

BioNOVA Introduction
Pharma 焯辉医药 May 2022

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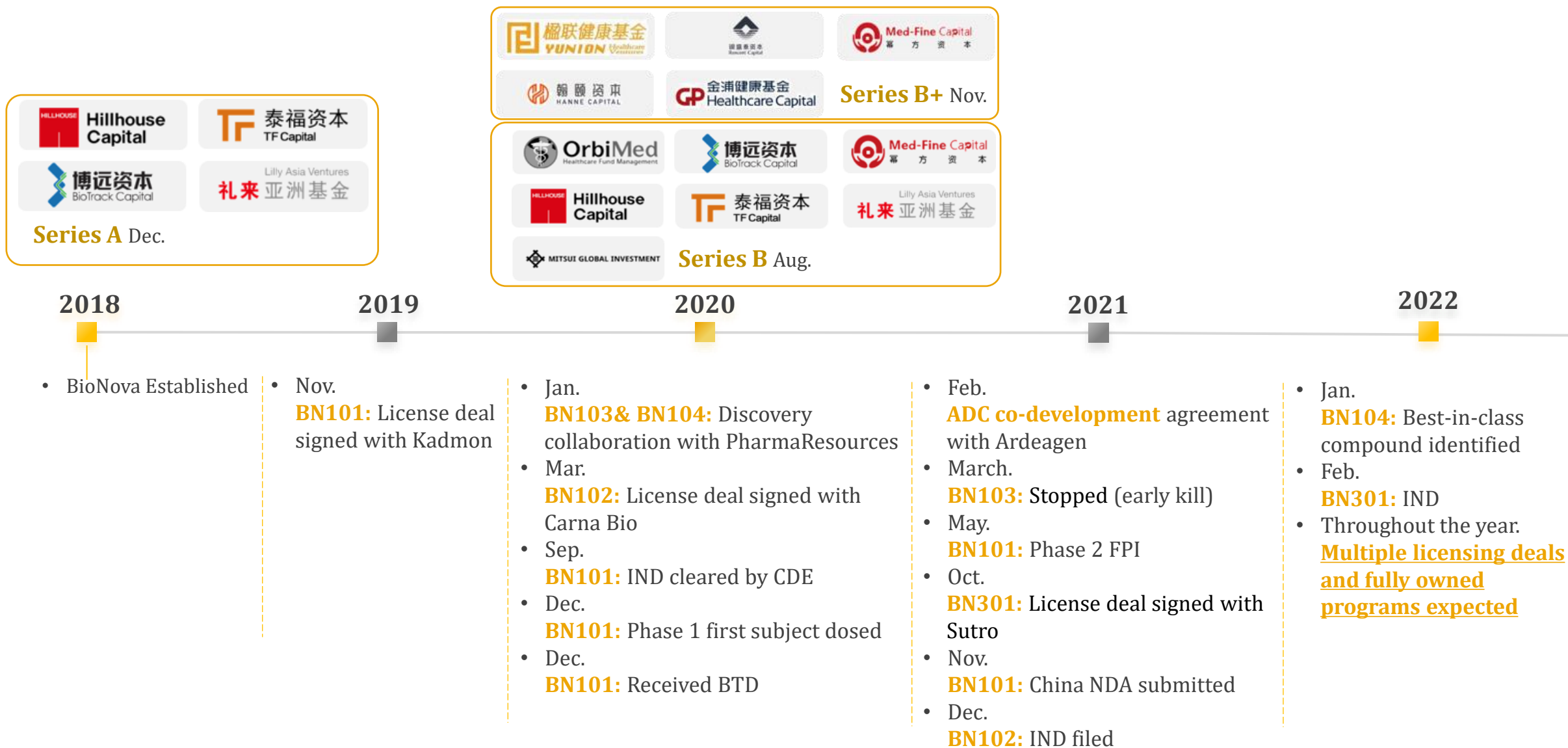
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I. Company Overview

TRACKING
RETINA PATH



BioNova Company History





Company Strengths and Vision

Execution

- ❑ Highly capable executive team with under-promise but over delivery mindset;
- ❑ Proven track records in BD, in-licensing with value creation and speedy clinical development;
- ❑ In-house cross-functional discovery team with efficient ongoing due diligence backed decisions for competitive advantage

Innovation

- ❑ Utilizing industrial best mastermind to architect differentiated in-house discovery and co-development pipeline
- ❑ Deep understanding in development and regulatory strategy to guide BioNova with timely development and high probability of success

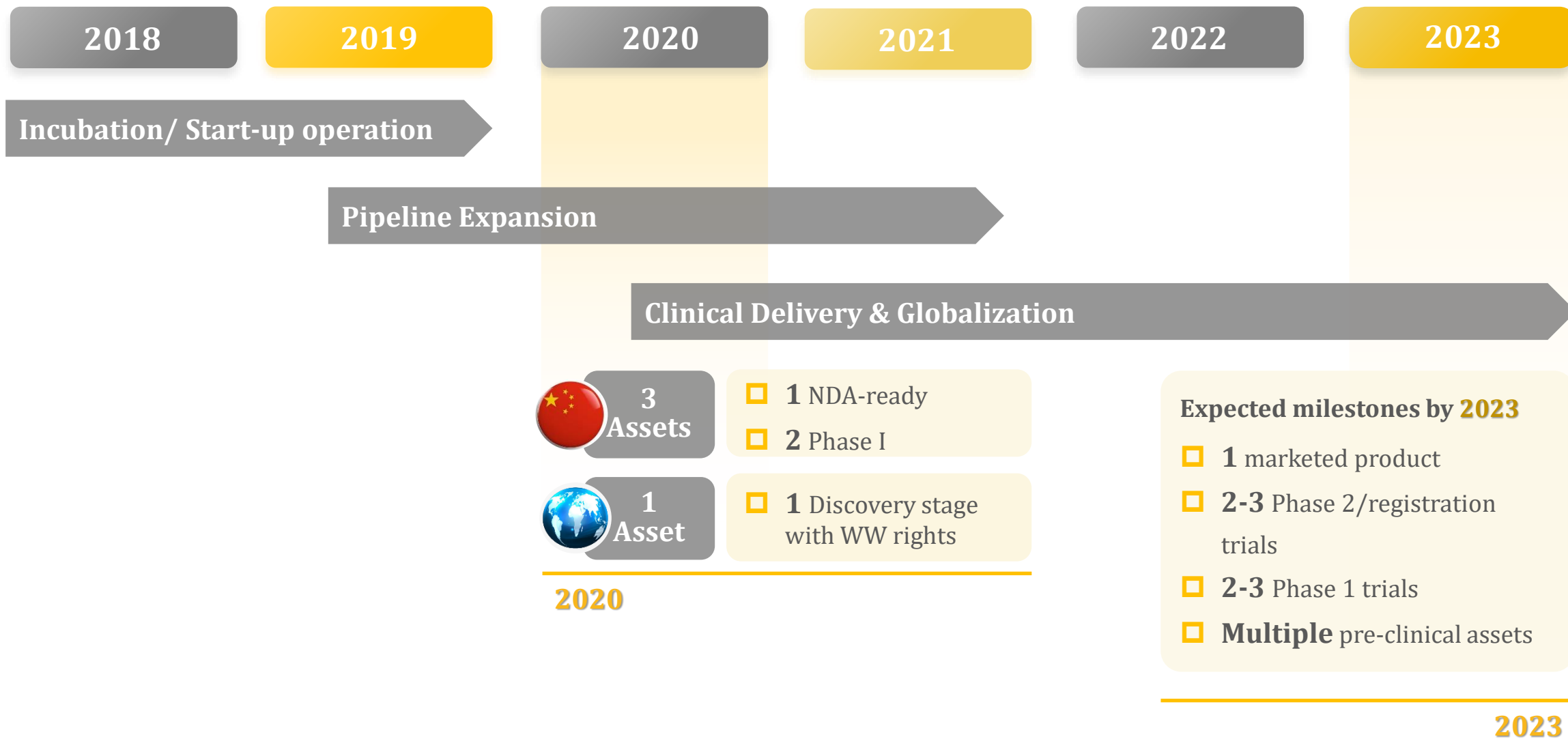
Globalization

- ❑ Global team with insight to position promising targets with competitive advantage
- ❑ Assets with worldwide rights of out-licensing potentials
- ❑ Clear company growth plan to go from China to worldwide

❑ BioNova is growing steadily with huge potential to become a top China biopharma with global footprint.



