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): **November 10, 2018**

SYNLOGIC, INC.

(Exact name of registrant as specified in its charter)

Delaware	**001-37566**	**26-1824804**
(State or other jurisdiction of incorporation)	(Commission File Number)	(IRS Employer Identification No.)

301 Binney St., Suite 402 **Cambridge, MA** (Address of principal executive offices)	**02142** (Zip Code)

Registrant's telephone number, including area code: **(617) 401-9975**

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 (see General Instruction A.2. below):

□ 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 (17 CFR §230.405) or Rule 12b-2 of the Securities Exchange Act of 1934 (17 CFR §240.12b-2).
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.

On November 10, 2018, Synlogic, Inc. ("Synlogic") conducted an investor webcast and presentation summarizing preclinical data from its Synthetic Biotic medicine clinical candidate, SYNB1891, which Synlogic is developing for the treatment of cancer. A copy of the presentation is furnished with this Current Report on Form 8-K as Exhibit 99.1.

The information in Item 7.01 of this Current Report on Form 8-K and Exhibit 99.1 attached hereto is intended to be furnished and shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934 (the "Exchange Act") or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933 or the Exchange Act, except as expressly set forth by specific reference in such filing.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

99.1 Investor presentation provided by Synlogic dated November 10, 2018

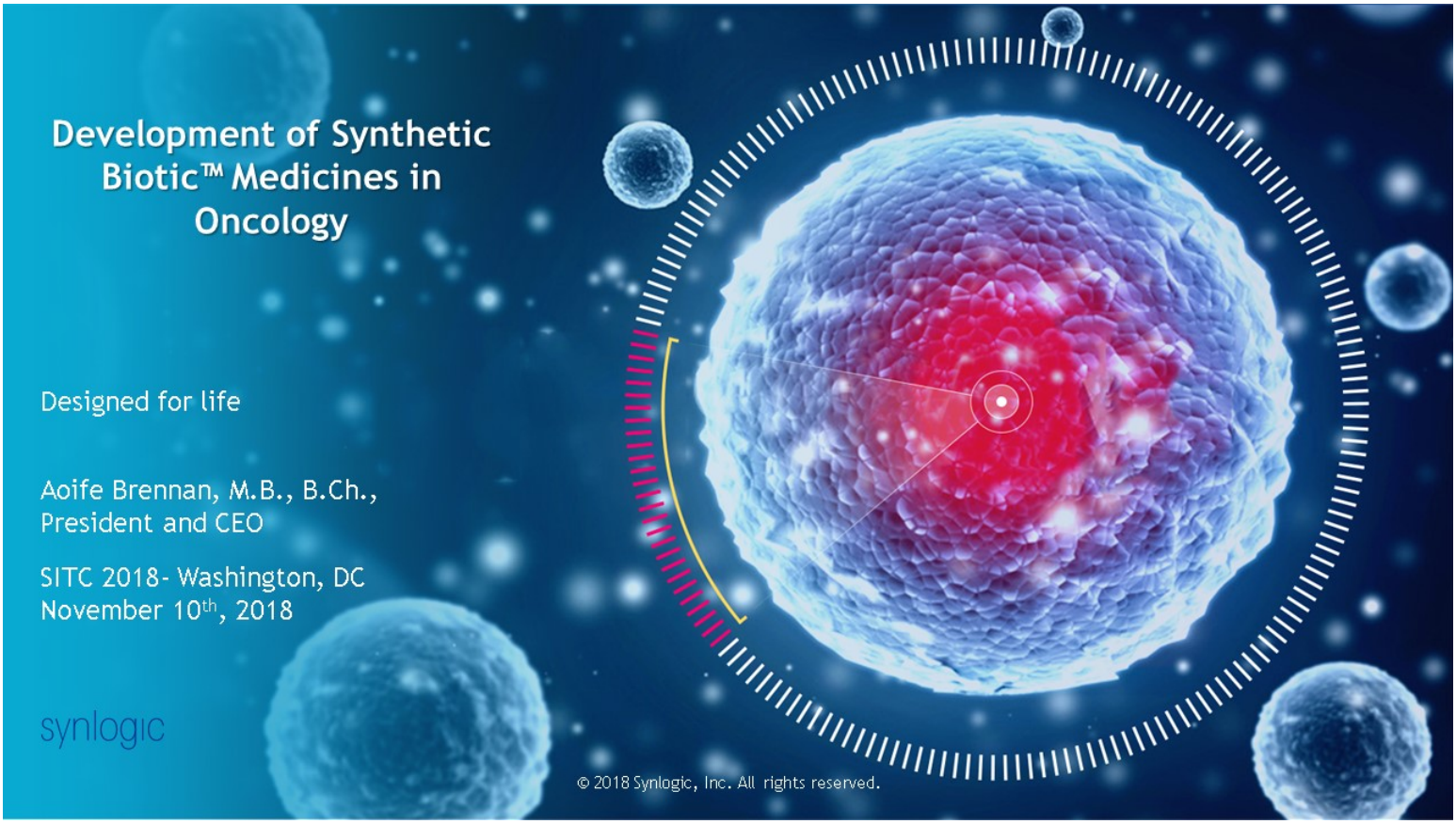
SIGNATURES

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.

SYNLOGIC, INC.

Date: November 12, 2018

By: /s/ Todd Shegog
Name: Todd Shegog
Title: Chief Financial Officer


Development of Synthetic Biotic™ Medicines in Oncology
Designed for life
Aoife Brennan, M.B., B.Ch.,
President and CEO
SITC 2018- Washington, DC
November 10th, 2018
synlogic
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Forward Looking Statements

This presentation contains "forward-looking statements" that involve substantial risks and uncertainties for purposes of the safe harbor provided by the Private Securities Litigation Reform Act of 1995. All statements, other than statements of historical facts, included in this presentation regarding strategy, future operations, future financial position, future revenue, projected expenses, prospects, plans and objectives of management are forward-looking statements. In addition, when or if used in this presentation, the words "may," "could," "should," "anticipate," "believe," "estimate," "expect," "intend," "plan," "predict" and similar expressions and their variants may identify forward-looking statements. Examples of forward-looking statements include, but are not limited to, the approach we are taking to discover and develop novel therapeutics using synthetic biology; statements regarding the potential of our platform to develop therapeutics to address a wide range of diseases including: inborn errors of metabolism, liver disease, inflammatory and immune disorders, and cancer; the future clinical development of Synthetic Biotic medicines; the potential of our technology to treat hyperammonemia and phenylketonuria; the expected timing of our anticipated clinical trial initiations; the benefit of orphan drug and fast track status; the adequacy of our capital to support our future operations and our ability to successfully initiate and complete clinical trials; the results of our collaborations; and the difficulty in predicting the time and cost of development of our product candidates. Actual results could differ materially from those contained in any forward-looking statement as a result of various factors, including, without limitation: the uncertainties inherent in the preclinical development process; our ability to protect our intellectual property rights; and legislative, regulatory, political and economic developments, as well as those risks identified under the heading "Risk Factors" in our filings with the SEC. The foregoing review of important factors that could cause actual events to differ from expectations should not be construed as exhaustive and should be read in conjunction with statements that are included herein and elsewhere, including the risk factors included in our quarterly Report on Form 10-Q filed with the SEC on August 9, 2018. The forward-looking statements contained in this presentation reflect our current views with respect to future events. We anticipate that subsequent events and developments will cause our views to change. However, while we may elect to update these forward-looking statements in the future, we specifically disclaim any obligation to do so. These forward-looking statements should not be relied upon as representing our view as of any date subsequent to the date hereof.

Agenda

12:30 pm - 12:40 pm	Introductions and intro to Synlogic platform and approach *Aoife Brennan, MB, ChB* *President & CEO, CMO,* *Synlogic Inc.*
12:40 pm - 12:55 pm	Unmet medical need in solid tumor immunotherapy *Filip Janku, MD, PhD* *MD Anderson Cancer Center*
12:55 pm - 1:10 pm	Role of Type I IFN in tumor immune recognition and therapy *Dmitriy Zamarin, MD, PhD* *Memorial Sloan Kettering Cancer Center*
1:10 pm 1:40 pm	Review of SYNB1891 data and program *Jose Lora, PhD* *VP, Research,* *Synlogic Inc.*
1:40 pm 2:00 pm	Q&A and closing remarks *Aoife Brennan*

Synthetic Biotic™ Medicines: A Novel Class of Living Medicines

Synthetic

Designed genetic circuits

Degradation of disease-causing metabolites

Production of therapeutic molecules

Biotic

Bacterial chassis

Non-pathogenic

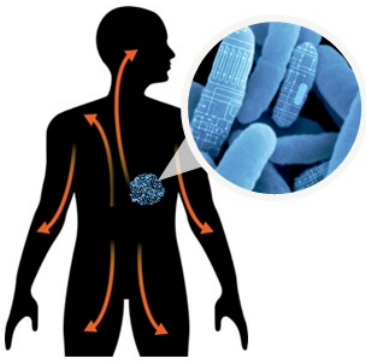
Amenable to genetic manipulation

PROGRAMMABLE POTENCY

Pathways, Combinations, Biomarkers

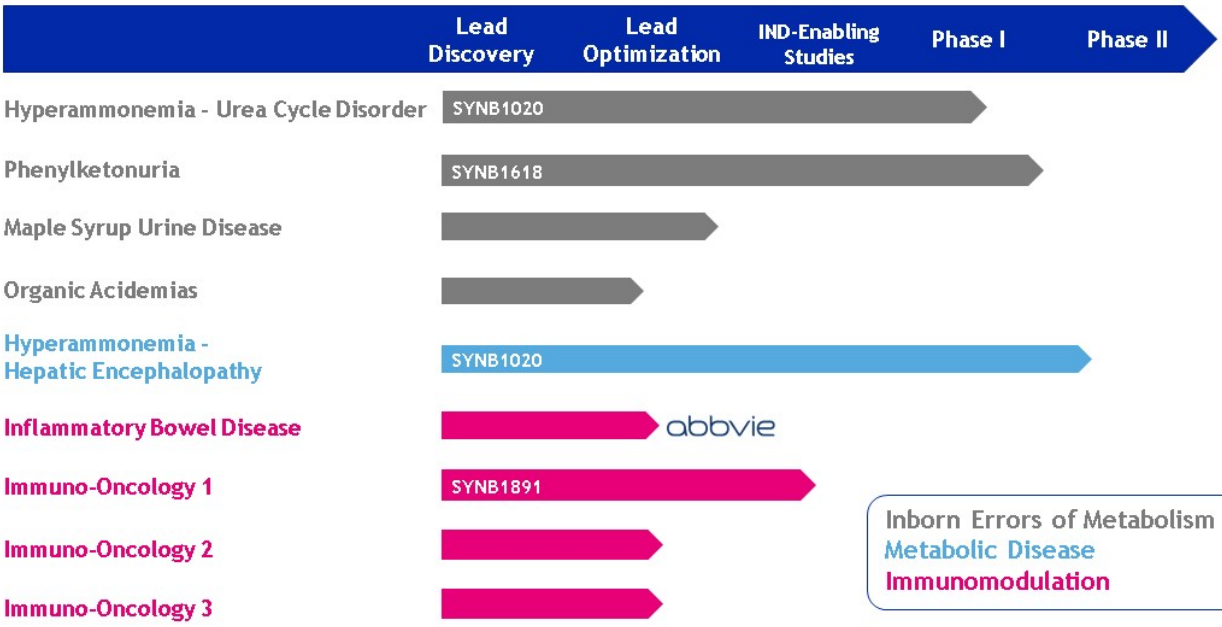
SWITCHES FOR CONTROL, TUNING

LOCAL, REDUCED SYSTEMIC TOXICITY



synlogic

Synthetic Biotic Platform Breadth and Potential: Pipeline Focused on Three Therapeutic Areas

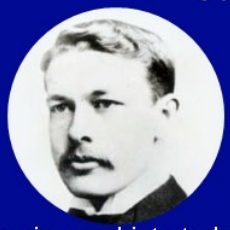


synlogic

Synlogic Immuno-Oncology Approach

Reimagining Early Immunotherapy for Combinatorial Effect

Bacteria Recognized as Earliest Immunotherapy



"Nature often gives us hints to her profoundest secrets, and it is possible that she has given us a hint in which, if we will but follow, may lead us on to the solution of this difficult problem."

