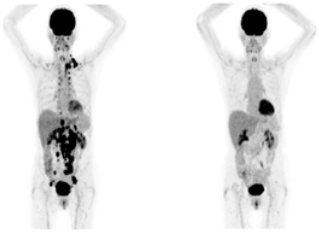
Mechanism



Kudo et al., "T lymphocytes expressing a CD16 signaling receptor exert antibody-dependent cancer cell killing," Cancer Res. 74:93-103 (2014)

Clinical Proof-of-Concept

Preliminary anti-tumor activity in the context of a favorable tolerability profile demonstrated with ACTR707+rituximab in CD20+ NHL (*Flinn et al. ASH 2018*)



Representative image of complete response in DLBCL subject treated with 5 prior therapies including ASCT

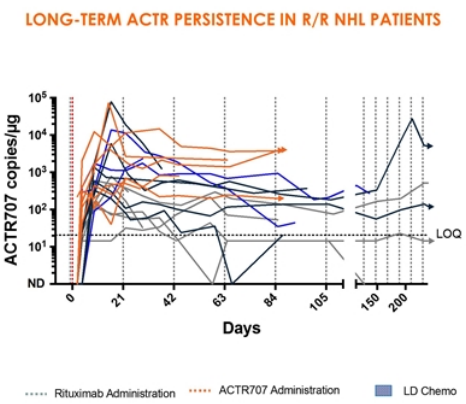
Proof Of Concept: Anti-tumor Activity R/R NHL

Potent anti-tumor activity including complete responses in initial cohorts of ATTCK-20-03

- Treatment with ACTR707 and rituximab generated robust clinical responses without T cell mediated toxicities of CRS or severe neurotoxicity
- Of 8 CRs, 6 were ongoing at last scan (4 >6 mos and 2 <6 mos); 2 progressed prior to the 6 month timepoint

BEST RESPONSE‡, n	Cohort 1 (n=6)	Cohort 2 (n=3)	Cohort 3 (n=5)	Cohort 4‡ (n=6)	Cohorts 1-4 (n=20)
Complete Response	**3**	**1**	**2**	**2**	**40% (8/20)**
Partial Response	**0**	**1**	**2**	**0**	**15% (3/20)**
Stable Disease	0	0	0	1	5% (1/20)
Indeterminate Response	1	0	0	0	5% (1/20)
Progressive Disease	2	1	1	3	35% (7/20)
Best Overall Response Rate	**50% (3/6)**	**67% (2/3)**	**80% (4/5)**	**33% (2/6)**	**55% (11/20)**
ACTR707+ T cells administered, median (range)	29M (23-38M)	35M (30-50M)	55M (45-55M)	83M (65-100M)	

** R/R defined as PD as best response to any line of prior therapy or relapse ≤ 12 months post ASCT*
‡ data cut Nov 2019



R/R NHL: Safety Data for Cohorts 1–4

- ACTR707 in combination with rituximab was well-tolerated in initial cohorts of ATTCK-20-03
- No dose-limiting toxicities (DLTs), no cytokine-release syndrome (CRS) or severe neurotoxicity events reported

ACTR707 Preliminary Phase 1 trial safety results in r/r NHL (Cohorts 1-4)

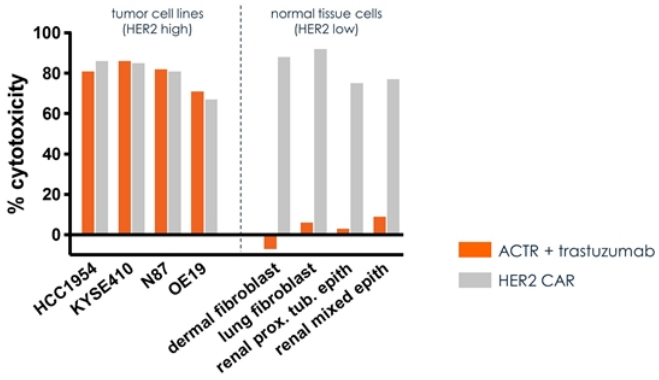
	Cohort 1 (n=6)	Cohort 2 (n=3)	Cohort 3 (n=5)	Cohort 4 (n=6)
Dose-limiting toxicities	0	0	0	0
Severe neurologic events (≥ Gr3)	0	0	0	0
CRS (any grade)	0	0	0	0
ACTR707-related SAEs	1	2	0	1
Febrile Neutropenia	1	1	0	1
Cytopenia	0	1	0	0

