

Corporate Overview

September 17, 2020



Nasdaq: ANAB

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This presentation and any accompanying oral presentation contain "forward-looking" statements within the meaning of the "safe harbor" provisions of the Private Securities Litigation Reform Act of 1995, including, but not limited to: the timing of the release of data from our clinical trials, including etokimab's week 16 data for the Phase 2 clinical trial in adult chronic rhinosinusitis with nasal polyps patients and imsidolimab's Phase 2 trials in GPP and PPP patients; the timing of initiation of imsidolimab's Phase 2 trials in EGFRi /MEKi and ichthyosis; the timing of a regulatory strategy update for GPP; the timing of an IND filing for ANB032; the milestones and success of our GSK collaboration; and our projected 2020 cash burn and cash runway. Statements including words such as "plan," "continue," "expect," or "ongoing" and statements in the future tense are forward-looking statements. These forward-looking statements involve risks and uncertainties, as well as assumptions, which, if they do not fully materialize or prove incorrect, could cause our results to differ materially from those expressed or implied by such forward-looking statements. Forwardlooking statements are subject to risks and uncertainties that may cause the company's actual activities or results to differ significantly from those expressed in any forward-looking statement, including risks and uncertainties related to the company's ability to advance its product candidates, obtain regulatory approval of and ultimately commercialize its product candidates, the timing and results of preclinical and clinical trials, the company's ability to fund development activities and achieve development goals, the company's ability to protect intellectual property and other risks and uncertainties described under the heading "Risk Factors" in documents the company files from time to time with the Securities and Exchange Commission. These forward-looking statements speak only as of the date of this presentation, and the company undertakes no obligation to revise or update any forward-looking statements to reflect events or circumstances after the date hereof.

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AnaptysBio: Clinical-Stage Novel Antibody R&D Engine



Advancing First-In-Class Immunology Therapeutics to Patients

Multiple Clinical Catalysts

Upcoming catalysts include GPP and PPP Phase 2 data (imsidolimab), EGFRi-mediated skin toxicity and ichthyosis Phase 2 initiation (imsidolimab), Phase 1 clinical data (ANB030) and new IND filing (ANB032)

Validated Platform

Rapid antibody R&D engine has advanced 7 internally-generated antibodies to clinical development since 2016

Novel Immunology Pipeline

Focused on first-in-class inflammation and immuno-oncology mechanisms, advancing 1 new program to IND each year

Accelerating Partnership Revenues

Approximately \$100MM in partnership revenues received to date, additional \$20MM milestone and royalties anticipated following dostarlimab US BLA approval in H2 2020

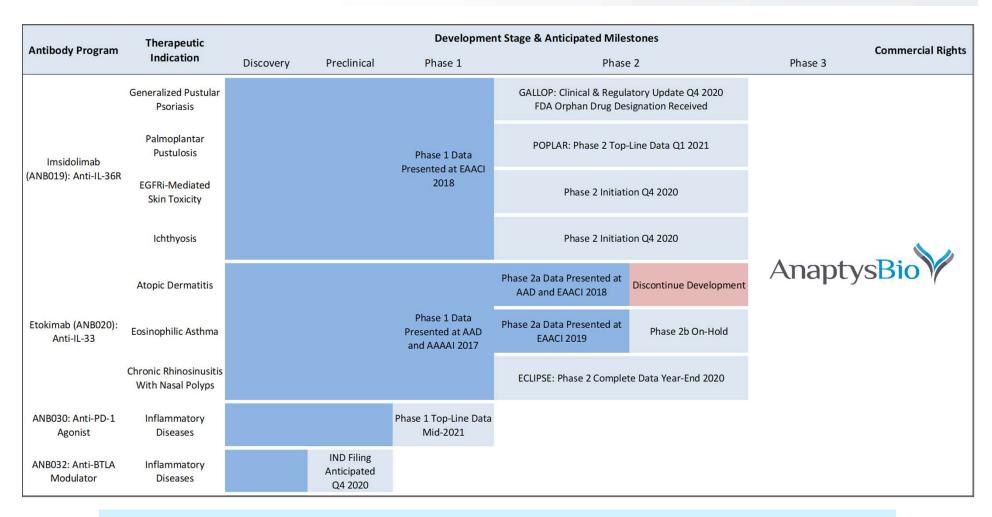
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Business Model

