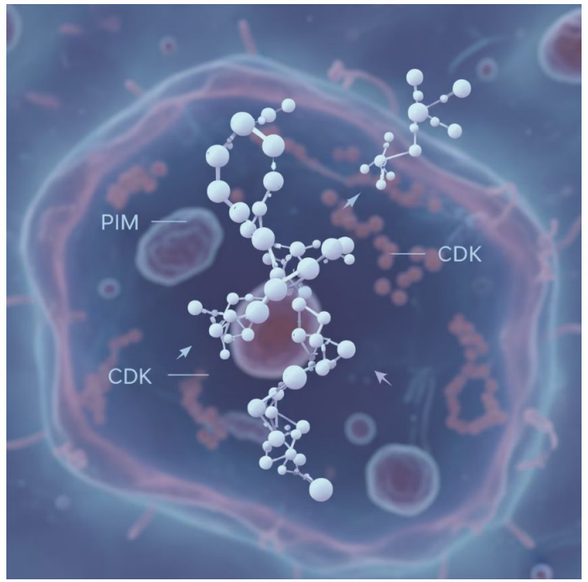
NB-001: First-in-Class Dual PIM and CDK Inhibitor

Program Overview

NB-001 represents a breakthrough in hematologic malignancies as the first dual inhibitor simultaneously targeting PIM1/2/3 and CDK2/4/6 kinases. This injectable small molecule addresses a **$15B market opportunity** in acute myeloid leukemia (AML) and non-Hodgkin lymphoma (NHL).

The program is currently advancing through Phase I clinical trials in both Australia and China, with **strong global patent protection extending beyond 2038** (PCT filed). This dual-action mechanism offers a differentiated approach to overcoming resistance mechanisms that limit current single-target therapies.



Key Attributes

- First-in-Class designation
- Injectable formulation
- Dual kinase inhibition
- Global clinical development

Dual Kinase Innovation

Targets PIM1/2/3 and CDK2/4/6 for a novel approach to overcome resistance.

Significant Market Potential

Addresses a $15B market in AML and NHL with a differentiated treatment.

Robust IP & Development

Strong global patent protection beyond 2038, advancing through Phase I trials.

NB-001: First-in-Class Dual PIM and CDK Inhibitor

Dual Pathway Inhibition

NB-001 simultaneously inhibits PIM1/2/3 and CDK2/4/6 kinases, disrupting downstream signaling pathways that drive tumor cell differentiation and proliferation. Pan-kinase PIM inhibition eliminates cross-compensation and reduces bypass resistance mechanisms.

Dose-Dependent Efficacy

In melanoma PDX models, NB-001 (ETH-155008) at 40mg/kg demonstrated superior tumor suppression compared to palbociclib at 100mg/kg, showcasing potent activity against CDK4/6 inhibitor-resistant strains with dose-dependent responses.

Enhanced Safety Profile

No drug-drug interactions or combination-related toxicity observed. Safety window equivalent to or better than gilteritinib and abemaciclib. Importantly, no QT interval prolongation detected, indicating superior cardiac safety compared to standard therapies.

Clinical validation is already emerging: **Complete response (CR) and partial response (PR) cases have been confirmed in both Australian and Chinese Phase I trials**, demonstrating early proof-of-concept in heavily pretreated patient populations.

Competitive Advantage: Genentech's PIM, AstraZeneca's CK2/PIM combination and Sanofi's PIM1/PIM2 programs all target single pathways and remain at preclinical stages, positioning NB-001 as the clinical leader in this therapeutic space.

NB-002: Best-in-Class Long-Acting Amylin Analog

Addressing the Obesity Epidemic

NB-002 is a best-in-class long-acting amylin analog targeting the **$15B metabolic disease market**, specifically obesity and type 2 diabetes mellitus (T2DM). This injectable hormone therapy acts as an amylin/calcitonin receptor agonist with weekly dosing convenience.

The program is currently in IND-enabling studies with **strong global patent protection**. NB-002's innovative formulation platform enables both monotherapy and combination approaches with GLP-1 receptor agonists, offering unprecedented flexibility in treating metabolic disorders.

