

ANTENGENE INVESTOR PRESENTATION

TREATING PATIENTS BEYOND BORDERS

MARCH 2023

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I. COMPANY OVERVIEW

Antengene is Executing on Our Strategy to Bring Transformative Medicines to Patients Around the World





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Global Management Team with Deep Local Experience and Insights Working Seamlessly Across Regions



Dual Engine of Growth Through Value-adding Partnerships and In-house Discovery







Antengene Has Executed and Delivered on Significant Milestones Since IPO



		November 20 th , 2020	November 20 th , 2022					
Commercialization	Product Approvals	0	塞利尼索片 🗙 POVIO [®] 🔅 🌾 🧐 Taiwan					
Registrational Trials	ATG-010 (Selinexor)	2 ongoing	4 ongoing; 1 completed					
Registrational Path	ATG-008 (Onatasertib)	No	Yes (Relapsed/Metastatic Cervical Cancer)					
Global Best-in-class Potential Assets in Clinical Stage		ATG-017 - ERK1/2 small molecule inhibitor	ATG-017 - ERK1/2 small molecule inhibitor ATG-101 - PD-L1/4-1BB bispecific antibody ATG-037 - CD73 small molecule inhibitor ATG-018 - ATR small molecule inhibitor ATG-022 - Claudin 18.2 ADC (IND submitted)					
Global First-in-class Potential Asset		0	ATG-031 – CD24 monoclonal antibody					
Cash Reserve		RMB 918 mm (immediately prior to IPO)	RMB 2,151 mm*					
Market Cap		USD 1,549 mm	USD 356 mm					

The Antengene Pipeline Includes Multiple Novel Immuno-oncology and Targeted Products – Allowing Broad Proprietary Combinations





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II. PIPELINE

Pipeline of Commercial or Near NDA Stage Drugs with First-in-Class/Best-in-**Class Potentials**



ANTENGENE

Assets	Target (Modality)	Indication	Pre-clinical	Phase I	Phase II	Phase III	NDA	Commercialization	Antengene Rights	Partner
			Combo with dexamethasone ((MARCH)			Mainl	and China NDA approved		
			Combo with dexamethasone (
		R/R Multiple Myeloma	Combo with bortezomib and d	examethasone <i>(BENC</i>	н)	*				
			Combo with bortezomib and d	examethasone <i>(BOST</i>	ON) - Partner's Pivotal 1	Trial in the US	US, EU, SG,	AU & TW sNDA approved		
			Combo with IMID/PI/CD38 mA	b and dexamethasone	(STOMP)					Karvopharm
ATG-010 ¹ (Selinexor)	XPO1 (Small molecule)		Monotherapy <i>(SEARCH)</i>		*	•				
		R/R Diffuse Large B-cell Lymphoma R/R NHL R/R T-cell & NK-cell Lymphoma	Monotherapy <i>(SADAL) - Partner's Pivotal Trial in the US</i> US , SG, SK & TW sNDA approved							ANTENGENE
			Combo with R-GDP <i>(DLBCL-03</i>)	0)	*					
			Combo with lenalidomide + rit	uximab <i>(SWATCH)</i>						
			Combo with ICE/GemOx/tisleli	zumab <i>(TOUCH)</i>	with 💆 BeiG	ene				
		Myelofibrosis	Monotherapy <i>(MF 035)</i>		*					
ATG-016 (Eltanexor)	XPO1 (Small molecule)	R/R MDS	Monotherapy <i>(HATCH)</i>							
ATG-008	mTORC1/2	Cervical Cancer and Other Advanced Solid Tumors	Combo with toripalimab (TORI	CH-2)*	W	vith ************************************			APAC ³	Celgene (III) Bristol Myers Squibb" Company
(Onatasertib)	(Small molecule)	R/R Diffuse Large B-cel Lymphoma	Combo with ATG-010 <i>(MATCH)</i>							
		Antenge	ne Trials ⁴	Partner Trials⁵	GI	lobal Trials in Collaborat	ion with Partner	Registrational T	rial in China	

1 (s)NDA approved by US FDA, China NMPA, Australia TGA, South Korea MFDS and Singapore HSA; China Hong Kong and China Taiwan NDA submissions are completed; ² Antengene has rights for Greater China (Mainland China, Hong Kong, Taiwan, Macau), Australia, New Zealand, South Korea, and the ASEAN Countries; ³ Antengene has rights for Greater China, South Korea, Singapore, Malaysia, Indonesia, Vietnam, Laos, Cambodia, the Philippines, Thailand and Mongolia; ⁴ Most advanced trial status in Antengene territories and the trials are responsible by Antengene;