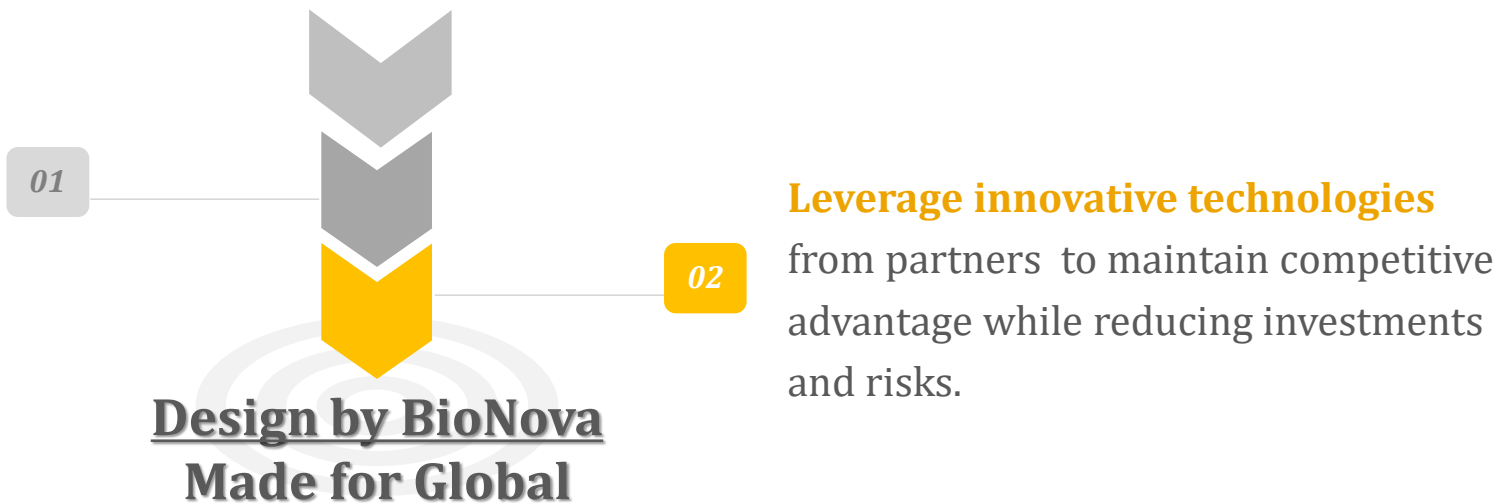
Execution - Robust Pipeline Progress in 3 Years, with Promising Expected Milestones





Innovation - In-house Discovery and Co-development Strategy

Fully utilize **BioNova knowledge and strength in hem/onc.** to build up in-house pipeline with synergistic potential for diseases with clear UMN;



In-house

□ “Home Grown” assets:

- Focusing on targets with clear mechanism and potential for quick registration;
- Leveraging in-house expertise and deep understanding of the landscape;
- Highly selective and cost-effective projects with clear criteria for early killing.



**Co-
development**

□ **Co-development** with master brains to own cutting-edge technologies:

- Leveraging innovative technologies from global partners;
- Cost-sharing approach to reduce investments and risks;
- Maintaining decision power and options for more upside.



Globalization - Overview and Development Plan

Growing into a Global Biopharma

01

- “Jump start” with acquired assets targeting huge unmet medical needs but less “crowded.”
- Carefully position discovery assets targeting proven biology and off to fast development potential.

02

- Speedy move to cutting-edge technologies from partners’ expertise and know-how.
- Innovative development strategy and regulatory pathways that add value to partners.

03

- “Home grown” novel target agents for global simultaneous development.
- Out-licensing opportunities for ex-China development and commercialization.

04

- Clear company growth plan to go from China to worldwide.
- Long-term global innovation company and asset acquisition plans.

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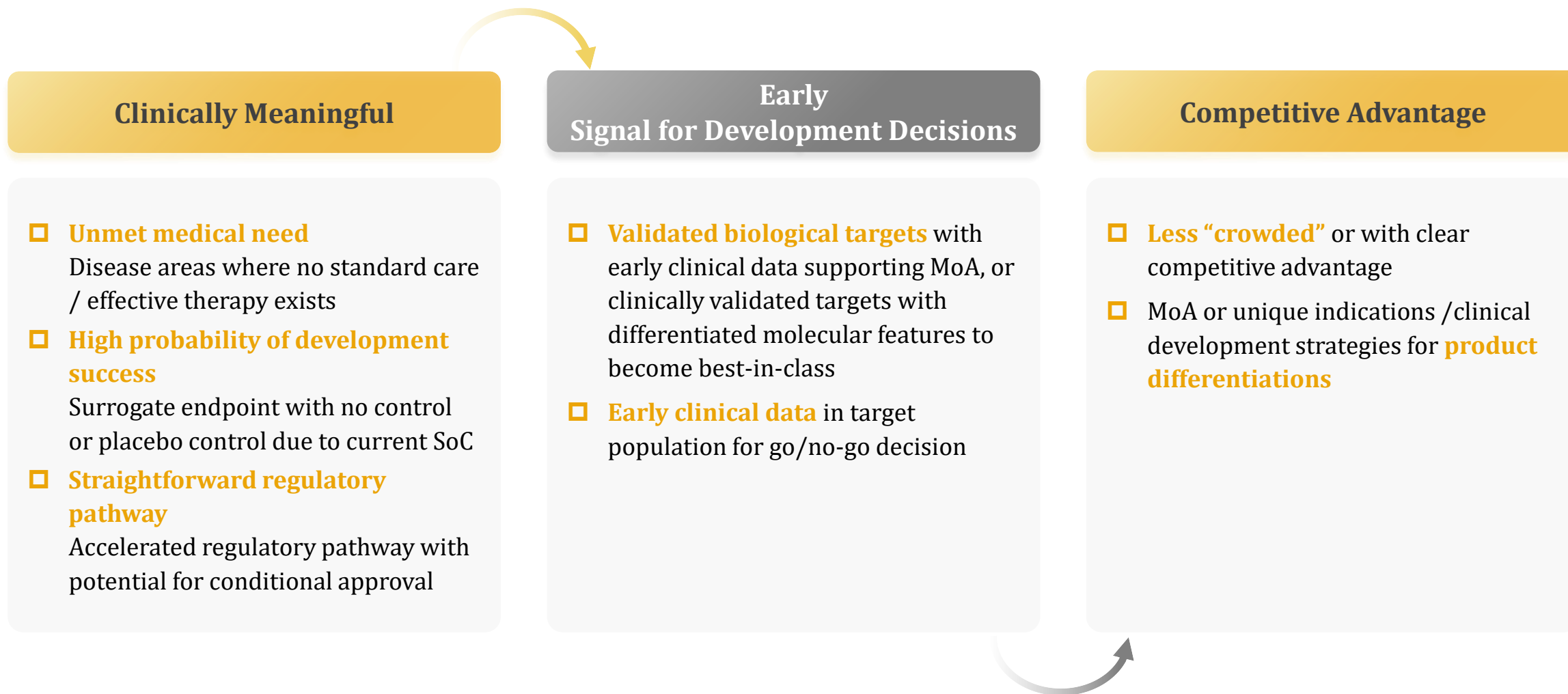
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III. Product Pipeline

