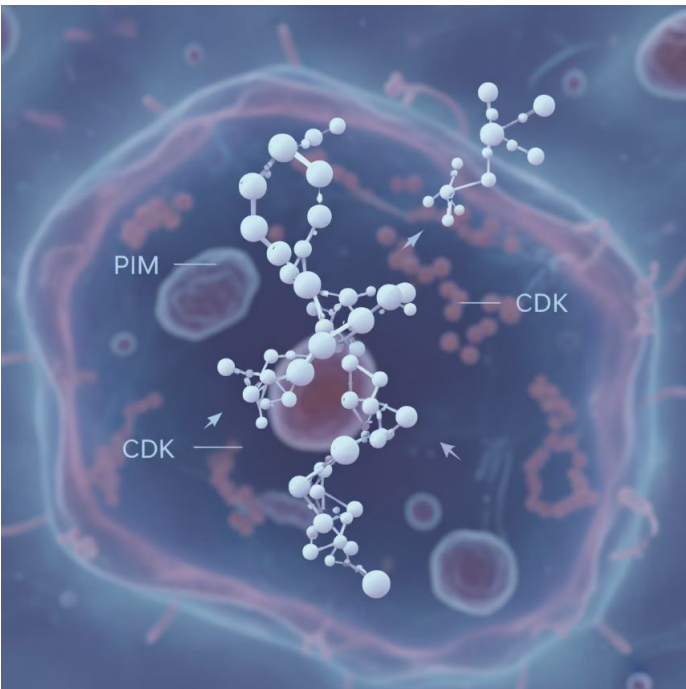


NB-001: First-in-Class Dual PIM and CDK Inhibitor

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

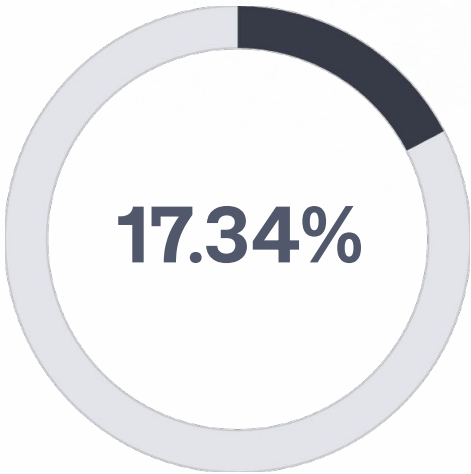
Combination Ready

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

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

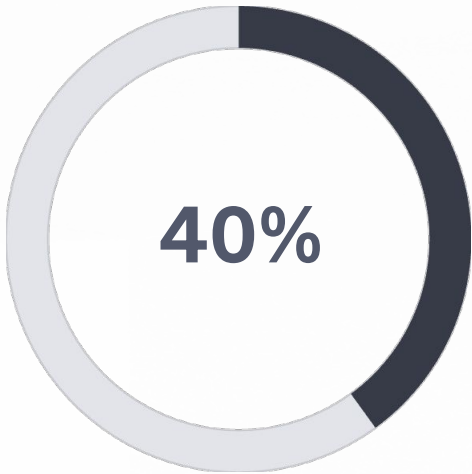
Innovative Formulation Platform

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

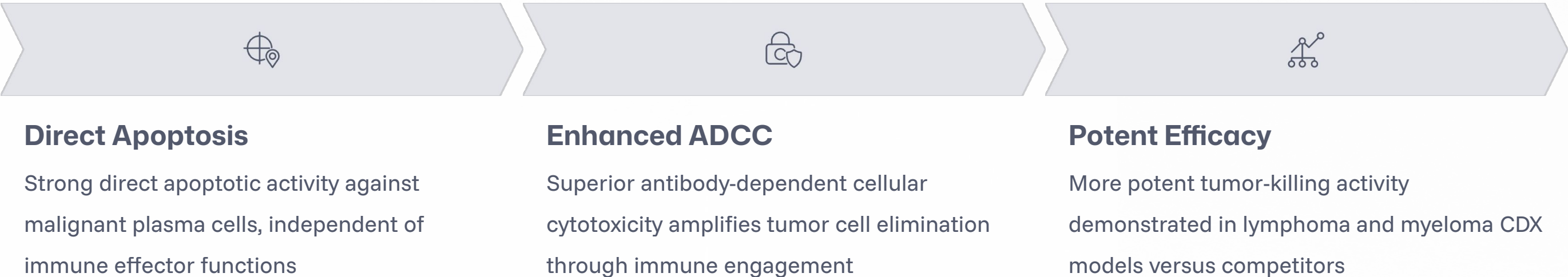
Enhanced ADCC

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

NB-003: Differentiated Mechanism, Superior Activity

Unique Epitope Recognition

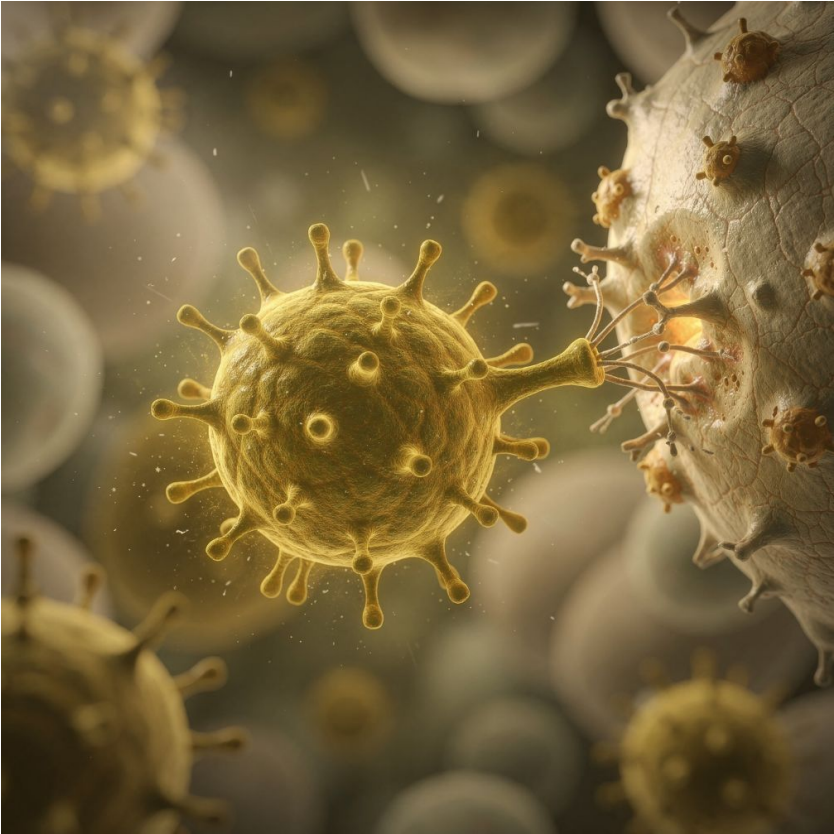
NB-003 is a humanized IgG1 monoclonal antibody that targets a **differentiated epitope of CD38** distinct from existing approved therapies. This unique binding profile results in negligible RBC binding compared to daratumumab and isatuximab, addressing a key limitation of current CD38-directed therapies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