⁵ Most advanced trial status in partner territories in the rest of the world and the trials are conducted by our licensing partners

* Investigator-initiated trials; R/R: relapsed/refractory; ND: newly diagnosed; MDS: myelodysplastic syndrome; CRC: colorectal cancer; PrC: prostate cancer; CAEBV: chronic active Epstein-Barr virus; NHL: non-Hodgkin lymphoma; Hem/Onc: hematological malignancies and solid tumors; R-GDP: Rituximab, Gemcitabine, Dexamethasone &Cisplatin;

AU: Australia; EU: Europe; SG: Singapore; SK: South Korea; TW: Taiwan; US: United States;

A Clinical Stage Pipeline with Transformational Potentials



Assets	Target (Modality)	Hits Discovery	Lead Nomination	<i>In vitro</i> efficacy	<i>In vivo</i> efficacy	CMC/Tox	IND	Phase I	Antengene Rights	Partner
ATG-017 (Tizaterkib) ¹	ERK1/2 (Small molecule)	Monotherapy <u>+</u> nivo	lumab for R/R Hem/On	c (ERASER)				with (^{III}) Bristol Myers Squibb		
ATG-101 ²	PD-L1/4-1BB (Bispecific)	Monotherapy for H	em/Onc (PROBE & PROE	BE-CN)						
ATG-037 ³	CD73 (Small molecule)	Monotherapy <u>+</u> pen	nbrolizumab for Hem/O	nc (STAMINA)				with 📀 MERCK		
ATG-018	ATR (Small molecule)	Monotherapy for H	em/Onc <i>(ATRIUM)</i>						Global Global	ANTENGENE
ATG-022	Claudin 18.2 (ADC)	Monotherapy for O	nc (CLINCH)							
ATG-031	CD24 (mAb)	Monotherapy for He	em/Onc (<i>PERFORM</i>)							

Antengene Trials

¹ Licensed from AstraZeneca and Antengene has obtained exclusive global rights to develop, commercialize and manufacture ATG-017;

² Licensed from Origincell and Antengene has obtained exclusive global rights to develop, commercialize and manufacture ATG-101;

³ Licensed from Calithera Biosciences and Antengene has obtained exclusive global rights to develop, commercialize and manufacture ATG-037



ATG-008 (mTORC 1/2 INHIBITOR)



Summary of ATG-008 (Onatasertib)

- Mammalian target of rapamycin (mTOR), a core component of two structurally distinct protein complexes (mTORC1 and mTORC2), regulates different cellular processes and is upregulated in multiple types of tumors
- When mTORC1 is inhibited, mTORC2 is upregulated. This increase in mTORC2 activity generates a surplus of phosphorylated PKB/AKT which, despite mTORC1 inhibition, inhibits apoptosis and promotes cell proliferation via alternative pathways
- mTORC1 and mTORC2 has to be inhibited simultaneously for good anti-tumor efficacy

First- and Best-in-Class Potential

- Second generation mTOR inhibitor, targeting both TORC1 and TORC2
- Demonstrated comprehensive mTOR inhibition, which could minimize development of resistance due to mTORC2 upregulation
- Encouraging initial clinical data in combination with anti-PD-1 mAb in the treatment of relapsed or metastatic cervical cancer



ATG-008 (Onatasertib) In Combination with Toripalimab

Potential Best-in-Class Treatment for 2L+ Cervical Cancer as Demonstrated in "TORCH-2"