CRS = Cytokine Release Syndrome
Data cutoff: Nov 2019

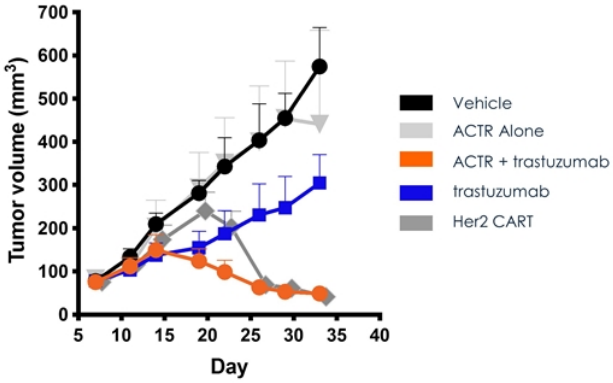
ACTR707: Optimized for Solid Tumors

ACTR707 was discovered by screening 100+ ACTR constructs for improved potential in solid tumors

Selective for on-tumor attack, sparing normal tissues with low levels of antigen expression



Potent, T cell-directed anti-tumor activity, versus trastuzumab alone

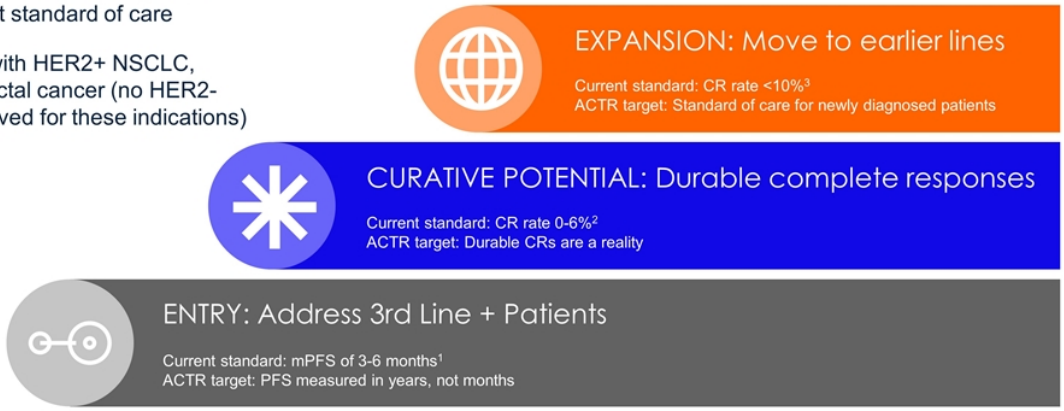


ATTCK-34-01: ACTR707 + TRASTUZUMAB in HER2+ Tumors

Significant unmet need in advanced HER2+ tumors, with opportunities for better treatment options

>10,000 patients (U.S.) with HER2+ breast or gastric cancers have exhausted current standard of care

>2,000 eligible patients (U.S.) with HER2+ NSCLC, bladder, head & neck, or colorectal cancer (no HER2-targeted agents currently approved for these indications)



[1] PFS = Progression-free survival. Range based on trastuzumab + chemo in 3rd+ line patients from SOPHIA, HER2CLIMB, and TH3RESA studies. [2] CR = Complete response. Range observed in SOPHIA, HER2CLIMB, TH3RESA, DESTINY-Breast01, and lapatinib studies. [3] Includes addition of EMILIA, CLEOPATRA, and MARIANNE studies

ATTCK-34-01: Phase 1 Trial of ACTR707 in HER2+ Tumors

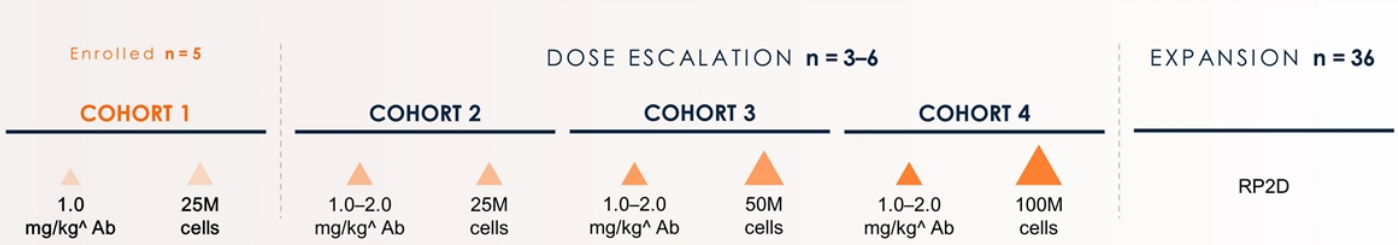
Open label phase 1 adaptive dose-escalating study to define optimal ACTR707 cell dose and trastuzumab dose; patients treated with 3 days of lymphodepleting chemotherapy (fludarabine + cyclophosphamide)

ELIGIBILITY
- Advanced HER2 (3+) solid tumor malignancy, adequate prior therapy including HER2 directed therapy if available

OBJECTIVES
- Demonstrate safety, tolerability, clinical response and determine recommended Phase 2 dose