Weekly Injectable

Convenient once-weekly administration improves patient compliance and treatment adherence

Dual Stability Platform

Unique formulations stable across broad pH ranges enable flexible combination strategies

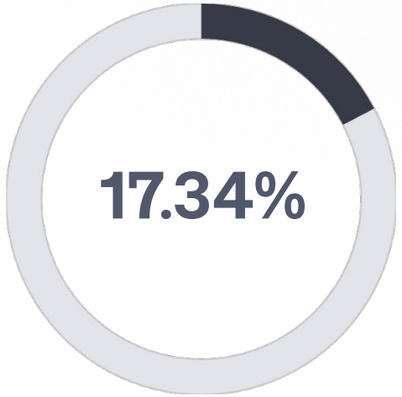
Combination Ready

Designed for synergistic combinations with GLP-1RAs like semaglutide and tirzepatide

NB-002: Superior Weight Loss and Extended Half-Life

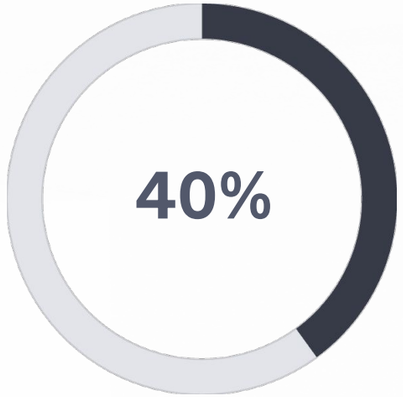
Innovative Formulation Platform

NB-002's diverse combination platform covers a broad pH range with two distinct formulation strategies: **acid-stable analogs (pH 3.0-5.0)** and **neutral-stable analogs (pH 6.0-7.5)**. This versatility enables optimal formulation with various combination partners, including market-leading GLP-1 receptor agonists.



Monotherapy Weight Loss

At 30 nmol/kg in DIO rats, exceeding Cagrilintide's 15.86% performance



Extended Half-Life

Longer duration observed versus Cagrilintide in cynomolgus monkey studies

Best-in-Class Combination Potential

NB-002 demonstrates **superior potential in weight management** both as monotherapy and in combination with GLP-1RAs such as semaglutide or tirzepatide. Preclinical studies show advantages over competing amylin analogs combined with identical GLP-1 partners, suggesting enhanced mechanistic synergy.

Comprehensive off-target toxicity and dose-ranging studies confirm an **excellent safety profile**, supporting confident dose escalation in clinical development and enabling exploration of higher, more efficacious doses that may be prohibited for competing molecules.

NB-003: First-in-Class CD38-Targeting Antibody

Program Overview

NB-003 is a first-in-class humanized monoclonal antibody targeting CD38 for the treatment of multiple myeloma (MM) and autoimmune diseases. With **FDA IND approval** secured, this program addresses an **$8B market opportunity** with strong global patent protection.

Differentiated Targeting

Unlike daratumumab and isatuximab, NB-003 recognizes and binds to a unique epitope on CD38 with **negligible red blood cell (RBC) binding**, potentially eliminating interference with blood banking procedures and reducing infusion-related reactions.

Multiple Myeloma

Primary indication leveraging potent tumor suppression and strong direct apoptotic activity

Autoimmune Expansion

Clinical validation of CD38 in lupus nephritis creates pathway for indication expansion

Enhanced ADCC

Superior antibody-dependent cellular cytotoxicity drives robust tumor-killing activity

NB-003: Differentiated Mechanism, Superior Activity

Unique Epitope Recognition

NB-003 is a humanized IgG1 monoclonal antibody that targets a **differentiated epitope of CD38** distinct from existing approved therapies. This unique binding profile results in negligible RBC binding compared to daratumumab and isatuximab, addressing a key limitation of current CD38-directed therapies that can interfere with blood compatibility testing.

Direct Apoptosis

Strong direct apoptotic activity against malignant plasma cells, independent of immune effector functions

Enhanced ADCC

Superior antibody-dependent cellular cytotoxicity amplifies tumor cell elimination through immune engagement