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

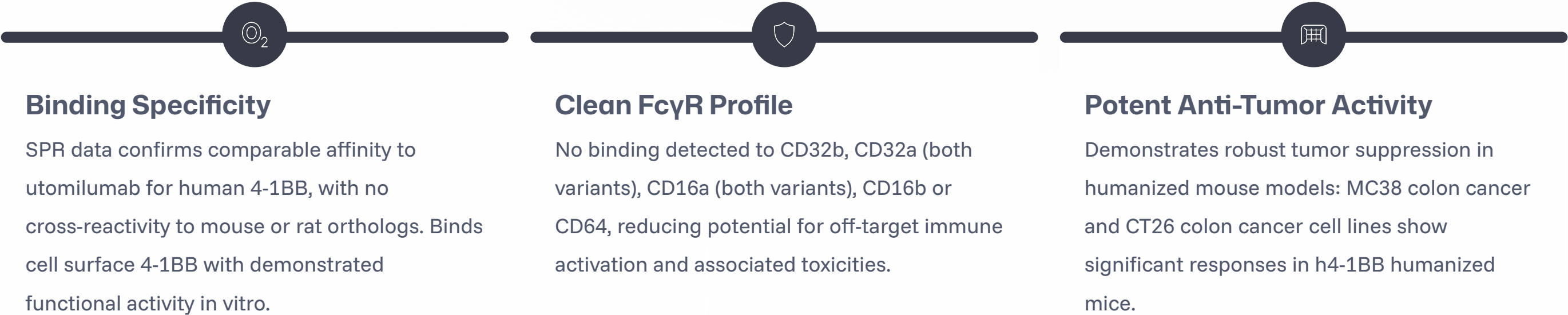
Clinical-Stage Safety

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.



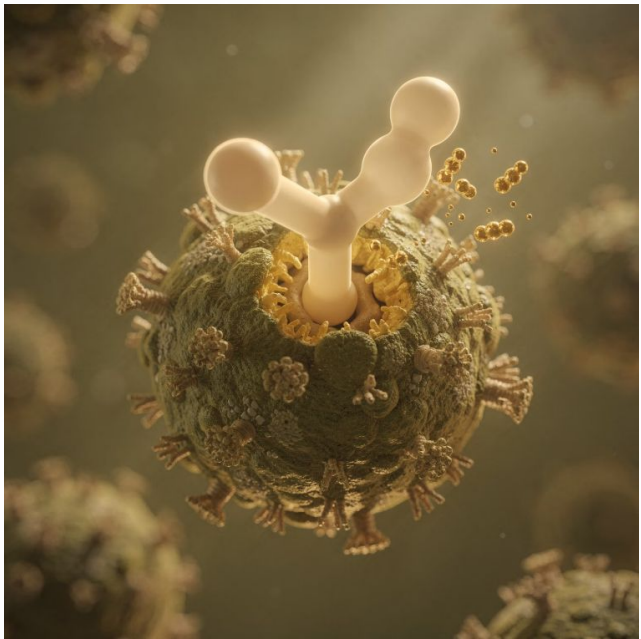
Favorable Pharmacokinetics

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

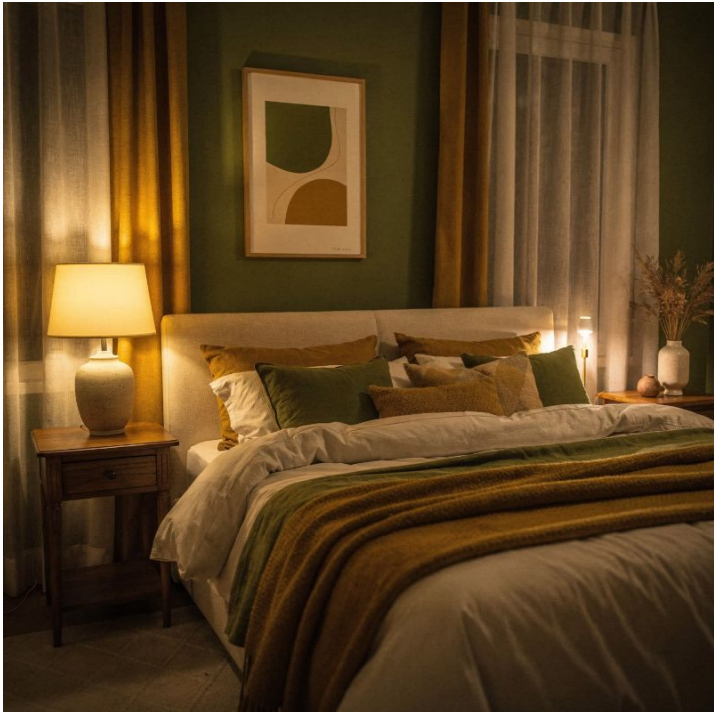
Preclinical studies utilizing cell-derived xenograft (CDX) and patient-derived xenograft (PDX) mouse models demonstrate **robust inhibitory effects on SCLC**. Importantly, NB-005 exhibits a favorable safety profile with no abnormal symptoms observed following injections at doses ranging from 50 to 100 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