Dr. William B. Coley
Immuno-Oncology Pioneer

Engineer a Living Solution: Synthetic Biotic Medicines

Rationally Designed for Combinatorial Effect

Locally Inflame the TME

Systemically Drive Tumor Antigen-Specific Immunity

In Situ Vaccination: Neo-antigen Priming and Sustained Immune Response

synlogic

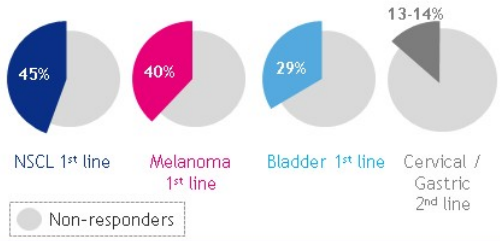
Synlogic Vision for Immuno-Oncology

Expand the benefits of immunotherapy broadly across tumor types

TREATMENT FAILURES

For indications where immune checkpoint inhibitors are indicated, only 13-45% of patients respond

ORR for Select FDA approved CPI Monotherapy



UNRESPONSIVE TUMORS

Other tumor types show little-to-no response to checkpoint inhibitors, for example:

Colorectal - MSS

Pancreatic

Prostate - castrate resistant

Breast - ER+, hormone therapy refractory

Enable broad response and remission through engagement of multiple immunomodulatory pathways to enhance tumor inflammation and promote robust T cell responses

THE UNIVERSITY OF TEXAS
MD Anderson Cancer Center
Making Cancer History®

Unmet medical need in solid tumor immunotherapy

Filip Janku, MD, PhD
Associate Professor
Clinical & Translational Research Center Medical Director

Investigational Cancer Therapeutics
(Phase I Clinical Trials Program)
MD Anderson Cancer Center
Houston, TX

Bench to Bedside & Back

FDA-approved Immune Checkpoint Inhibitors*

Drug	Immune Checkpoint(s)	FDA-approved tumor-type**
Ipilimumab	CTLA-4	Melanoma
Nivolumab	PD-1	Melanoma
		Non-small cell lung cancer
		Small cell lung cancer
		Renal cell carcinoma
		Classical Hodgkin lymphoma
		Squamous cell carcinoma of the head and neck
		Urothelial carcinoma
		Hepatocellular carcinoma
		Mismatch repair deficient and microsatellite instability high metastatic colorectal cancer
Pembrolizumab	PD-1	Melanoma
		Non-small cell lung cancer
		Squamous cell carcinoma of the head and neck
		Classical Hodgkin lymphoma
		Urothelial carcinoma
		Gastric or gastroesophageal junction
		Microsatellite instability-high or mismatch repair deficient solid tumors
		Cervical cancer
Atezolizumab	PD-L1	Urothelial carcinoma
		Non-small cell lung cancer
Durvalumab	PD-L1	Urothelial carcinoma
		Non-small cell lung cancer
Avelumab	PD-L1	Merkel cell carcinoma
		Urothelial carcinoma
Nivolumab with Ipilimumab	PD-1 and CTLA-4	Melanoma
		Renal cell carcinoma
		Microsatellite instability-high or mismatch repair deficient metastatic colorectal cancer

*List of FDA-approved immune checkpoint inhibitors as of September 14, 2018.

Adapted from: https://www.fda.gov/Drugs/InformationOnDrugs/ApprovedDrugs/ucm279174.htm

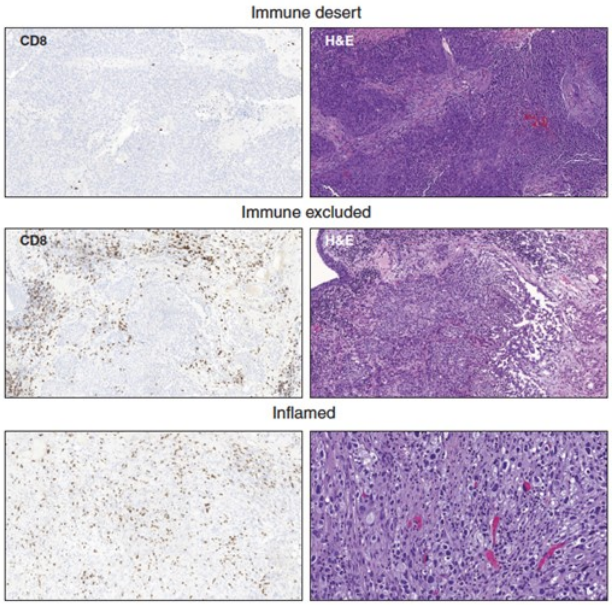
**Tumor type must meet the criteria listed in the above-mentioned website.

Response rates to checkpoint inhibitors in approved indications

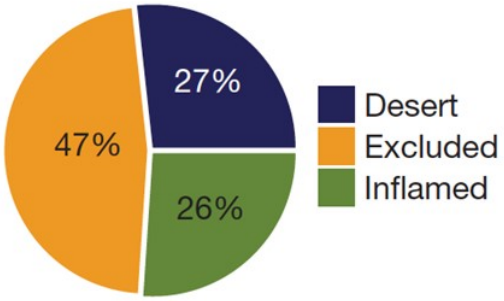
- **Melanoma**
  - Pembrolizumab: RR ~ **30%**
  - Nivolumab/ipilimumab: RR ~ **50%**
- **Non-small lung cancer**
  - Pembrolizumab: RR ~ **20%-40%**
  - Nivolumab: RR ~ **20%**
- **SCC of head and neck**
  - Pembrolizumab: RR ~ **18%**
  - Nivolumab: RR ~ **13%**
- **Urothelial cancer**
  - Pembrolizumab: RR ~ **21%**
  - Nivolumab: RR ~ **28%**
  - Atezolizumab: RR ~ **15%-26%**

Robert NEJM 2015
Wolchok NEJM 2013
Garon NEJM 2015
Reck NEJM 2016
Ferris NEJM 2016
Chow J Clin Oncol 2016
Bellmunt 2017
Rosenberg 2016

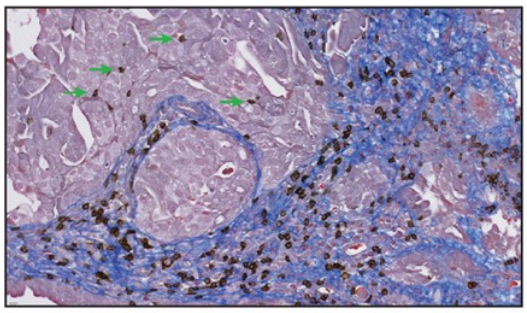
Classification by tumor immune phenotype in urothelial cancers



Mariathasan S. Nature 2018



Immune excluded (CD8 trichrome stain)



Immunotherapy: unmet need

Estimated New Cases

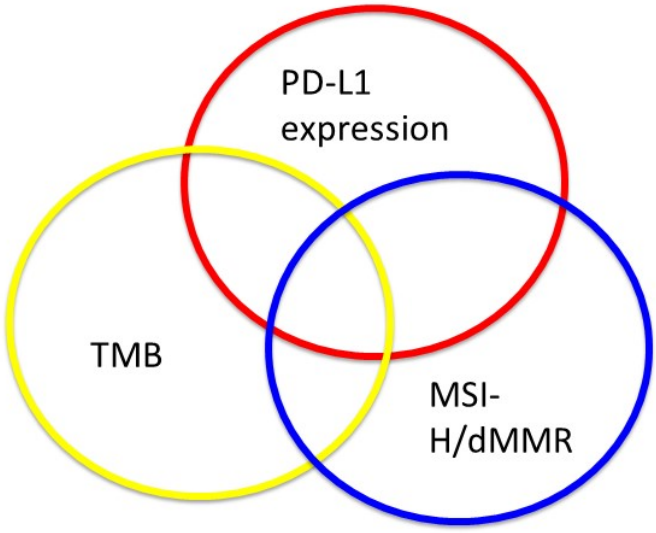
Males				Females		
Prostate	164,690	19%		Breast	266,120	30%
Lung & bronchus	121,680	14%		Lung & bronchus	112,350	13%
Colon & rectum	75,610			on & rectum	64,640	7%
Urinary bladder	62,380			rine corpus	63,230	7%
Melanoma of the skin	55,150			roid	40,900	5%
Kidney & renal pelvis	42,680			anoma of the skin	36,120	4%
Non-Hodgkin lymphoma	41,730			-Hodgkin lymphoma	32,950	4%
Oral cavity & pharynx	37,160	4%		Pancreas	26,240	3%
Leukemia	35,030	4%		Leukemia	25,270	3%
Liver & intrahepatic bile duct	30,610	4%		Kidney & renal pelvis	22,660	3%
All Sites	**856,370**	**100%**		**All Sites**	**878,980**	**100%**

MSI-H: ~5% of mCRC

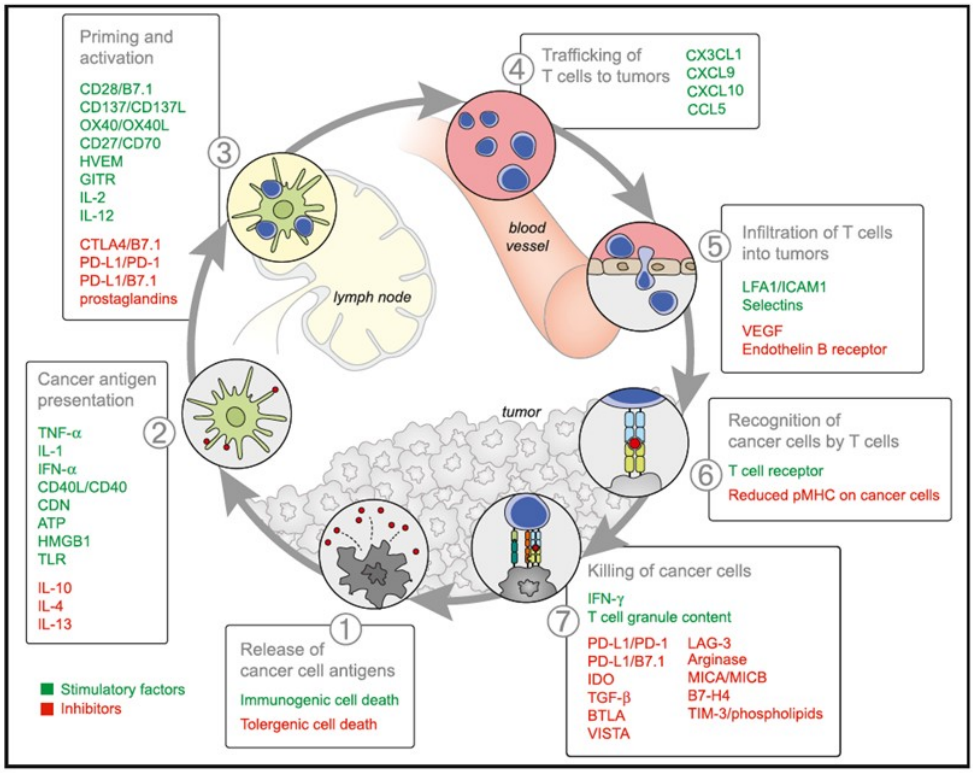
- Ovarian cancer
- Soft tissue sarcomas
- Glioma
- Myeloma