	ATG 008 + Toripalimab (Data from "TORCH-2")	Pembrolizumab (Global Standard of Care)	AK104 (Only CPI Approved by CDE)	Sintilimab + Anlotinib
Mechanism of Action (MoA)	mTORC 1/2i + PD-1 mAb	PD-1 mAb	PD-1/CTLA-4 BsAb	PD-1 mAb + VEGFRi
Number of Patients	21 (ITT)	98 (ITT) 100 (FAS, ITT		39 (EE, ITT 42)
Prior Treatment Lines	≤2 (52.4%); ≥3 (47.6%)	≤2 (69.4%); ≥3 (30.6%)	≤2 (100%)	≤2 (78.6%); ≥3 (21.4%)
PD-L1	N, TPS≥1% (42.8%)	N, CPS≥1 (83.7%)	Ν	Y, CPS≥1 (100%)
ORR	52.4%; 77.8% (TPS≥1%)	12.2%	33%	59%
DCR	94.4%	30.6%	52%	94.9%
PFS (months)	5.45; 9.63 (15 mg cohort)	2.1	3.75	9.4
OS (months)	NE	9.4	17.5	NE
Response in CPI treated	1/2	N/A	N/A	N/A
Response in AdCa	1/2	1/5	NE	0/6

AdCa: Adenocarcinoma

Key Takeaways of ATG-008 (Onatasertib) Clinical Programs







Pre-IND consultation with CDE planned for a pivotal study that will define the regulatory path for ATG-008, with advanced cervical cancer as the lead indication



The **TORCH-2 trial is still enrolling patients** to further evaluate the role of ATG-008 & anti-PD-1 combination in patients who have failed prior CPI treatments



GLOBAL RIGHTS PROGRAMS

Global-Rights Pipeline Comprised of Clinical Stage and IND-Ready Assets with First and/or Best-in-Class Potential



	ATG-017 (Tizaterkib)	ATG-101	ATG-037	ATG-018	ATG-022	ATG-031
Target	ERK1/2	PD-L1/4-1BB	CD73	ATR	Claudin 18.2	CD24
Modality	Small Molecule	Bispecific Antibody	Small Molecule	Small Molecule	ADC	Monoclonal Antibody
Indication	 RASm NSCLC, Pancreatic cancer, CRC, and Melanoma I/O combinations 	 Re-sensitize prior CPI responders (NSCLC, SCLC, GI, H/N SCC, melanoma) Disease with previously limited CPI activity Multiple combination opportunities 	 Monotherapy where immune suppressed TME is critical Broad opportunities both as monotherapy and combination with existing / future I/O 	Hematological Malignancies / Solid Tumors	Solid Tumors	Hematological Malignancies / Solid Tumors
Differentiation	 Higher potency and dual IoC and PoA activity with slow off- rate kinetics Lower efficacious dose with a higher max absorbable dose/dose ratio Broad therapeutic potential (targeting RAS/MAPK pathway) Multiple combination opportunities 	 PD-L1 cross-linking dependent activation of 4-1BB to avoid unwanted 4-1BB signaling in normal tissue and minimize risk of hepatotoxicity Demonstrated significant anti- tumor activity in animal models of resistant tumors as well as those that progressed on anti-PD-1/L1 treatment Displayed an excellent safety profile in GLP toxicology studies 	 Orally bioavailable small molecule that completely overcomes 'hook effect' common in other anti-CD73 antibodies Tissue penetrance not achievable with mAbs Promising preclinical efficacy as a monotherapy and strong combination potential 	 Better <i>in vivo</i> efficacy compared with benchmark in pre- clinical CDX tumor models Orally available 	 High affinity antibody (pM); Strong <i>in vivo</i> efficacy pre-clinically in Claudin 18.2 low expression PDX models Demonstrated an excellent safety profile in GLP toxicology studies 	 First in class target No clinical competitor Showed mono- therapy <i>in vivo</i> efficacy and synergy with chemotherapy, rituximab and CPI
Status	Enrollment ongoing in Australia for continuous dosing and intermittent dosing cohorts; Combo with nivolumab to initiate enrollment in 2023	Phase 1 clinical trial "PROBE" ongoing in Australia (4 th cohort), first patient to be dosed in the US; "PROBE-CN" ongoing in China (3 rd cohort)	Phase 1 clinical trial "STAMINA" ongoing in Australia, currently enrolling for the 2 nd cohort	Phase 1 clinical trial "ATRIUM" ongoing in Australia, currently enrolling for the 3 rd cohort	Australian HREC approved in December 2022	IND planned for early Q1 2023

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ATG-017 (Tizaterkib) May Enhance the Activity of Checkpoint Inhibitors or Reverse Resistance Mechanisms



Through inhibiting ERK1/2 activity, ATG-017 may enhance the activity of checkpoint inhibitors or reverse resistance mechanisms

ERK activation contributes to hyper-progressive disease induced by anti-PD-1 therapy



- PD-1/PD-L1 expression on tumor cells inhibit tumor cell growth through deregulation of canonical signaling pathways, including the AKT and ERK1/2 pathways, and prevent the interaction with PD-1-expressing T cells
- Clinically available antibodies targeting PD-1 (blue) or PD-L1 (cyan) enhance tumor cell growth via activation of AKT and ERK1/2 in the absence of adaptive immunity, which may be associated with hyper-progressive and pseudo-progressive disease in the clinic.