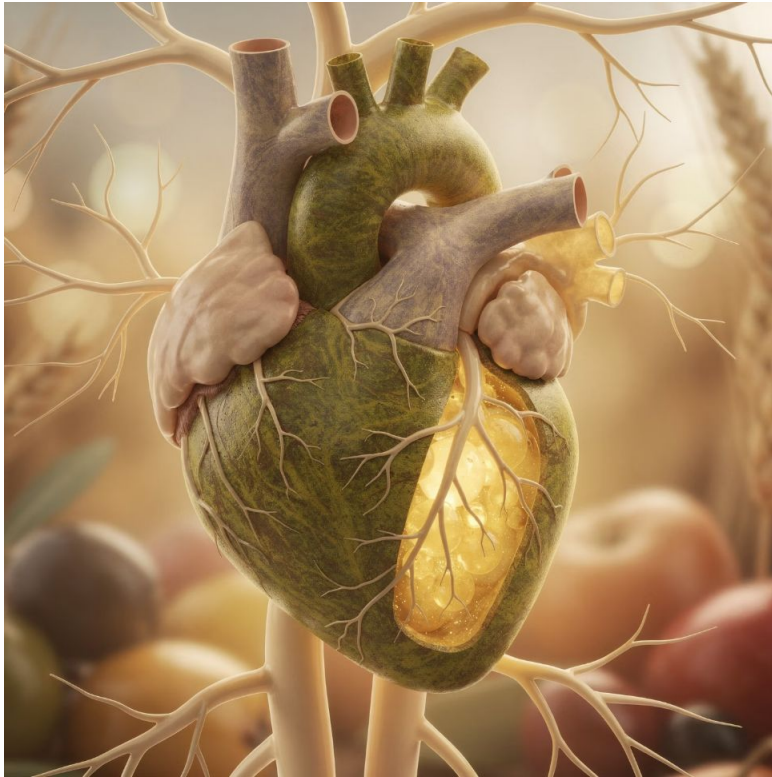
Delays Resistance

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

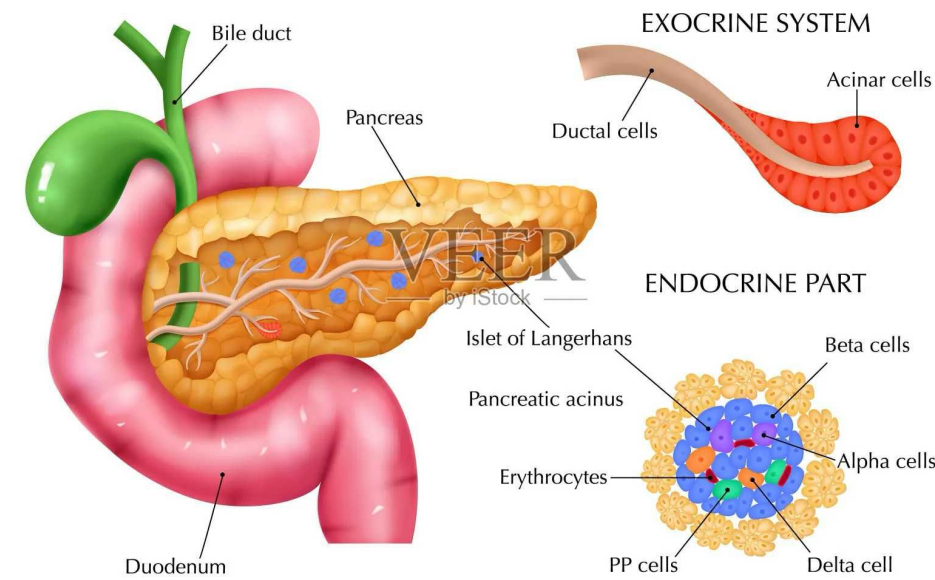
Demonstrates excellent safety profile coupled with potent PCSK9 inhibition. Reduces PCSK9 mRNA levels, suggesting potential disease-modifying effects beyond current injectable biologics that only inhibit protein function.

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

PANCREAS ANATOMY



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.

NB-010: Anti-PD-L1 ADC

The Opportunity

IND-ready with a differentiated preclinical package: dual mechanism, superior pharmacology, stable linker, favorable PK and a wide safety margin — positioning for a compelling clinical development program.

Molecule Design

DAR8 with Hydrophilicity-Enhanced Linker

Maximizes drug loading while maintaining antibody-like biophysical properties; identical HIC retention time for ADC and parental antibody.

Dual-Function Payload

Top1 inhibition + DDX5 degradation delivers synergistic cytotoxicity with a novel mechanism distinct from MMAE and DXd.

Dual Mechanism of Action

Simultaneous PD-L1 checkpoint blockade and targeted intracellular cytotoxicity — immune activation + tumor killing in one molecule.

Species Cross-Reactivity

002mAb retains potent PD-L1 affinity in human and cynomolgus, enabling direct IND-enabling study translation.

Minimized FcγR Engagement

Preserves immune effector function while maintaining FcRn recycling for favorable PK.

Validated Target, Open Landscape

PD-L1 is expressed in 63% of solid tumors across 15,000+ patients. Only two αPD-L1 ADCs (Pfizer/Seagen Phase III; Henlius Phase II) have reached advanced development — far less crowded than HER2 or TROP2.

Significant Market Potential

Global PD-(L)1 market reached \$58B in 2025; ADC sales exceeded \$16B. Peak sales potential for a differentiated αPD-L1 ADC: \$6–15B across NSCLC, TNBC, gastric, esophageal, urothelial, and other indications.

First-Mover Advantage

Blue-ocean competitive positioning with room to differentiate through superior molecule design, payload innovation, and clinical execution.

NB-010: Anti-PD-L1 ADC

Preclinical Data Package

NB-010 has demonstrated a best-in-class preclinical profile across pharmacology, PK, safety, and developability — supporting advancement into IND-enabling studies.



