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

ADURO BIOTECH, INC.
(Exact name of Registrant as Specified in its Charter)

Delaware	**001-37345**	**94-3348934**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(I.R.S. Employer Identification No.)**

740 Heinz Avenue
Berkeley, California 94710
(Address of Principal Executive Offices) (Zip Code)

Registrant's telephone number, including area code: (510) 848-4400

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 communication 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, par value $0.0001 per share	**ADRO**	**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 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by 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 8.01. Other Events.

Attached hereto as Exhibit 99.1 is an investor presentation that Aduro Biotech, Inc. plans to present during the 38th Annual J.P. Morgan Healthcare Conference commencing on January 13, 2020.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Aduro Biotech, Inc. Investor Presentation.

SIGNATURE

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.

ADURO BIOTECH, INC.

Dated: January 13, 2020

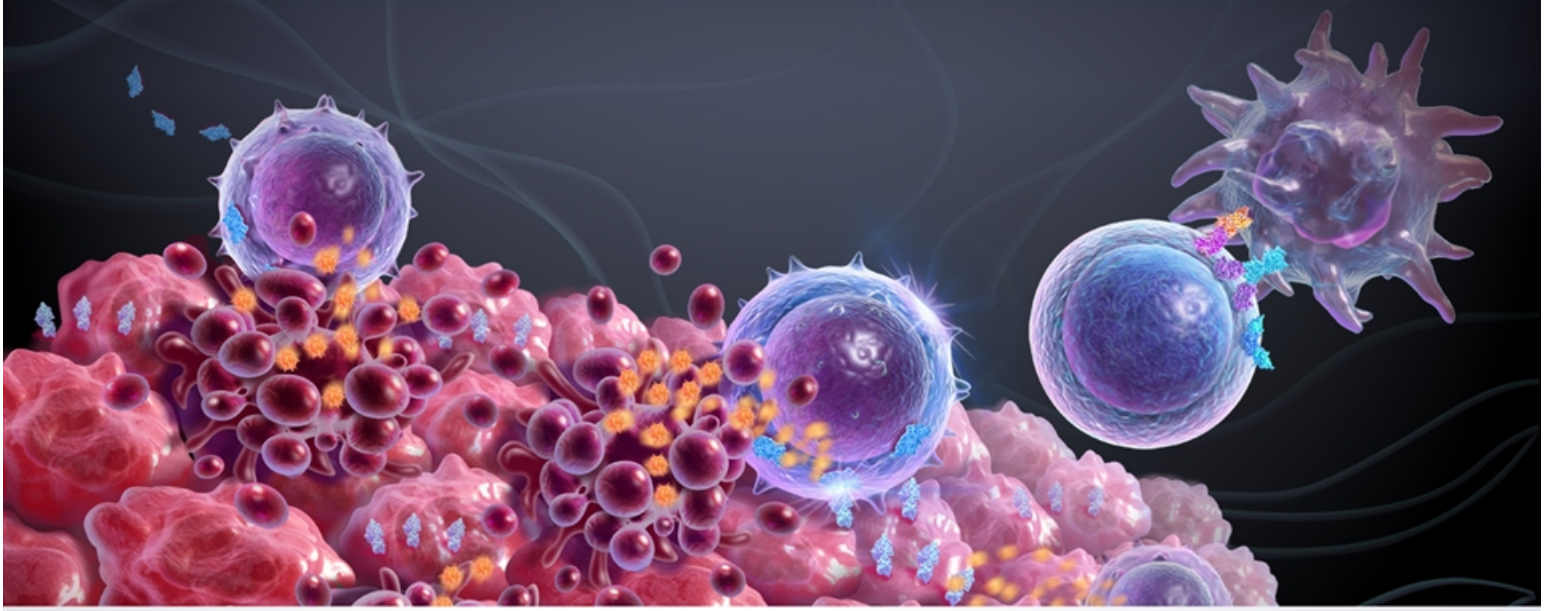
By: /s/ Celeste Ferber
Name: Celeste Ferber
Title: SVP, General Counsel and Secretary

Exhibit 99.1

Corporate Presentation

January 2020

ADURO BIOTECH™



DISCOVERING & DEVELOPING THERAPIES FOR CHALLENGING DISEASES

Note Regarding Forward-Looking Statements

This presentation and the accompanying oral presentation include express and implied forward-looking statements regarding the current intentions, expectations, estimates, opinions and beliefs of Aduro Biotech, Inc. ("Aduro") that are not historical facts. These forward-looking statements include statements regarding Aduro's expectations for its product candidates (including their therapeutic and commercial potential, anticipated future development activities, anticipated timing of development activities, including initiation of clinical trials and presentations of clinical data and the indications Aduro and its collaborators plan to pursue), future results of operations and financial position, including current funds providing operating capital into 2022 and the recent restructuring resulting in reductions in operating expenses and an extended cash runway, business strategy, strategic collaborations, any royalty or milestone payments and Aduro's ability to obtain and maintain intellectual property protection for its product candidates. Such forward-looking statements may be identified by words such as "believes," "may," "will," "expects," "endeavors," "anticipates," "intends," "plans," "estimates," "projects," "should," "objective" and variations of such words and similar words. Forward-looking statements are not guarantees of future performance and are subject to risks and uncertainties that could cause actual results and events to differ materially from those anticipated, including, but not limited to, Aduro's history of net operating losses and uncertainty regarding its ability to achieve profitability, Aduro's ability to develop and commercialize its product candidates, Aduro's ability to use and expand its technology to build a pipeline of product candidates, Aduro's ability to obtain and maintain regulatory approval of its product candidates, Aduro's ability to operate in a competitive industry and compete successfully against competitors that have greater resources than it does, Aduro's reliance on third parties, Aduro's ability to execute on its corporate strategy, including the success of the restructuring and Aduro's ability retain senior management and other highly qualified personnel, and Aduro's ability to obtain and adequately protect intellectual property rights for its product candidates. Aduro discusses many of these risks in greater detail under the heading "Risk Factors" in its most recent Quarterly or Annual Report on Form 10-Q or Form 10-K filed with the Securities and Exchange Commission. Any forward-looking statements that Aduro or any presenter makes in this presentation and the accompanying oral presentation speak only as of the date of these presentations. Except as required by law, Aduro assumes no obligation to update its forward-looking statements whether as a result of new information, future events or otherwise, after the date hereof.

Nothing contained in this presentation is, or should be construed as, a recommendation, promise or representation by the presenter or Aduro or any director, employee, agent or adviser of Aduro. This presentation does not purport to be all-inclusive or to contain all of the information you may desire.

Developing Therapies Targeting STING & APRIL Pathways for Oncology, Autoimmune & Inflammatory Diseases

Leaders in cGAS-STING & APRIL Pathways

- STING agonist in oncology
- cGAS-STING inhibitors in autoimmune & inflammatory diseases
- Anti-APRIL antibody in IgA nephropathy
- Comprehensive preclinical characterization

Strategic Alliances

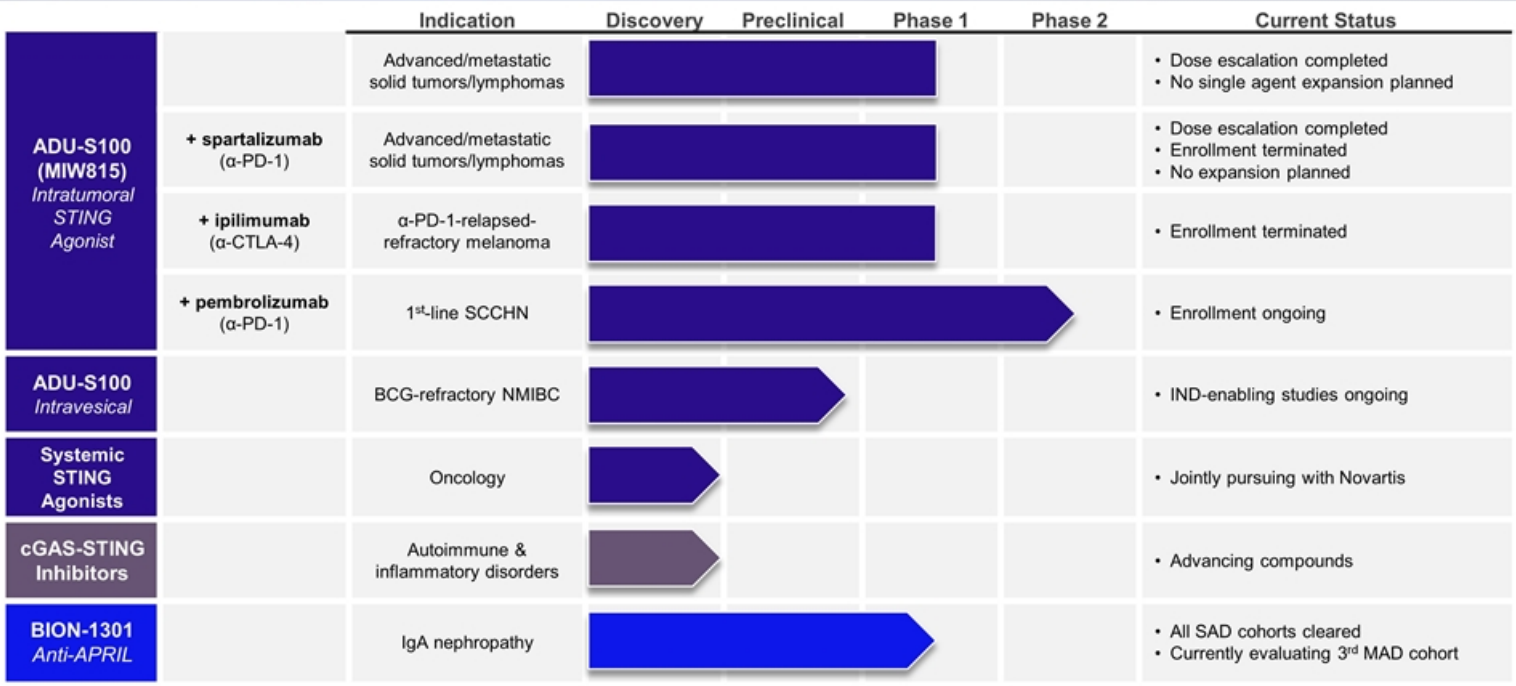
- Co-development and co-commercialization partnership with NVS
- Potential near-term development milestones