- Multiple lines of research suggests that ERK1/2 activation contributes to:
 - **Tumor-associated macrophage infiltration and M2 macrophage polarization**, causing an immunosuppressive microenvironment and reduced efficacy of anti-PD-1 therapy

Enrollment Ongoing in Australia for Continuous Dosing and Intermittent Dosing Cohorts; Combo with Nivolumab to Initiate Enrollment in 2023

ATG-017 (Tizaterkib): Potentially Best-in-Class ERK1/2 Inhibitor



Summary of ATG-017 (Tizaterkib)

ERK1/2: RAS/MAPK signaling pathway drives cell survival and proliferation; dysfunction in the signaling pathway is a major trigger for the development of most cancer types



Best-in-Class Potential

Potent and selective small molecule extracellular signal-regulated kinases 1 and 2 (ERK1/2) inhibitor with best-in-class potential

Leading in Clinical Development

- First-in-human Phase I trial investigating safety and preliminary efficacy among patients with solid tumors and hematological malignancies
- Currently in the 6th cohort of monotherapy continuous dosing in solid tumors of the Phase I "ERASER" trial
- Preliminary efficacy observed in current monotherapy dose escalation study
- Combo cohort with Nivolumab planned for early 2023

Broad Therapeutic Potential in Cancer

- Great clinical potential as a monotherapy or combination in multiple solid tumors and hematological malignancies that harbor activating alterations in the RAS-MAPK pathway
 - E.g. RASm NSCLC, Pancreatic, CRC, and Melanoma

Source: F Liu et al. Acta Pharmaceutica Sinica B2018; 8(4); 552-652. Targeting ERK, an Achilles' Heel of the MAPK pathway, in cancer therapy Note: RAS= renin-angiotensin system, SOS=son of sevenless; GRB-2=growth factor receptor bound protein 2; RTK=receptor tyrosine kinase; RAF=renin-angiotensin system; MEK=MAPK/ERK kinase; MRK=mantle cell lymphoma; RSK=ribosomal S6 kinase; MNK=MAPK-interacting kinase; MSK=mitogen-activated and stress-activated protein kinase.

ATG-101 Has the Potential to Optimize PD-L1 Antagonism and Tumor-specific 4-1BB Agonism



- Efficacy of PD-1/PD-L1 targeting is well-demonstrated over the past decade
- 4-1BB is a T cell co-stimulatory receptor, the benefits of which have yet to be realized in the clinic
- The concept of PD-L1×4-1BB bispecific antibody like ATG-101 has demonstrated promising activity in early clinical trials with an acceptable safety profile (GEN1046, NCT03917381)
- Novel design of ATG-101: high affinity of PD-L1 and conditional activation of 4-1BB, results in **4-1BB agonist activity only when crosslinked by PD-L1-positive tumor cells**
- Biodistribution murine model confirms PD-L1 drug localization¹



Enrollment Ongoing in Australia (4th Cohort) and China (3rd Cohort)

Excellent Safety Profile

- High affinity of PD-L1 and conditional activation of 4-1BB results in 4-1BB agonist activity only when crosslinked by PD-L1-positive tumor cells, reducing risk of 4-1BB related liver toxicity
 - No liver toxicity observed in GLP toxicology study in cynomolgus monkeys with dose up to 100 mg/kg

Broad Therapeutic Potential in Cancer

- Demonstrated potent in vivo efficacy in anti-PD-1/PD-L1 resistant and relapsed mouse tumor models
- Activates exhausted T cells *in vitro*, suggesting a potential in reversing T cell dysfunction and exhaustion
- Increases the infiltration, proliferation and activation of CD8+ T cells and the infiltration of Natural Killer T cells in the tumor microenvironment, thus rendering "cold" tumors "hot"

ATG-037 Can Reverse Adenosine-Mediated Immunosuppression



The adenosine axis plays a well-established and critical role in suppression of the immune response and ATG-037 can reverse adenosine-mediated immunosuppression