Superior Endocytosis & Cytotoxicity

Outperforms both clinical-stage α PD-L1 ADC competitors in receptor-mediated internalization. Potent, concentration-dependent tumor cell killing with minimal impact on immune effector cells, preserving the checkpoint inhibition mechanism.



Bystander Killing & MDR Resistance

Comparable bystander effect to Competitor 1 ADC across 4 heterogeneous tumor co-culture models. Full cytotoxic activity maintained in P-gp/BCRP efflux-competent MDR cell lines (HCT15, DMS53) — unlike MMAE and DXd-based ADCs.



Robust In Vivo Efficacy

Complete tumor regression in EBC-1 (lung SCC) CDX model at 10 mg/kg. Potent tumor growth inhibition in Karpas 299 (ALCL) model at doses as low as 3 mg/kg.



Plasma Stability & PK

Linker stable across human, cynomolgus, rat, and mouse plasma at 37°C for ≥ 21 days. Rat IV PK: $T_{1/2} = 162$ h; TAb and ADC curves nearly identical (MRT 215 h) — confirming exceptional in vivo linker integrity.



Favorable Safety Profile

Tolerated at ≥ 120 mg/kg in rats and ≥ 20 mg/kg in cynomolgus monkeys. Only mild, reversible AEs (hair loss, loose stools) consistent with ADC class effects. Dose escalation ongoing with no dose-limiting toxicities identified.



Excellent Developability

High purity, high thermostability, low hydrophobicity (identical HIC RT for DAR8 ADC and parental antibody), and low non-specific binding — supporting CMC scale-up and standard cold-chain storage.

NB-011: Tumor MicroEnvironment Activated

Validated Platform

The Tumor MicroEnvironment Activated (TMEA) platform addresses a fundamental challenge in oncology: conventional drugs exert less than 1% of their effects in tumors, causing systemic toxicity including cardiotoxicity, hematotoxicity and immunotoxicity. Legumain-activated drugs accumulate and activate selectively in the tumor microenvironment, dramatically reducing systemic toxicity.

Biologics

Conditionally activated antibodies releasing active biologics to engage immune cells

ADC

Antibody-drug conjugates activated only in the tumor microenvironment

Albumin-Drug

Albumin conjugates delivering active payloads selectively to tumors

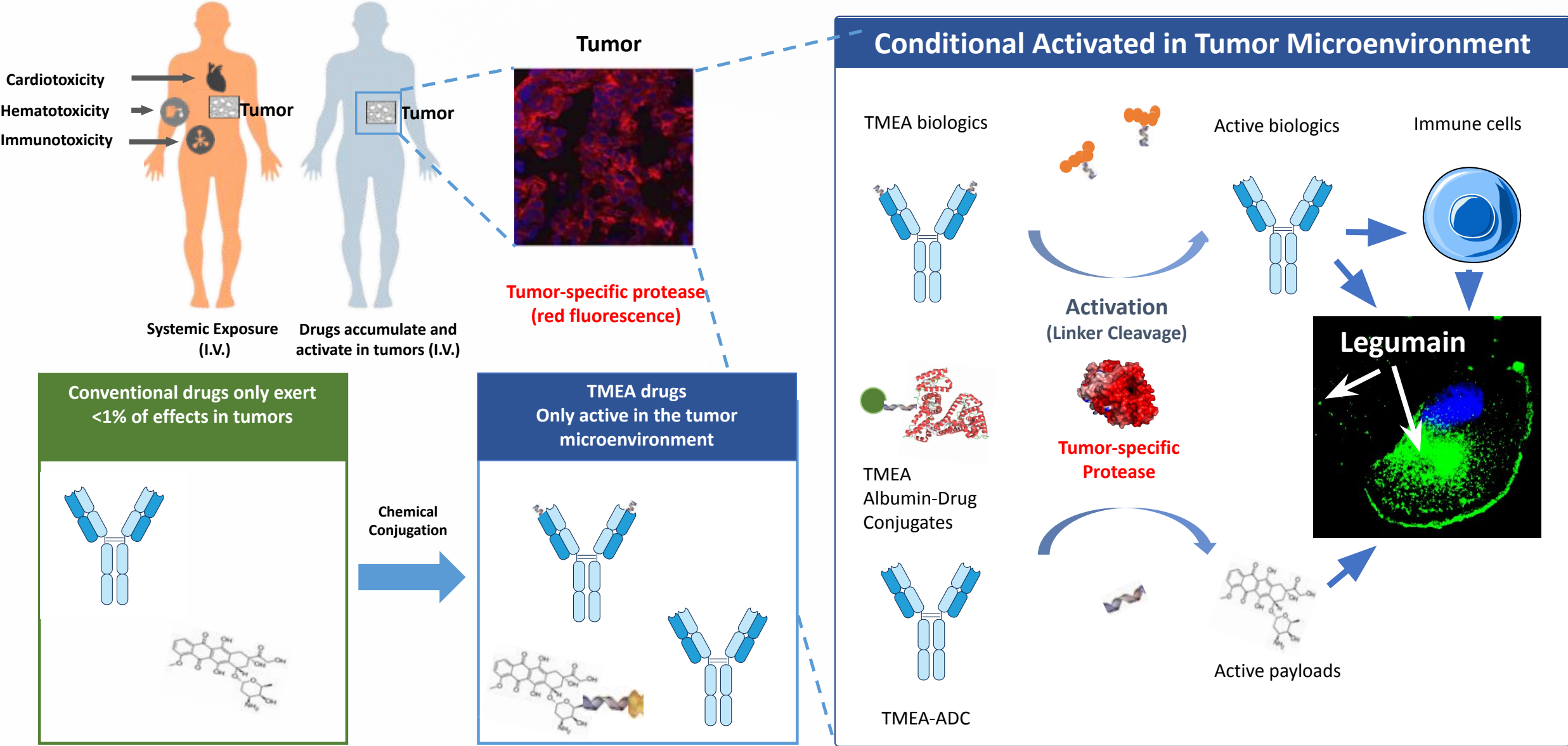
De-Risked Asset

Legubicin has completed a Phase II/III trial vs. doxorubicin, demonstrating superior PFS, OS, ORR, and DCR, with >20-fold reduction in Grade $\geq$ 3 cardiac toxicity and 43.8% vs. 1.9% treatment discontinuation due to cardiotoxicity. NDA filing underway in China.