MERCK Lilly NOVARTIS

Financial Strength

- $235M at end of Q3 2019
- Operating capital into 2022

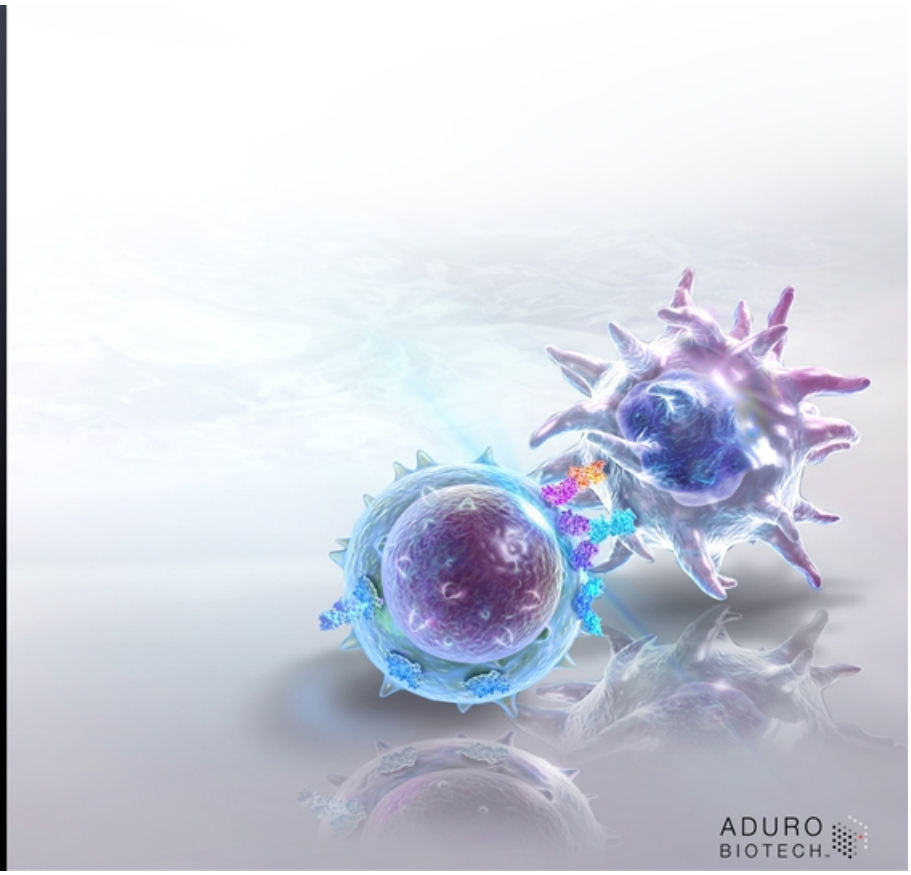
Aduro Pipeline



 SCCHN = squamous cell carcinoma of the head and neck; BCG = Bacillus Calmette-Guerin; NMIBC = non-muscle invasive bladder cancer

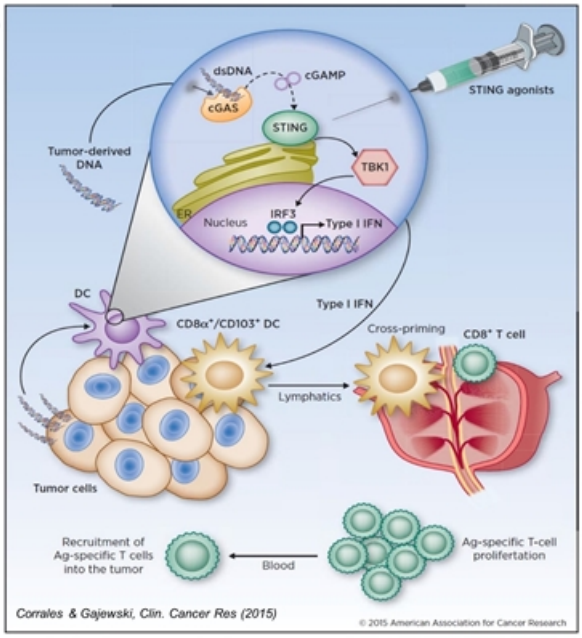
ADU-S100 (MIW815)

Intratumoral STING agonist



ADU-S100 (MIW815) Demonstrated Preclinical Anti-Tumor Activity

- STING activation in the hematopoietic compartment drives ADU-S100 (MIW815) dependent anti-tumor immunity
  - Type I IFN is required for optimal anti-tumor immune response
- ADU-S100 (MIW815) induces both local innate immune activation in injected tumors (cytokine production) and activates tumor specific CD8+ T cells
  - Bridging innate to adaptive immunity
- Dose level impacts induction of ADU-S100 (MIW815) tumor-specific T cell response
  - Immunogenic dosing regimens induce systemic and durable adaptive tumor immunity
- **ADU-S100 (MIW815) is an effective combination agent with checkpoint inhibition to enhance anti-tumor efficacy and durable immunity**



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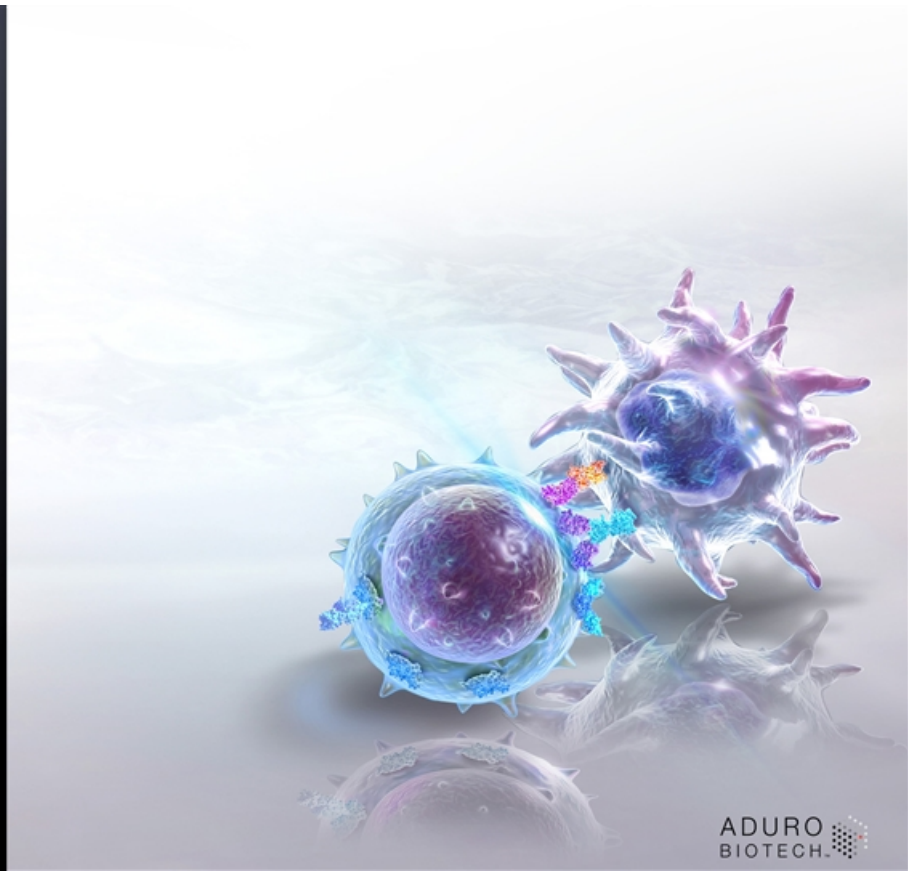
ADU-S100 (MIW815)
Monotherapy