Cash and existing partnerships anticipated to extend runway into 2023, \$392MM in cash (end Q2 2020) with projected 2020 net burn of ~\$60MM

Wholly-Owned Product Pipeline



7 AnaptysBio-Generated Antibodies Advanced to Clinical Development Since 2016

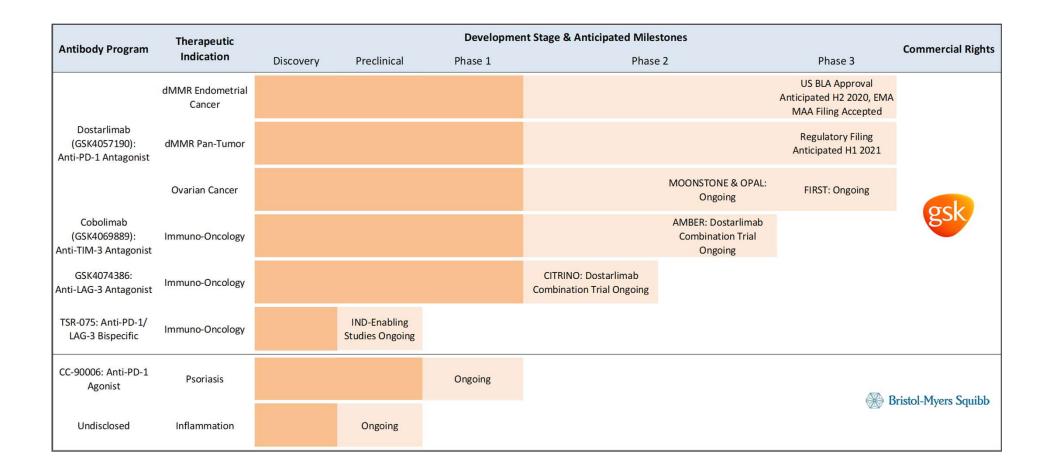


All programs generated internally using AnaptysBio's proprietary antibody platform technology

Partnered Product Pipeline



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Anticipated Wholly-Owned Pipeline Catalysts



Program	Clinical Catalyst	Anticipated Timing
	GALLOP: GPP Phase 2 Trial	Additional clinical data and regulatory strategy update anticipated in Q4 2020
Imsidolimab	POPLAR: PPP Phase 2 Trial	Top-line data anticipated in Q1 2021
(ANB019, anti-IL-36R)	EGFRi Mediated Skin Toxicity Phase 2 Trial	Phase 2 trial initiation in Q4 2020
	Ichthyosis Phase 2 Trial	Phase 2 trial initiation in Q4 2020
Etokimab (ANB020, anti-IL-33)	ECLIPSE: Adult Chronic Rhinosinusitis with Nasal Polyps Phase 2 Trial	Complete data by year-end 2020
ANB030 (anti-PD-1 Agonist)	Healthy Volunteer Phase 1 Trial	Top-line data anticipated in mid-2021
ANB032 (anti-BTLA Modulator)	IND Filing	Anticipated Q4 2020



Wholly-Owned Pipeline: Imsidolimab (ANB019, Anti-IL-36R)

Generalized Pustular Psoriasis Palmoplantar Pustulosis EGFRi-Mediated Skin Toxicity Ichthyosis

IL-36 Dysfunction Mediates Severe Inflammatory Disease





Normal Individuals

IL-36 cytokine signaling balanced by function IL-36 receptor antagonist

Inflammatory Response

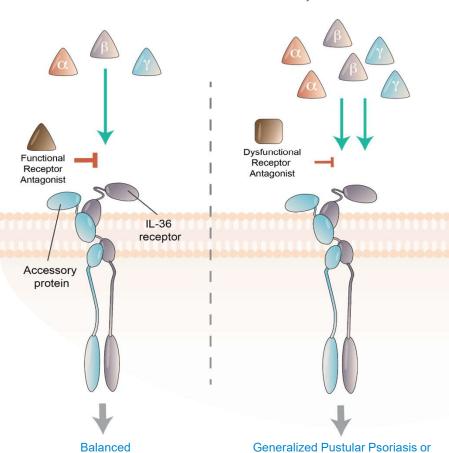
Disease Patients

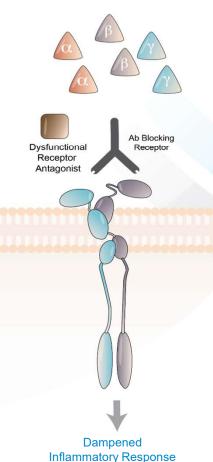
Uncontrolled signaling due to dysfunctional IL-36 receptor antagonist or elevated IL-36 cytokine levels