TRACKING
RETINA PATH



Pipeline Build-up Strategy





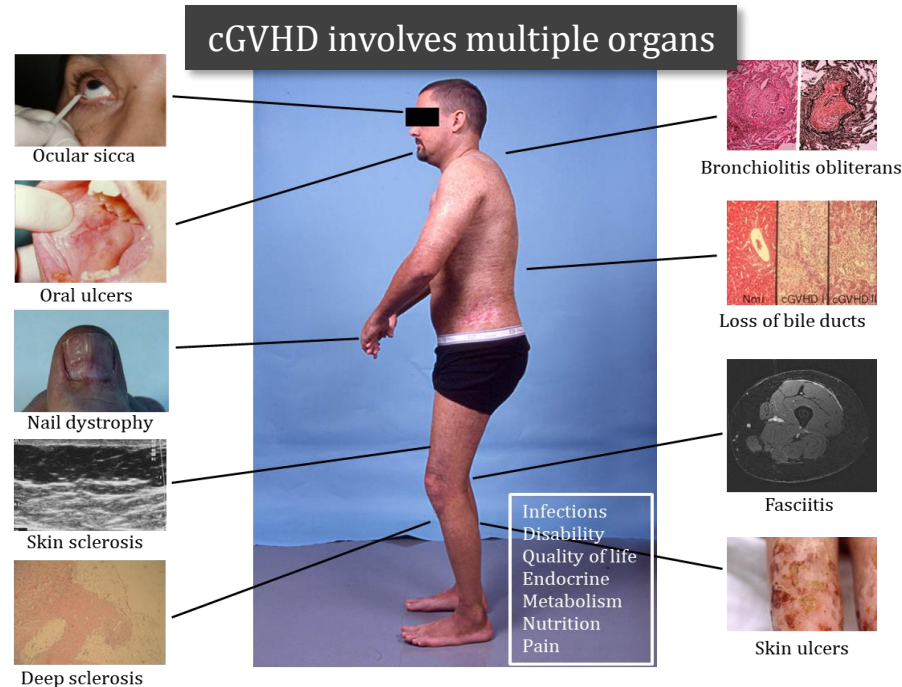
Current Pipeline

Product Candidate	Target	Indication	Discovery	IND Enabling	Phase 1	Phase 2	Phase 3	Commercial Rights
In-licensed pipeline								
BN101	ROCK2	cGVHD				NDA		China
BN102	BTK (reversible)	CLL/SLL, MCL, WM, MZL						China
BN301 (ADC)	CD74	NHL, AML, MM						China
In-house R&D pipeline								
BN104	Non disclosure	AML	PCC					WW

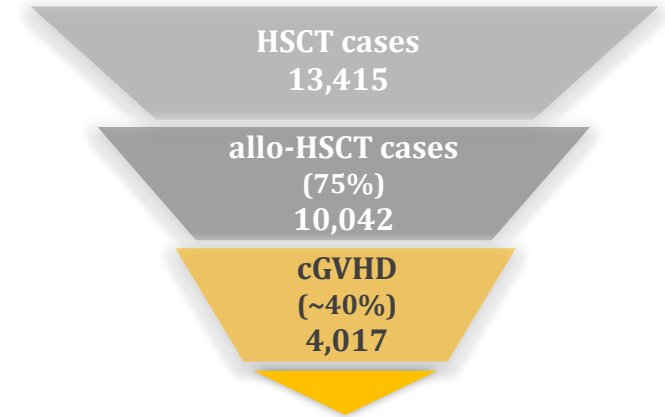


Chronic GVHD (cGVHD)

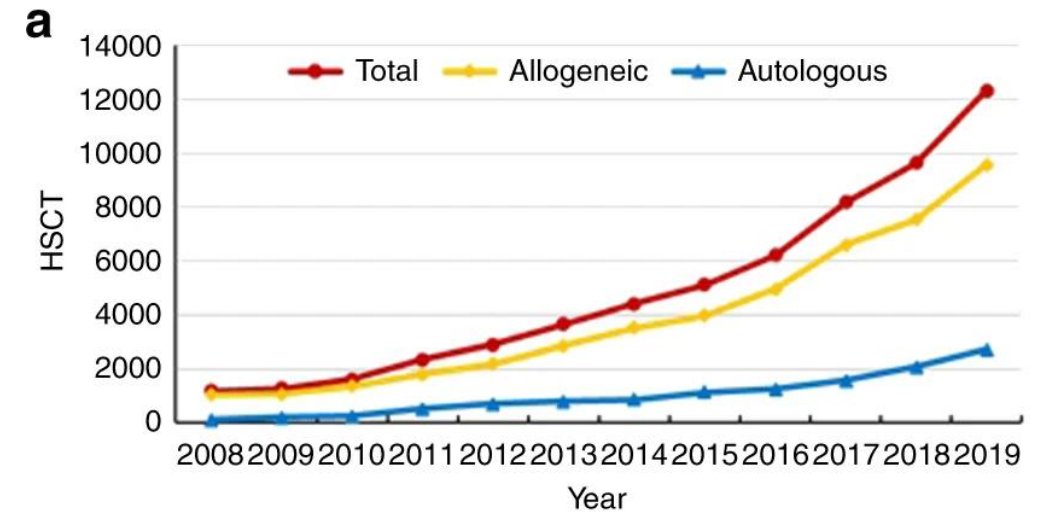
- **Graft-versus-host disease (GvHD)** is the common complication following allogeneic hematopoietic stem cell transplantation (allo-HSCT) which is the most desirable therapy of curative potential for leukemia and lymphoma patients. It is a systemic disorder that occurs when the graft's immune cells recognize the host as foreign and attack the recipient's body cells, leading to inflammation and fibrosis in multiple tissues.
- **Chronic GVHD (cGVHD)** with a high incidence of 30%-70% in GvHD, is the major cause of late non-relapse death after HSCT. cGVHD may manifest simultaneously from acute GvHD (aGVHD), develop after the treatment of aGVHD, or may occur *de novo*. Classical cGVHD occurs 100 days after transplantation but may also overlap with aGVHD.