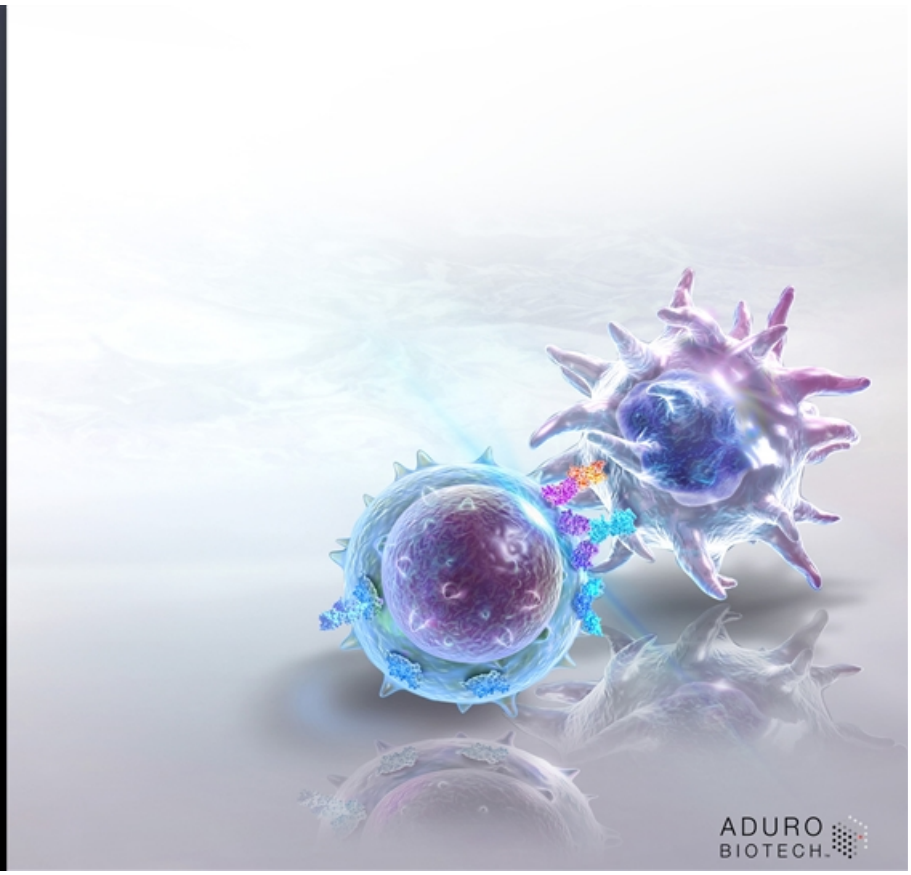
Data Highlights from Phase 1 ADU-S100 (MIW815) Monotherapy Trial 50 µg – 3200 µg

Good safety profile	Well tolerated in heavily pre-treated, heterogenous patient population
Preliminary signs of clinical and biomarker activity	• 2 of 41 patients treated had a PR, one patient received prior anti-PD-1 therapy • 11 patients achieved SD
Target engagement	Observed dose-dependent increase in IFN-ß after ADU-S100 (MIW815) administration, indicating target engagement and activation of the STING pathway

Data Cut-off: August 16, 2018

Meric-Bernstam F, et al. SITC 2018 poster presentation: abstract P309.

ADU-S100 (MIW815)
+ spartalizumab
(α-PD-1)



Preliminary Anti-tumor Activity

ADU-S100 (MIW815) 3-weeks-on/1-week-off

Five confirmed responses (4 PR + 1 CR)

- Two PR and one CR in patients with IO-naive TNBC
  - At time of data cut, these patients were on treatment for >6 months
  - Two of these patients expressed PD-L1 levels of >1% at baseline (data from third TNBC patient not available)
- Two PR in patients with previously IO-treated melanoma

Additional 12 patients achieved SD

- Tumor types: Sarcoma, melanoma, SCC skin, breast, lymphoma, and head and neck

ADU-S100 (MIW815) monthly

No patients achieved a response; however, **six patients achieved SD**

- Tumor types: Ovarian, breast, uveal melanoma, head and neck, and cutaneous melanoma
- Four of whom maintained SD for ≥6 months

CRC, colorectal cancer; SCC, squamous cell carcinoma.

Data cut-off: April 5, 2019

Meric-Bernstam F, et al. ASCO 2019 oral presentation: abstract 2507.

Data Highlights from Phase 1 ADU-S100 (MIW815) + Spartalizumab 50 µg – 1600 µg

Good safety profile

- ADU-S100 (MIW815) + spartalizumab was generally well tolerated in patients with solid tumors or lymphomas, with no DLTs reported as of the data cut-off
- The MTD was not reached

Preliminary signs of clinical and biomarker activity

- The combination has demonstrated anti-tumor activity in PD-1–naive TNBC and PD-1–relapsed/refractory melanoma
- Three out of eight evaluable anti-PD-1-naïve-TNBC patients had a response (2 PRs + 1 CR)
- Two out of 25 evaluable melanoma patients had a response (2 PRs)

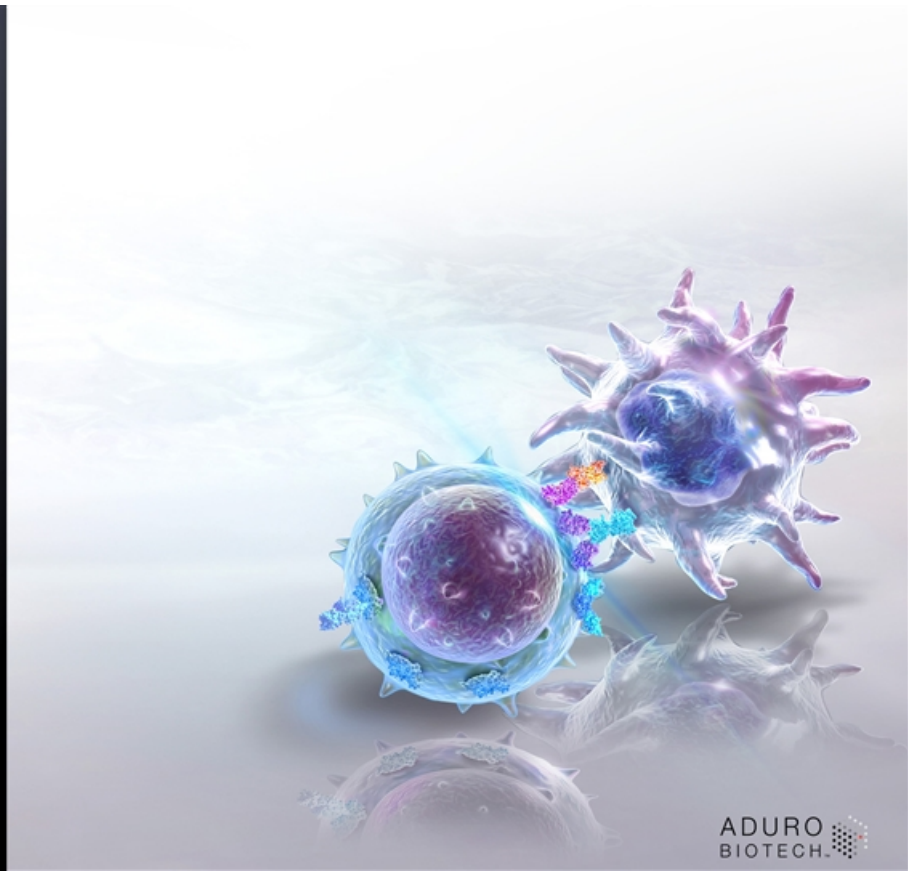
Target engagement

- ADU-S100 plasma exposure increases dose-proportionally
- IFN-β concentrations appeared to increase with increasing exposure to ADU-S100 (MIW815)

Data Cut-off: April 5, 2019

Meric-Bernstam F, et al. ASCO 2019 oral presentation: abstract 2507.