ATG-037: Orally Available Small Molecule Inhibitor of CD73 with Best-in-Class Potential



Summary of ATG-037

Functions to inhibit CD73 - the ecto-5'-nocleotidase that catalyzes the conversion of AMP to adenosine, a key immune suppressive molecule in the tumor microenvironment

Best-in-Class Potential

- Completely blocks CD73 activity and overcomes "hook effect" commonly seen in anti-CD73 antibodies
- Promising pre-clinical efficacy as monotherapy or in combination with standard of care (SoC) in both solid and liquid tumors
- Rescues T-cell functions in high AMP conditions

Excellent Safety Profile

- No ATG-037 related toxicity identified in GLP toxicology studies
 - Potential large therapeutic window
- No inhibition of CD39 and other related targets (up to 10 mM)

Broad Therapeutic Potential in Multiple Tumor Types

 Pancreatic, esophageal, gastric, non-small cell lung, colorectal, prostate, head and neck etc.







NK

Adenosine Induces Immunosuppressive Cell Types and Enhances Their Function

ATG-018 is an Oral and Highly Selective Small Molecule Inhibitor of ATR that may Improve on Benchmark ATR Inhibitors



- Many patients with malignant tumors carry genetic alternations which correlate with functional loss or deregulation of key DDR proteins, most notably p53 and ATM
- These tumors **extensively rely on ATR** for DNA repair
- ATG-018 can inhibit DNA damage repair, release tumor cells from cell cycle arrest and induce synthetic lethality in ATM/p53-deficient tumor cells
- ATG-018 Demonstrated superior in vivo efficacy, compared with clinical benchmark in pre-clinical CDX models



ATG-022 is a High Affinity Anti-Claudin 18.2 ADC with Potential Activity Even in Tumors with Very Low Level Expression of the Target







Australian HREC Approved in December 2022



ATG-022: An Anti-Claudin 18.2 ADC with Potent *In Vivo* Efficacy in Claudin 18.2 Very Low-Expression Tumors



Summary of ATG-022

- Claudin 18.2 is a tumor-associated antigen overexpressed particularly in gastric, esophageal and pancreatic cancers, occasionally in other tumors including lung, ovarian, and head & neck malignancies
- Clinical ADC candidate with vc-MMAE as linker payload (DAR4)

Best-in-Class Potential

- High affinity antibody (pM grade) against Claudin 18.2 allows targeting of patients with low expression of Claudin 18.2
- Strong *in vivo* efficacy pre-clinically in PDX models with various Claudin 18.2 expression levels, including in tumors with extremely low Claudin 18.2 expression

Excellent Safety Profile

- Demonstrated an excellent safety profile in GLP toxicology studies
 - Induced complete tumor regression (tumor-free) in pre-clinical PDX model without affecting the body weight of the animal
- Displayed high specificity in Retrogenix's Cell Microarray Technology Experiment
 - ATG-022 mAb specifically interacted with Claudin 18.2, the primary target, on both fixed and live cells



Christina Peters, Stuart Brown Antibody-drug conjugates as novel anti-cancer chemotherapeutics

ATG-031: First-in-Class CD24 Antibody to Inhibit the "Don't Eat Me" Signal



Summary of ATG-031

- CD24 is a novel "don't eat me" target not expressed in healthy erythrocytes, thus potentially overcoming the anemia issues commonly seen in CD47
- First-in-class humanized CD24 mAb inhibits the "don't eat me" signal by blocking CD24-Siglec10 pathway and enhances macrophage-mediated phagocytosis of cancer cells
- CDx antibody successfully developed in-house for patient selection
- Potent single agent in vivo efficacy and synergy with chemotherapy or CPI





Clinical Development Timeline Spanning 2023 to 2025 Encompassing a Series of Clinical Data Readouts