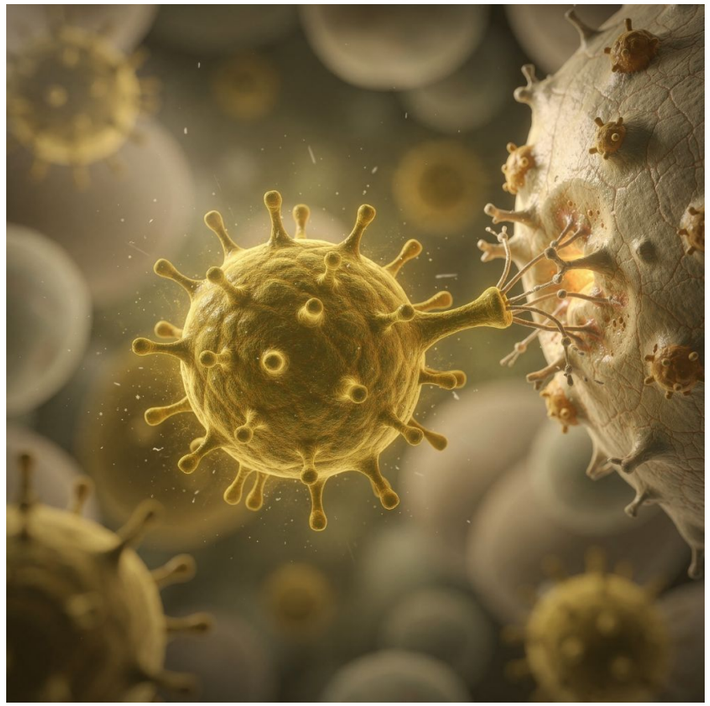
Potent Efficacy

More potent tumor-killing activity demonstrated in lymphoma and myeloma CDX models versus competitors

Expanded Therapeutic Potential

Beyond multiple myeloma, CD38 represents a **clinically validated target for autoimmune diseases**, as demonstrated by daratumumab's efficacy in lupus nephritis. NB-003's superior safety profile—particularly its minimal RBC binding—positions it as an exceptionally strong candidate for indication expansion into autoimmune disorders where chronic dosing and safety considerations are paramount.

NB-004: Best-in-Class 4-1BB Agonist Antibody



Immuno-Oncology Innovation

NB-004 is a best-in-class recombinant humanized antibody targeting 4-1BB (CD137) for advanced and metastatic solid tumors. This injectable therapeutic addresses a **$6B market opportunity** with **FDA IND approval** and strong global patent protection.

The 4-1BB pathway represents a validated immuno-oncology target for T cell activation, but previous clinical attempts have been limited by dose-limiting hepatotoxicity. NB-004's unique mechanism addresses this critical safety challenge while maintaining potent anti-tumor activity.

Unique Epitope Binding

Overlaps with endogenous 4-1BB ligand binding site, mimicking natural T cell activation

Balanced Activation

Optimal T cell stimulation while blocking excessive CD137 ligand-mediated activation

Clinical-Stage Safety

Designed to avoid hepatotoxicity that limited previous 4-1BB agonist development

NB-004: Optimized T Cell Activation with Superior Safety

Biomimetic Mechanism of Action

NB-004 binds to a **unique epitope of 4-1BB that overlaps with the endogenous 4-1BB ligand**, creating a biomimetic activation pattern. This binding paradigm mimics natural CD137 ligand activity for T cell activation while simultaneously blocking excessive ligand-mediated stimulation, achieving optimal 4-1BB axis activation without toxicity.

Binding Specificity

SPR data confirms comparable affinity to utomilumab for human 4-1BB, with no cross-reactivity to mouse or rat orthologs. Binds cell surface 4-1BB with demonstrated functional activity in vitro.

Clean FcγR Profile

No binding detected to CD32b, CD32a (both variants), CD16a (both variants), CD16b or CD64, reducing potential for off-target immune activation and associated toxicities.

Potent Anti-Tumor Activity

Demonstrates robust tumor suppression in humanized mouse models: MC38 colon cancer and CT26 colon cancer cell lines show significant responses in h4-1BB humanized mice.

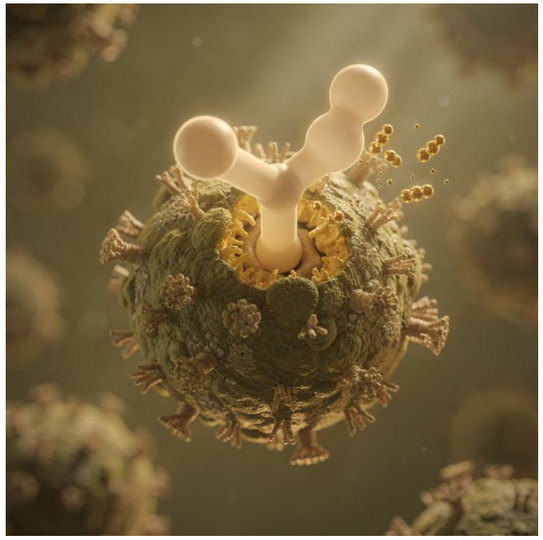
Favorable Pharmacokinetics

Cynomolgus monkey studies reveal **linear pharmacokinetic profiles** across the dose range of 0.2-20 mg/kg, with dose-dependent Cmax and drug exposure. This predictable PK profile supports straightforward clinical dose escalation and enables confident translation to human studies.