Siegel CA J Cancer Clin 2018

Predictive factors for response to immune checkpoint inhibitors

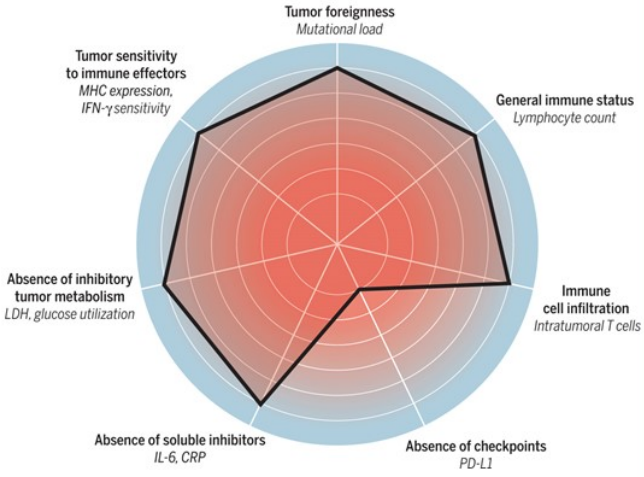


Stimulatory and Inhibitory Factors in the Cancer-Immunity Cycle

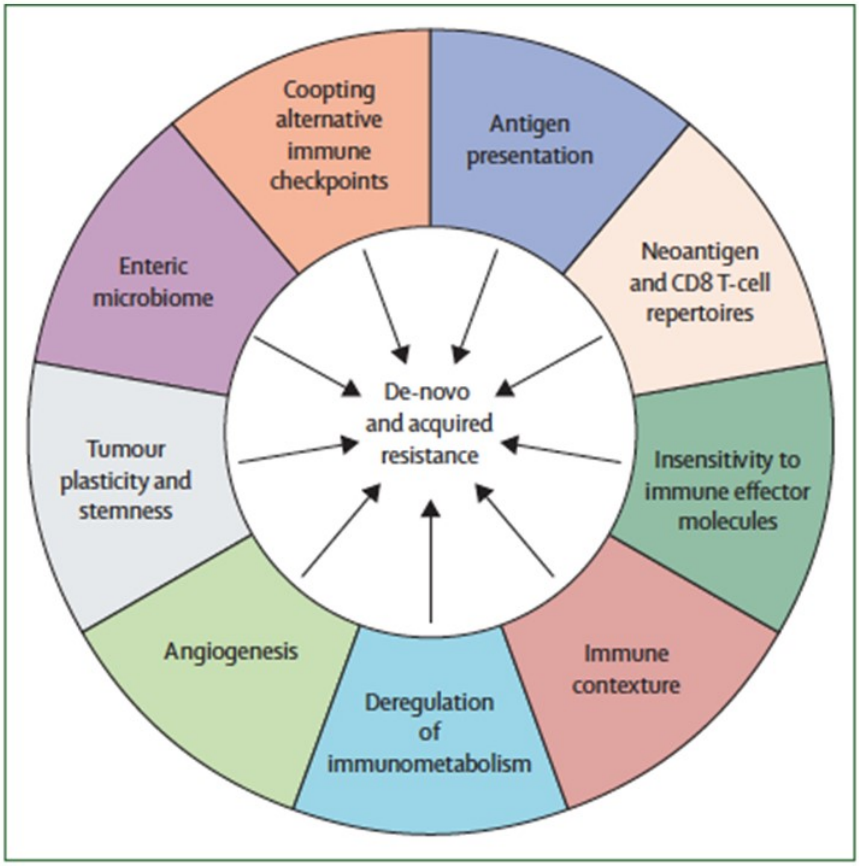


Chen DS. Immunity 2013

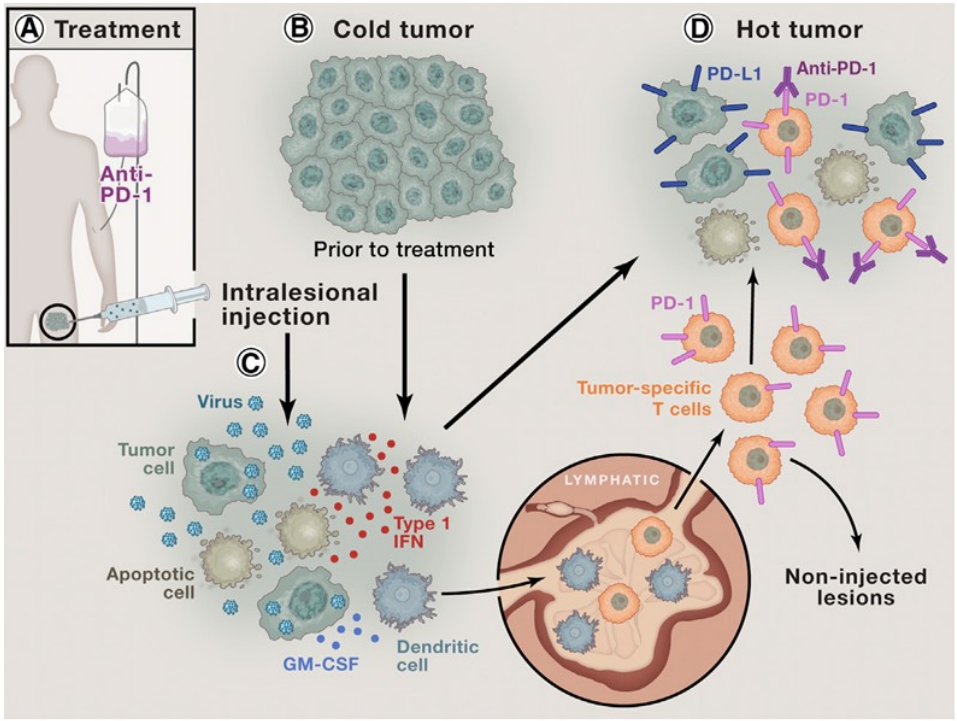
Mechanisms of immunoresistance



Syn Lancet Oncol 2017
Blank Science 2016

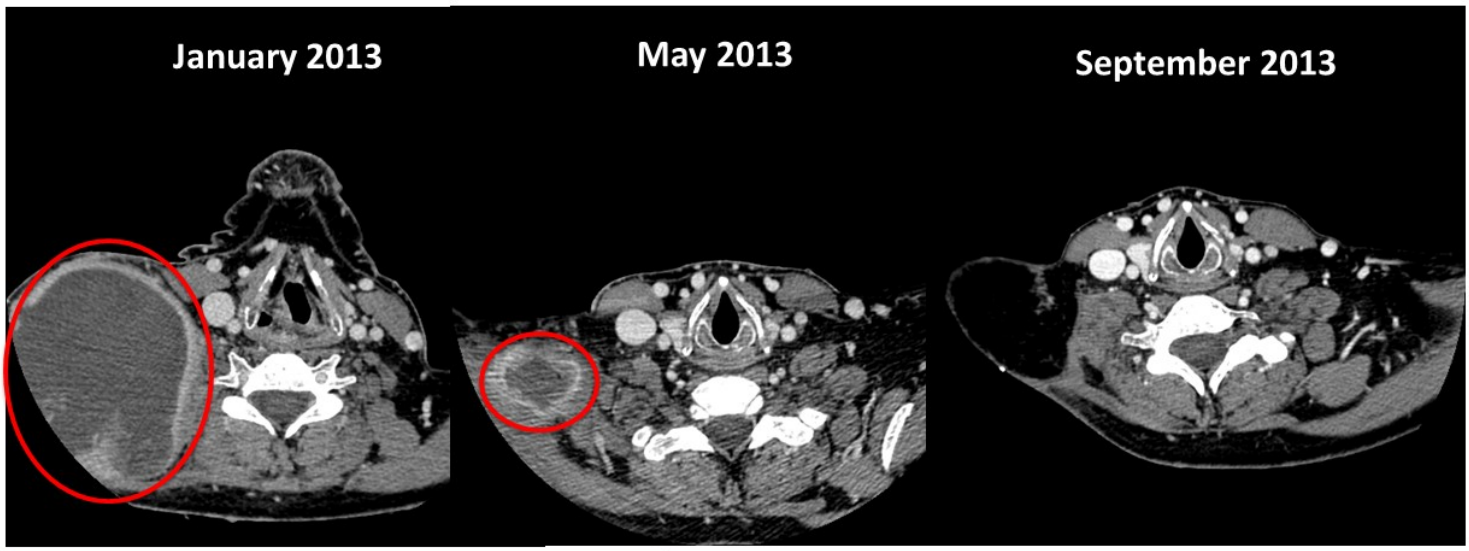


Converting Cold Tumors into Hot



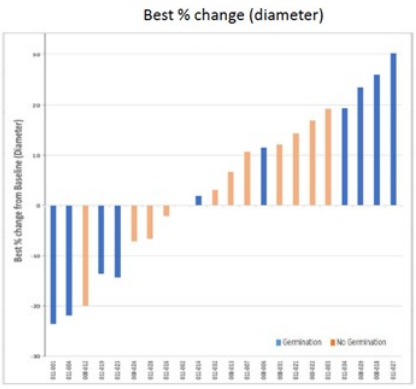
Haanen Cell 2017

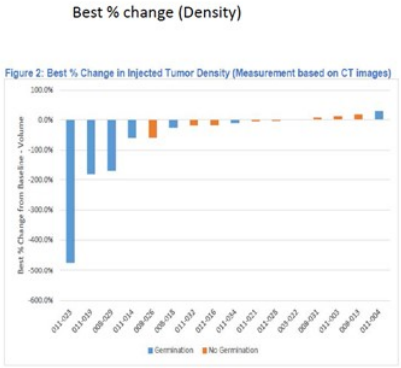
Patient with MPNST (sarcoma) with spontaneous remission after prolonged infection with coagulase negative staphylococcus and *Klebsiella Pneumoniae*

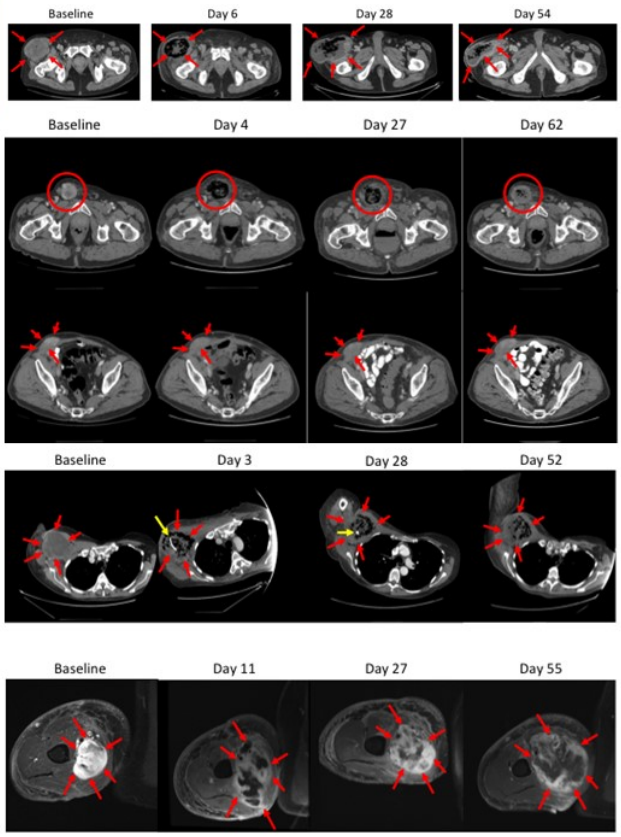


Prolonged infection with bacteremia
January to February 2013

Anticancer activity of single intratumor injection of *Clostridium Novyi-NT*







Janku
CRI-CMIT-EATI-AACR 2018

Cytokine response after single intratumor injection of *Clostridium Novyi-NT*

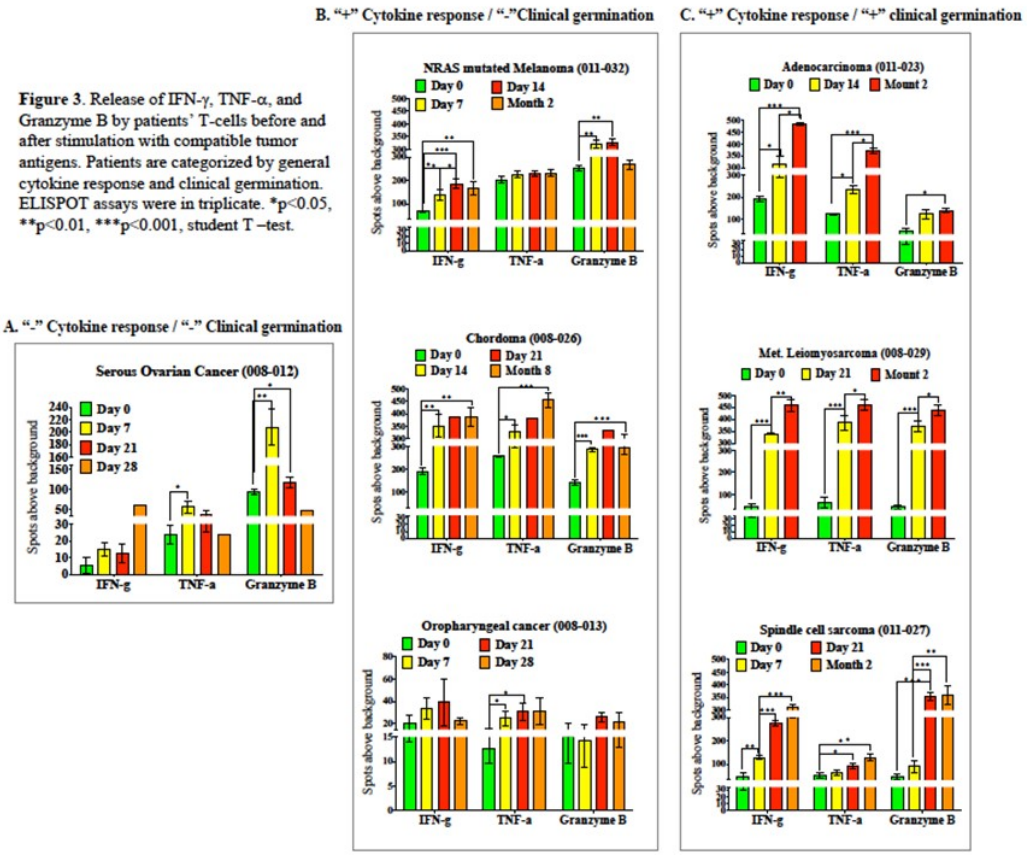
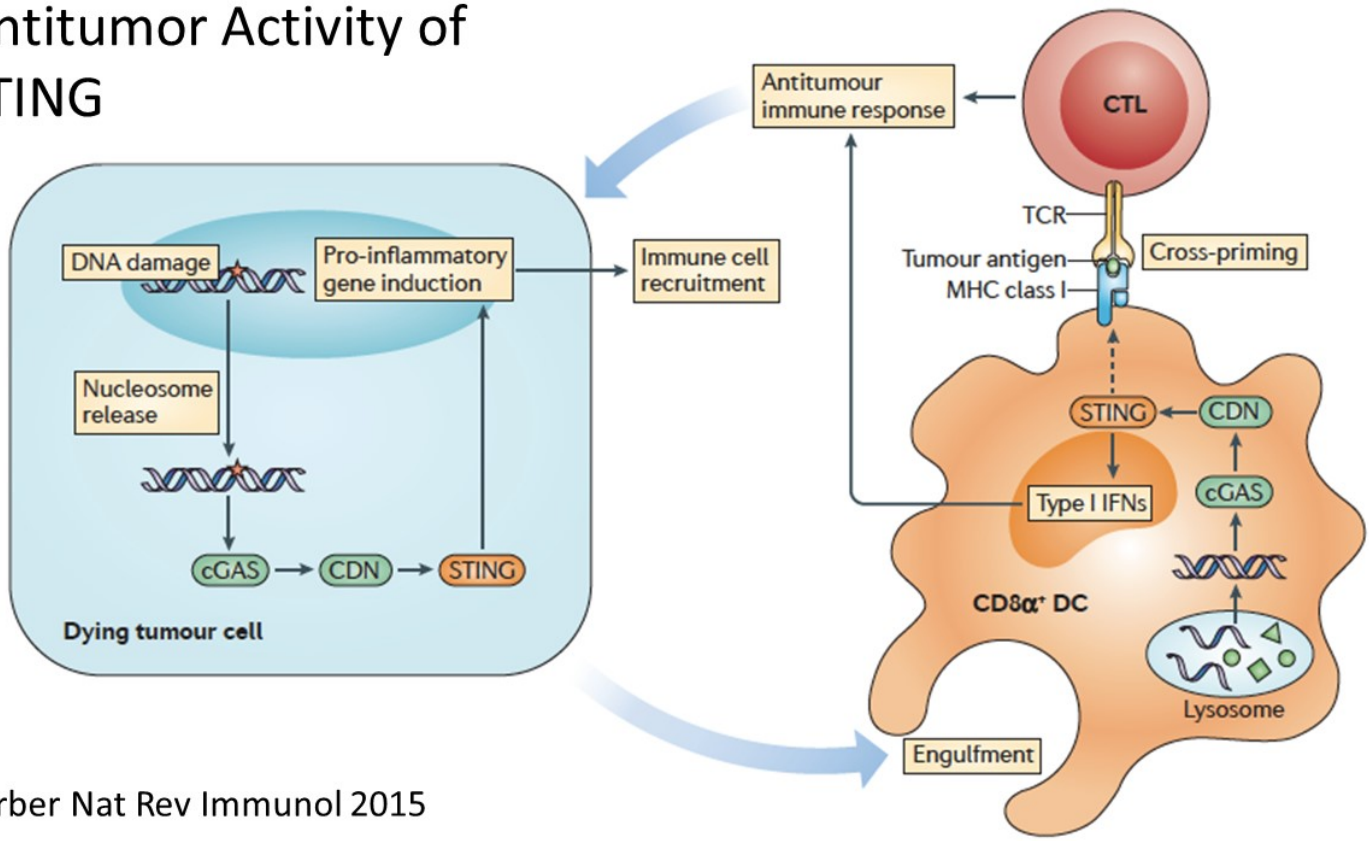


Figure 3. Release of IFN-γ, TNF-α, and Granzyme B by patients' T-cells before and after stimulation with compatible tumor antigens. Patients are categorized by general cytokine response and clinical germination. ELISPOT assays were in triplicate. *p<0.05, **p<0.01, ***p<0.001, student T –test.