Palmoplantar Pustulosis

Treated Patients

Anti-IL-36R antibody has the potential to dampen disease by blocking the IL-36 receptor





Generalized Pustular Psoriasis (GPP)

Orphan Disease Associated with IL-36 Receptor Antagonist Mutations



- GPP is a systemic, life-threatening inflammatory disease characterized by widespread pustules
 - Patients have a high fever and elevated levels of serum CRP and inflammatory cytokines (e.g. IL-8)
- Severe GPP patients can die from cardio-pulmonary failure, exhaustion, toxicity and infection
 - No approved therapies for treatment of GPP
- Affects approximately 3,000 patients in the United States
- FDA has granted Orphan Drug Designation to imsidolimab for the treatment of GPP

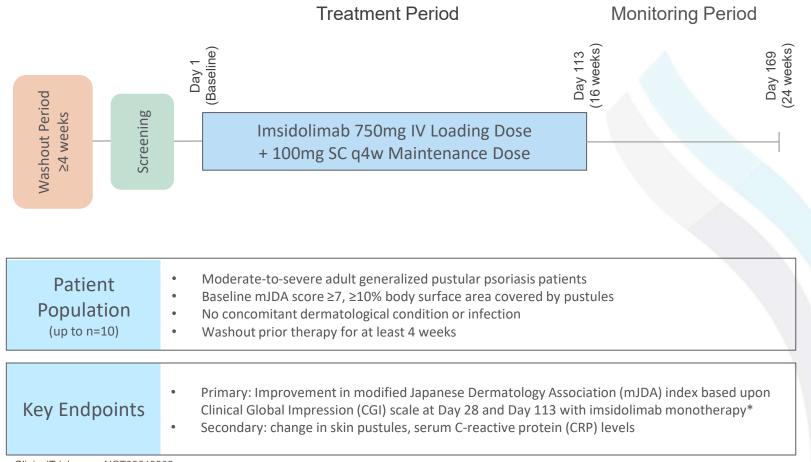




GALLOP: Imsidolimab Moderate-to-Severe GPP Phase 2 Trial



Trial Design



ClinicalTrials.gov: NCT03619902

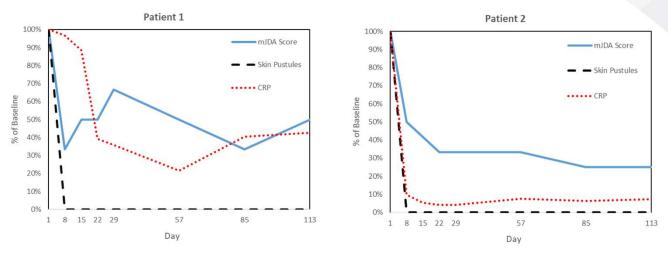
^{*} Rescue therapy available if no improvement by Day 28, however use of rescue therapy disqualifies patient from achieving primary endpoint

Imsidolimab Monotherapy Resulted in Rapid and Sustained Improvement



Interim Analysis Conducted Using First Two Patients To Complete Day 113

Endpoint	Baseline (average)	Result (average, where applicable)
Primary endpoint		Achieved by both patients
Rescue therapy use		None
mJDA score (combines dermatological and systemic inflammation markers)	9	Reduced by 58% at Day 8 and 63% at Day 113
Skin pustules (dermatological hallmark of GPP)	27%	Complete clearance (100% reduction) from Day 8 through Day 113
CRP (systemic inflammation marker)	34 mg/L	Reduced by 47% at Day 8 and 75% at Day 113
Genotype		Wildtype, unmutated IL36RN, CART14 and AP1S3 alleles
Anti-Drug Antibodies		None detected



Another patient dropped out of the trial due to *Staphylococcal aureus* bacteremia diagnosis on Day 3 post-imsidolimab dosing, which was deemed a possibly drug related SAE. AnaptysBio believes this event is unlikely to be drug related due to prior patient history and symptoms of infection prior to imsidolimab dosing.