Estimated 2020 cGVHD incidence in China



HSCT trend in China during 2008-2019

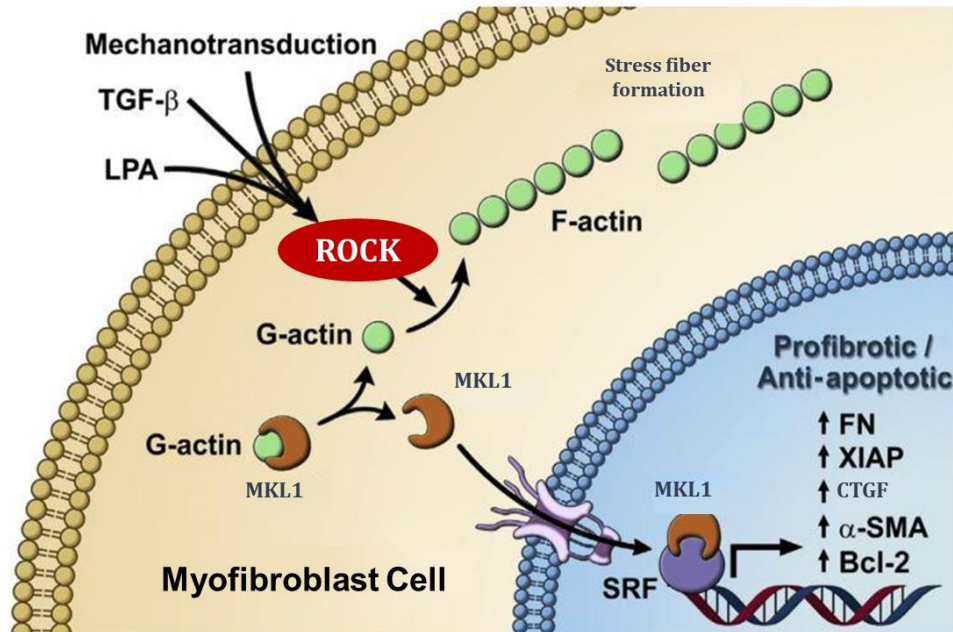




BN101: A FIC Selective ROCK2 Inhibitor for cGVHD

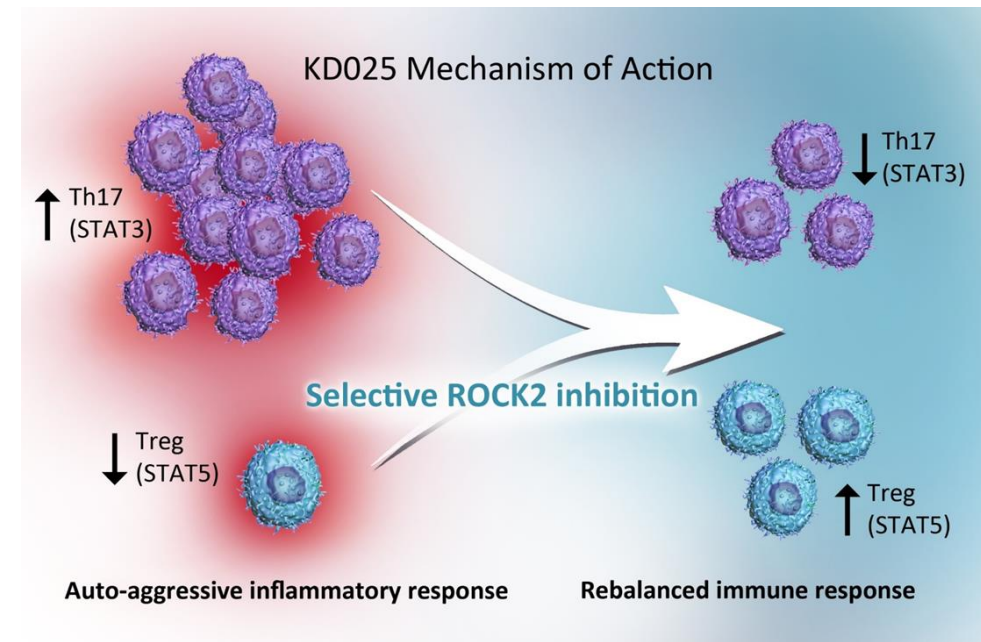
ROCK: an Intracellular Integrator of Pro-fibrotic Signal

- ❑ ROCK regulates multiple profibrotic processes, including myofibroblast activation
 - ROCK is downstream of major pro-fibrotic mediators
 - ROCK regulates fibroblast differentiation to myofibroblasts, a pathological cell type in fibrosis
 - ROCK mediates stress fiber formation
 - ROCK regulates transcription of pro-fibrotic genes



BN101/KD025: A FIC Selective ROCK2 Inhibitor

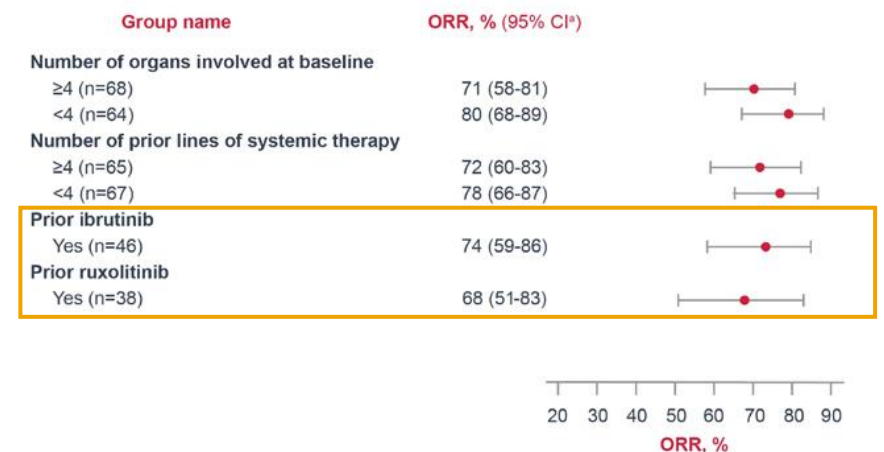
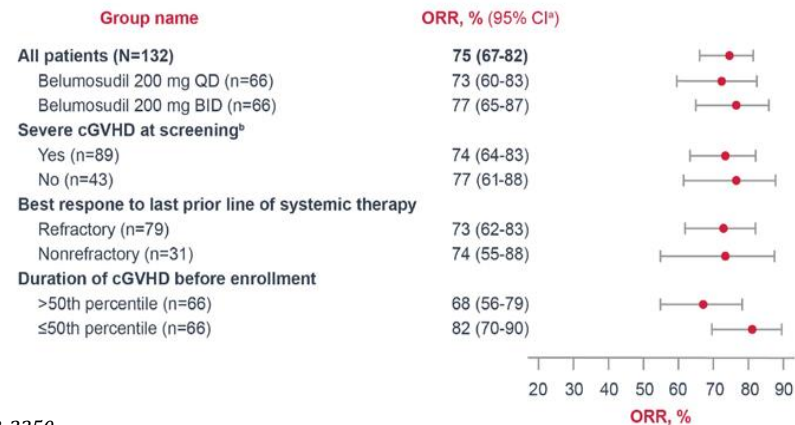
- ❑ ROCK2 inhibition downregulates pro-inflammatory Th17 responses and increases Treg function
 - Reduces STAT3 phosphorylation
 - Increases STAT5 phosphorylation
- ❑ ROCK2 inhibition re-establishes immune homeostasis



BN101: Superior Product Profile for cGVHD

		KD025 ¹	Ibrutinib ^{2*}	Ruxolitinib ^{3*}
Dosing		QD	QD	BID
Indication		cGVHD 3L+	cGVHD 2L+	aGVHD 2L+
Efficacy (ORR/CR)		73%	67%	76.4%
Key Patient Characteristics	Median prior line	3	2	< 2 lines
	>=4 organ involved	50%	7% (72% <= 2)	NA
	Moderate/severe cGVHD	27%/70%	52%/40%	40.6%/58.8%
Serious AEs (Gr 3/4 >=5%)	Infection	8%	36% (1 grade 5)	19.4%
	Anemia	0%	2%	12.7%
	Thrombocytopenia	0%	0%	15.2%
	Neutropenia	0%	0%	8.5%
	Hypertension	6%	0%	4.8%
	Atrial fibrillation		2%	

High response rate of BN101
regardless of prior treatment
with ibrutinib or ruxolitinib



1. ASH 2020
 2. Miklos D. et al. BLOOD, 23 November 2017; 130(21): 2243-2250
 3. Robert Z. et al. NEJM, 15 July 2021: 228-238
 * Not approved for cGVHD in China



BN101 Global Development Timeline



- 5 Oct, Orphan Drug Designation for cGVHD

- 9 Sep, IND approval in China
- 30 Sep, Rolling submission of cGVHD NDA to FDA - 30 Nov, NDA submission completed
- 26 Nov, Ph 1 trial in China
- 18 Dec, CDE Breakthrough Therapy Designation

2015

- cGVHD IND in US

2017

2018

- 16 Oct, FDA Breakthrough Therapy Designation

2020

- Feb, China Ph1 completed
- Apr, BN101-201 clinical trial
- 16 Jul, FDA approval in US
- China NDA filed in Nov 2021