ADU-S100 (MIW815)
+ pembrolizumab (α-PD-1) in First-Line SCCHN



Phase 2 Ongoing:
ADU-S100 (MIW815) + Pembrolizumab in First-Line SCCHN

- Shift from heavily pre-treated, heterogenous patient populations to earlier lines of treatment for patients with specific tumor type
- Increasing evidence that potential benefit from immunotherapies may be greater in patients with fewer prior therapies
- Advance to N = 34 based on ORR for N = 20 (Simon 2-stage criteria)
- Looking for an approximate **doubling** of the response rate with pembrolizumab alone:
  - ORR for pembrolizumab in KEYNOTE-048:
    - 19% in patients with CPS $\geq$1
    - 23% in patients with CPS $\geq$20

Merck & Co., Inc. (2019, June 11). *FDA Approves Two New Indications for Merck's KEYTRUDA® (pembrolizumab)* [Press Release]. Retrieved from https://investors.merck.com/news/press-release-details/2019/FDA-Approves-Two-New-Indications-for-Mercks-KEYTRUDA-pembrolizumab/default.aspx

Phase 2 Clinical Trial Design: ADU-S100 (MIW815) + Pembrolizumab in First-Line SCCHN

Phase 2 (N=34)

Patients: Adults with recurrent or metastatic SCCHN, first-line setting

Primary Objective: Evaluation of clinical activity by ORR per Response Evaluation Criteria in Solid Tumors (RECIST) v1.1

Key Inclusion Criteria:
- Histological or cytological confirmation of recurrent or metastatic SCCHN
- Measurable disease as defined by RECIST v1.1
- PD-L1 positive (CPS $\geq$1)

Key Exclusion Criteria:
- Diagnosis of recurrent or metastatic carcinoma of the nasopharynx, squamous cell carcinoma of unknown primary histology; or salivary gland or non-squamous histologies
- Disease amenable to local therapy with curative intent
- Prior systemic anti-cancer therapy for the treatment of recurrent or metastatic HNSCC

Screening Period

Treatment Period

ADU-S100: Fixed dose IT injection, up to two lesions

Pembrolizumab: 200 mg IV infusion

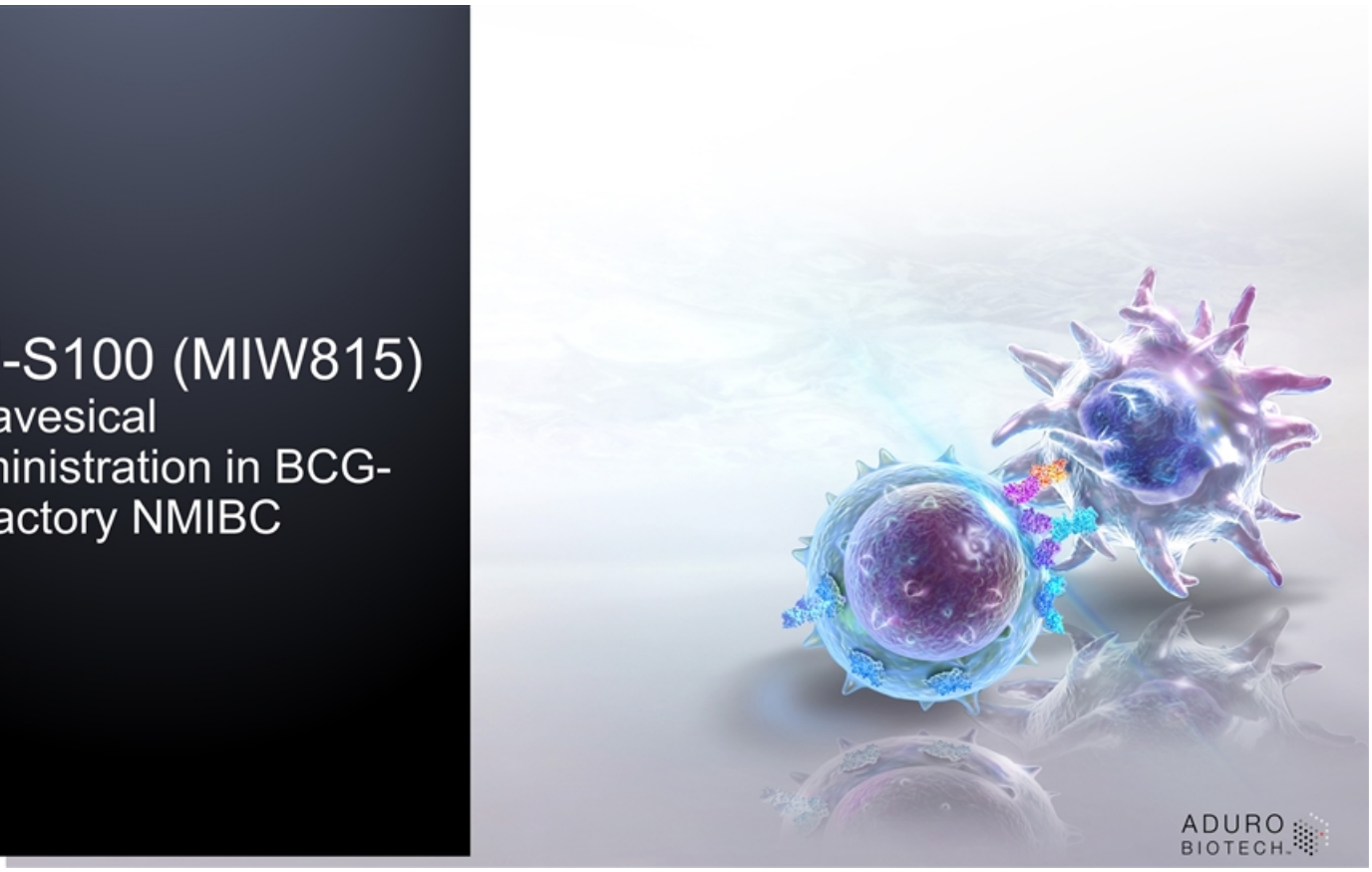
Follow-up Period

Safety reporting
Subsequent cancer treatment
Survival

Patients are treated until confirmed disease progression, unacceptable toxicity or 35 cycles

ADU-S100 (MIW815)

Intravesical administration in BCG-refractory NMIBC



Rationale for Intravesical ADU-S100 in NMIBC

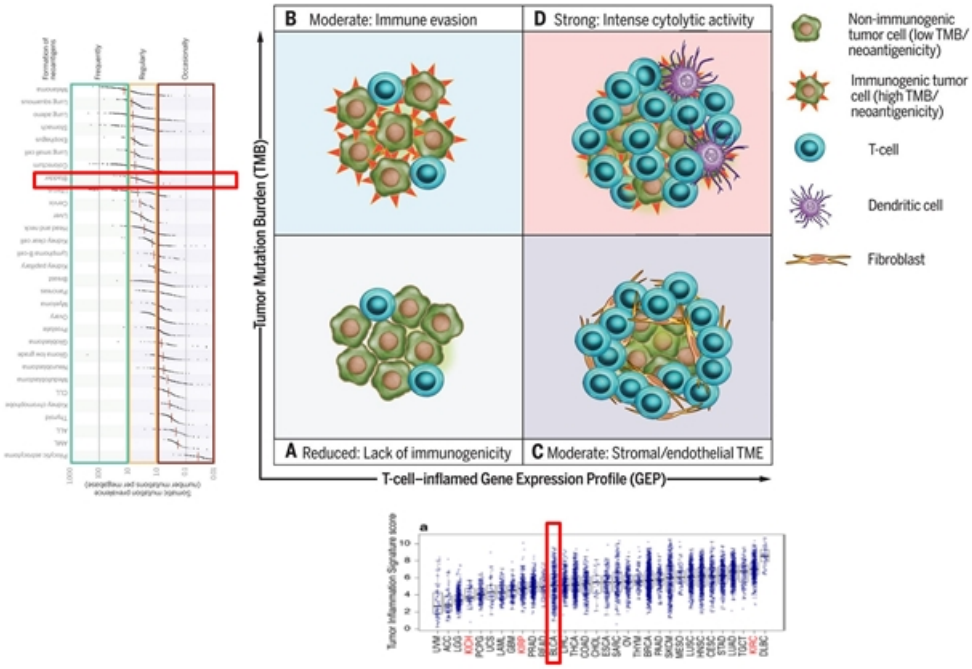
Scientific

- Local, superficial disease in which local control drives disease outcome
  - Ablation of the tumor may be sufficient to control disease, anenestic response less important
- Similar to BCG, ADU-S100 is a small molecule with potential to induce a local innate immune response
  - BCG refractory patients have a TME enriched with ADU-S100 targets
- Mechanisms leading to BCG refractory state may not interfere with response to ADU-S100, e.g., M2 macrophages, receptor loss

Clinical Development

- Potential for single agent therapeutic application of ADU-S100
- Intravesical administration attractive alternative to systemic therapy
- BCG-unresponsive patients constitute a high unmet medical need recognized by the FDA with approval pathway outlined in guidance

Bladder Cancer is a “Hot” Tumor with High TMB & Inflammatory GEP



High mutational burden rate for potential immunogenicity

Schumacher et al. Science. 2015.