NB-011: Tumor MicroEnvironment Activated

The Problem: Systemic Toxicity vs. Tumor Targeting

TMEA biologics and TMEA-ADCs are conditionally activated in the tumor microenvironment by tumor-specific proteases such as Legumain. Linker cleavage releases active biologics that engage immune cells, and active payloads that exert cytotoxic effects, all localized within the tumor.



NB-012: AKT-1 ASO for Hepatocellular Carcinoma

First-in-class clinical-stage ASO therapeutic targeting AKT-1

World's first clinical-stage ASO therapeutic targeting AKT-1, delivered via the QTsome™ LNP platform. With IND clearances in the US and China, active Phase I and Phase II trials, and FDA Orphan Drug Designation, NB-012 represents a differentiated and potentially practice-changing approach to hepatocellular carcinoma.



First-in-Class

The world's first clinical-stage ASO therapeutic targeting AKT-1, delivered via the QTsome™ LNP platform



Global Clinical Program

Active Phase I and Phase II trials with IND clearances in both the US and China; enrolling sites in Mainland China, Hong Kong and the US



FDA Orphan Drug Designation

Received FDA Orphan Drug Designation for hepatocellular carcinoma, underscoring unmet medical need



Combination Potential

Pre-clinical data shows superior tumor suppression in combination with TKIs (Sorafenib, Lenvatinib, Cabozantinib) and anti-PD-1 therapy

NB-012: AKT-1 ASO for Hepatocellular Carcinoma

Clinical Highlights

0

No DLTs at 0.1, 0.3 or 0.6 mg/kg/week dose group.
Infusion-related reactions (IRRs, Grade 2) occurred in 2 patients at 1.0 mg/kg/week, leading to a withdrawal..
MTD - 0.6 mg/kg/wk

9.4 mo

Median PFS across evaluable Phase I patients

15.9 mo

Median stable disease duration in Phase I

93.55%

Tumor Growth Inhibition with NB-012 + anti-PD-1 in pre-clinical models

3-Stage

Phase II design: monotherapy escalation → combo escalation → combo expansion

Phase I Takeaways

- MTD established at 0.6 mg/kg/week
- 1 confirmed partial response (11.5 months) in heavily pre-treated NSCLC patient
- Favorable tolerability; no withdrawals due to AEs

Phase II Status (as of Dec 2025)

- 10 subjects enrolled in monotherapy escalation (0.6–1.3 mg/kg/week)
- 3 subjects in combination cohort (0.6 mg/kg/week + Sorafenib)
- Zero DLTs; all Grade 1–2 TRAEs; no study terminations

NB-013: TLR9 Agonist CpG-ODN LNP for Solid Tumors

A Clinical-Stage Intratumoral Immunotherapy

An intratumoral TLR9 agonist designed to potently activate cellular immunity within the tumor microenvironment. With dual IND approvals from both the FDA (January 2025) and CDE (March 2025), it is poised to enter first-in-human trials in the near term across multiple advanced solid tumor indications including HNSCC, Sarcoma, and Osteosarcoma. Phase 1 first-in-human trials are imminent, with Phase 2 initiation anticipated in late 2026.

- Preclinical data demonstrate potent activation of cellular immunity, converting immunologically "cold" tumors to "hot" tumors
- Designed as a potential PD-(L)1 combination therapy to improve checkpoint inhibitor response rates
- Phase II clinical trial initiation anticipated in late 2026



Core Active Ingredient

CpG-ODN (Qtolimod) — a potent TLR9 agonist that activates innate and adaptive cellular immunity for tumor immunotherapy



QTsome Delivery System

Proprietary lipid nanoparticle platform for intratumoral injection, significantly improving local retention and systemic safety profile



Dual IND Approvals

FDA IND approved Jan 18, 2025; CDE IND approved March 2025 — enabling parallel US and China clinical development

NB-013: TLR9 Agonist CpG-ODN LNP for Solid Tumors

A Clinical-Stage Intratumoral Immunotherapy

A proprietary lipid nanoparticle (LNP) formulation of CpG-ODN (Qtolimod), a potent TLR9 agonist designed for intratumoral injection in patients with advanced solid tumors. By activating innate and adaptive cellular immunity within the tumor microenvironment, NB-013 converts immunologically "cold" tumors to "hot," and is designed as a combination partner for PD-(L)1 checkpoint inhibitors.

Proven Preclinical Efficacy

Robust tumor growth inhibition demonstrated in three syngeneic models (MC38, B16-F10, K7M2); survival advantage confirmed vs. vehicle controls

Favorable Safety Profile

30-fold safety window above max intended clinical dose; localized PK with drug essentially undetectable in blood and peripheral tissues