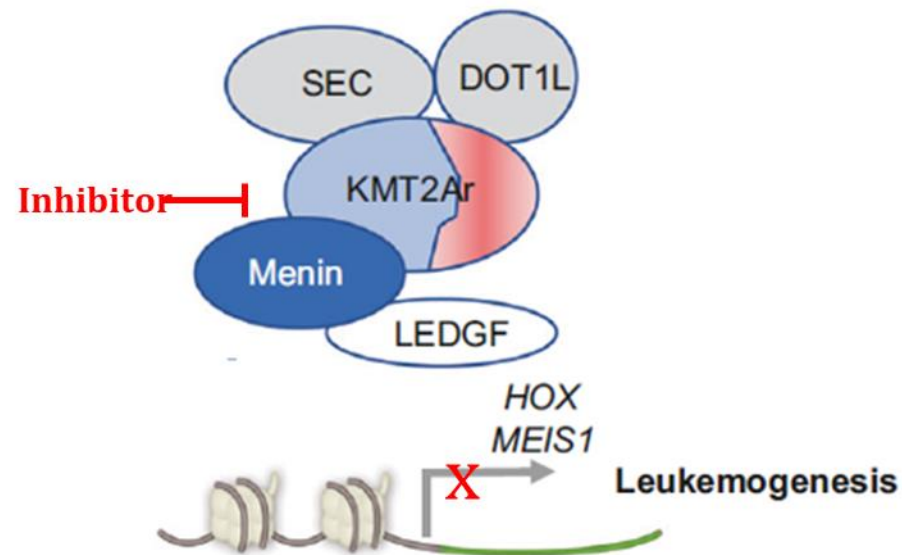
2021



BN104 - Menin Fusion: A Validated Target for Both MLLr (KMT2Ar) and NPM1c Mutant

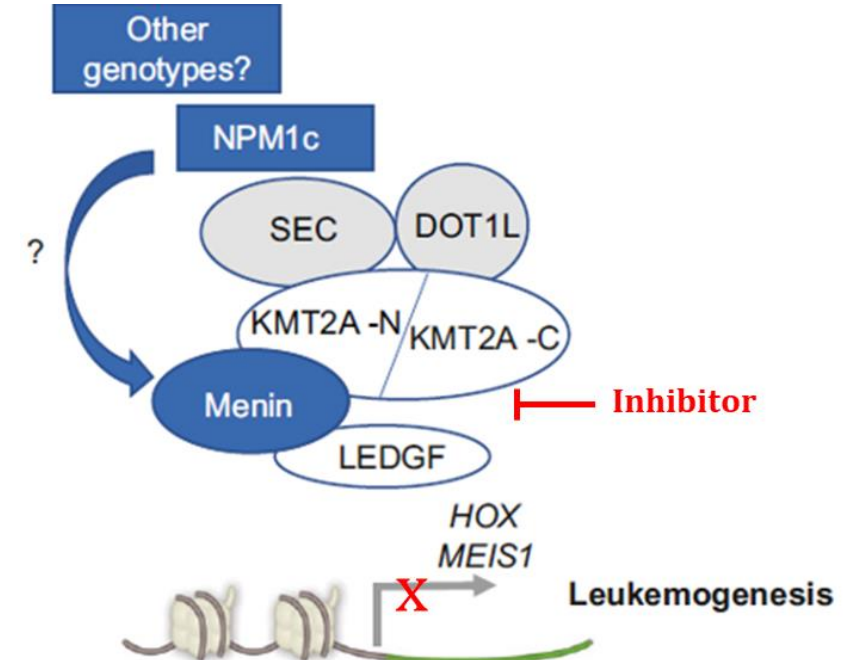
MLLr acute leukemias

- Annual global incidence 5,000-7,000
- 4-10% AML
- 10-15% ALL (80% of infant ALL)
- 15% therapy-related AML (70% for topo II)
- 5-year OS for adult MLLr <25%



NPM1c AML

- Annual global incidence ~20,000
- 20-30% AML
- 5-year OS for adult NPM1c 50%



Issa et al., *Leukemia*, 2021, 35, 2482-2495 ; Dohner, H. et al. *Blood*, 2017, 129(4), 24-447; Falini, B. et al. *Blood*, 2011, 117(4), 1109-1120;



BN104 – A Potential Best-in-Class Menin Inhibitor



Abstract #3943

Discovery of A Novel Menin-MLL Inhibitor for Potential Treatment of MLLr Leukemias and NPM1c AML

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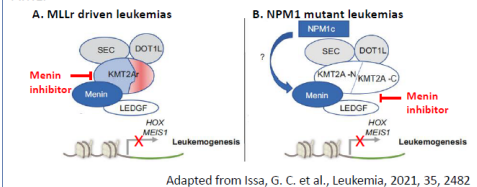
Abstract

Patients with MLL rearranged (MLLr) acute leukemias often have poor prognosis, and there is no targeted therapies available for this subtype of leukemias. The protein protein interaction (PPI) between MLLr and menin is critical for the pathogenesis of MLLr-driven leukemias. And it has been well demonstrated in both preclinic and clinic that blockade of this PPI could have therapeutic implications in the treatment of menin-MLL dependent leukemias. Herein we report the discovery of BNM-1192, a small molecule menin inhibitor. BNM-1192 is a potent and highly selective menin inhibitor with optimized drug-like properties and showed excellent efficacy in MV4-11 xenograft mouse model. Furthermore, BNM-1192 has low risk in QTc prolongation given the fact it is an extremely weak hERG inhibitor with IC₅₀ of greater than 100 μM. It also demonstrated favorable toxicological profile in preliminary tox studies.

Background

Rearrangement of the mixed lineage leukemia (MLL, also known as MLL1 or KMT2A) gene occurs in about 10% of acute leukemias, and is particularly prevalent in infant acute leukemias, accounting for up to ~70% of infant acute lymphocytic leukemia (ALL) cases. More than 80 partner genes are implicated in MLL fusions, and six main partner genes make up about 80% of cases, which include AF4, AF6, AF9, AF10, ENL and ELL. MLL fusion proteins enhance proliferation and block hematopoietic differentiation, ultimately driving the development of leukemia by dysregulation of the HOXA and MEIS1 genes. MLLr leukemia is one of the high-risk types of leukemia with aggressive nature, resistance to therapy, and high frequency of early relapse, and with a 5-year survival rate of only approximately 35%.

The interaction between menin and MLLr is critical to the pathogenesis of MLLr-driven leukemias. Recent studies also revealed the importance of the menin-MLL1 wild-type (wt) interaction in NPM1 mutant AML. And blocking the menin-MLL interaction has proved to be a viable therapeutic strategy for the treatment of MLLr associated acute leukemias and NPM1 mutant AML.



BNM-1192 is a potent and selective menin inhibitor



Figure 1. Left, Menin-MLL peptide cocrystal (PDB 4GQ6). Menin shown as ribbon in cyan, and MLL peptide as sticks in magenta. Right, docking pose of BNM-1192 binding to menin. BNM-1192 binds to the same menin pocket as MLL N-terminal peptide. And a hydrogen bond formed directly between small molecule and Glu363 of menin was revealed.

Table 1. Antiproliferative activities against leukemia cell lines.

	Cell lines	BNM-1192	SNDX-5613
IC ₅₀ (nM)	MV-4-11 (AF4 fusion)	3.5±1.2 (n=8)	9.2±3.0 (n=7)
	MOLM13 (AF9 fusion)	12	26
	OCI-AML3 (NPM1 mutant)	11	75
	HL-60 (MLL wild type)	>10000	~8000

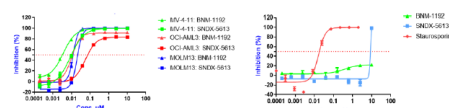


Figure 2. Dose response curve. Left, targeted cell lines; right, HL-60, control cell line.

- BNM-1192 is very potent against leukemia cell lines with MLL-fusion protein and NPM1 mutant.
- ~1000-fold selectivity over control cell line, HL-60, observed.

BNM-1192 leads to menin protein degradation

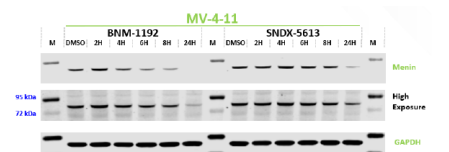


Figure 3. Western blot to determine menin protein. MV-4-11 cells were treated with BNM-1192 and SNDX-5613 at 10 μM for 2, 4, 6, 8, 24 hours, respectively, with DMSO as the control. Transfer: iBlot P0-9min; Sample: cell lysate; Total Protein: 30ug (BCA) 4-12% BT Gel & MOPS; M: Marker (Beyotime# P0069)

BNM-1192 demonstrated decent PK properties

Table 2. Cross-species PK

PK parameters	Mouse	Rat	Dog
CL (mL/min/kg) ^a	64	90	22
t _{1/2} (h) ^a	1.6	2.4	3.9
V _{ss} (L/kg) ^a	4.5	13	3.8
AUC ₀₋₂₄ (ng*h/mL) ^b	1272	272	6186
F (%) ^b	50	16 (175 ^c)	79

^a iv 1mg/kg; ^b po 10 mg/kg; ^c po 100 mg/kg;

- Good to excellent bioavailability in mouse and dog; improved exposure at higher dose for rat.