NB-005: Best-in-Class DLL3-Targeted ADC

Program Overview

NB-005 is a best-in-class antibody-drug conjugate (ADC) targeting DLL3 for small cell lung cancer (SCLC) and other neuroendocrine tumors. This injectable therapeutic addresses a **$5B market opportunity** and is currently in IND-enabling studies with strong global patent protection.



DLL3 Targeting Advantage

DLL3 is highly expressed on SCLC tumor cells but minimally present on normal tissues, providing an exceptional therapeutic window. This Notch pathway protein represents one of the most validated targets in neuroendocrine malignancies.

DLL3 Antibody

Highly selective antibody component binds tumor-associated DLL3 with high affinity

Novel Linker

Proprietary linker platform with exceptional plasma stability and optimized drug release kinetics

TOP1 Inhibitor

Designed to avoid hepatotoxicity that limited previous 4-1BB agonist development

NB-005: Revolutionary Linker Technology Drives Differentiation

Best-in-Class Linker Platform

NB-005 leverages a **novel and patented linker platform** that demonstrates transformational stability advantages. In plasma stability studies, only **0.0002% of free payload** is released after 30 days of incubation in both human and mouse plasma—substantially lower than the 0.2% release rate typical of conventional linkers, representing a **1,000-fold improvement** in stability.

0.0002%

Payload Release

After 30 days in plasma, versus 0.2% for conventional linkers

1000X

Stability Improvement

Over standard ADC linker technologies currently in clinical use

This proprietary linker efficiently mitigates payload hydrophobicity and ensures exceptional systemic circulation stability, directly addressing key limitations of first-generation ADCs that experienced premature payload release and associated toxicity.

Robust Preclinical Efficacy and Safety

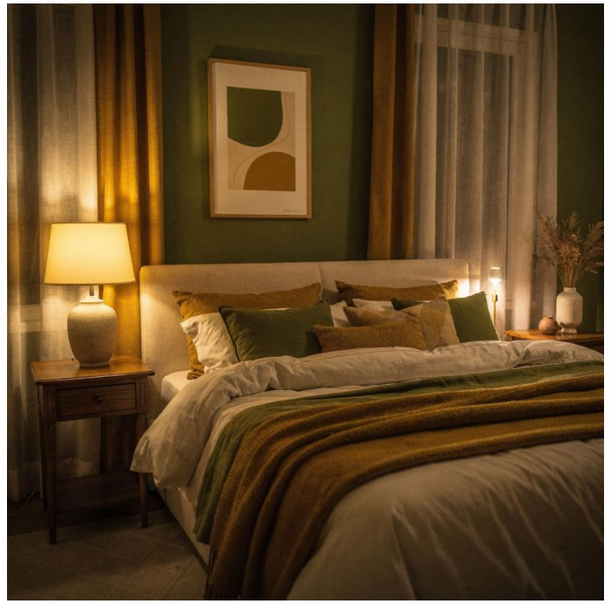
Preclinical studies utilizing cell-derived xenograft (CDX) and patient-derived xenograft (PDX) mouse models demonstrate **robust inhibitory effects on SCLC**. Importantly, NB-005 exhibits a favorable safety profile with no abnormal symptoms observed following injections at doses ranging from 50 to 100 mg/kg, supporting a wide therapeutic window and enabling aggressive clinical dosing strategies.

NB-006: Best-in-Class Ramelteon Modified-Release

Addressing Insomnia Limitations

NB-006 is a best-in-class modified-release formulation of ramelteon for insomnia characterized by difficulty with sleep onset and early awakening. This oral tablet targets the **$5B insomnia market** with **FDA and NMPA IND approval** and robust patent protection (CN: 202110950477.6; US18/288382).

The program has completed three pilot PK studies with results meeting expectations, and successfully manufactured a scale-up batch of 150,000 tablets, demonstrating manufacturing feasibility and commercial readiness.