Early data on safety, PK, PD through 2023 with ATG-017, ATG-101 and ATG-037

	2023			2024					20	2026				
\sim	Q1	Q2	Q3	Q4	Q1	Q2	Q3	Q4	Q1	Q2	Q3	Q4	Q1	Q2
ATG-017 (ERK 1/2)	1 st data read out, RP2D	Start co malign	ombination anti PI ancies cohorts; Re	D-1, start hema ecruit expansion	tological n cohorts	Define & s	start pivotal prog	gram						
ATG-101 (PD-L1/4-1BB)	Dose-escalatio	n, determine RP2I	D, 1 st data read ou	t	Expansion co	horts, Ph II combi	nations	Defi	ne & start pivotal program					
ATG-037 (CD73)	Monothe 1 st da	rapy dose escala ata read out, RP2I	tion;	C	ombination dose	escalation and ex	pansion		Define & start pivot program	al				
ATG-018 (ATR)		1 st dat	a read out, RP2D			Expansior	n cohorts, Ph II co	ombinations						
ATG-022 (Claudin 18.2)			1st data read ou	ıt, RP2D			Expansi	on cohorts, Ph II	combinations					
ATG-031 (CD24)	IND			1st data i	read out, RP2D									



III. COMMERCIAL STAGE ASSET UPDATE



ASEAN NDA Schedule

H2 2024

XPOVIO® (selinexor) XPOVIO®





• rrMM - XPOVIO[®] in combination with bortezomib and dexamethasone (XVd)

(selinexor)^{20 mg}

- rrMM XPOVIO[®] in combination with dexamethasone (Xd)
- rrDLBCL XPOVIO[®] as monotherapy (X)

Expected Approval in Malaysia

Expected Approval in Thailand H2 2024

- rrMM XPOVIO[®] in combination with bortezomib and dexamethasone (XVd)
- rrMM XPOVIO[®] in combination with dexamethasone (Xd)
- rrDLBCL XPOVIO[®] as monotherapy (X)

Expected Approval in Indonesia H2 2024

- rrMM XPOVIO® in combination with bortezomib and dexamethasone (XVd)
- rrMM XPOVIO[®] in combination with dexamethasone (Xd)
- rrDLBCL XPOVIO[®] as monotherapy (X)

ATG-010 (Selinexor) - First-in-class and Only-in-class SINE Compound with Unique MoA and Differentiated Profile





Key Highlights

- 1st and only XPO1 inhibitor approved by FDA for treating R/R MM and R/R DLBCL
- 1st and only FDA-approved drug for the treatment of both R/R MM and R/R DLBCL
- Only single-agent, oral therapy approved by the FDA to treat R/R DLBCL
- Recommended by NCCN and CSCO guidelines for R/R MM and R/R DLBCL treatment



SINE + mTORi

Selinexor + ATG-008 in diffuse large B-cell lymphoma (MATCH)

■ SINE + I/O:

Selinexor + ATG-101 in solid tumors and lymphoma

Clinical Benefits Validated by Selinexor's Completed and Ongoing Studies in Multiple Myeloma and DLBCL





Source: Dimopoulos M, et al. ASCO 2020. Abstract 8501; Gasparetto C, et al. ASH 2020. Abstract 1366.; Gasparetto C, et al. ASH 2020. Abstract 8510.; Chen C, et al. ASH 2020. Abstract 726.; White D, et al. ASH 2020. Abstract 1393.; Kyprolis Package Insert; Study PX-171-003 A1; Lonial et al. Lancet 2016.; Pomalyst Package Insert.; Stewart et al. (Results of the Pivotat STORM Study (Part 2) in Pentidomide). Pomalyst* (pomalidomide). Pomalyst* (pomalidomide). Pomalyst* (pomalidomide). Chari A, Vogi DT, Dimopoulos M, et al. Results of the Pivotat STORM Study (Part 2) in Penti-Refractory Multiple Myeloma (MM): Deep and Durable Responses with Oral Selinexor Plus Low Dose Dexamethasorne in Patients with PentaMM. Blood 2018; FDA label for XPOVIO* (selinexor); Kalakonda N, et al. Carrently in press and publication expected in the near term (Lancet Haematology 2020).

*Some of the information in this presentation is from third-party medical professionals and for academic purposes only. Antengene is not responsible for the contents published by such external sources. **Data shown for SDd and SPd in STOMP are from patients not previously exposed to D and patients dosed at RP2D respectively.