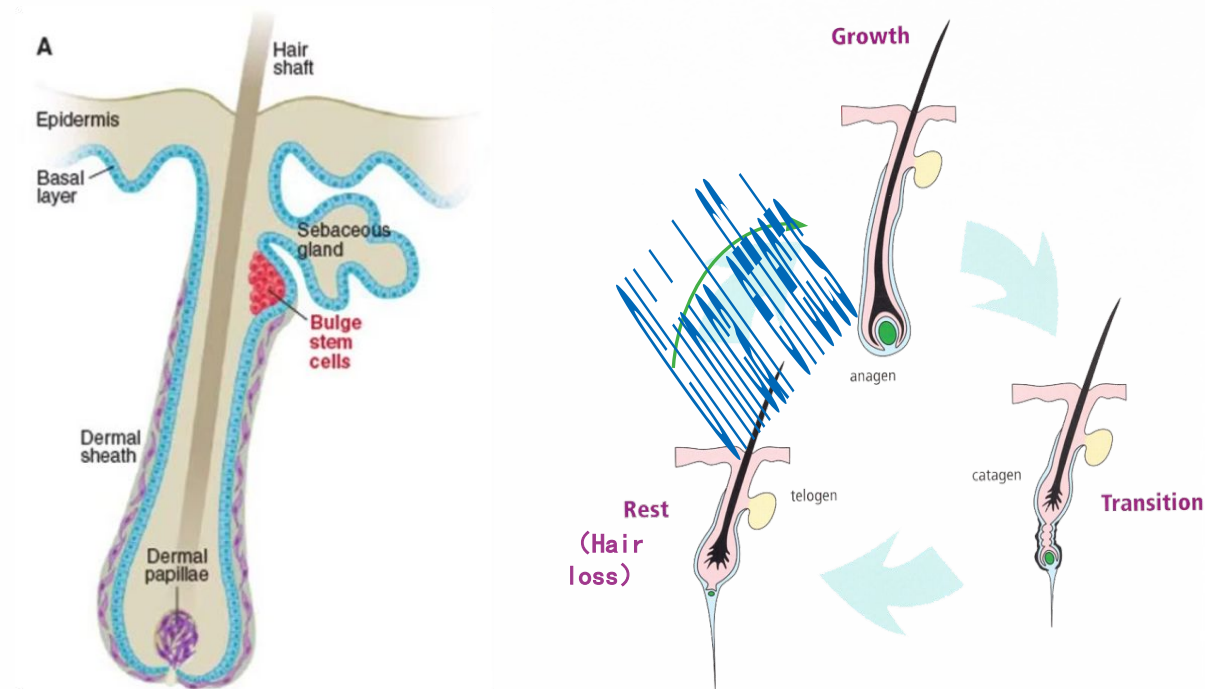
Near-Term Clinical Milestones

Phase 1 FPI (China, Dec 2025), US site startup underway; Phase 2 planned late 2026

NB-014: Topical Thyromimetic for Androgenetic Alopecia; Phase 2

First-in-class Thyromimetic MOA; activates hair follicle stem cells

Targets androgenetic alopecia by inducing resting (telogen) hair follicles to re-enter the active growth (anagen) phase. As a new chemical entity with a well-characterized mechanism of action, it has demonstrated the ability to promote hair growth across multiple preclinical models, including rodent, primate and human scalp organ culture, with excellent efficacy and a favorable safety profile.



Clinical Development Plan

The program holds broad intellectual property coverage through PCT filings spanning China, the US, Japan and the EU and is targeting NDA/MAA submission by 2030.

The clinical development timeline spans roughly a decade across four phases. Phase 1 first-in-human studies (SAD and MAD) were completed during 2021–2022, followed by Phase 2a proof-of-concept work from 2022–2024, which included 6/9-month toxicology, dose-range finding and the Phase 2a trial itself.

The program is currently in the Phase 2b window (2025–2027), running a one-year trial with End-of-Phase-2 regulatory authority meetings planned. The final stage targets a Phase 3 multi-center trial from 2028–2030, culminating in the regulatory submission.

Molecular Effect

First receptor signal activation interacts with the "hairless" gene, modulating gene expression, energy metabolism and tissue remodeling.

Cellular Effect

Activates hair follicle stem cell function in the bulge niche, driving cell growth and differentiation.

Clinical Effect

Promotes hair regeneration from resting follicles, restoring normal hair density and quality.

NB-014: Topical Thyromimetic for Androgenetic Alopecia; Phase 2

Patient Population & Market Overview

Disease Burden

Androgenetic alopecia (AGA) is a slowly progressive, patterned, non-scarring hair loss — commonly known as male or female pattern baldness — typically occurring after puberty. It is characterized by an abundance of resting (telogen) follicles and progressive follicular miniaturization. The prevalence in men **exceeds 50% by middle age** and affects nearly as many women.

Global market for AGA-approved pharmaceuticals currently exceeds **\$1B**, with significant untapped potential due to limited efficacy of existing therapies.

Market Landscape

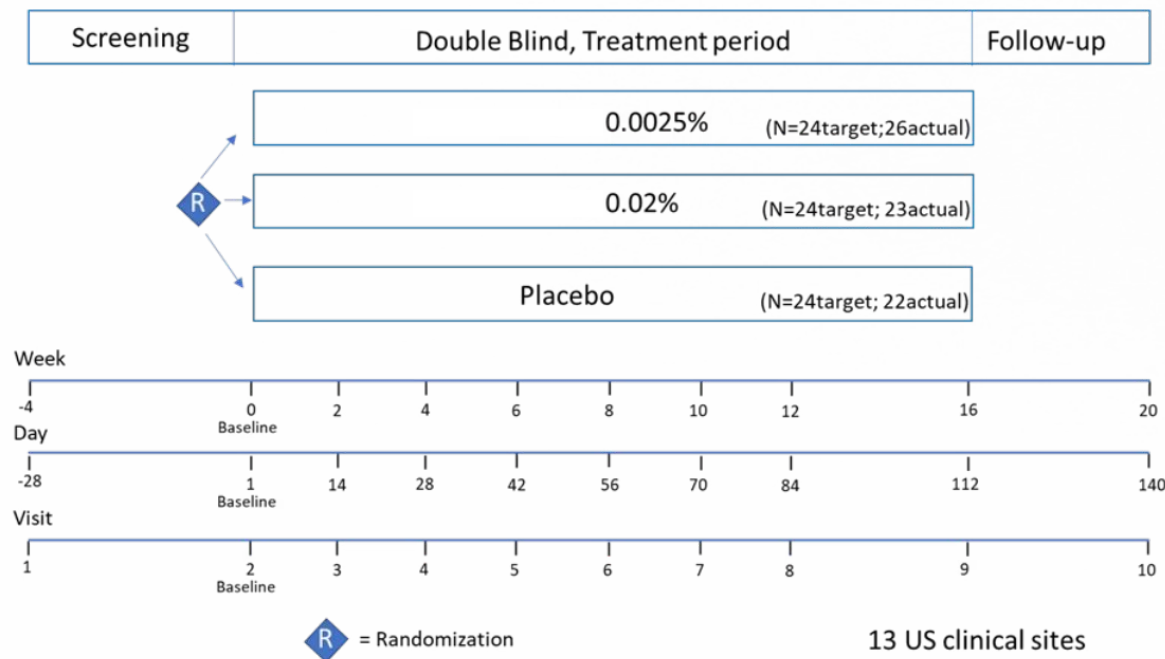
Minoxidil and **Finasteride** are the only FDA-approved drugs for AGA treatment. The global minoxidil market was valued at **\$951.8M in 2020**, with a projected **CAGR of 4.8%** through 2027. The Finasteride market is expected to grow from >\$110M (2020) to >\$130M (2026) at a CAGR of 2.5–3.0%.