Janku
CRI-CMIT-EATI-AACR 2018

Antitumor Activity of STING



Barber Nat Rev Immunol 2015

Phase I: Intratumor STING agonist MK-1454 +/- pembrolizumab

- Phase I: Accelerated Titration Design -> modified Toxicity Probability Interval
- Endpoints
  - Primary: safety, dose
  - Secondary: PK/PD
  - Exploratory: objective response
- DLTs:
  - Monotherapy (26 patients): G3 vomiting (1)
  - Combination (25 patients): G2 erythema multiforme (1), G3 injection site pain (1), G3 skin/tumor necrosis (1)
- AEs:
  - Pyrexia (65.2%/42.9%), chills (39.1%/25%), injection site pain (47.8%/10.7%), fatigue (34.8/25%)

Harrington ESMO 2018

Phase I: Intratumor STING agonist MK-1454 +/- pembrolizumab

EFFICACY

- Monotherapy
  - Myoepitehlial carcinoma > - 30% (not confirmed as PR)
  - 2 patients with shrinkage of injected lesions

- Combination
  - Partial response: 6 (TNBC, 1; HNSCC, 3; ATC, 2)
  - Shrinkage of injected and noninjected lesions observed
  - PRs were durable (>6 months)
  - Median 83% reduction in size of target lesions for responders

Harrington ESMO 2018

SITC 2018: MIW815 STING agonist

- 41 pretreated patients with solid tumors or lymphomas
- No DLTs
- The most common AEs: pyrexia (7; 17.1%), injection site pain (6; 14.6%), headache (6; 14.6%).
- Grade 3/ 4 AEs: increased lipase (2; 4.9%), elevated
- amylase, tumor pain, dyspnea, respiratory failure,
- and injection site reaction (1 each; 2.4%).
- On-treatment tumor biopsies showed increases in CD8 T cells infiltrating the injected tumors in a subset of patients.
- PR: Merkel cell (CPI naïve), Parotid gland (CPI pretreated), both response appear to be durable

Meric-Bernstam. SITC 2018

STING agonists in the clinic

- MK-1454: early data for monotherapy and combination with pembrolizumab presented at ESMO 2018
- MIW815: early data for monotherapy presented at SITC 2018
- MK-2118: clinical trial ongoing (monotherapy and combination with pembrolizumab)

Conclusions

- Immunotherapy with immune checkpoint inhibitors can be effective in subsets of patients with melanoma, lung cancer and other tumor types
- Immunotherapy with immune checkpoint inhibitors has not shown enough activity resulting in FDA approval in many common cancers including breast cancer, prostate cancer, ovarian cancer, MSS colorectal cancer and sarcomas, which creates unmet need for novel therapeutic approaches
- Turning cold tumors into hot with activators of innate immunity such as STING agonists (and others) offers a new promising approach to increase efficacy of cancer immunotherapy

Memorial Sloan Kettering Cancer Center

Role of type I IFN in tumor immune recognition and therapy

Dmitriy Zamarin MD PhD
Assistant Attending Physician
Translational Research Director
Gynecologic Medical Oncology Service
Immunotherapeutics Service
Memorial Sloan Kettering Cancer Center

Memorial Sloan Kettering Cancer Center

Type I IFN: the first cytokine

Alick Isaacs Jean Lindenmann

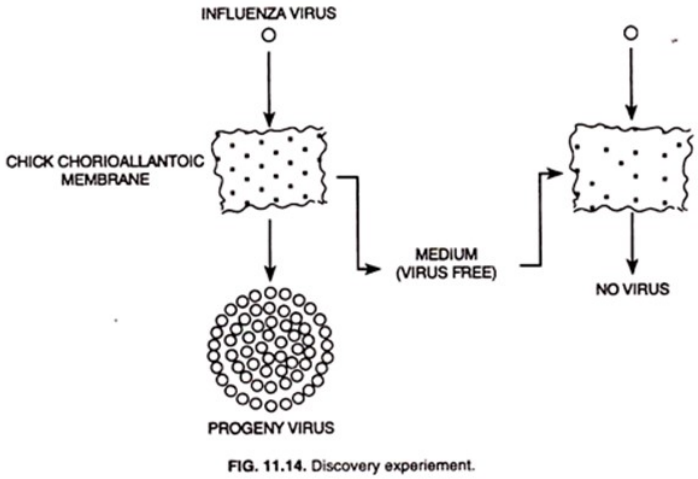
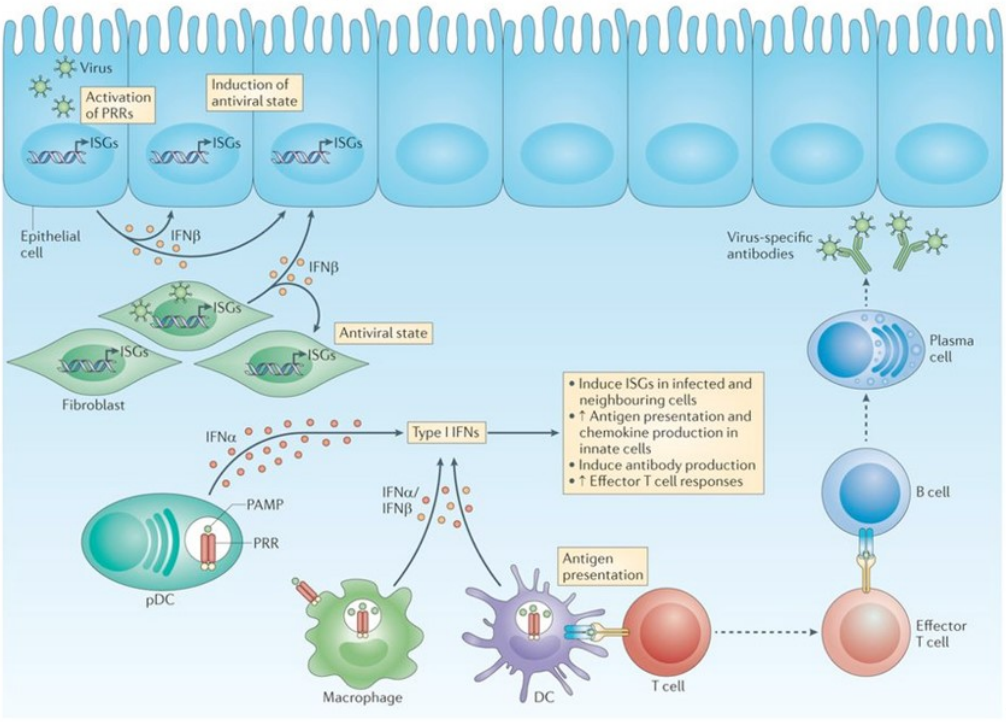


FIG. 11.14. Discovery experiement.

Isaacs, A., and Lindenmann, J., *Proc. Roy. Soc.*, B, **147**, 258 (1957)

Memorial Sloan Kettering Cancer Center

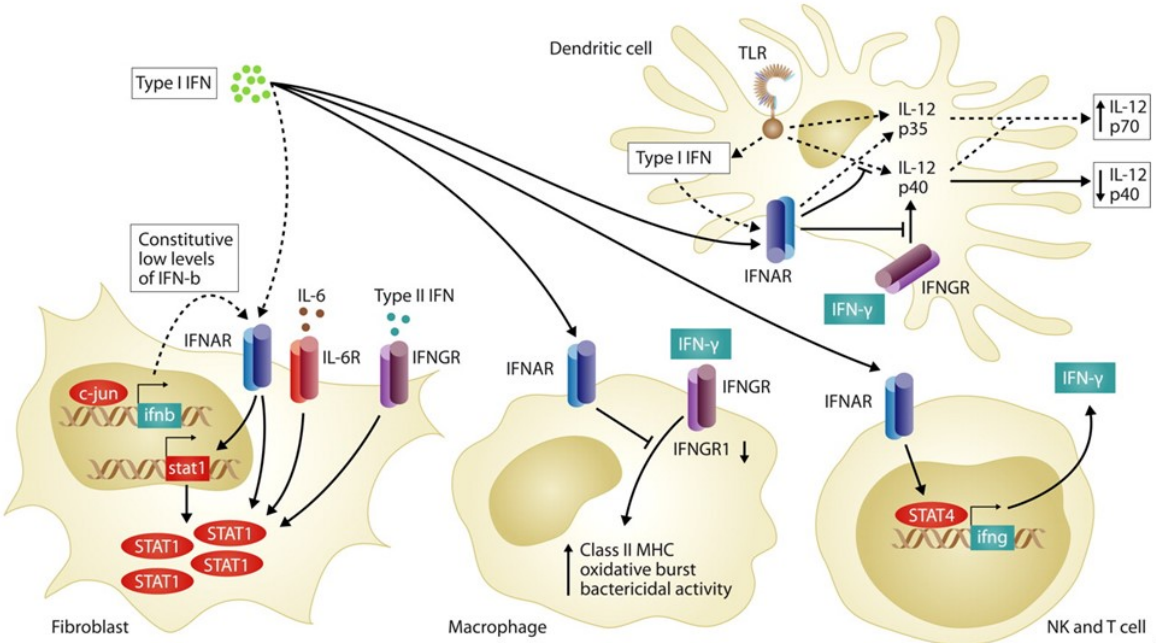
Functions of type I IFN in infection



Ivashkiv et al. Nat Rev Immunol 2013

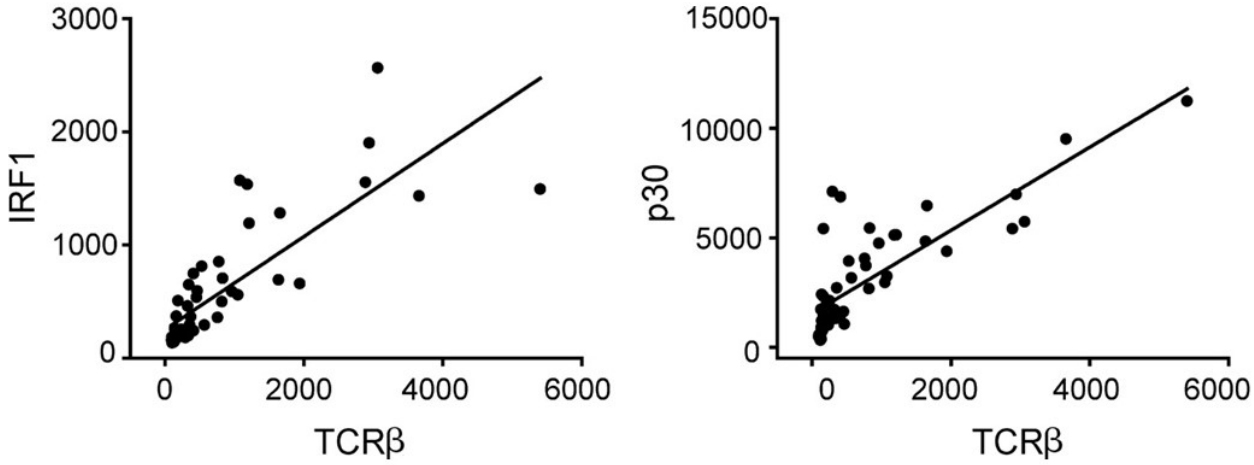
Memorial Sloan Kettering Cancer Center

Cross-talk between type I IFN and adaptive immunity



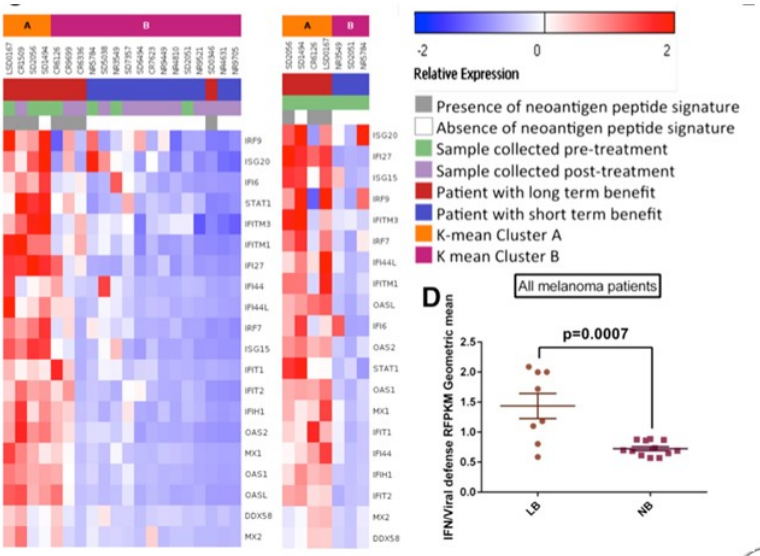
Trinchieri G, JEM 2010.