Multiple Selinexor Containing Regimens Recommended by NCCN, ESMO, CSCO, and CMDA-CMA Guidelines

National Comprehensive Cancer Network[®]

Multiple Myeloma

1-3 Prior Therapies

- SVd QW
- SDd
- SPd
- SKd

> 3 Prior Therapies (whose disease is refractory to at Least Two PIs, IMIDs, and an anti-CD38 mAb)

• Sd

Diffuse Large B-cell Lymphoma

3L+ (including patients with disease progression after transplant or CAR T-cell therapy)

S monotherapy



European Society for Medical Oncology

Multiple Myeloma

2L Option After VRD

- R sensitive (SVd)
- R refractory (SVd)
- V sensitive (SVd)

2L Option After DaraRD

- R sensitive (SVd)
- R refractory (SVd)

2L Option After DaraVMP or DaraVTD

• V sensitive (SVd)

Second or Subsequent Relapse

- R refractory and PI sensitive (SVd)
- Triple-class refractory (Sd)

CSCO

Multiple Myeloma

Relapsed/Refractory



Diffuse Large B-cell Lymphoma

Relapsed/Refractory

S monotherapy — Upgraded to Level 2 Recommendation



Chinese Medical Doctor Association

ANTENGENE

Chinese Medical Association

Multiple Myeloma

Relapsed/Refractory

SVd
SPd
SDd
SKd

* Some of the information in this presentation is from third-party studies and only provided to medical research professionals for academic purpose. Antengene is not responsible for the contents published by such external sources. ** Approved for RRMM by the US FDA, European EMA, Chinese NMPA, Korean MFDS, Singaporean HSA, Australian TGA, and Taiwan TFDA. Approved for RRDLBCL by the US FDA, Korean MFDS, Singaporean HSA, and Taiwan TFDA. As of Nov 14, 2022. *** Indications for Selinexor undergoing clinical trial / planned study include: PTCL, NKTL, AML, MDS, MF

Dose Reduction can be Used to Manage Patients, while Optimizing Outcomes with PFS of 16.6 Months



The median dosage of XPOVIO in the BOSTON trial was 80 mg (range: 30-137 mg) taken once weekly¹

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Key efficacy endpoints for patients (N=195) with and without XPOVIO dose restrictions in the BOSTON trial²

	ITT Patient Population	Patients with Dose Reduction
Patient population	N = 195	n=126
% of ITT arm	100	65
mPFS, mo	13.9 (95% CI: 11.7, NE)	16.6 (95% CI: 12.9, NE)
ORR, %	76.4	81.7
≥VGPR, %	44.6	51.6
mDOR, mo	20.3 months (95% Cl: 12.6, NE)	Not evaluable (95% CI: 13.8, NE)

Limitation of Subgroup Analyses:

- This post hoc analysis was exploratory in nature, not included in the study objectives, and does not control for type 1 error
- This post hoc analysis was not powered or adjusted for multiplicity to assess PFS across other prespecified subgroups
- This post hoc analysis was underpowered to detect clinically meaningful differences in treatment effect

65% of patients in the XVd arm had an XPOVIO dose reduction (126/195 patients) vs 35% of those without a dose reduction (65/195 patients)²

Source: Karyopharm Investor Presentation dated December 8th, 2021

1. XPOVIO. Prescribing information. Karyopharm Therapeutics Inc; 2021. 2. Jagganath, et al. ASH 2021

*** Approved for RRMM by the US FDA, European EMA, Chinese NMPA, Korean MFDS, Singaporean HSA, Australian TGA, and Taiwan TFDA. Approved for RRDLBCL by the US FDA, Korean MFDS, Singaporean HSA, and Taiwan TFDA. As of Nov 14, 2022.

^{*} Response was evaluable in 125 of 126 patients receiving a dose modification for selinexor and 66 of the 69 patients who did not receive a dose modification for selinexor. Overall response rate is the proportion of patients who achieve a partial response or better, before IRC-confirmed progressive disease or initiating a new MM treatment or crossover. CI=confidence interval, IRC=independent review committee; ITT=intent to treat; mDOR=median duration of response; mo=month; mPFS=median progression-free survival; NE=not evaluable; ORR=overall response rate; VGPR=very good partial response.

^{**} Some of the information in this presentation is from third-party studies and only provided to medical research professionals for academic purpose. Antengene is not responsible for the contents published by such external sources.

XPOVIO Evolving into a Standard of Care with Dose and Schedule Redefined Over Time to Improve Efficacy and Patient Experience



From the STORM trial to the BOSTON trial to the STOMP trial, XPOVIO dosing has been continually refined to help optimize the patient experience



Source: Karyopharm Investor Presentation dated February 8th, 2022

* STOMP was designed to study selinexor in combination with other MOAs across multiple triplet and quadruplet regimens, including XVd. MM=multiple myeloma; MOA=mechanism of action; RRMM=relapsed or refractory multiple myeloma.