Surgical hair restoration and low-level laser devices offer alternative or supplemental options, but overall market penetration remains limited due to inadequate efficacy of available pharmacological solutions.

NB-014: Topical Thyromimetic for Androgenetic Alopecia; Phase 2

Efficacy and Safety of Topical Solution Administered QD for 16 Weeks



Study Design	
Phase	Phase 2, Randomized, Double-Blind, Placebo-Controlled
Sites	Multicenter (USA)
Population	Males 18–55 yrs, Mild-to-Moderate AGA (IIIv, IV, Modified Norwood-Hamilton Scale)
Treatment	Daily topical application for 16 weeks
Endpoint	Change from Baseline in non-vellus target area hair count at Week 16

Topline Results

Efficacy

Positive treatment response demonstrated. Change from Baseline in non-vellus target area hair count at Week 16:

- 0.02% NB014 = +24.3 (PoC)
- 0.0025% NB014 = +20.3
- Placebo = +14.0.

Safety

Well tolerated. 32 TEAEs in 21 subjects (29.6%); no SAEs; no treatment discontinuations; no clinically significant lab, vital sign, or ECG changes.

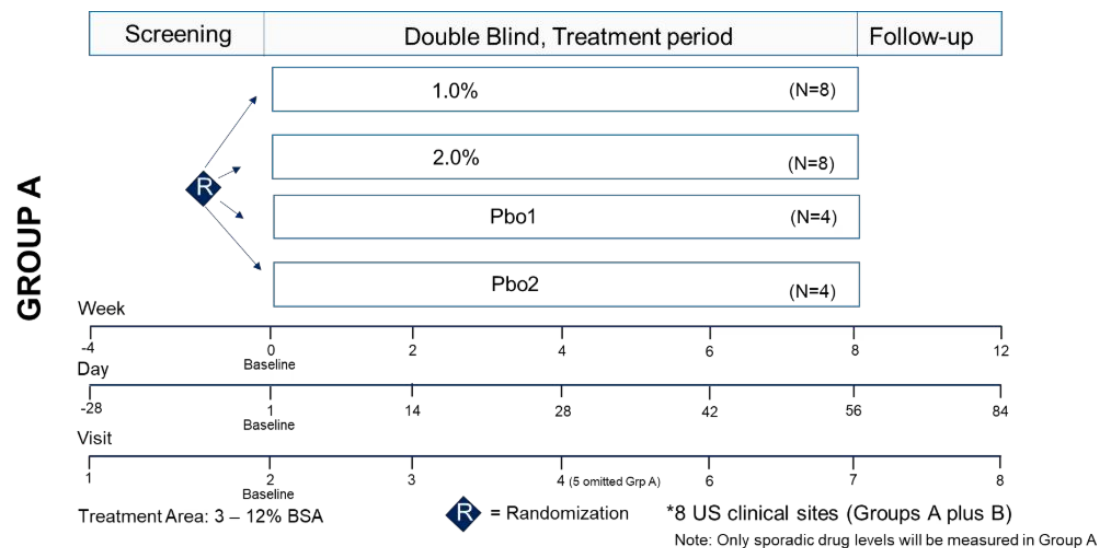
Pharmacokinetics

Extremely low to no systemic exposure — nearly all samples below LLOQ (5 pg/mL), confirming favorable local activity with minimal systemic absorption.

NB-015: Topical JAK1/Tyk2 Dual Inhibitor for Atopic Dermatitis

Targets both TH1 and TH2 pathways

Targets both the TH2 and TH1 pathways, the primary drivers of AD pathology. This differentiated dual-inhibitor approach is designed to deliver broader and more durable therapeutic response than single-pathway agents.



Study Design	
Phase	Phase 2, Randomized, Double-Blind, Placebo-Controlled study with open-label PK sub-study
Sites	Multicenter (USA)
Population	Male/female patients ≥18 years with mild-to-moderate AD (m-EASI score 5–16; AD history ≥2 years).
Treatment	Daily topical application for 16 weeks
PK Cohort	Open-label PK cohort: 6 patients treated with NB-015 2.0%

Efficacy

- Positive dose response demonstrated on the registration secondary endpoint (vIGA-AD 0/1 with ≥2-point improvement)
- m-EASI-75/90 clearly achieved in both active treatment groups at Week 8
- BSA improvement clearly achieved in the high-strength 2% group
- Itch (WI-NRS) improvement noted in the 2% NB-015 group
- m-EASI improvement time course most pronounced in the 2% high-strength group across Weeks 2, 4, 6, and 8

Safety

- Well-tolerated overall
- TEAEs: 1 patient on placebo withdrew due to flare at application sites
- No SAEs reported in any group
- No significant Local Site Reaction signal in actives vs. placebo
- No clinically significant changes in lab tests (chemistry, hematology, urinalysis) or vital signs
- No ECG trends suggesting treatment-related changes

Pharmacokinetics

- Limited systemic exposure — favorable for treatment of larger BSA
- Only 2 of 6 patients in the OL 2% cohort (mean BSA 5.2%) had measurable plasma levels, majority under 1 ng/mL

NB-015: Topical JAK1/Tyk2 Dual Inhibitor for Atopic Dermatitis

Patient Population & Market Overview

Disease Burden

Atopic dermatitis/eczema is a chronic, recurrent inflammatory skin disease commonly associated with food allergy, allergic rhinitis, and asthma. Incidence in children reaches 15–20% with a rising trend; adult incidence is 1–3%.

China alone has >20 million affected children, with a comparable burden in the US. Over 90% of patients present with mild-to-moderate severity, the target population for topical therapies.

Global market for atopic dermatitis/eczema drugs exceeds \$4B, with robust growth driven by demand for safer, more effective topical therapies.