Interim Analysis Summary



Anticipate Additional Clinical Data and Regulatory Strategy Update in Q4 2020

- Both patients achieved the primary endpoint of disease score improvement at Day 29 and Day 113 without requiring rescue therapy
 - Patients demonstrated rapid and sustained mJDA score improvement, with reduction of 58% at Day 8 and 63% at Day 113
 - Complete clearance of pustules was achieved by Day 8 and through Day 113, while CRP decreased to nearly normal levels in both patients
- Imsidolimab was generally well-tolerated by both patients included in interim analysis
 - No serious or severe adverse events were reported
- Based upon results of interim analysis, enhancing enrollment with:
 - Increased clinical sites and geographies
 - Curtailed washout period requirement for enrollment

Palmoplantar Pustulosis (PPP)





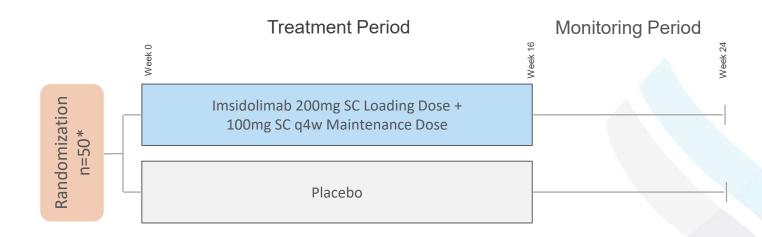
- Severe inflammation of hands and feet
 - Significant pain and inability to stand, walk or work
- No approved therapeutic options in this indication
- PPP is an orphan disease that affects approximately 150,000 patients in the United States



POPLAR: Imsidolimab Palmoplantar Pustulosis Phase 2 Trial



Top-Line Data Anticipated Q1 2021



Patient Population	Adult Moderate-to-Severe Palmoplantar Pustulosis
Key Endpoints	PPPASI Score Improvement
Week 16	Safety

ClinicalTrials.gov: NCT03633396

^{*} Enhancing enrollment with increased clinical sites and geographies

New Indication: EGFRi-Mediated Skin Toxicity

Translational Data Suggests IL-36 Signaling Drives EGFR/MEK Inhibitor Papulopustular Rash

- Papulopustular rash is the most frequent clinically significant dermatological toxicity associated with EGFR/MEK inhibitor solid tumor treatment
- Majority of patients experience doselimiting skin toxicity and/or discontinuation of EGFR/MEK inhibitor therapy
- Recent human translational data indicates elevated IL-36 signaling is the key driver for this skin toxicity
 - Associated with IL-8 release and neutrophilia
- Approximately 60,000 patients are treated annually with EGFR/MEK inhibitors
- Anticipate initiating Phase 2 trial of imsidolimab for the treatment of EGFRimediated skin toxicity in Q4 2020



Gerber et al. Eur J Med Res. 2012; 17(1):4

New Indication: Ichthyosis

Orphan Disease Associated With Excess IL-36 Signaling



- Ichthyosis is a rare, orphan dermatological indication with high medical unmet need
- Patients suffer from dry, scaly skin, often leading to itch and painful cracking
- Translational studies have demonstrated high IL-36 cytokine expression levels in patient skin biopsies
- Approximately 6,000 adults diagnosed with moderate-to-severe ichthyosis in the United States
- Initiation of imsidolimab Phase 2 trial in ichthyosis anticipated in Q4 2020





Wholly-Owned Pipeline: Etokimab (ANB020, Anti-IL-33)

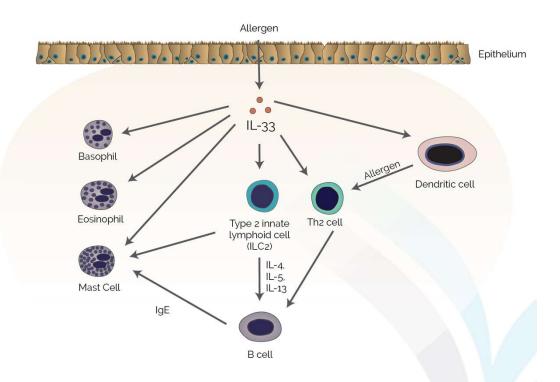
Chronic Rhinosinusitis with Nasal Polyps Moderate-to-Severe Eosinophilic Asthma