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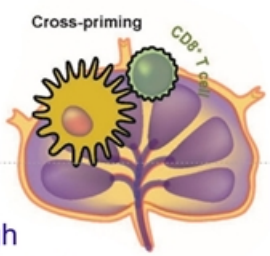
Scientific Rationale for ADU-S100 in BCG refractory NMIBC

1. BCG refractory patients have increased macrophages, S100 targets and turns on **cytokine production**

2. ADU-S100 is a strong innate immune stimulant that may drive the **exfoliation response** induced by infection/BCG

3. Bladder tumor cells have increased expression of a transporter for ADU-S100 compared to normal cells, may cause selective **tumor killing**



4. Relatively high mutational burden rate, potential immunogenicity **CD8+ T cell activation**

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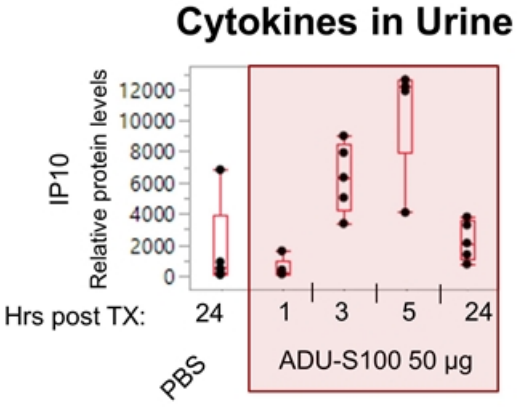
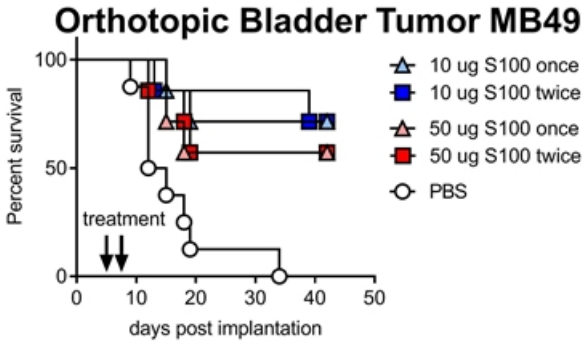
ADU-S100 Activates an Immune Response in the Bladder after Intravesicular Delivery and Controls Tumors in Preclinical Models

Preclinical efficacy in standard orthotopic model, MB49 bladder tumor

- Observe local cytokine induction with intravesicular delivery of ADU-S100
- Preliminary data demonstrates anti-tumor efficacy





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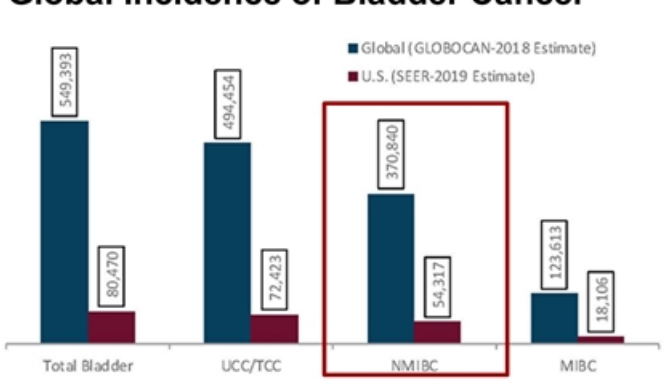
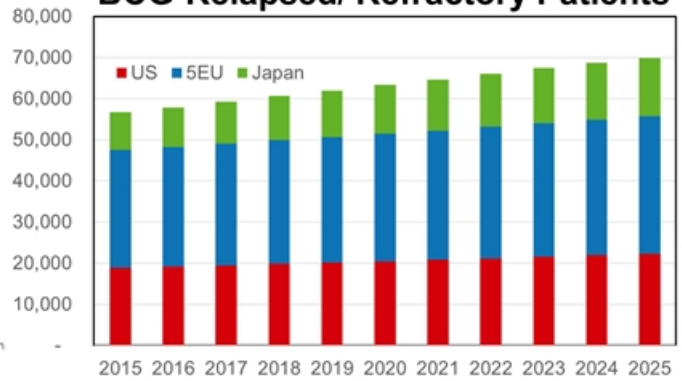
Major Unmet Need in BCG-Unresponsive NMIBC

Global Incidence of Bladder Cancer



BCG-Relapsed/ Refractory Patients*



- BCG treatment will eventually fail in up to 50% of patients
- Due to worldwide shortage, only 50% of patients who need BCG receive treatment

Kamat, A., Flaig, T., Grossman, H. et al. Consensus statement on best practice management regarding the use of intravesical immunotherapy with BCG for bladder cancer. Nat Rev Urol 12, 225–235 (2015) doi:10.1038/nrurol.2015.58

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Valrubicin is the Only Approved Option for BCG Refractory CIS when Cystectomy is Not an Option and Few Ongoing Clinical Studies

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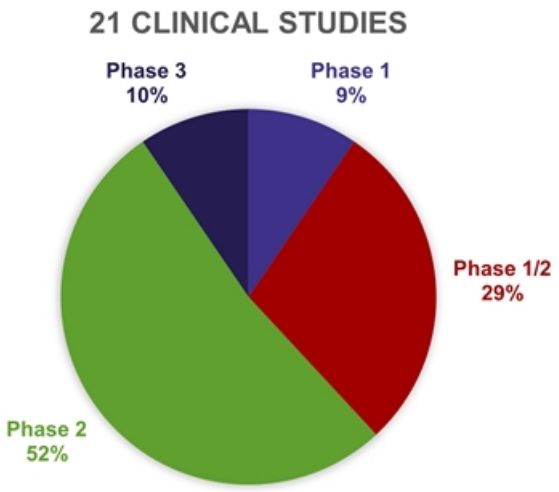
Vol. 163, 761–767, March 2000
Printed in U.S.A.

EFFICACY AND SAFETY OF VALRUBICIN FOR THE TREATMENT OF BACILLUS CALMETTE-GUERIN REFRACTORY CARCINOMA IN SITU OF THE BLADDER

GARY STEINBERG, ROBERT BAHNSON,* STANLEY BROSMAN, RICHARD MIDDLETON, ZEV WAJSMAN,† MICHAEL WEHLE AND THE VALRUBICIN STUDY GROUP‡

From the University of Chicago Hospitals, Chicago, Illinois, Ohio State University, Columbus, Ohio, Santa Monica Urologic Group, Santa Monica, California, University of Utah Medical Center, Salt Lake City, Utah, and University of Florida Medical College, Gainesville and Department of Urology, Mayo Clinic, Jacksonville, Florida

- Single-arm study of patients with BCG failure
  - 70% with ≥2 prior BCG courses
  - 80% with 2 – 24 month interval between last treatment and valrubicin
- DFR 6 months = 21%
- Suboptimal salvage therapy for BCG failure



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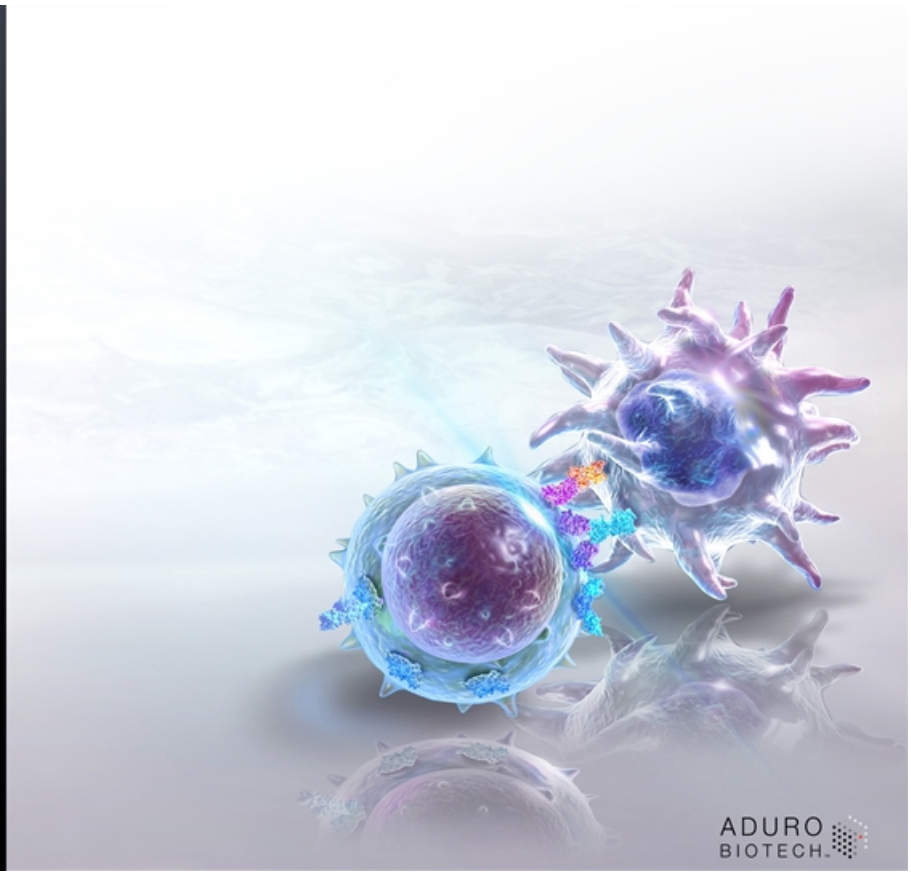
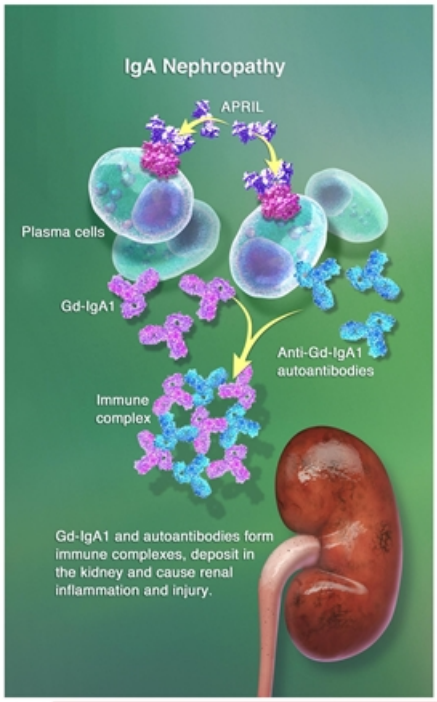
BION-1301
Anti-APRIL Antibody