Type I IFN- related transcripts correlate with T cell infiltration in tumors



Fuertes M.B. et al., JEM. 208:2005-16 (2011)

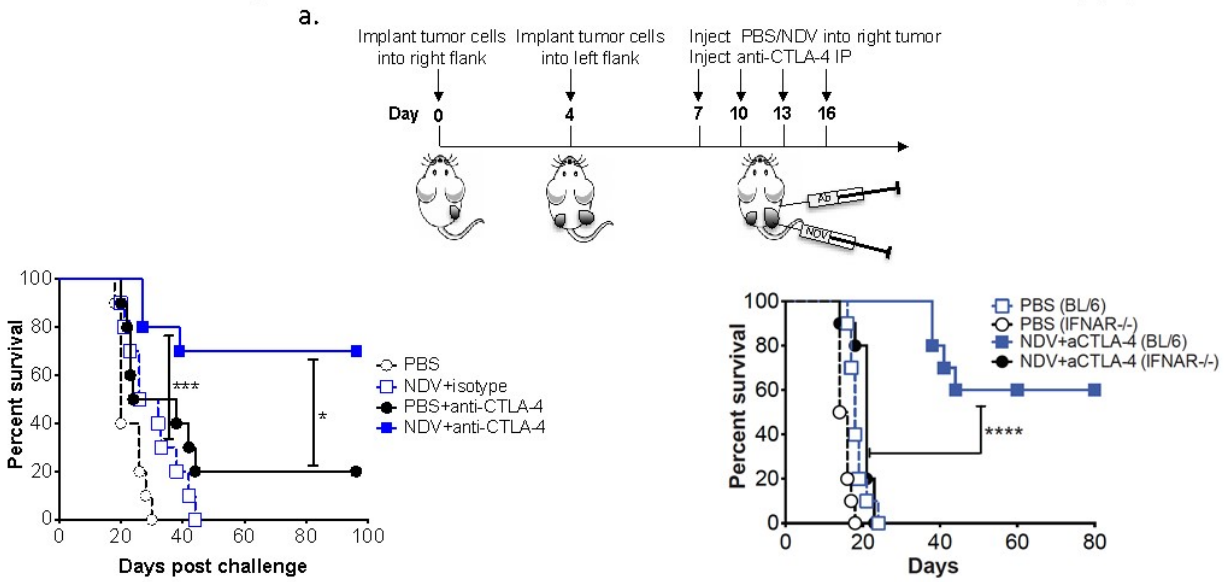
Type I IFN signature is associated with clinical benefit from ipilimumab in melanoma



Chiappinelli et al., Cell 2015

Memorial Sloan Kettering Cancer Center

Type I IFN pathway is essential for the efficacy of cancer immunotherapy

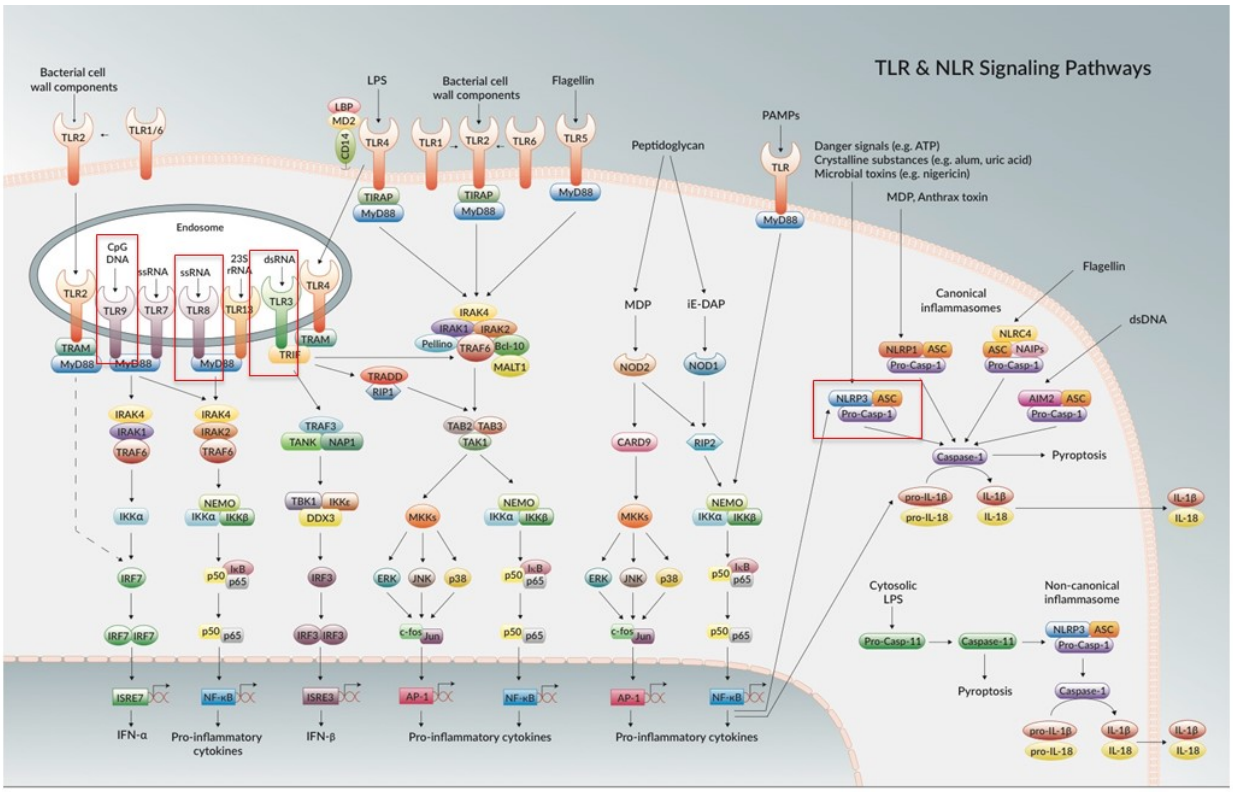


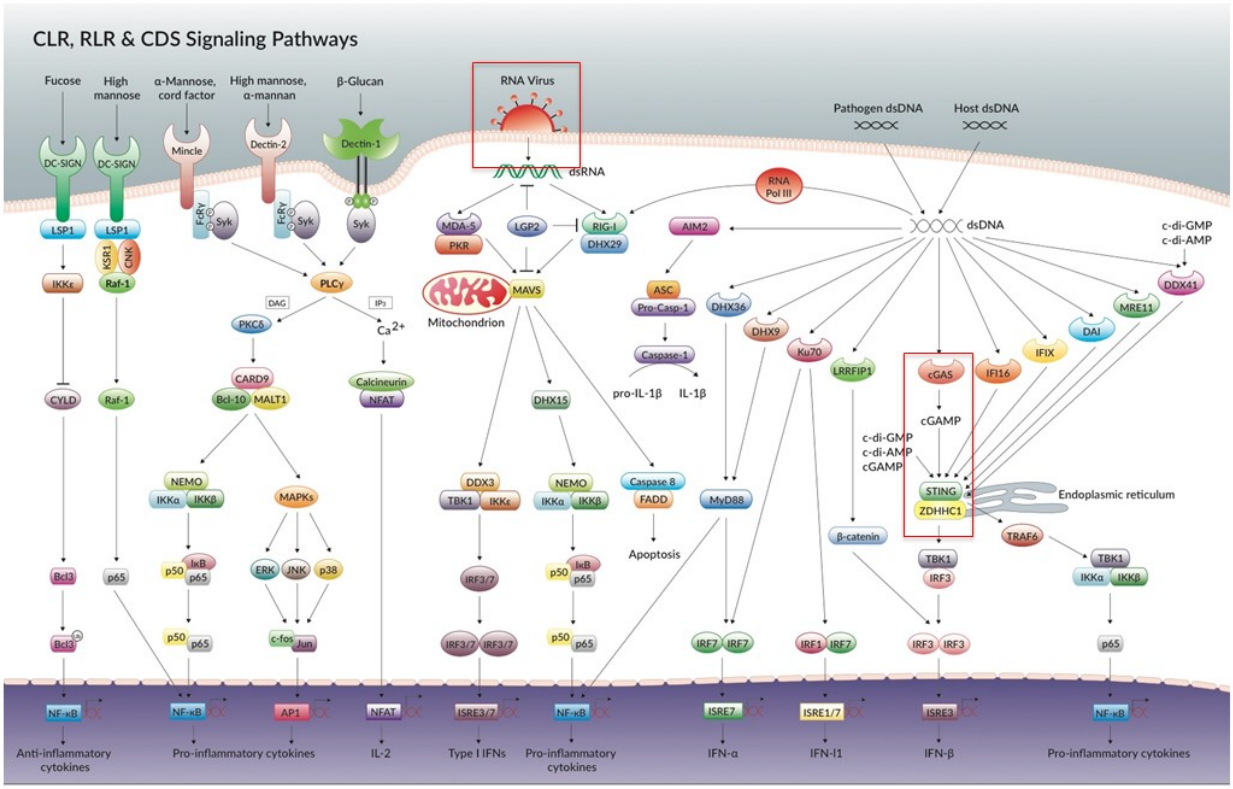
Zamarin D, Wolchok JD, Allison JP. *Sci Transl Med*. 2014 5:226ra

Memorial Sloan Kettering Cancer Center

Mechanisms of activation of type I IFN pathway

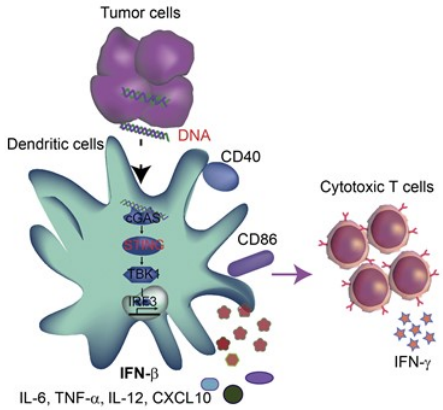
Memorial Sloan Kettering Cancer Center

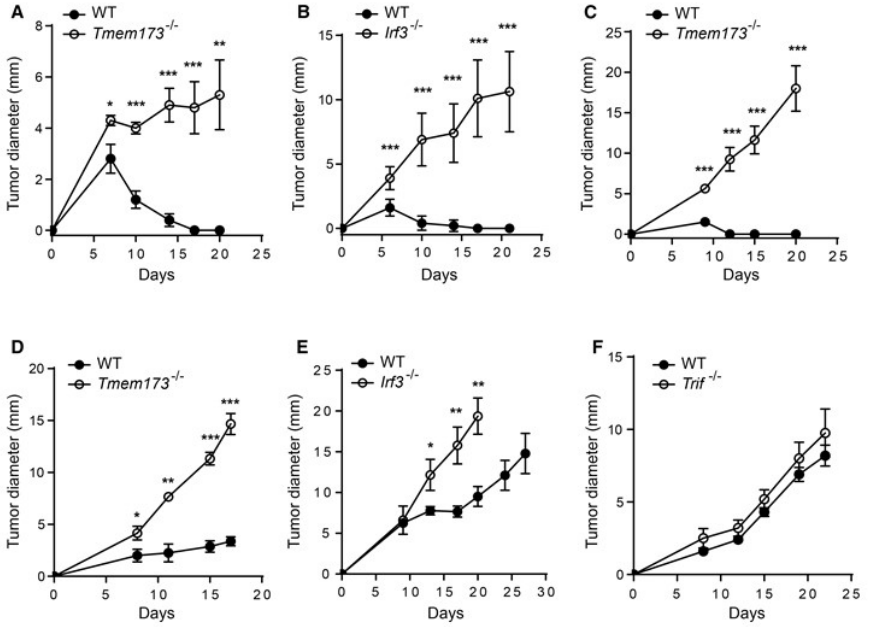




n Kettering
-

STING pathway is required for immune recognition and elimination of tumors.