Dual-Release Profile

Two pulse-like releases promote sleep onset and prevent early awakening, better meeting clinical needs than immediate-release formulations

Once-Daily Dosing

Single dose achieves effect of multiple administrations, improving compliance and patient convenience

Non-Controlled

Not a controlled substance, ensuring strong market accessibility and prescriber confidence

NB-006: Superior Profile Versus Takeda's Rozerem

Better Therapeutic Effect

The innovative dual pulse-like release profile of NB-006 addresses both sleep onset and maintenance, preventing early awakening that limits immediate-release ramelteon (Rozerem). This biphasic release more closely matches physiological sleep architecture and better fulfills clinical needs.

Enhanced Safety Profile

No drug dependence with minimal side effects distinguishes NB-006 from benzodiazepines and Z-drugs. The short half-life ensures minimal next-day hangover effects, addressing a major limitation of competing therapies and improving patient quality of life.

Improved Patient Compliance

Once-daily dosing achieves the therapeutic effect of multiple administrations, significantly reducing medication burden. This simplified regimen enhances adherence, particularly important in chronic insomnia management where compliance often determines treatment success.

Superior Pharmacokinetics

NB-006 demonstrates a **better PK profile than Takeda's Rozerem**, with optimized absorption kinetics, improved bioavailability, and controlled-release characteristics that sustain therapeutic levels throughout the night while clearing rapidly in the morning.

Market Advantage: As a non-controlled substance with demonstrated safety and efficacy advantages, NB-006 is positioned to capture significant market share from both prescription sleep aids and over-the-counter products.

NB-007: First-in-Class Dual FLT3/CSF1R Inhibitor

Program Overview

NB-007 is a first-in-class small molecule dual-targeting inhibitor of FLT3 and CSF1R for acute myeloid leukemia (AML) and myelodysplastic syndromes (MDS). This injectable therapeutic addresses a **$6B market opportunity** with **NMPA IND approval** in China and strong global patent protection beyond 2041 (PCT filed).

Dual Mechanism Advantage

Simultaneous inhibition of FLT3 (driving tumor cell proliferation) and CSF1R (modulating tumor-associated macrophages) creates a differentiated therapeutic approach that addresses both tumor cells and the immunosuppressive microenvironment.

Tumor Cells

FLT3 inhibition blocks malignant proliferation

Macrophages

CSF1R inhibition reprograms tumor microenvironment

Synergy

Combined action enhances anti-tumor efficacy

NB-007: Superior Single-Agent Activity and Safety

Dual-Pathway Innovation

Simultaneous inhibition of CSF1R and FLT3 pathways enables NB-007 to **act on macrophages and tumor cells concurrently**, improving the tumor microenvironment while directly suppressing malignant cell growth. This dual mechanism enhances anti-tumor effects beyond what either pathway inhibition could achieve alone.

High Selectivity

Better kinase selectivity profile than gilteritinib, reducing off-target effects and improving therapeutic window

Superior Single-Agent Activity

Outperforms mainstream drugs in AML/MDS when used as monotherapy, driven by dual mechanism

Avoids Combination Toxicity

Single dual-target agent eliminates DDI concerns and toxicity superposition from drug combinations

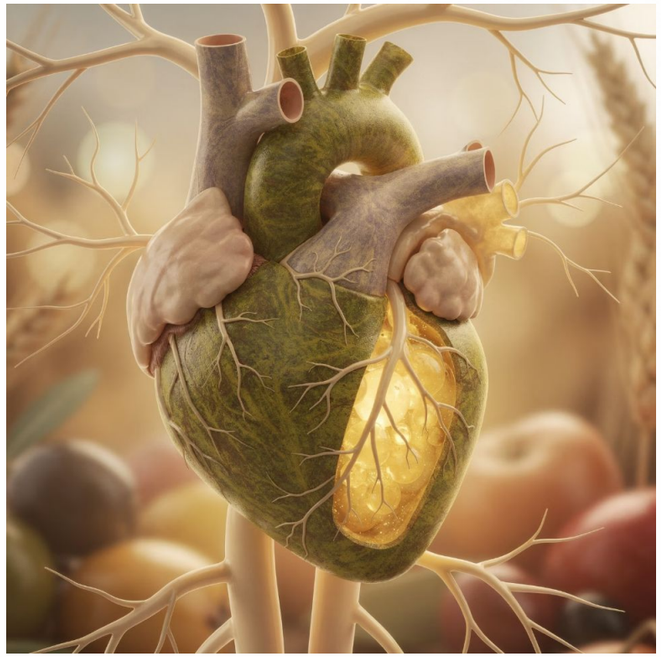
Delays Resistance

Dual targeting reduces likelihood of resistance development through single pathway mutations

Optimal Pharmacokinetic Profile

NB-007 exhibits a **long half-life meeting once-daily dosing requirements**, enhancing patient convenience and compliance. The extended PK profile maintains sustained target inhibition throughout the dosing interval, maximizing therapeutic benefit while minimizing pill burden in a population often managing multiple medications.