IgA Nephropathy: Biology



IgA nephropathy (IgAN) is a chronic autoimmune disease associated with inflammation in the kidneys that diminishes their ability to filter blood

↓

A key breakthrough was identification of IgA1 molecules lacking glycosylation at the hinge region, often referred to as galactose-deficient IgA1 (Gd-IgA1)

↓

In patients with IgAN, Gd-IgA1 gives rise to autoantibody production

↓

Gd-IgA1–autoantibody complexes deposit in the kidneys, resulting in complement activation, inflammation and subsequent renal damage

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IgA Nephropathy: Epidemiology

- **IgAN is the most frequent biopsy-proven primary glomerular disease, but the geographic distribution varies widely**
  - Clinical findings that trigger biopsy vary by country
  - Biopsy registry data **underestimate disease burden** as patients with mild disease may not undergo biopsy, and in countries lacking screening programs, disease may not be detected
- **Because the diagnosis of IgAN requires a kidney biopsy, the exact disease prevalence remains difficult to establish**
- **US prevalence is estimated to be ~130,000 – 150,000**
- **UK prevalence is estimated to be ~10,000 – 15,000**
- **Asian populations have a higher incidence rate of IgAN, with ~75,000 new cases diagnosed annually in China**

Schena FP, Nistor I, Semin Nephrol 2018, 38(5):435-442; Magistroni et al., Kidney Intl (2015) 88:974-89;
Ilyas M. Pediatric IgA nephropathy. Medscape. 2017. https://emedicine.medscape.com/article/981516-overview.

IgA Nephropathy: Diagnosis & Disease Progression

Diagnosis

- Patients often diagnosed in their 20s or 30s
- Diagnosis, **which requires biopsy**, often made following GI or respiratory infection with observation of macroscopic hematuria
- When screened, up to half of patients with microscopic hematuria found to have IgAN
- Routine screening of asymptomatic patients results in a high % of patients with IgAN

Disease Progression

- 30% to 40% of patients with IgAN progress to ESRD within approximately 20 years
- Key risk factors for progression are blood pressure, proteinurea, kidney histology and eGFR
- High-risk patients – based on blood pressure, proteinuria and eGFR – have significantly increased mortality

IgA Nephropathy: Treatment

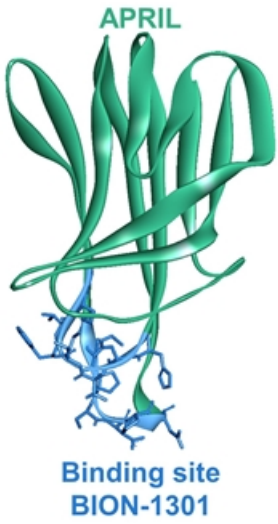
- **There are no approved treatments for IgAN**
- **Current standard of care treatment includes:**
  - Controlling hypertension (via renin-angiotensin blockade), which can reduce proteinurea and slow disease progression
  - Immunosuppressive drugs (e.g., corticosteroids for 6 months), which have very severe and long-lasting side effects and inconclusive long-term benefits
  - Fish oil and tonsillectomy (in Japan), which have shown inconclusive benefit
- **A significant portion of patients will progress despite blood pressure control**
- **No treatment available that selectively reduces the production of Gd-IgA1 and the anti-Gd-IgA autoantibodies that give rise to IgAN**

The need for an effective treatment for IgA Nephropathy is clear

APRIL and its Role in IgAN

A Prolife**r**ation **I**nducing **L**igand, or **APRIL:**

- TNF-family ligand implicated in regulation of B cell-mediated immune responses
- Soluble factor that functions via binding to TACI and BCMA receptors
- Critically drives → IgA class switching through TACI
  → Survival of IgA-producing plasma cells through BCMA



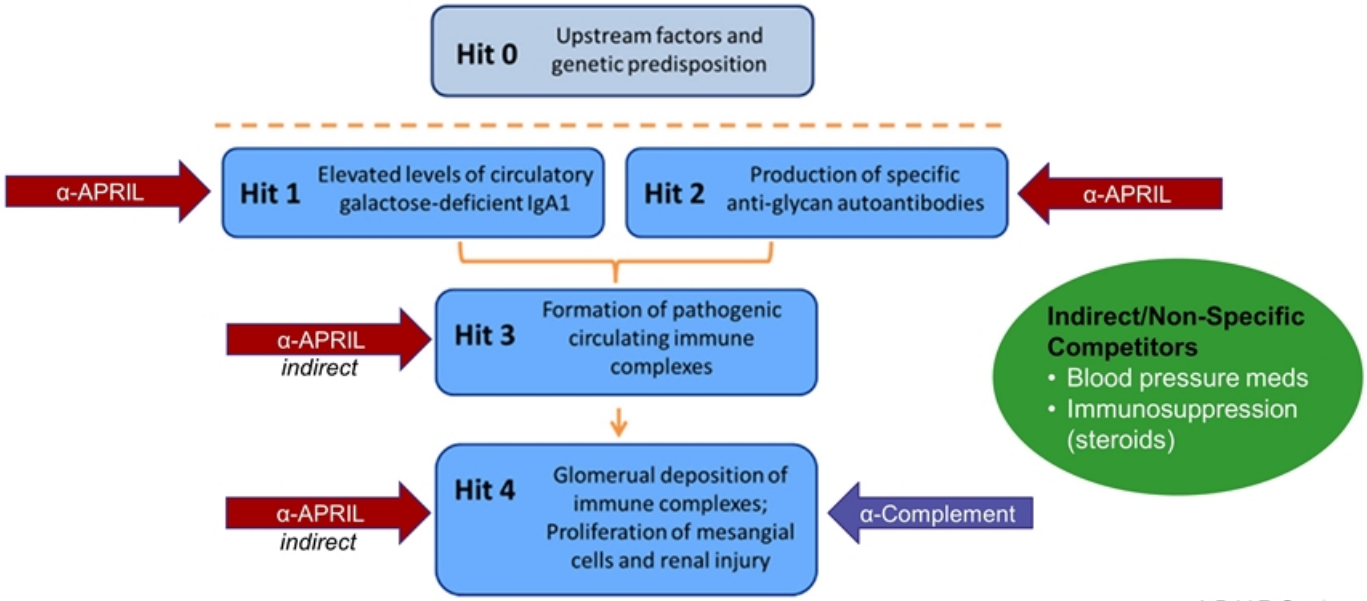
- Patients with IgAN have significantly higher levels of APRIL than healthy controls
- Higher APRIL levels in IgAN patients correlate with poor prognosis:
  - ↑ Gd-IgA1, ↑ proteinuria and ↓ eGFR
- When APRIL is added to lymphocytes from IgAN patients, Gd-IgA1 significantly ↑
- A polymorphism in the APRIL gene confers IgAN susceptibility

Blocking APRIL is a distinct approach to reduce circulating levels of IgA, Gd-IgA, anti-Gd-IgA autoantibodies and immune complex formation