BNM-1192 showed excellent efficacy in MV-4-11 xenograft mouse model

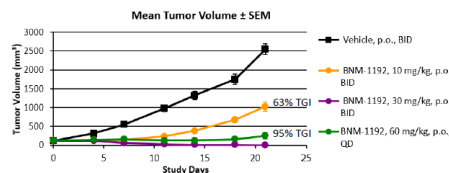


Figure 4. MV-4-11 xenograft mouse model. Mice were administered orally either vehicle or compound for 21 days dosed QD or BID as indicated.

Table 3. PK of mice with tumor burden

Dose (mg/kg)	C _{max} (ng/mL)	C _{sh} (ng/mL)	AUC ₀₋₂₄ (ng.hr/mL)
10	68	7.2	250
30	564	43	1680
60	1575	154	7166

- BNM-1192 showed dose-dependent efficacy
- BNM-1192 at 30 mg/kg BID resulted in tumor regression
- BNM-1192 at 60 mg/kg QD also showed good tumor inhibition (95%).
- Duration of coverage above IC₅₀ (IC₅₀) seems more important than exposure for efficacy

BNM-1192 has low risk in QTc prolongation

Table 4. In vitro Early safety data

	BNM-1192	SNDX-5613
hERG (IC ₅₀ μM)	>100	9.6
Mini Ames (TA98/TA100/TA1535/TA1537; WP2 uvrA (pKM 101))	Negative	ND

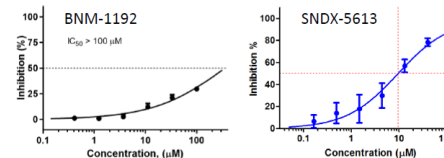


Figure 5. Concentration-dependent blockade of hERG channel. Left, BNM-1192; right, SNDX-5613.

BNM-1192 showed favorable tox profile

- A 7-day preliminary tox studies in rat was done
- No significant findings
- The high dose, 1000 mg/kg, identified as the HNSTD
- >500-fold safety margin based on exposure (AUC)

Conclusions

- BNM-1192 is a low nanomolar menin inhibitor. As high as 1000-fold selectivity was observed for targeted cell lines over mechanistically irrelevant cell line.
- BNM-1192 resulted in tumor regression at 30 mg/kg, BID in MV-4-11 xenograft mouse model. And QD dosing is a promising alternative dosing regimen.
- BNM-1192 is an extremely weak hERG inhibitor, indicating very low potential in QTc prolongation.
- BNM-1192 demonstrated favorable profile in early safety and toxicology.
- IND-enabling studies of BNM-1192 is ongoing, and Phase 1 study is expected in early 2023.



BN102 - Resistance to Covalent BTK Inhibitors Creates a New High Unmet Medical Need

B-cell Malignancies

- ❑ **B-cell malignancies** include chronic lymphocytic leukemia (CLL)/small lymphocytic lymphoma (SLL) and B-cell non-Hodgkin's lymphoma (NHL), such as diffuse large B-cell lymphoma (DLBCL), mantle cell lymphoma (MCL), follicular lymphoma (FL), marginal zone lymphoma (MZL) and lymphoplasmacytic lymphoma/Wahrenheit's macroglobulinemia (LPL/WM)
- ❑ In 2021, CLL and NHL together accounted for 5.4% of all new cancer cases in the United States and 4.1% of all cancer deaths.^{1,2}
- ❑ GLOBOCAN data shows that in 2020 there will be **92,834 new cases of NHL in China**, accounting for 2.0% of all new tumor cases, and an increasing trend year by year, with 54,351 deaths, accounting for 1.8% of all tumor deaths.³
- ❑ In China, B-cell NHL accounts for approximately **75% of all NHL**.

Reversible BTK Inhibitors

- ❑ **BTK (Bruton's Tyrosine Kinase)** plays a key role in B cell antigen receptor (BCR) signal transduction
 - BCR signal transduction is essential for the survival and proliferation of leukemia cells in many B-cell malignancies
- ❑ **Current therapeutics:** Covalent BTK inhibitors such as ibrutinib have been approved for the treatment of CLL/SLL, MCL, MZL and WM
 - 2020 ibrutinib global sales exceeded 10 billion USD. However, no treatment exists once patients progress on covalent BTK inhibitors
- ❑ **Next-generation, reversible BTK** inhibitors have demonstrated very promising efficacy in patients progressed on covalent BTK inhibitors.⁴

1. <https://seer.cancer.gov/statfacts/html/nhl.html>

2. <https://seer.cancer.gov/statfacts/html/clyl.html>

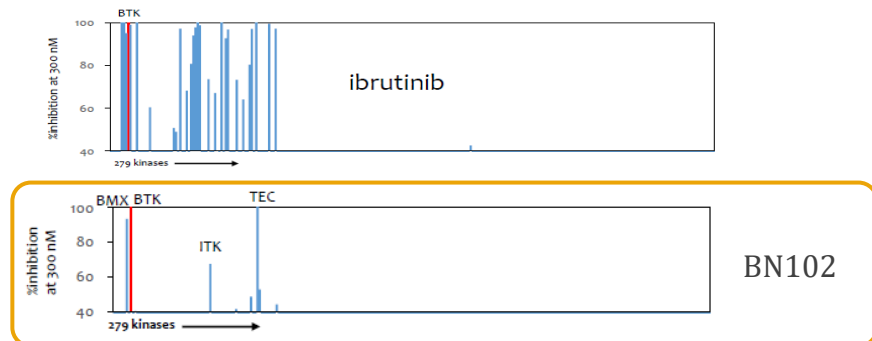
3. <https://gco.iarc.fr/today/data/factsheets/populations/160-china-fact-sheets.pdf>

4. [ASH 2021](#)



BN102 - A Highly Selective, Potent Reversible BTK Inhibitor

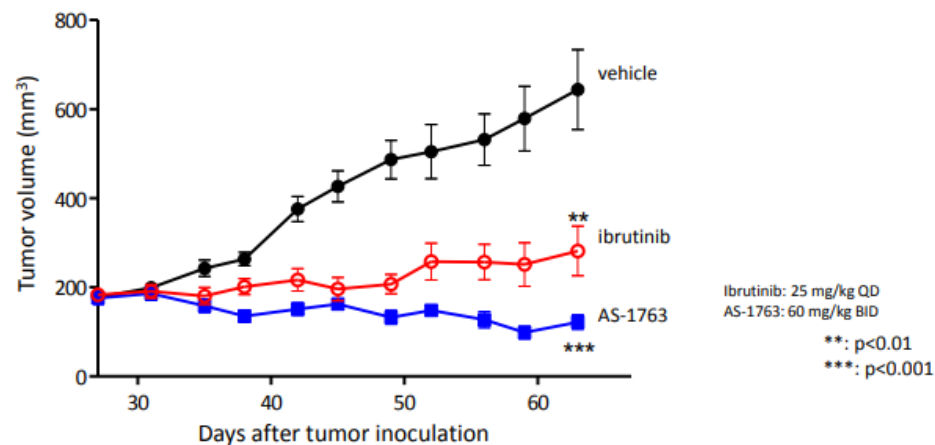
High kinase selectivity



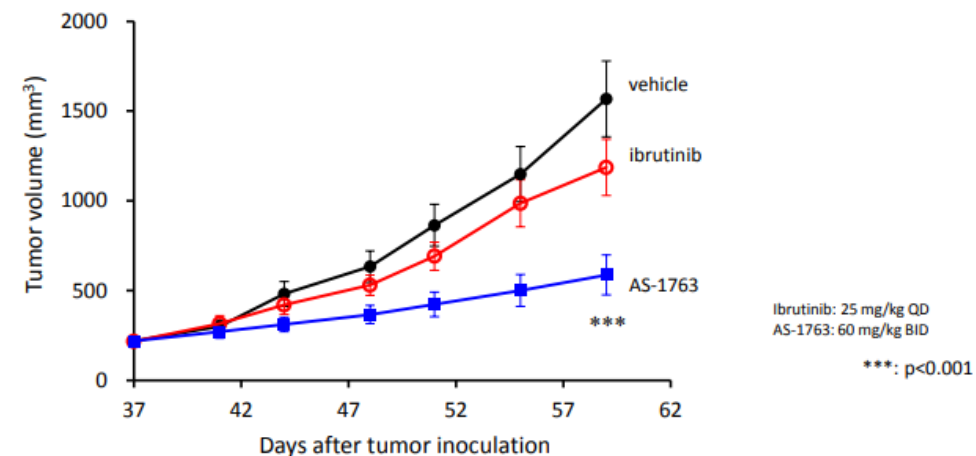
In vitro pharmacological activities of BN102