NB-008: Best-in-Class Oral PCSK9 Inhibitor



Oral Small Molecule Innovation

NB-008 is a best-in-class oral small molecule PCSK9 inhibitor for primary hyperlipidemia, familial hypercholesterolemia, cardiovascular disease and potentially oncology. This oral tablet addresses a **$6B market opportunity** currently dominated by injectable biologics.

The program is in IND-enabling studies with **strong global patent protection beyond 2041** (PCT filed). As the first oral small molecule PCSK9 inhibitor, NB-008 offers transformative advantages over existing injectable monoclonal antibodies.

Oral Bioavailability

Small molecule enables convenient oral dosing versus bi-weekly or monthly injections

Cost Advantage

Dramatically lower drug costs compared to biologics expand patient access and payor adoption

Patient Preference

Oral administration eliminates injection site reactions and improves treatment acceptance

NB-008: Overcoming Statin Limitations with Dual Indications

Addressing Unmet Medical Needs

NB-008's oral small molecule approach delivers **significant advantages** over currently approved PCSK9 macromolecules, including substantially lower drug costs and convenient oral administration that transforms the patient experience. This accessibility advantage is particularly important for chronic cardiovascular disease management requiring lifelong therapy.

Overcomes Statin Limitations

Addresses statin intolerance, inadequate LDL reduction, and drug-drug interactions that limit current standard-of-care approaches. Provides alternative mechanism for patients who cannot achieve lipid goals with statins alone.

Competitive Differentiation

Current oral PCSK9 inhibitors in clinical development are limited to peptides and alkaloids with bioavailability challenges. NB-008's small molecule structure offers superior pharmaceutical properties and development advantages.

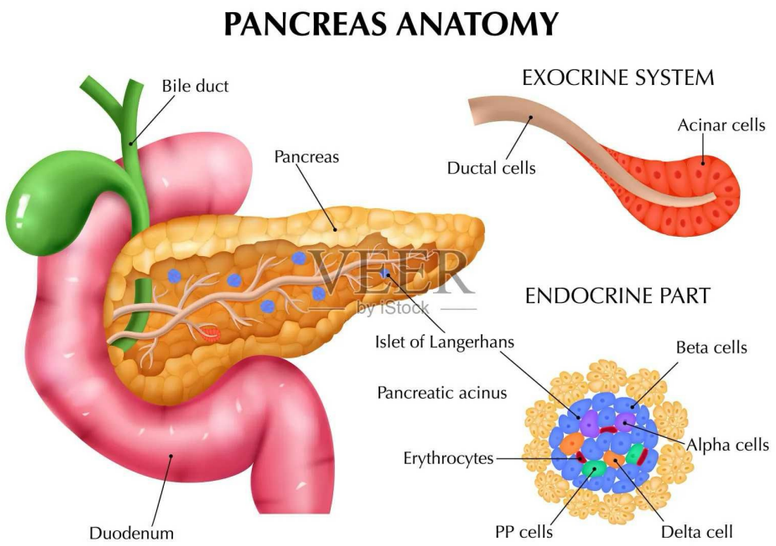
High Safety and Activity

Demonstrates excellent safety profile coupled with potent PCSK9 inhibition. Reduces PCSK9 mRNA levels, suggesting potential disease-modifying effects beyond current injectable biologics that only inhibit protein function.

Dual Indication Potential

Beyond cardiovascular disease, emerging evidence supports PCSK9's role in cancer biology, creating a **second major indication opportunity** in oncology. NB-008's oral formulation and safety profile make it particularly attractive for combination with cancer therapies, potentially opening entirely new therapeutic applications and market segments.

NB-009: First-in-Class Pan RAS Inhibitor



Polysaccharide-Drug Conjugate (PSDC) Innovation

A first-in-class Polysaccharide-Drug Conjugate targeting Ras Mutant tumors via micropinocytosis. This novel Pan Ras inhibitor addresses a market 8 times the size of the current one for KRAS 12C inhibitors, i.e. potentially up to **$30B market opportunity**.