STATUS
- Completed enrollment in Cohort 1
- Completing additional site activations

NEXT STEPS
- Report enrollment status, preliminary safety data 1Q 2020
- Report dose escalation safety/clinical response data during 2020



[1] PFS = Progression-free survival. Range based on trastuzumab + chemo in 3rd+ line patients from SOPHIA, HER2CLIMB, and TH3RESA studies. [2] CR = Complete response. Range observed in SOPHIA, HER2CLIMB, TH3RESA, DESTINY-Breast01, and lapatinib studies. [3] Includes addition of EMILIA, CLEOPATRA, and MARIANNE studies

ATTCK-34-01: Planned Efficacy/Biomarker Readouts

EFFICACY MEASURES

- Overall response rate per iRECIST, duration of response, progression-free survival, overall survival

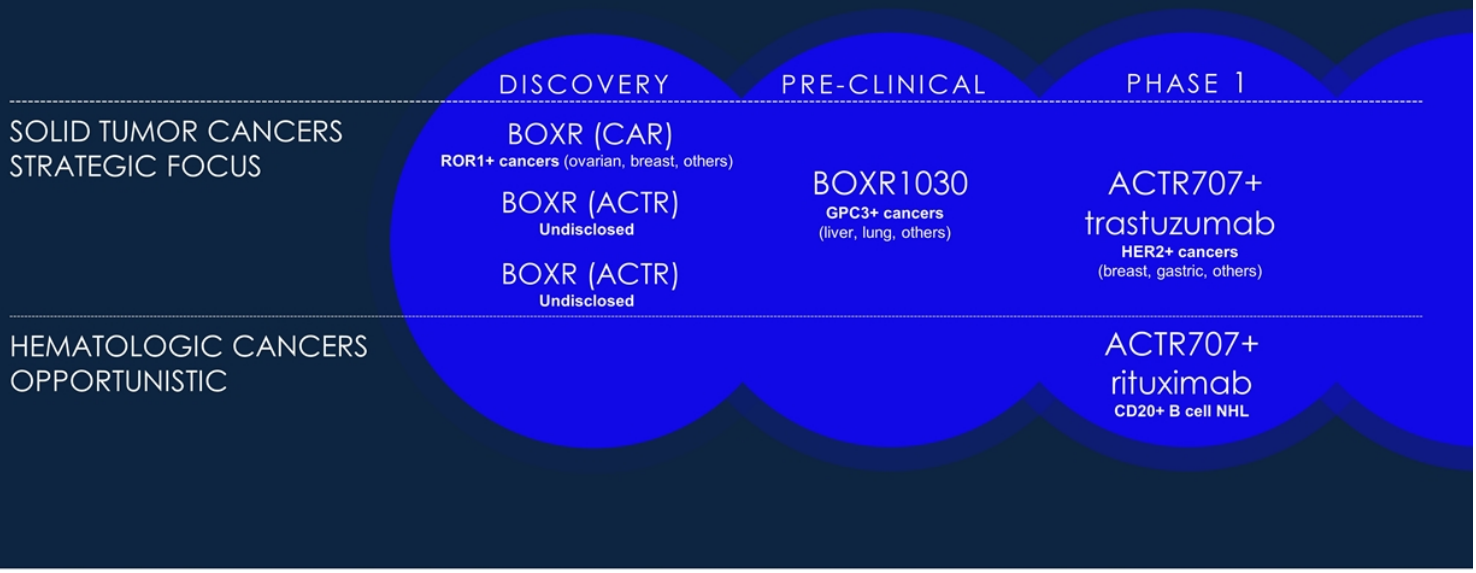
BIOMARKERS

- Persistence of ACTR by flow cytometry and qPCR
- ACTR phenotype and function
- Inflammatory markers and cytokines/chemokines after ACTR T cell product administration
- Trastuzumab pharmacokinetics (PK)
- Post-treatment biopsy analysis

Value-Creating Milestones

PROGRAM	INDICATION	EVENT	TIMING
ACTR707/087	r/r NHL	Safety/efficacy data from Phase 1 trials (ATTCK-20-2, ATTCK-20-03)	✓
ACTR707	HER2+ tumors	Preliminary safety data from Cohort 1 from Phase 1 trial (ATTCK-34-01)	**1Q 2020**
ACTR707	r/r NHL	Cohorts 5 & 6 data from Phase 1 trial (ATTCK-20-03)	**2020**
ACTR707	HER2+ tumors	Safety/efficacy data from dose cohorts from Phase 1 trial (ATTCK-34-01)	**2020**
BOXR1030	GPC3+ tumors	File Investigational New Drug (IND) application	**4Q 2020**
BOXR/ACTR	Solid tumors	Advance new preclinical programs	**2020**

A Focused Plan to Treat More Cancers



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Thank you

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