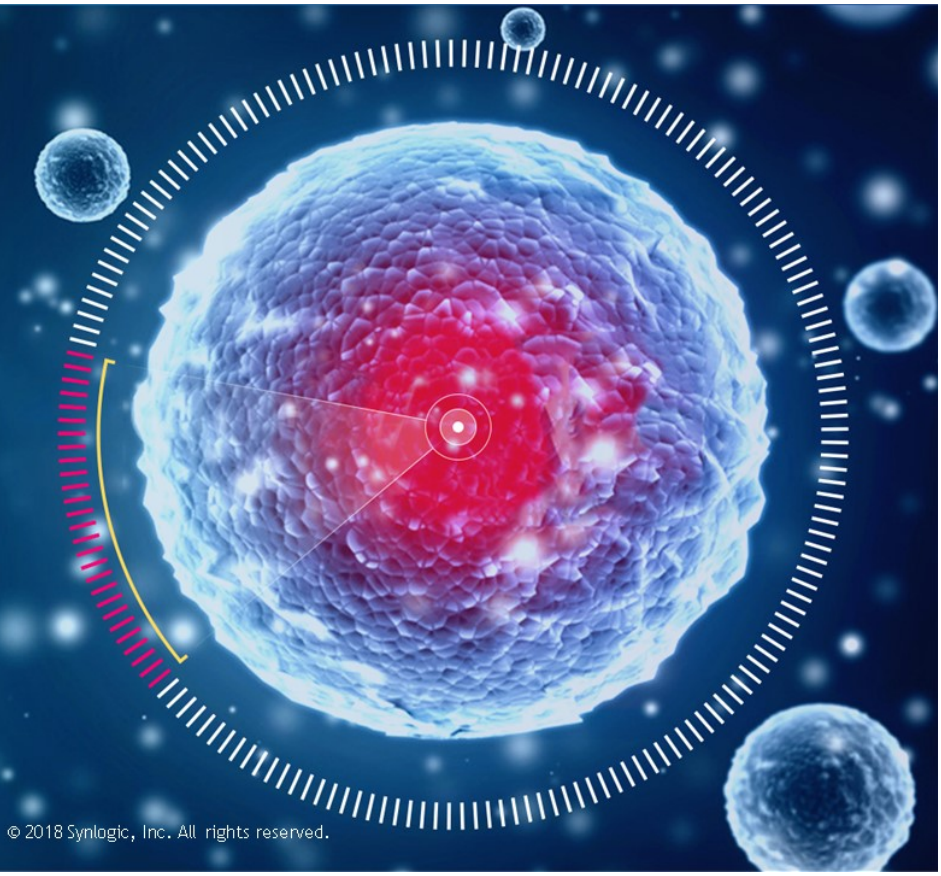
Woo …. Gajewski. Immunity 2015.

Memorial Sloan Kettering Cancer Center

Therapeutic strategies to target type I IFN pathway in cancer

- TLR agonists
- STING agonists
- Viruses
- Bacteria
- Engineered viruses and bacteria

Development of a STING Agonist-producing Synthetic Biotic™ Medicine to Activate Innate and Adaptive Immunity and Drive Antitumor Immune Responses



Designed for life

Jose M. Lora, PhD
Vice President, Research

SITC 2018- Washington, DC
November 10th, 2018

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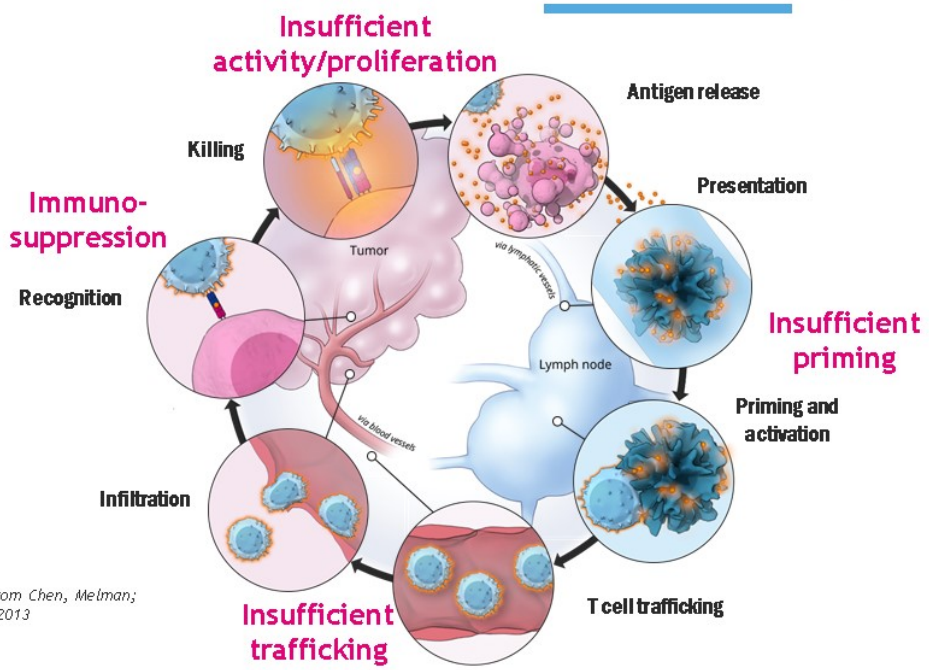
A Tumor Can Evade Multiple Critical Aspects of the Cancer-Immunity Cycle



Adapted from Chen, Melman; Immunity 2013

Monotherapies Often Fail to Overcome Tumor Evasion Mechanisms

Recognized Need to Combine Mechanisms to Broaden the Benefit of Immunotherapy

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Synthetic Biotic Medicines Engineered for Efficacy

Rational Design of Key Immunostimulatory Mechanisms in a Bacterial Chassis

Relieve Immunosuppression

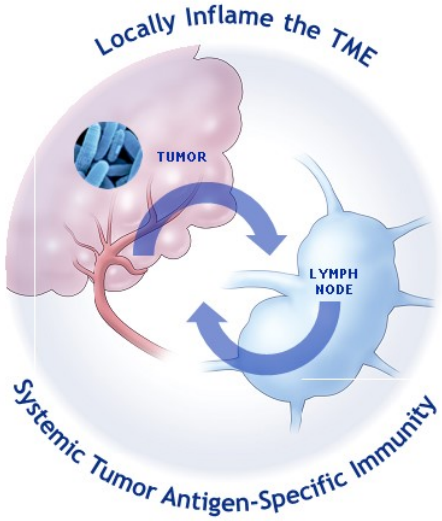
Consume immunosuppressive metabolites

Produce checkpoint inhibitors

Promote Trafficking

Chassis effect

Produce cytokines/chemokines



Promote and Sustain Immune Activation / Proliferation

Produce Immunostimulatory Molecules

Promote Immune Cell Survival and Activity

Prime for Tumor Antigen-Specific Vaccination

Chassis effect

Produce lytic factors

Produce agonists for immune cell activation

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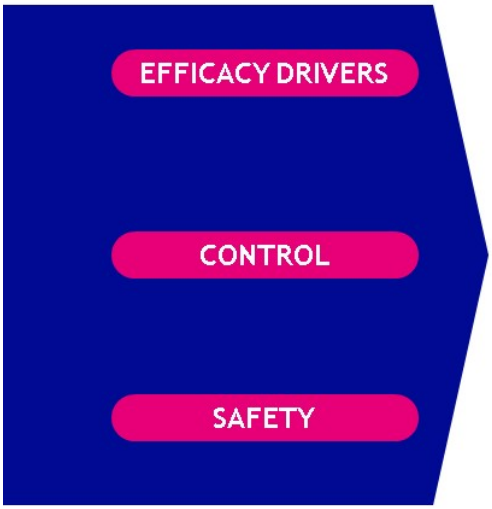
Synthetic Biotics Medicines Attributes

Platform Flexibility to Maximize Efficacy, Control, and Safety



Sustained Payload Delivery: Persistence in TME
Multiple Mechanisms: Large gene insertion capacity
Cellular Bioreactors: Enzymatic activity

Large Engineering Toolkit: Design to sense / respond to inducer
Manufacturability: No mammalian cell culture

Low Systemic Risk: Initial programs intratumoral
Non Pathogenic: Probiotic chassis, antibiotic deactivation

Intra-tumoral Injection of Synthetic Biotic Chassis: Tumor Colonization Without Leakage; Local Innate Immunity

Chassis Distribution

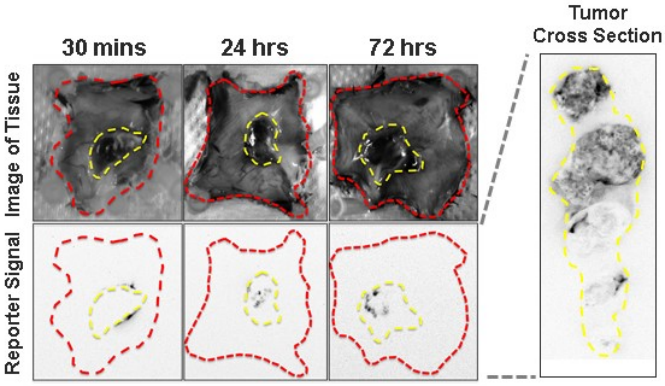
Robust proliferation in tumor.
No significant leakage to tissues

Survival/proliferation in tumors
10-15 days post-single dose.
Potential for limited injections

Elicits **innate responses** (e.g., IL-6, TNFα)
in the tumor, not in circulation

Behavior within TME

in B16.F10 Mice



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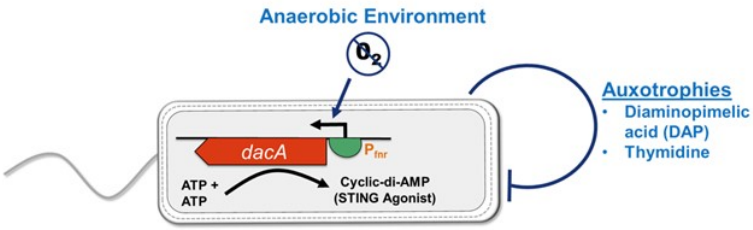
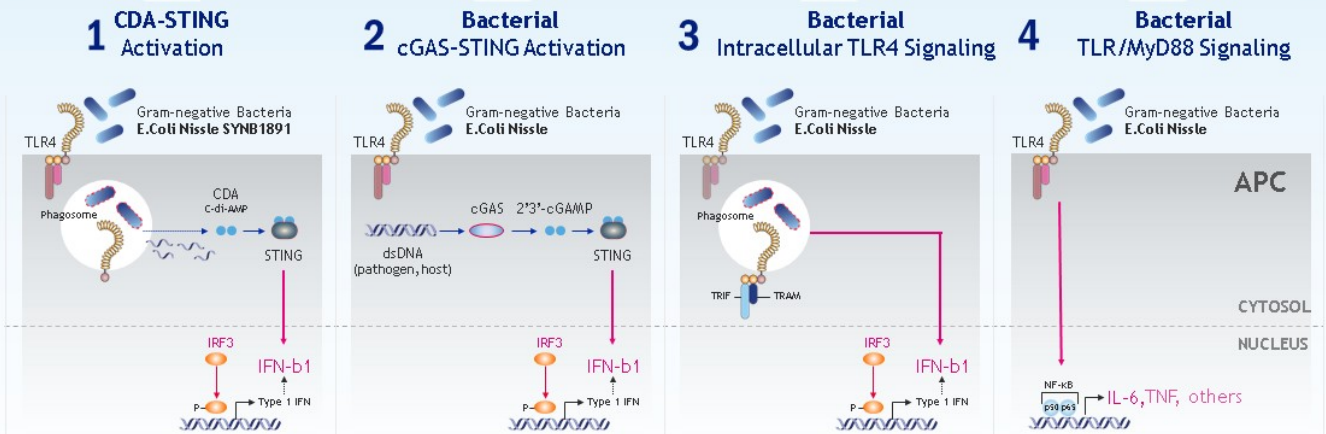
Dual Innate Immune Activator
Synthetic Biotic Medicine Producing STING Agonist: SYNB1891



- Synthetic biology applied to IO programs to confer activities for efficacy and control for safety
- SYNB1891 designed as a dual innate immune activator: Combined benefit of bacterial chassis and STING agonist
- *dacA* gene: Integrated into the genome under the control of inducible promoter (P_{fnr}) to produce c-di-AMP (CDA)
- Dual biosafety feature via auxotrophies
- Learnings inform future combinations

Dual Innate Immune Activator

TUMOR



	Promotes Trafficking, Immune Activation/Proliferation, Priming			
SYN-STING	✓	✓	✓	✓
Naked STING Agonist	✓	✓ ⊗	⊗	⊗

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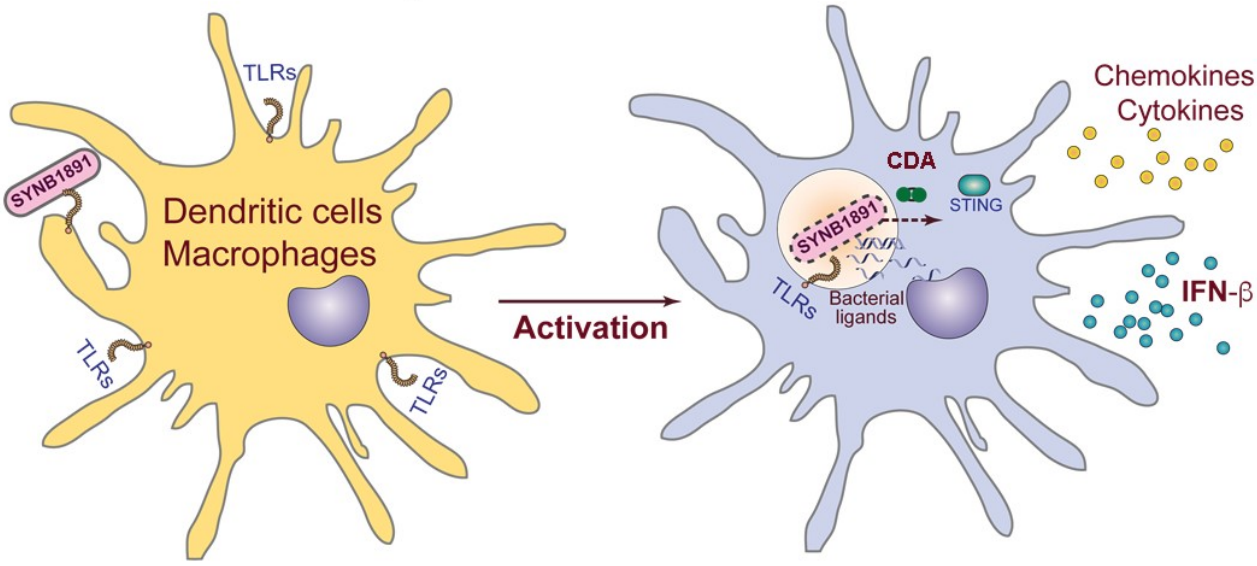
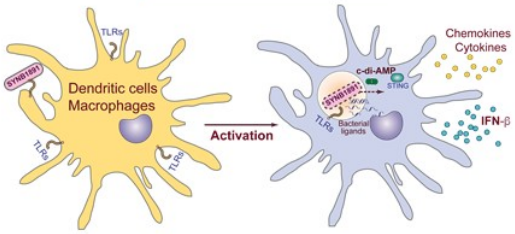
SYNB1891 Leverages Natural Phagocytic Activity of Antigen Presenting Cells