	IC50(nM)	
	BN102	ibrutinib
Autophosphorylation BTK (Ramos)	1.4	1.1
CD69 activation (Human whole blood)	11	8.1
Cancer cell growth (OCI-Ly10 cells)	1.8	0.75
Cancer cell growth (OCI-Ly10 [BTK C481S] cells)	20	1030
Normal cell growth (HEL299 cells)	6370	6870

In vivo antitumor effects of BN102 on human B-cell non-Hodgkin lymphoma cell line, OCI-LY10 tumor xenograft mouse model (n=8-10)



In vivo antitumor effects of BN102 on ibrutinib-resistant BTK^{C481S} knock-in OCI-LY10 tumor xenograft mouse model (n=11)



Ramos: human Burkitt lymphoma cell line
OCI-Ly10: human B-cell non-Hodgkin lymphoma cell line
OCI-Ly10 [BTK C481S]: BTK[C481S] knock-in OCI-Ly10 cells
HEL299: human embryo lung cell line

J Med Chem. 2021 Oct 14;64(19):14129-14141.



BN102 China Development Timeline





BN301 - Potential First-in-Class ADC for Patients with NHL and MM

CD74 Expression in cancers

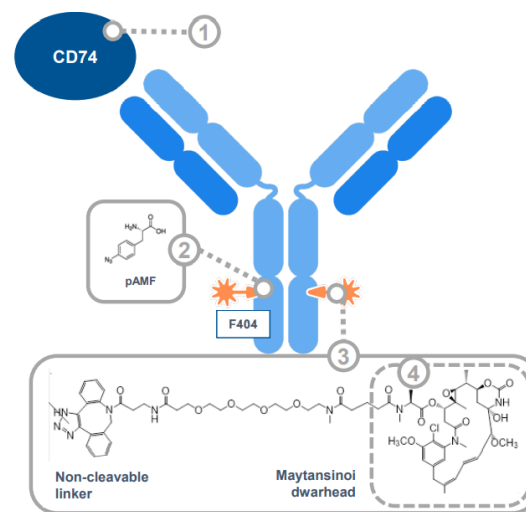
- CD74 is expressed in ~90% of B-cell cancers including myeloma and lymphoma
- CD74 also is expressed in nonhematopoietic cancers, such as gastric, renal, urinary bladder, non-small cell lung cancers, certain sarcomas, and glioblastoma

BN301: Potential First-in-Class CD74 Targeting ADC

- BN301/STRO-001 is a homogeneous antibody drug conjugate (ADC) with a drug-antibody ratio (DAR) of 2, targeting CD74:
 - CD74 is expressed in many hematological cancers and rapidly internalized
 - Conjugation through precisely positioned non-natural amino acids p-azidomethyl-L-phenylalanine, at positions F404 on the heavy chain
 - Comprises two non-cleavable linker-warheads that are **stable in circulation**
 - The active warhead, maytansinoid derivative, efficiently kills tumor cells following internalization of the ADC and was designed to **minimize bystander effects**

Frequent CD74 Expression in Multiple B-cell NHL Subtypes by IHC

	CD74 positive	%
B cell lymphoma – total samples	404/423	96
Follicular lymphoma	148/151	98
Grade 1 and 2	90/91	99
Grade 3 A and B	58/60	97
Diffuse large B-cell lymphoma	135/140	96
Extranodal marginal zone lymphoma	22/24	92
Splenic marginal zone lymphoma	4/5	80
Nodal marginal zone lymphoma	6/6	100
Mantle cell lymphoma	19/21	90
SLL/CLL	36/36	100
Lymphoplasmacytic lymphoma	5/5	100



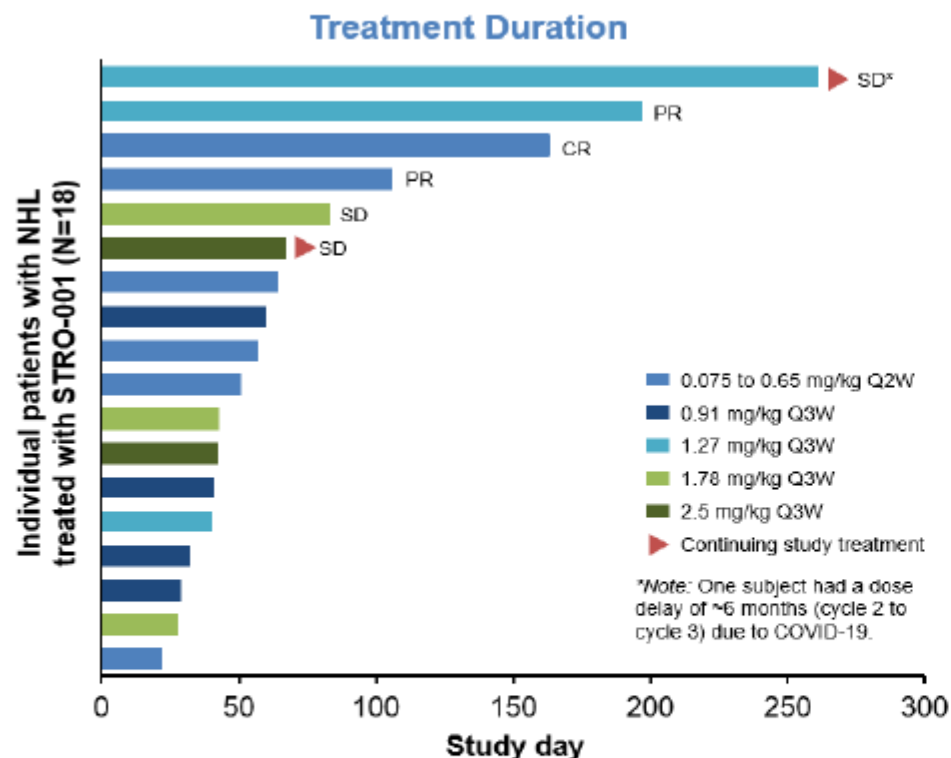
Competitive landscape

Owner	Immunomedics Gilead Sciences	Sutro Biopharma
Product	Milatuzumab	STRO-001
Status	Phase II	Phase I
Indication	GvHD, CLL, MM, NHL, SLE	Lymphomas, MM



3030 Preliminary Results of an Ongoing Phase 1 Dose Escalation Study of the Novel Anti-CD74 Antibody Drug Conjugate (ADC), STRO-001, in Patients with B-Cell Non-Hodgkin Lymphoma

626. Aggressive Lymphoma (Diffuse Large B-Cell and Other Aggressive B-Cell Non-Hodgkin Lymphomas)—Results from Prospective Clinical Trials *Poster Session III on Monday, December 7, 2020, 7:00 AM–3:00 PM PT*