Etokimab: First-in-Class Anti-IL-33 Antibody

Development Focused on Respiratory Indications



- IL-33 is genetically associated with asthma
 - IL-33 pathway loss-of-function mutations protect against asthma, while gain-offunction mutations increase asthma incidence
 - Translational studies have demonstrated IL-33's role as pro-inflammatory cytokine released upon allergen contact with epithelium
- Etokimab is a potentially first-inclass anti-IL-33 cytokine antibody
 - Etokimab development focused on respiratory indications, including chronic rhinosinusitis with nasal polyps and moderate-to-severe eosinophilic asthma
 - Estimate 400,000 adults diagnosed with chronic rhinosinusitis with nasal polyps, and 1.1 million adults diagnosed with severe asthma, are inadequately controlled by standard of care in the US



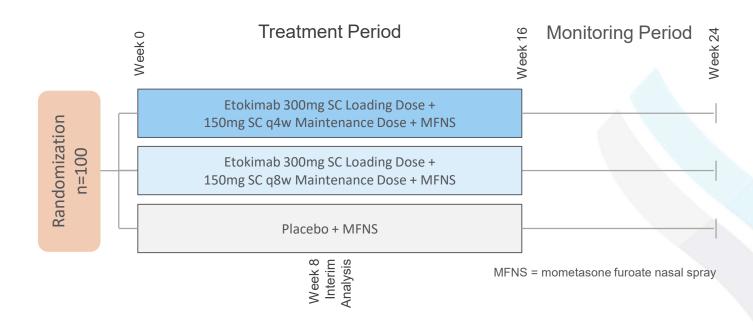
IL-33 is believed to act as a gatekeeper of allergic response with demonstrated activity in the initiation (activation of ILC2 cells)¹, propagation (activation of allergen-specific T and B cells)² and amplification (degranulation of mast cells and basophils)³.

- 1. Cayrol et al. Curr Opin Immunol (2014) 31:31
- 2. Peine et al. Trends Immunol (2016) 37(5):321
- 3. Saluja et al. Clin Transl Allergy (2015) 5:33

ECLIPSE: Etokimab CRSwNP Phase 2 Trial

Complete Data Anticipated by Year-End 2020





Patient	Adult Chronic Rhinosinusitis with Nasal Polyps
Population	Baseline NPS≥4 and SNOT-22>16
Key Endpoints	Primary: change in NPS and SNOT-22 relative to baseline at week 16 Secondary: FEV1, ACQ and blood eosinophil in asthma subsets

ClinicalTrials.gov: NCT03614923

ECLIPSE: Etokimab CRSwNP Phase 2 Trial

Interim Analysis Data Announced August 10th 2020



- Etokimab q4w and q8w treatment arms failed to achieve NPS and SNOT-22 statistical significance over placebo at this week 8 interim analysis
 - Both arms demonstrated statistical significance over baseline
- Secondary analyses demonstrated NPS and blood eosinophil level improvement in both asthma and nonasthma comorbid patients versus placebo in each etokimab-dosed arm, while ACQ-5 scores were improved in the asthmatic subset
- Blood eosinophil reduction achieved statistical significance over baseline in both etokimab treatment arms
- Etokimab was generally well-tolerated and demonstrated an acceptable safety profile
- AnaptysBio to assess path forward for the etokimab program after reviewing week 16 primary endpoint data by year-end 2020

Endpoint	Parameter	Etokimab q4w (n=35)	Etokimab q8w (n=35)	Placebo (n=35)
NPS	Baseline	5.4	5.2	5.7
	Week 8	-10%	-11%	-4%
	p-value vs placebo	0.3348	0.3042	N/A
	p-value vs baseline	0.0286	0.0243	0.4288
SNOT-22	Baseline	51.4	53.9	56.9
	Week 8	-23%	-23%	-19%
	p-value vs placebo	0.9927	0.9275	N/A
	p-value vs baseline	<0.0001	<0.0001	<0.001
Blood Eosinophils (cells/ microliter)	Baseline	440	350	430
	Week 8	-23%	-23%	7%
	p-value vs baseline	0.004	<0.001	0.358



Wholly-Owned Pipeline: Anti-PD-1 Agonist (ANB030) Anti-BTLA Modulator (ANB032)