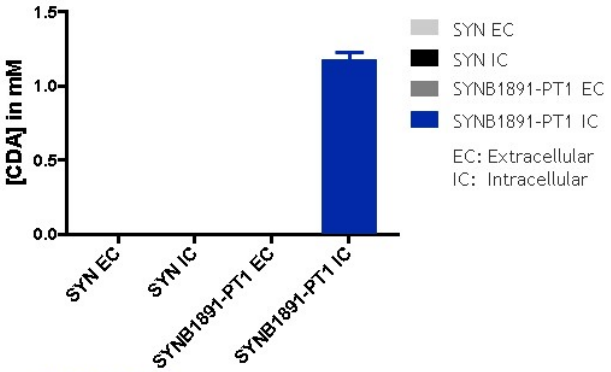
Agonizes STING in Natural Context



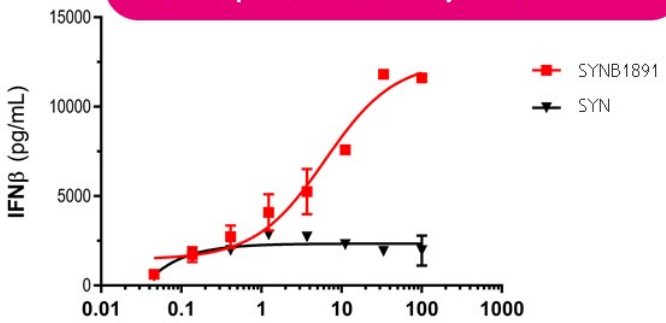
In Vitro Characterization of SYNB1891



STING Agonist Not Released to Extracellular Space



Dose-dependent Activity in Mouse APCs



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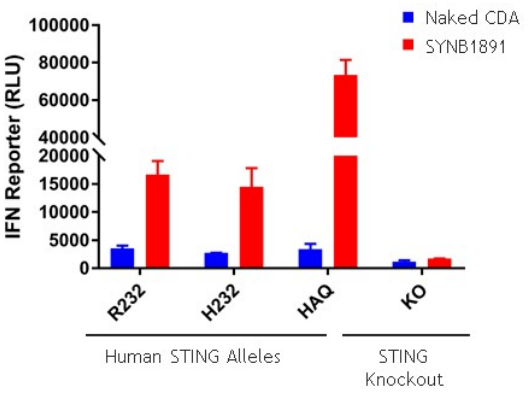
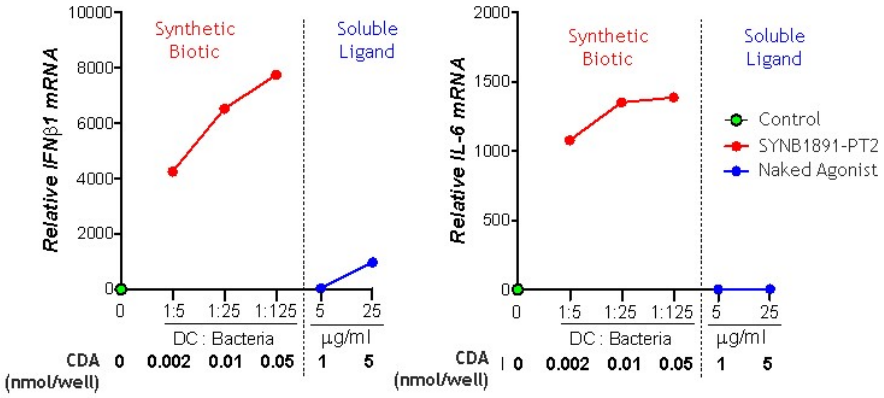
In Vitro Characterization of SYNB1891

Interferon Production Across Multiple Human STING Alleles Greater than Naked STING Agonist
Additional Proinflammatory Pathways Engaged

Reporter THP-1



Primary DCs



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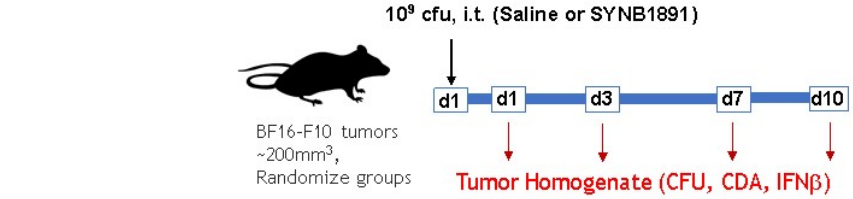
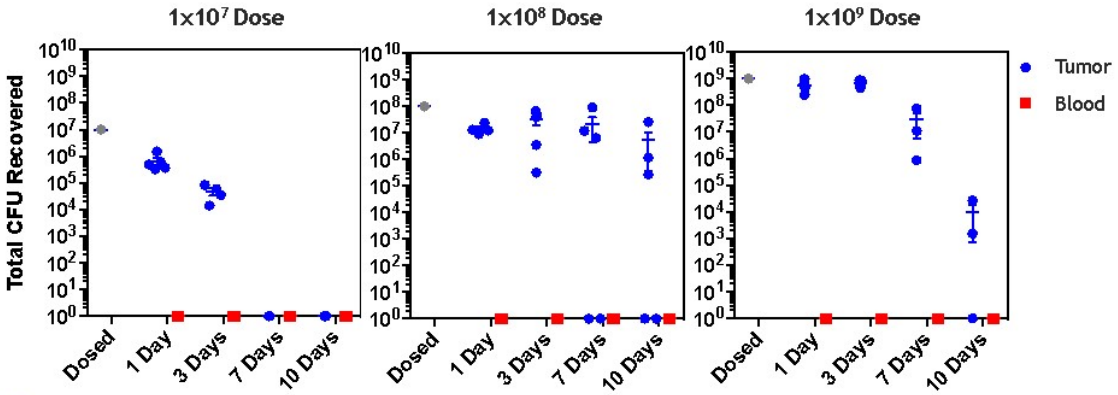
In Vivo Bacterial Kinetics of SYNB1891

Restricted to Tumor and Cleared Quickly

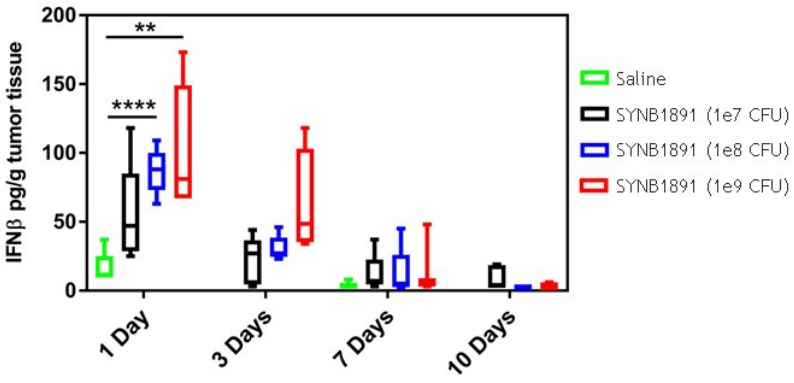




Pharmacodynamic Characterization of SYNB1891

Dose-dependent Increases in Tumoral IFNß and Other Innate Immune Markers

Dose Dependent IFNß Following 1 Injection



Additional Innate Immune Markers

Administration of SYNB1891 Results in Dose-dependent Increase in Tumoral CDA along with Innate Cytokines

Prototype Elicits Inflammation-related Gene Signature in Injected Tumors

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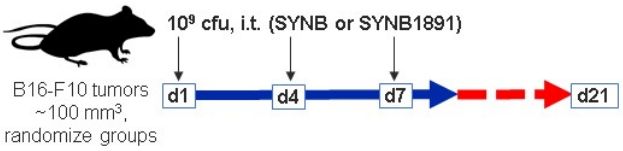
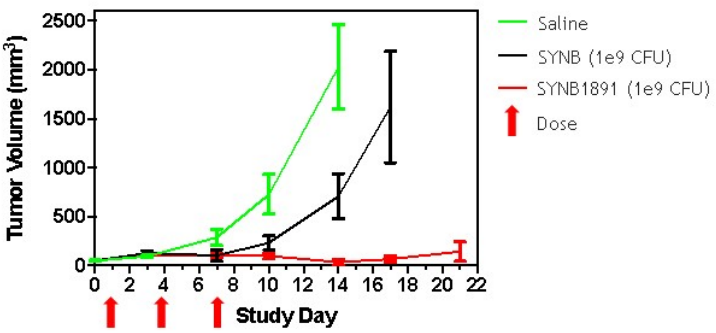
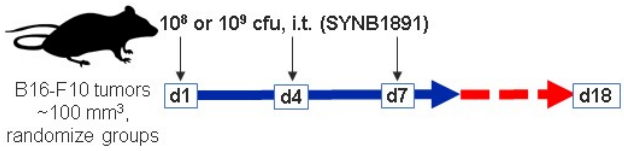
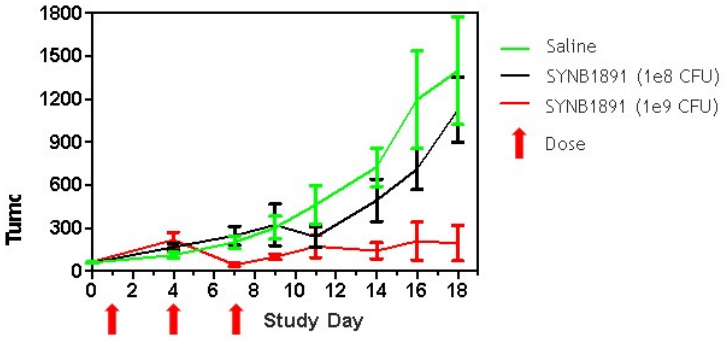
In Vivo Characterization of SYNB1891

Delivers Anti-tumor Activity as a Single Agent



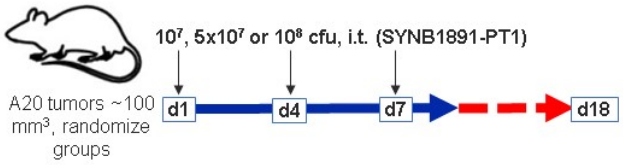


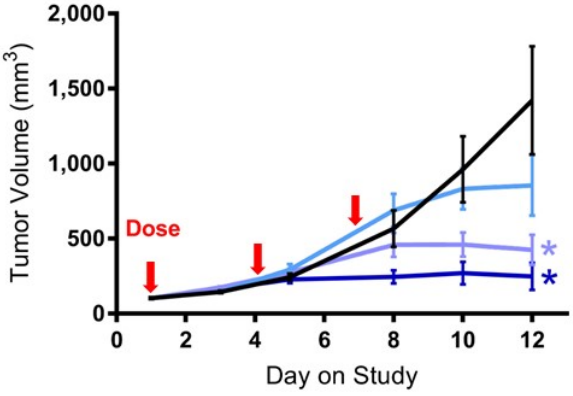


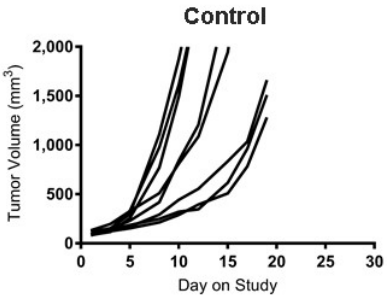


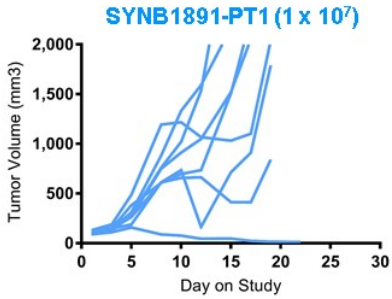
In Vivo Characterization of SYNB1891

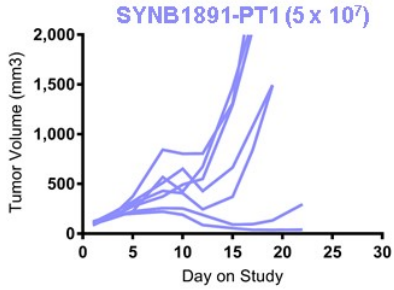
Dose-dependent Anti-tumor Activity of SYNB1891 Prototype Strain as a Single Agent