Dulos J, et al. Blood 2016 128:2112;

APRIL Pathway Agents Uniquely Positioned in 4 Hit Hypothesis on the Pathogenesis of IgA Nephropathy

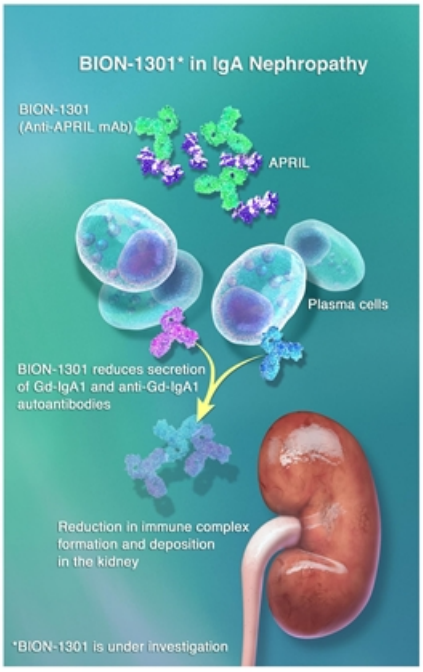
BION-1301 should impact all four categories of IgAN pathogenesis, and in the future could potentially be used in combination with drugs that have different MOAs



Adapted from: Suzuki, Clinical and Experimental Nephrology (2018), Barratt et al. Pediatr Nephrol (2018), Penfold et al. International Journal of Nephrology and Renovascular Disease 2018 modified.

BION-1301: First-in-Class Fully Blocking Anti-APRIL Antibody

- **First-in-class monoclonal antibody blocking APRIL binding to both of its natural ligands, the BCMA and TACI receptors**
- **No toxicology findings observed in 1-month toxicology study**
- **Well-tolerated in clinical studies** (NCT03340883, NCT03945318)
  - Well-tolerated up to 2700 mg IV in a Phase 1 safety and PK/PD study of late stage multiple myeloma patients
- **Animal studies demonstrate that treatment with BION-1301 reduces production of IgA, IgG and IgM**



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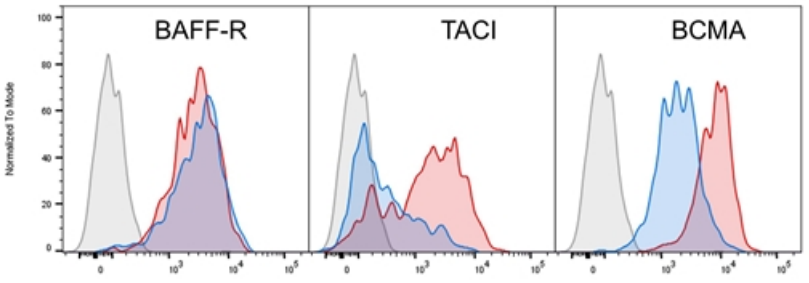
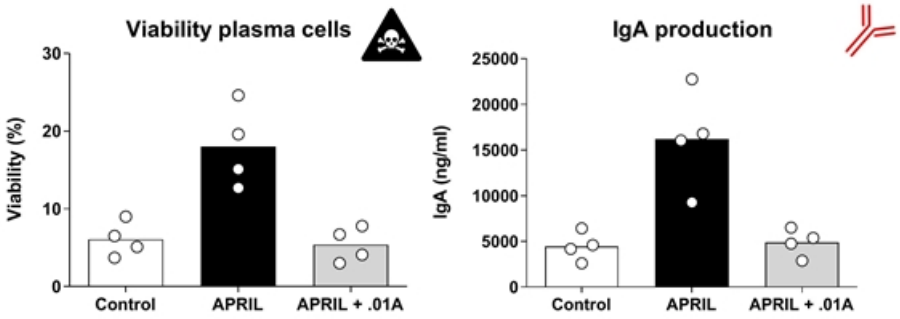
Human IgA+ Plasma Cells Express High TACI and BCMA Levels and Depend on APRIL for Survival and IgA production

- In vitro-generated IgA+ plasma cells selectively express high BCMA and TACI
- In vitro-generated IgA+ plasma cells require APRIL to survive and produce IgA
- Anti-APRIL (.01A) inhibits plasma cell survival and IgA production



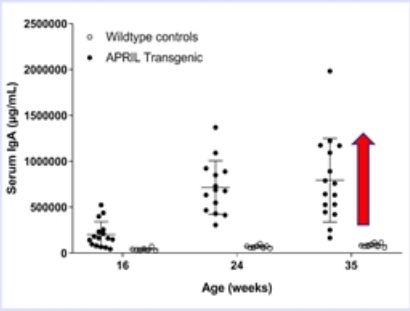


ADURO BIOTECH

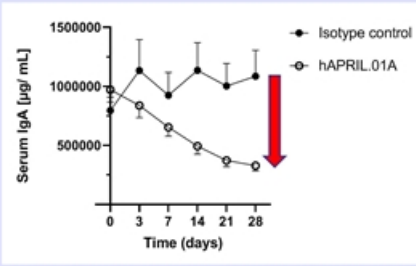
Preclinical Data in Mouse Models Provide Compelling Rationale for BION-1301 in IgA Nephropathy

hAPRIL Transgenic Mouse Model

Increased IgA production seen in hAPRIL Tg mice

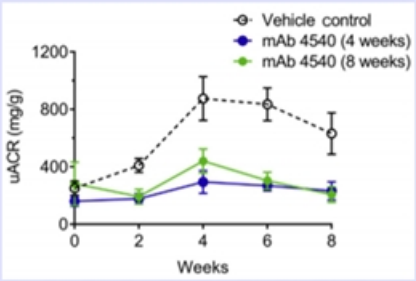


The BION-1301 parental antibody hAPRIL.01A inhibits IgA production in hAPRIL Tg mice



ddY IgAN Mouse Model

Treatment with anti-APRIL mAb 4540 suppresses proteinuria in IgAN ddY mice*



*Myette JR et al. A Proliferation Inducing Ligand (APRIL) targeted antibody is a safe and effective treatment of murine IgA nephropathy. Kidney International (2019) 96, 104–116

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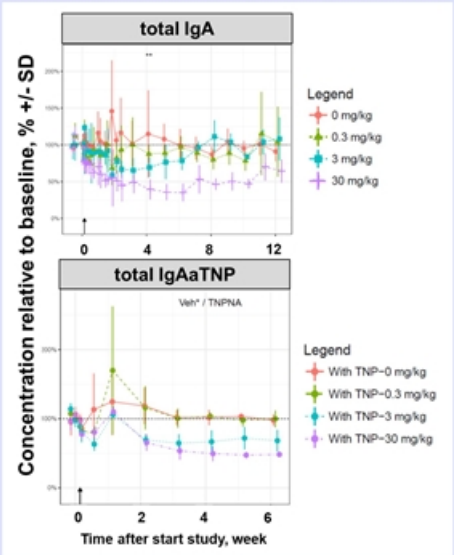
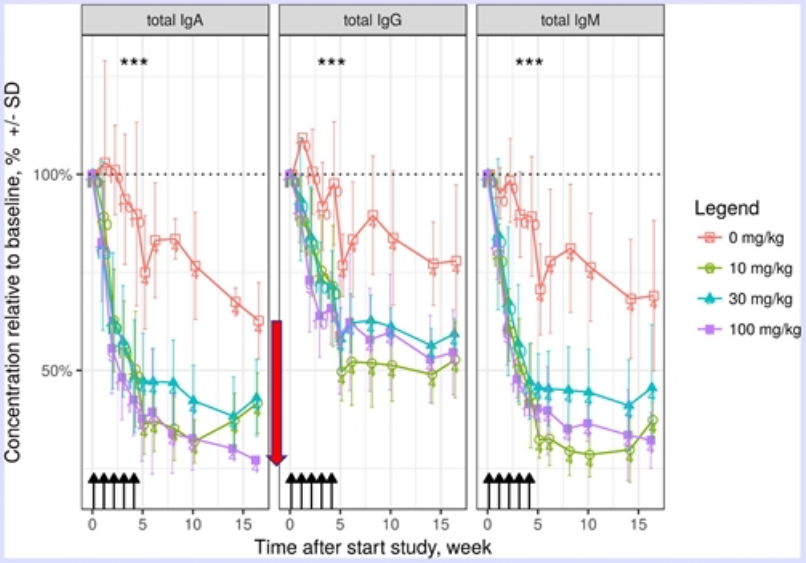
Preclinical Data from Tox Studies in Non-Human Primates (NHPs) Provide Compelling Rationale for BION-1301 in IgA Nephropathy

BION-1301 Dose Response in NHPs



BION-1301 Reduces Serum IgA, IgG and IgM levels in NHPs



Dulos J, et al. ASH 2017 poster presentation: abstract 1827.