Inflammatory Diseases

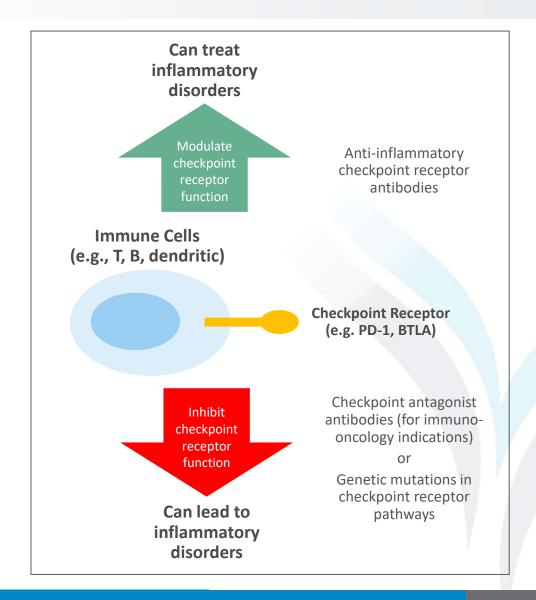
Anti-Inflammatory Checkpoint Receptor Antibodies





Anti-inflammatory checkpoint receptor antibodies have unique binding properties that are challenging to generate using traditional antibody technologies

AnaptysBio's technology platform has successfully discovered a portfolio of anti-inflammatory checkpoint receptor antibodies, which are advancing to clinical trials



ANB030: PD-1 Agonist Antibody





- PD-1 is a key inhibitory immune checkpoint receptor responsible for down-regulating T-cell mediated immune responses
- Insufficient PD-1 activity is associated with human inflammatory diseases
 - Genetic mutations in the PD-1 pathway can increase susceptibility to various inflammatory conditions*
- We hypothesize that augmenting PD-1 signaling through ANB030 treatment has the potential to suppress T-cell driven human inflammatory diseases
 - Designed to down-regulate autoreactive T cells by mimicking the function of PD-L1
- Preclinical translational data presented in March 2020
- Healthy volunteer Phase 1 trial ongoing
 - Anticipate top-line data in mid-2021

Antigen Presenting Cell Activated T Cell Healthy Individuals PD-1 PD-L1 **Negative Signal** *Inflammatory* Disease Patients **Insufficient Signal Anticipated** ANB030 Effect **Negative Signal ANB030**

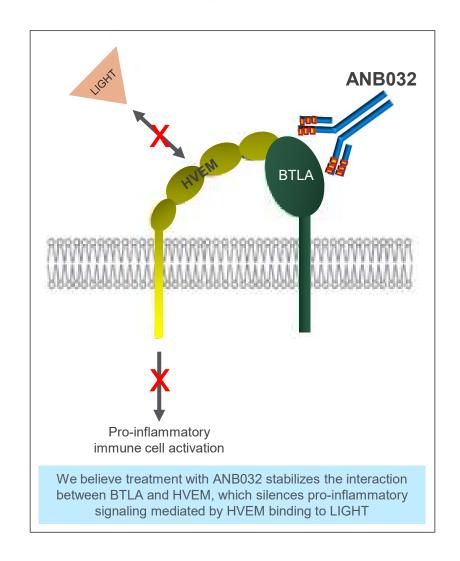
^{*} Okazaki and Honjo. Intern Immunol. 2007

ANB032: BTLA Modulator Antibody

Emerging Lymphoid and Myeloid Immune Control Mechanism Broadly Applicable to Inflammatory Disease



- BTLA is an inhibitory checkpoint receptor responsible for regulating activation of lymphoid (T and B) cells and myeloid (dendritic) cells
- Genetic defects in the BTLA pathway are associated with enhanced susceptibility to inflammatory diseases*
- ANB032 is an anti-inflammatory antibody targeting the BTLA pathway
 - Anticipate ANB032 may be broadly applicable to inflammatory disease due to breadth of BTLA expression across immune cell types
 - ANB032 has demonstrated robust *in vivo* efficacy in animal models of GVHD
- ANB032 IND filing anticipated in Q4 2020