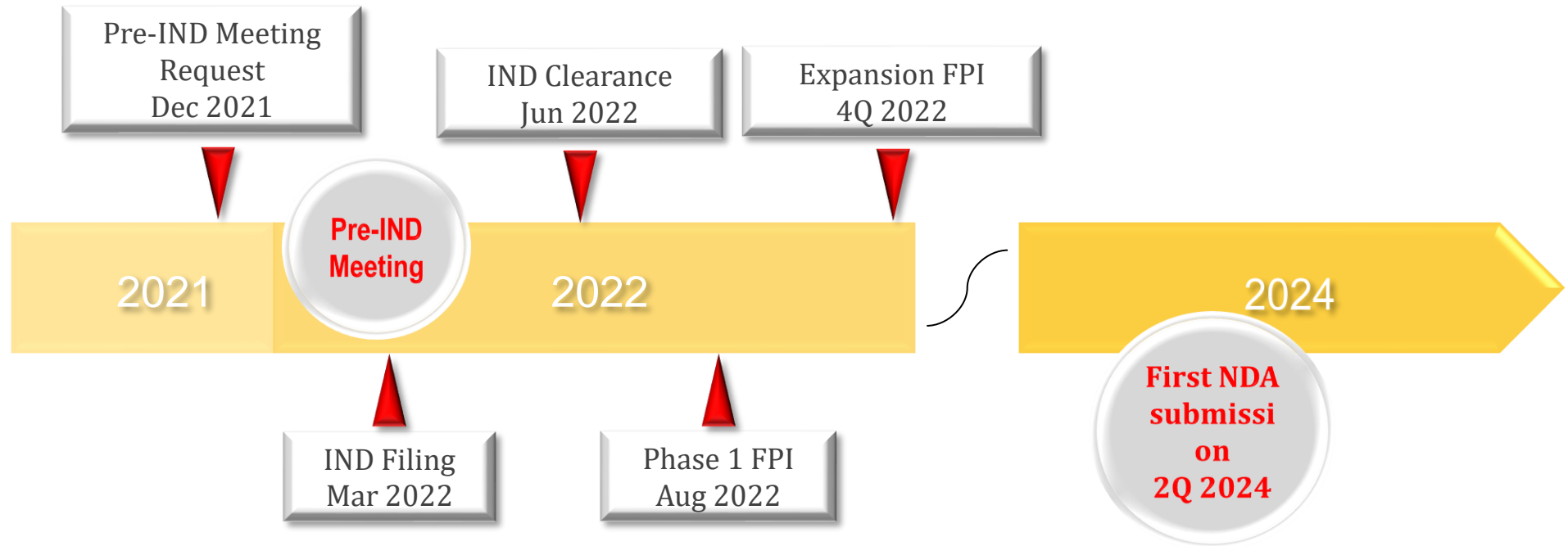


- STRO-001 was generally well tolerated, most AEs were Grade 1 & 2
- No ocular toxicity signals have been observed
- Median number of prior therapies is 4 (range 1-12)
- MTD has not been reached; next planned dose level is 3.5 mg/kg
- Preliminary anti-tumor activity has been observed in this heavily pre-treated patient population, including two DLBCL patients who had previously progressed after CAR-T

Dose level, mg/kg	Demographics and diagnosis	Prior Therapies	Best Response	Doses received	Duration of Treatment
0.075	82-year-old man with Stage III DLBCL, non-GC type diagnosed in 2015	- R-CHOP-R, - Rituximab/lenalidomide - Bendamustine/rituximab - Obinituzumab + gemcitabine + oxaliplatin	CR after 2 cycles (4 doses)	12	24 weeks (PD after 12 doses)
0.65	64-year old man diagnosed with double-hit Stage IV DLBCL in August 2017	- R-CHOP x 1 and EPOCH x 6 (2017) - RICE with IT prophylaxis (2017/2018) - Rituximab and XRT (2018) - Rituximab, gemcitabine + oxaliplatin with radiotherapy (2018) - Axicabtagene ciloleucel (CAR-T) (May 2018) - Rituximab and lenalidomide (Nov 2018)	PR at cycle 3	8	15 weeks (PD after 8 doses)
1.27	68-year old woman stage IV extranodal DLBCL, non-GC diagnosed in Feb 2018	- R-CHOP - RICE x 2 - DHAP x 2 - CAR-T (May 2019) - Lenalidomide (Nov 2019)	PR at cycle 3	10	27 weeks (PD at cycle 10)
1.27	51-year old woman, stage III marginal zone lymphoma diagnosed in May 2017	- Obinutuzumab	SD	10	45 Weeks Ongoing (Cycle 15)
1.78	36-year old man with stage IIIA follicular lymphoma diagnosed in June 2014	- Flt3L-vaccine immunotherapy - Rituximab - Pneumococcal conjugate vaccine immunotherapy - polyICLC (TLR-3 agonist) – immunotherapy - Pembrolizumab	SD	4	12 weeks (PD after Cycle 4)
2.50	74 year old man with stage IV follicular lymphoma	- Rituximab/fludarabine/Cytosine - Ifosfamide/carboplatin, etoposide - Auto SCT	SD	8	24 weeks (PD after Cycle 8)



BN301 China Development Timeline





BioNova Company Highlights

- BioNova is a clinical-stage biopharmaceutical company with **global vision and execution**.
- The company applies industry leading **innovative strategies** in every aspect of drug development.
- Extremely **capital efficient** with a laser focus on valuation-creation.



- **Experienced Founding Team** with deep understanding of development and regulatory strategy, as well as global insight to maximize the product value;
- **Proven Execution Excellence** in product development and successful track records in corporate management.



- **In-house Cross-functional Discovery Team** with highly selective and cost-effective principles and disciplines, supporting and driving a differentiated discovery and co-development strategy;
- **Innovative Pipelines** positioning products with competitive advantage in disease areas with high unmet clinical need and expedited regulatory pathway.
(Current pipeline including 1 NMPA NDA asset, 2 phase 1 assets and 1 discovery-stage assets with global rights)

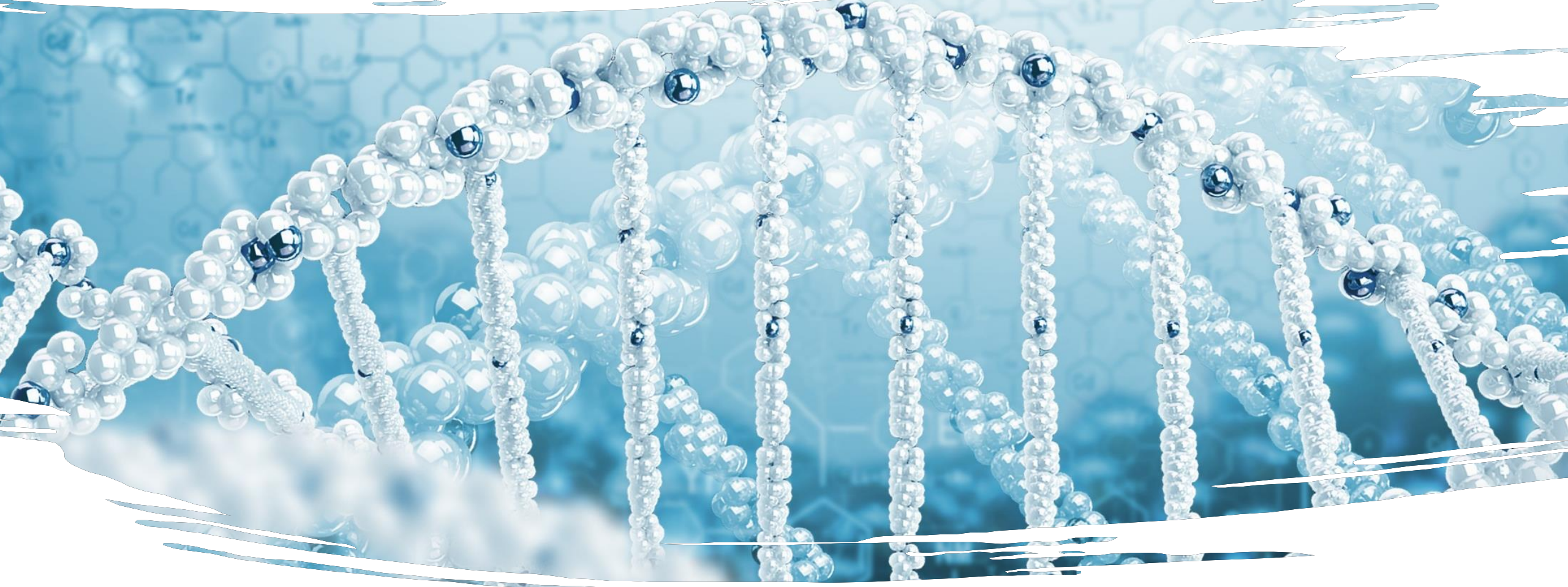


- Clear company growth plan to **go from China to worldwide**;
- **US Expansion in 2022** to bring products to value inflection point with a highly efficient US development team;
- **Global Partner** after clinical POC to unlock the value of proprietary assets for global markets.



- **Strong Endorsement by Top Tier Life Science VC Funds**, supporting the robust expansion of the company.





Thank You

November 2021