** Combinations other than XVd and Xd are not promoted by Karyopharm, but may be considered for future indication updates.

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****** Indications for Selinexor undergoing clinical trial / planned study include: PTCL, NKTL, AML, MDS, MF

Broad and Deep Potential for Selinexor / SINE Beyond Multiple Myeloma





Source: Antengene research

* Investigator Initiated Trials (IIT)

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Antengene is Focused on Markets with Greatest Commercialization Potential





Commercialization strategy focuses on selection of 6 core stage 1 markets defined by 3 parameters for success:

- High unmet need
- Reimbursed markets
- High GDP/capita



Initial focus is to build Antengene presence in the core markets



Ensure successful commercial launch of Xpovio®



Expand portfolio in core markets and expand Antengene presence to other stage 2 ASEAN markets

Antengene is Expanding into Stage 2 ASEAN Markets with Significant Future **Commercialization Potential**





Building XPOVIO® Launch Momentum with Regulatory Approvals Across Core Markets

ANTENGENE



Commercial Team with a Proven Track Record of Success





Fewer Multiple Myeloma Medicines Approved in China/Reimbursed in Australia Compared to the US – Launching with Less Competition Outside the US





Successful Commercial Launch of XPOVIO® in Mainland China

Approved Indication:

XPOVIO[®] in combination with dexamethasone (Xd) in Relapsed / Refractory Multiple Myeloma (rrMM)

Treatment Guideline Recommendations in China

- R/R Multiple Myeloma:
- **CSCO Guidelines** for the Diagnosis and Treatment of Hematologic Malignancies 2022
- CMDA & CMA Guidelines for the Diagnosis and Management of Multiple Myeloma in China (2022 revision)

R/R Diffuse Large B-cell Lymphoma:

CSCO Guidelines for the Diagnosis and Treatment of Lymphomas 2022

Initial Observations for XPOVIO[®] Launch in China Market - XPOVIO[®] Being Prescribed in Earlier Lines of Therapy in a Range of Combination Regimens

Physician Testimonials Highlighting XPOVIO's Differentiated Profile

"MARCH, BOSTON trial data indicates that Selinexor combo regimens bring more innovative therapeutic options and better treatment outcomes for relapsed/refractory, multidrug resistance, metastasis and/or high-risk MM patients."

> KOL, Dr. Jun Ma, Chief Supervisor of CSCO, Harbin Hematology and Oncology Institute

"Selinexor is more convenient and likely leads to higher compliance because it is an **oral regimen**. The efficacy of Selinexor is proven in a number of clinical trials. Besides being used as a monotherapy, Selinexor could also be **combined with a number of drugs such as chemo, target therapy, I/O, etc.**"

> KOL, Dr. Zhiming Li, Sun Yat-sen University Cancer Center

Asia Pacific Markets - Executing on XPOVIO® Launch Plans

- Total number of XPOVIO[®] treated patients **doubled in 3 months** (between July to October)
- First multiple myeloma indication (Xd regimen) included for reimbursement on 1st September
 - XPOVIO[®] achieved that in 180 days; whereas oncology medicines are listed in 496 days on average
- Xd achieved >50% new patient share of available penta-refractory patients
- Reimbursement of XVd regimen anticipated in H1 2023

Other Asia Pacific Markets

- Reimbursement anticipated in Taiwan and South Korea in 2024
- Building of KOL advocacy and XPOVIO[®] experience:
 - >250 patients treated with XPOVIO[®] via pre approval access program
 - o Pre-reimbursement Patient Familiarization Program activated
- ASEAN markets expansion commencing with NDA submissions in Thailand and Malaysia in 2022, and Indonesia in H1 2023

Limited Availability of Reimbursed Triplet Regimens in APAC Markets Compared to the US

IV. INVESTMENT HIGHLIGHTS

Steady Stream of Catalysts Continue to Drive Value for Investors

Focused on Execution and Key Priorities to Drive Value for Investors in 2023

ANTENGENE CORPORATION LIMITED (SEHK: 6996.HK)

MARCH 2023

THANK YOU

TREATING PATIENTS BEYOND BORDERS