^{*} Lin et al. J Biomed Sci. 2006



Partnered Pipeline: GSK Immuno-Oncology Collaboration

Dostarlimab (GSK4057190, anti-PD-1 Antagonist) Cobolimab (GSK4069889, anti-TIM-3 Antagonist) TSR-033 (GSK4074386, anti-LAG-3 Antagonist)

GSK Immuno-Oncology Collaboration

Dostarlimab Approaching US Regulatory Approval



dMMR Endometrial	BLA and MAA Accepted US Approval Anticipated in H2 2020	GARNET (n=125) RUBY (n=470)
dMMR Pan-Tumor	Regulatory Filing Anticipated H1 2021	GARNET (n=125)
Colorectal		GARNET (n=48)
		FIRST (n=912)
Ovarian	MOONSTONE (n=150)	
	OPAL (n=41)	
NSCLC	JASPER (n=142)	
	ATOMICC (n=132)*	
Cervical	STAR (n=66)*	
Liver	n=42*	
Rectal	n=30*	
Melanoma	n=56*	
Sarcoma, Clear Cell	n=16*	
HNSCC	n=23*	
All-Comer/	AMBER (n=873)	
Undisclosed	CITRINO (n=200)	
	DI 0	21 2

Phase 2 Phase 3

Dostarlimab (anti-PD-1 Antagonist)

Dostarlimab +

Dostarlimab + TSR-033 (anti-LAG-3 Antagonist)

Aggregate Financial Terms

- \$1.1B in aggregate milestone payments
 - \$432MM in potential development & regulatory milestones
 - \$660MM in potential commercial milestones
- 4-8% royalty upon global sales

Dostarlimab Regulatory Filing and Approval Milestones

\$15MM Received of \$90MM Total

- ✓ dMMR Endometrial: 10MM upon 1st FDA BLA Acceptance (Received H1 2020)
- ✓ dMMR Endometrial: \$5MM upon 1st MAA acceptance by EMA (Received H1 2020)
- dMMR Endometrial: \$20MM upon 1st US Regulatory Approval (Anticipated In H2 2020)
- dMMR Endometrial: \$10MM upon 1st EU Regulatory Approval
- dMMR Pan-Tumor: 10MM upon 2nd FDA BLA Acceptance
- dMMR Pan-Tumor: \$5MM upon 2nd MAA acceptance by EMA
- dMMR Pan-Tumor: \$20MM upon 2nd US Regulatory Approval
- dMMR Pan-Tumor: \$10MM upon 2nd EU Regulatory Approval

^{*} Investigator sponsored trial dMMR = mismatch repair deficient



Proprietary Technology Platform

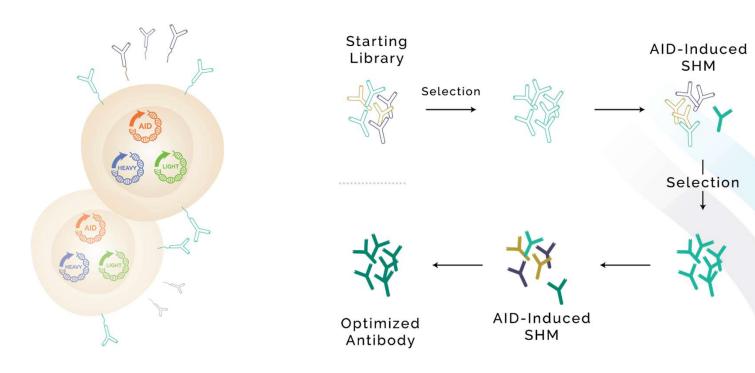
Somatic Hypermutation (SHM) Platform





In Vitro SHM

Iterative Antibody Evolution



In vitro SHM permits access to biological targets that have been difficult to address with prior antibody technologies

Somatic Hypermutation (SHM) Platform

Advantages Over Competing Antibody Technologies



- Unprecedented antibody diversity through SHM
 - In situ antibody diversity generation outside of the constraints of an in vivo environment
- High potency & functional activity
 - Only small doses may be required to convey therapeutic effect in vivo
- Reliable manufacturability
 - Increased probability of successful clinical and commercial manufacturing
- Speed: ~2.5 years from novel target to IND (or equivalent) filing
 - Enables rapid development of potentially first-in-class therapeutic antibodies to emerging target biology

7 AnaptysBio-generated antibodies have advanced to clinical development since 2016



Summary

Anticipated Wholly-Owned Pipeline Catalysts



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