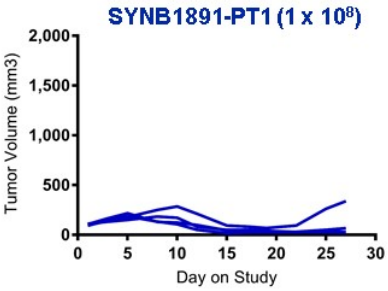












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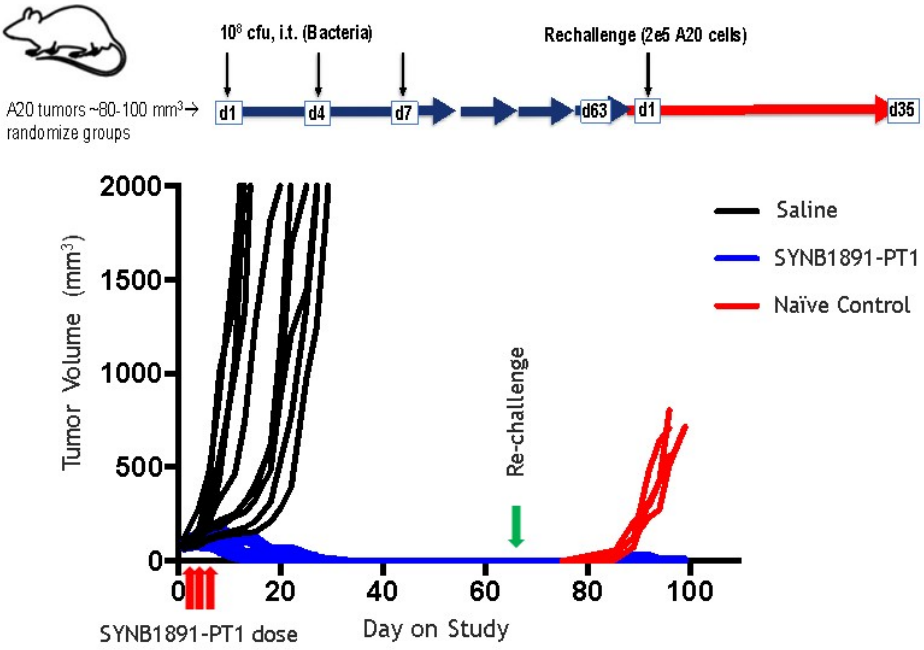
In Vivo Characterization of SYNB1891

SYNB1891 Prototype Strain Leads to Systemic Anti-tumor Immunity



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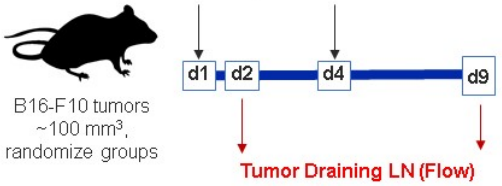
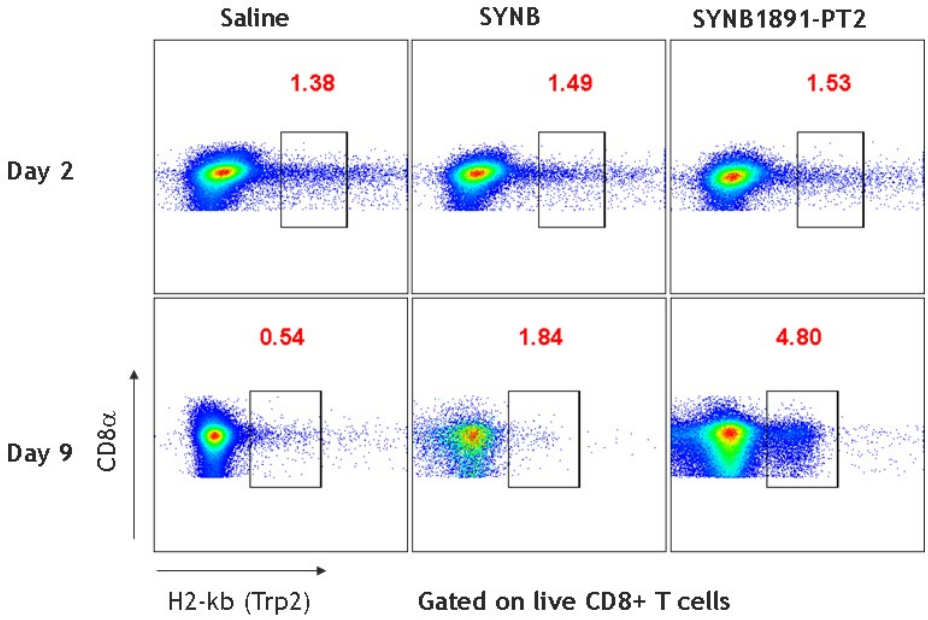
In Vivo Characterization of SYNB1891

SYNB1891 Prototype Strain Leads to Generation of Tumor Antigen-specific T Cell





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Dual Innate Immune Activator SYNB1891

A STING Agonist-producing Synthetic Biotic Designed to Locally Inflame the TME and Systemically Drive Tumor Antigen-Specific Immunity

Progress Towards the Clinic

Tumor Colonization without Leakage

Enhanced Activity vs. Naked STING Agonist

Intracellular Activation of STING and Bacterial-Induced Immune Pathways Within APCs

Dose-dependent Anti-tumor Activity

Immunological Memory

IND Submission 2H19

Promise Over Other Approaches

STING Agonism in Natural Context

Activation of Multiple Innate Immune Pathways

Low Systemic Risk

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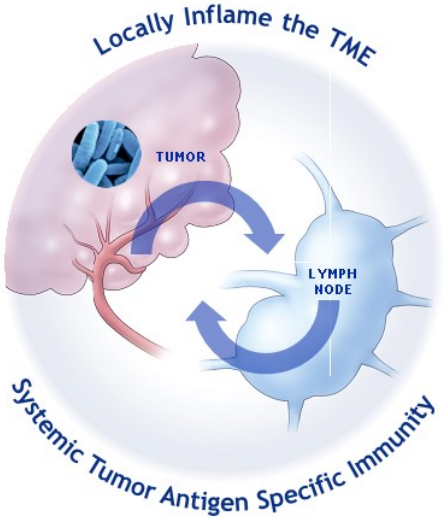
Dual Innate Immune Activator SYNB1891



NEXT STEPS

IND-Enabling Studies On-going

IND Submission 2H19

Pipeline of Synthetic Biotic Effectors Poised to Deliver

Relieve Immunosuppression

Kyn Consumption

Ade Consumption

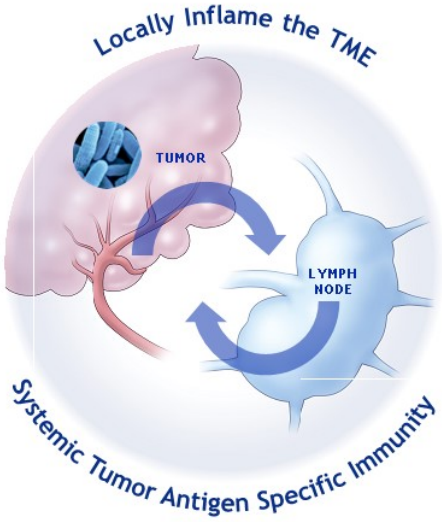
αPD-1 scFv

Promote Trafficking

Chassis effect

CXCL10

Hyaluronidase



Promote and Sustain Immune Activation / Proliferation

IL-15; IL-12

Arg Production

4-1BBL

OX40L

Prime for Tumor Antigen-Specific Vaccination

Chassis effect

5FC→5FU

STING

αCD40 scFv/CD40L

TNFα

IFNγ

αCD47 ScFv / Sirpα

GM-CSF

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Development of Synthetic Biotic™ Medicines in Oncology

Designed for life

Aoife Brennan, M.B., B.Ch.,
President and CEO

Jose M. Lora, PhD
Vice President, Research

SITC 2018- Washington, DC
November 10th, 2018

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Promise Over Other Approaches

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Low Systemic Risk

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Broad Ambitions in Immuno-Oncology

Vision: Expand and Exceed the Effect of Cancer Immunotherapies

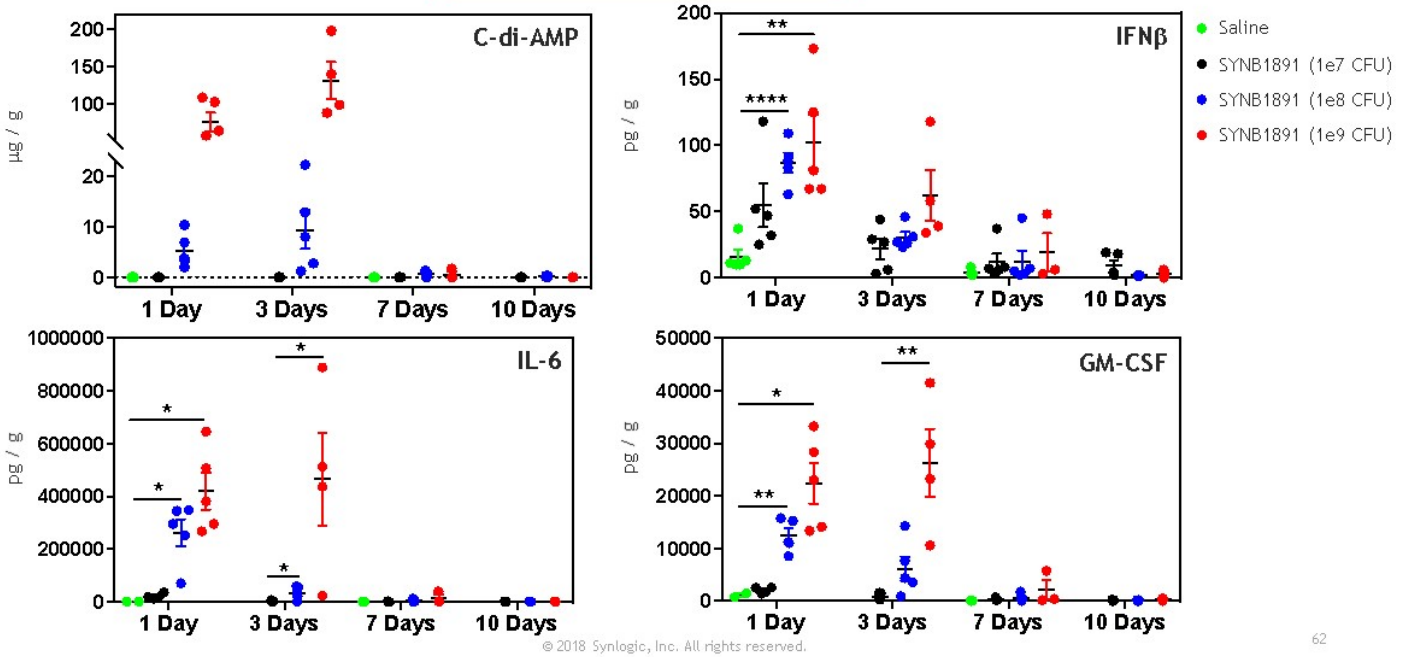
Table of Synthetic Biotic Strains

Strain	Genetic Content
SYN	Un-engineered *E. coli* Nissle:Abx+
SYNB	DAP/Thy dIn EcN (no dacA insert):Abx+
SYNB1891-PT1	DAP dIn EcN:$dacA_{plasmid}$:FnR-inducible:Abx+
SYNB1891-PT2	DAP/Thy dIn EcN:$dacA_{integrated}$:FnR-inducible:Abx+
SYNB1891	DAP/Thy dIn EcN:$dacA_{integrated}$:FnR-inducible:Abx-

Pharmacodynamic Characterization of SYNB1891

Administration of SYNB1891 Results in Dose-dependent Increases in Tumoral CDA, Cytokines

Production in Tumor Tissue



Pharmacodynamic Characterization of SYNB1891

Prototype Elicits Inflammation-related Gene Signature in Injected Tumors

B16-F10 or CT-26 tumors ~100 mm^3, randomize groups

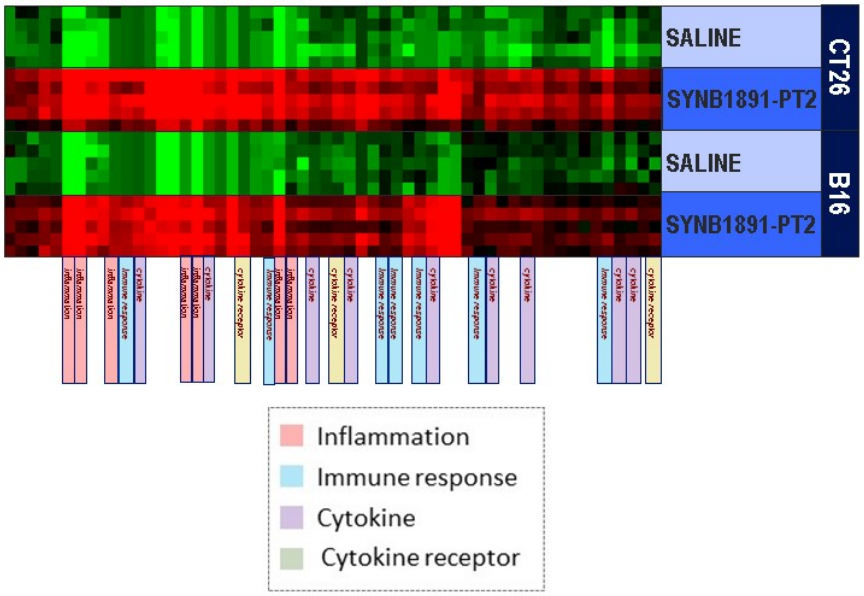
Saline or 10^9 cfu SYNB1891-PT2, i.t.

d1 — d4

Harvest Tumor Tissue

Isolate Tumor Infiltrating Lymphocytes

Perform RNAseq on Purified T cells



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