Treatment Landscape

Mild–Moderate (>90%)

Topical steroids, calcineurin inhibitors, PDE4 inhibitors, JAK inhibitors, antihistamines, moisturizers — but side effects and insufficient efficacy remain key unmet needs.

Severe (<10%)

Biological agents (Dupixent/anti-IL-4R), oral JAK inhibitors (Upadacitinib, Abrocitinib), phototherapy — effective but associated with systemic risks and high cost.

NB-016: First-in-class small-molecule MyD88 inhibitor

Novel mechanism targeting innate immune signaling at its molecular origin

MyD88 is the master switch of innate immunity, the single adaptor protein through which 13 of 14 Toll-Like Receptor sub-families signal. Blocking it addresses the root cause of the world's most prevalent chronic diseases. No approved drug does this. NB-016 is the first.

The Problem

- Non-infectious inflammatory diseases are the #1 cause of chronic morbidity globally
- No existing therapy, biologic or small-molecule, is curative
- Biologics target single cytokines; steroids suppress broadly; neither addresses the signaling origin
- The TLR/MyD88 pathway drives autoimmune, oncological, neurological, metabolic and ischemic disease

The Solution

- NB-016 blocks MyD88 dimerization, interrupting the cascade at its molecular origin
- 1.1 New Chemical Entity; AI-assisted drug design; 19 patents granted (US, EU, China)
- World's first MyD88 inhibitor in clinical-stage compassionate use
- 100% complete remission across all IIT dermatology cases
- >90% radical cure rate; 4+ year relapse-free follow-up confirmed
- Dual IND approval: China NMPA + US FDA; Phase II underway

100%

Remission Rate

IIT Dermatology

19

Patents Granted

US · EU · China

2x

Dual IND Approval

NMPA + FDA

NB-016: First-in-class small-molecule MyD88 inhibitor

Why Invest Now

De-risked, differentiated and development-ready. Three programs. One mechanism. A clear path to NDA and IPO by 2027–2028.

466

Molecules Screened

Rigorous discovery program yielding 7 candidate drugs and 19 granted patents.

100%

IIT Remission Rate

Every enrolled patient achieved complete clinical remission across all dermatitis indications.

>90%

Radical Cure Rate

Durable, relapse-free outcomes confirmed at 4+ years — unprecedented in psoriasis.

3

Clinical Indications

Dermatology (Phase II), Ophthalmology (IND prep), IRI/IV (IND prep). One mechanism drives all three.

De-Risked: Dual IND approval (NMPA + FDA). Phase I completed with clean safety profile. IIT clinical data in hand.

Differentiated: No approved MyD88 inhibitor exists globally. NB-016 is first-in-class with no competitor near clinical use.

Near-Term Catalysts: Phase II interim data, IPO (HKEX 2027), NDA submission (2028), global licensing discussions underway.

NB-017: Best-in-Class Bispecific ADC Targeting PD-L1 & B7H3

Novel mechanism targeting innate immune signaling at its molecular origin

A novel anti-PD-L1/B7H3 bispecific antibody-drug conjugate (BsADC, DAR8) engineered to overcome the key limitations of single-target ADCs and IO-refractory disease.

Dual-Target Rationale

PD-L1 + B7H3 co-expression in 70–90% of solid tumors; avidity-driven binding overcomes antigen heterogeneity and resistance

Best-in-Class Payload

Highly hydrophilic linker + Exatecan payload; efflux pump resistant; strong bystander effect; minimal immune cell toxicity

Clinical-Ready Profile

Favorable PK in rats; well-tolerated in cynomolgus monkeys at ≥ 30 mg/kg (Q3W $\times$ 3); Fc-silenced to eliminate Fc γ R toxicity

NB-017: Best-in-Class Bispecific ADC Targeting PD-L1 & B7H3

Preclinical Data Summary

Key Efficacy Highlights

- Complete responses at 3 mg/kg in EBC-1 CDX model; outperforming DB-1419 analogue and HLX43 analogue at the same dose
- Potent TGI in MDA-MB-231, A375, and H1975 CDX models; superior to all mono-target comparators
- Dose-dependent tumor growth inhibition at 1 and 5 mg/kg in triple-negative breast cancer model
- Synergistic dual-targeting effect confirmed across multiple tumor types

Differentiation vs. Competitors

- **vs. DB-1419 analogue:** Superior TGI across all CDX models tested; stronger bystander killing
- **vs. HLX43 analogue (PD-L1 ADC):** Enhanced internalization, broader tumor coverage via B7H3 co-targeting
- **vs. vc-MMAE class:** Efflux pump resistance confirmed via tariquidar inhibition assay
- **Immune safety:** Minimal cytotoxicity on myeloid dendritic cells; PD-1/PD-L1 blockade activity preserved

9.25nM

PD-L1 Affinity (KD)

Moderate, avidity-optimized binding

1.14nM

B7H3 Affinity (KD)

High-affinity second arm

30mg/kg

Cyno Tolerability

Well-tolerated Q3W×3, no DLTs

DAR8

Drug-Antibody Ratio

High payload load; hydrophilic linker

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	

Additional Pipeline Assets (2/2)

Drug Candidate	Indication	Mechanism	Stage	Key Feature
TDM-181064	Psoriasis (PsO) & Inflammation	Oral prodrug; multiple small molecule candidates	IND-enabling studies complete; FIH late 2026	Both topical and oral routes viable
TDM-003	Systemic Lupus Erythematosus (SLE)	Selective Tyk2 inhibitor (JH1 domain); oral	Candidate nominated; exploratory tox in progress	Immune modulator; IFN signal blockade
Scar Program	Scarring / Wound Repair	Novel kinase inhibitor; YAP pathway	SAR lead optimization; U of Michigan collaboration	First-mover in scarring biology

Let's Connect

For more information about partnership opportunities and to join us on this exciting journey of bringing transformative therapies to patients worldwide, please reach out to:



Lawrence Hu, PhD

**Chief Technology Officer
Head of Business Development**

Phone: 973-967-9949

Email: lawrence@navikabio.com



Accelerating Innovation to Improve Patients' Lives

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