The program is in IND-enabling studies with strong global patent protection beyond 2040 (PCT filed). As the first Polysaccharide-Drug Conjugate using “Click Chemistry “ (Nobel-Prize-winning technology) , NB-009 as a Pan Ras inhibitor is a breakthrough treatment for High Mortality Cancers with high unmet need and poor prognoses, such as:

- Pancreatic Cancer (where KRAS mutations occur in ~88% of cases)
- Colorectal Cancer (~50%)
- Non-Small Cell Lung Cancer (~32%)

Broader Coverage Advantages

Significant inhibition of KRAS-driven tumor growth, including KRAS wild-type , G12C, G12D, G12V, G12R, etc.

Higher Antitumor Activity

Outperformed Revolution Medicine’s RMC-6236—the leading pan-RAS anti-tumor agent—in lung cancer models.

Higher Safety

Excellent safety at a high dose of 15 mg/kg, representing a 2.5-fold improvement over free docetaxel with only minimal liver exposure.

NB-009: Robust Anti-Pan-KRAS Tumor Activity via Macropinocytosis

No Pan-RAS inhibitor approved for commercial sale yet; ~23% to 25% of malignant tumors have KRAS mutations. This translates to roughly 2.7M new patients globally each year.

NB-009 PSDC inhibits tumor growth by suppressing the PI3K-AKT , the p38 pathway and reduced proliferation (Ki-67). PSDC turn tumor cell membranes from natural “barriers” into “carriers” and enrich the drug inside tumor cells. Modified polysaccharides with negatively charged glutamic acid are non-immunogenic, fully biodegradable and avoid clearance by the reticuloendothelial system and markedly reduces uptake by normal cells.

First Click Chemistry Application

World- first to apply "click chemistry" to polysaccharide–drug conjugation. The reaction is simple, rapid, and highly specific.

- High yield (85-95%)
- High drug loading（30%）
- Allow coupling of a wide variety of drug molecules
- Capable of large-scale industrial production

Outstanding Efficacy

In RAS-driven lung cancer model, NB-009 at a dose of 15 mg/kg significantly inhibited H358 /KRAS G12C tumor growth, demonstrating in vivo antitumor efficacy 5.56-fold greater than RMC-6236 from Revolution Medicine and 9.4-fold greater than Adagrasib from BMS

Unique Biodistribution Profile

Selectively accumulating in tumor sites while sparing normal organs—unlike existing cancer therapies such as small molecules, macromolecules (including antibodies and ADCs) and nanoparticle formulations. Resulted in less drug resistance and minimal accumulation in liver and better safety profile

PSDC Platform Potential

Difunctionalized polysaccharides with two different functional groups are the first one able to link both targeting ligand and drug in the world. Expansion to Additional Drug Classes: Small molecule, oligonucleic acids such as siRNA ASOs, protein, etc for various therapies.

Additional Pipeline Assets

ALDC (albumin drug conjugates, except for Legutaxel)	Chemotherapy	TMEA-Doxorubicin	China Pivotal phase II/III Est. NDA filing in 2025Q4	Targeted Chemotherapy, to revolutionize traditional chemotherapy
		TMEA-Paclitaxel	China Phase I/II	
		TMEA-DXd	China Phase I	
		TMEA-AXL/VEGFR inhibitor	IND filing 2025Q3	
TMEA ADC (antibody drug conjugates)	ADC therapy	PD-L1-TLR7/8 iSAC	IND filing in 2026Q1/2	New generation Linker-payloads
		CLDN6-DXd	IND filing in 2025Q4	
		EGFR/TROP2-Dual Payloads	IND filing in 2026Q2	
		ROR1-Dual Payloads	IND filing in 2026Q1	
		EGFR/cMet-ADC	Tox batch production	
TMEAbody (Antibodies and Cytokines)	Cytokine Related	IL-2 TMEAbody	Australia / China Phase I	BIC Immunotherapy: New generation
		PD-1-IL2 TMEA	CMC development	
	I/O Therapy	CTLA-4 TMEAbody	Australia / China Phase I	
		CD47 TMEAbody	IND filing in 2025Q4	
		CLDN18.2-CD3 TMEA	IND filing in 2026Q3	
		CLDN6-CD3 TMEA	IND filing in 2026Q3	
		CDH17-CD3 TMEA	IND filing in 2026Q1/2	