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Phase 1 Study of BION-1301 in Healthy Volunteers & IgA Nephropathy Patients Ongoing

Part 1 in Healthy Volunteers:
Double-blind, Placebo-controlled, Single Ascending Dose

Up to 5 Dose Cohorts

Primary Objectives:

- Assess safety profile in healthy volunteers (HVs) & IgAN patients
- PK/PD relationship in HVs & IgAN patients
- Establish proof-of-mechanism:
  - Reduction in IgA in HVs and/or reduction in Gd-IgA in IgAN patients

Part 2 in Healthy Volunteers:
Double-blind, Placebo-controlled, Multiple Ascending Dose

3 Dose Cohorts

Part 3 in IgAN Patients:
Open Label, Multiple Dose

1 Dose Cohort

ADURO BIOTECH

FDA Co-Authored Article Supporting Reduction in Proteinuria as a Surrogate Endpoint for eGFR for Accelerated Approval

Proteinuria

- As of January 2019, FDA officially agrees to proteinuria as surrogate endpoint for eGFR in IgA Nephropathy
- Expect other global health authorities to follow suit
- General consensus among KOLs (several involved in Ph3 discussions with FDA) that a 30% reduction in proteinuria is necessary for approval

Estimated Glomerular Filtration Rate (eGFR)

- Ongoing discussion between industry and FDA on exactly how to measure a reduced rate of eGFR as endpoint
- Expecting publication to address this in early 2020

Proteinuria Reduction as a Surrogate End Point in Trials of IgA Nephropathy

Aliza Thompson,[1] Kevin Carroll,[2] Lesley A. Inker,[3] Jürgen Floege,[4] Vlado Perkovic,[5] Sonia Boyer-Suavet,[6] Rupert W. Major,[7] Judith I. Schimpf,[4] Jonathan Barratt,[8] Daniel C. Cattran,[9] Barbara S. Gillespie,[10] Annamaria Kausz,[11] Alex W. Mercer,[12] Heather N. Reich,[9] Brad H. Rovin,[13] Melissa West,[14] and Patrick H. Nachman[15]

Abstract

IgA nephropathy (IgAN) is an important cause of ESKD for which there are no approved therapies. A challenge for evaluating treatments for IgAN is the usual long time course for progression to ESKD. The aim of this Kidney Health Initiative project was to identify surrogate end points that could serve as reliable predictors of a treatment's effect on long-term kidney outcomes in IgAN and be used as a basis for approval. Proteinuria was identified as the most widely recognized and well studied risk factor for progression to ESKD in IgAN. The workgroup performed a critical review of the data on proteinuria reduction as a surrogate end point for a treatment's effect on progression to ESKD in IgAN. Epidemiologic data indicate a strong and consistent relationship between the level and duration of proteinuria and loss of kidney function. Trial-level analyses of data from 13 controlled trials also show an association between treatment effects on percent reduction of proteinuria and treatment effects on a composite of time to doubling of serum creatinine, ESKD, or death. We conclude that data support the use of proteinuria reduction as a reasonably likely surrogate end point for a treatment's effect on progression to ESKD in IgAN. In the United States, reasonably likely surrogate end points can be used as a basis for accelerated approval of therapies intended to treat serious or life-threatening conditions, such as IgAN. The clinical benefit of products approved under this program would need to be verified in a postmarketing confirmatory trial.

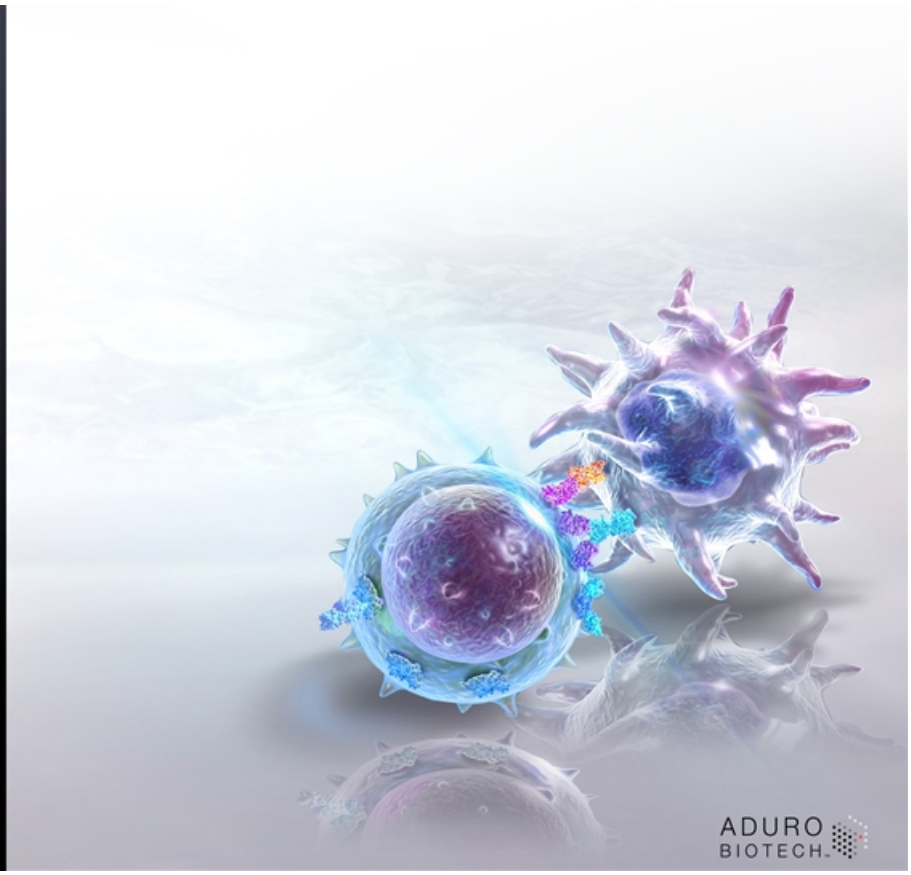
Clin J Am Soc Nephrol 14: ●●●–●●●, 2019. doi: https://doi.org/10.2215/CJN.08600718

ADURO BIOTECH

Several Ongoing Toxicology Studies to Support Long-Term Subcutaneous Dosing

- **1-Month intravenous (IV) toxicology study completed prior to IND filing**
  - No tox findings noted
- **1-Month subcutaneous (SC) local tolerability toxicology study completed**
  - Will support initiation of Phase 1 IV/SC bridging study in healthy volunteers
- **3-Month IV toxicology study completed**
  - Will support extending treatment of IgAN patients to 3 months in ongoing Phase 1 study
- **6-Month IV toxicology study**
  - To support Phase 2 study start

Business Overview



Aduro Strategic Collaborations and Partnerships

NOVARTIS	Lilly	MERCK
ADU-S100 and other STING agonists	cGAS-STING pathway inhibitor program	Anti-CD27 agonist
Oncology	Autoimmune & Inflammatory	Oncology
• $700M upfront & potential milestones • $50M equity • Co-development & co-commercialization	• $12M upfront • $620M potential development and commercial milestones per product • Research funding	• $447M potential milestones • Global license
• Aduro leads US sales • Profit/expense share US, major EU and Japan • Royalties ROW	• Lilly responsible for global commercialization • Single to low-double digit royalties • Option to co-fund clinical development in exchange for increased royalties	• Mid single-digit to low teens royalties

Strong Financial Position and Broad Intellectual Property Portfolio

Q3 2019 Financials

Cash, cash equivalents and marketable securities	$235.4M
R&D expenses	$15.5M
G&A expenses	$8.7M
Shares outstanding as of September 30, 2019	80.5M

Extensive Patent Portfolio

Global Rights (includes in-licensed patents)

- >150 issued composition and methods patents
- >250 pending applications

Nominal Expiration

- STING: 2025-39
- APRIL: 2030-36

Corporate restructuring in Q1 2020 is expected to reduce operating expenses & extend cash runway

Potential Upcoming Catalysts

		Indication	Catalyst	Expected Timing H1 2020	Expected Timing H2 2020
ADU-S100 (MIW815) *Intratumoral STING Agonist*	**+ spartalizumab** (α-PD-1)	Advanced/metastatic solid tumors or lymphomas	Report complete Ph 1 dose escalation & enrichment results		●
	+ pembrolizumab (α-PD-1)	1st-line SCCHN	Report interim study results		●
ADU-S100 (MIW815) *Intravesical*		Relapsed/refractory NMIBC	Initiate Ph 1b dose escalation study		●
BION-1301 *Anti-APRIL Antibody*		IgA nephropathy	Report Ph 1 data in healthy volunteers	●	
			Report Ph 1 data in IgA nephropathy patients		●

SCCHN = squamous cell carcinoma of the head and neck

NMIBC = non-muscle invasive bladder cancer

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