VisionSearch
Bio

SIMULATION-DRIVEN ONCOLOGY

MilanoQ

A simulation-validated therapeutic platform for resistant cancers

Immune-compatible · mutation-targeted · built from safe, natural compounds

Validated across 30+ advanced in-silico simulations

Investor & Partner White Paper

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EXECUTIVE SUMMARY

The efficacy of chemotherapy, without the toxicity.

MilanoQ is a next-generation, immune-compatible, mutation-targeted therapeutic system designed to treat resistant cancers — beginning with colorectal cancer driven by NRAS/KRAS Q61H mutations. It is built entirely from safe, natural compounds and has been validated across more than 30 advanced simulations spanning quantum pharmacology, immune-system mapping, and whole-body microdosing. To our knowledge, MilanoQ is one of the most deeply modeled non-toxic oncology platforms in preclinical development.

The results below are **computationally validated in silico**. They represent a strong, internally consistent modeling package that de-risks the biology ahead of wet-lab work — not clinical outcomes. VisionSearch Bio is now raising to convert these simulations into CRO-led preclinical evidence and IND-enabling studies.

~11.5 uM	~4.1x	~72%	<4%
IC50 — tumor apoptosis	CD8+ immune reactivation	Long-term non-relapse	Mutation-escape rate

THE PROBLEM & THE MARKET

Resistant cancers outrun today’s medicine.

Current treatments for advanced, mutation-driven cancers offer poor durability, high toxicity, and frequent relapse. Conventional chemotherapies such as FOLFOX and FOLFIRI suppress the immune system, cause severe organ stress, and fail to prevent long-term resistance or recurrence. Patients are left with a stark trade-off between efficacy and quality of life.

The opportunity is large and underserved. The global colorectal cancer market is estimated at roughly **$13.7 billion in 2025**, with colorectal cancer drug sales projected to approach **$20 billion by 2033**. KRAS mutations appear in roughly **40% of colorectal cancers** and NRAS in a further 1–6%. Yet the recent wave of approved KRAS inhibitors — sotorasib (approved for colorectal use in January 2025) and adagrasib — target only the **G12C** variant. The **Q61H** mutations MilanoQ is designed for remain largely unaddressed, leaving a clear clinical and commercial white space.

THE MILANOQ PLATFORM

A four-step mechanism, designed to work with the body.

01 — Target	Engages resistant driver mutations (NRAS/KRAS Q61H) directly, rather than relying on broad cytotoxicity.
02 — Induce	Drives programmed tumor-cell death (apoptosis) at an IC50 of ~11.5 µM in modeling.
03 — Reactivate	Restores CD8+ T-cell activity ~4.1× — immune engagement normally reserved for biologics and checkpoint inhibitors.
04 — Protect	Suppresses mutation escape (<4%) to deliver durable, long-term disease control.

SIMULATION-VALIDATED RESULTS

What the models show.

The following endpoints were produced by MilanoQ's in-silico validation stack. Each is a computational result intended to guide and de-risk subsequent laboratory confirmation.

Endpoint	Result (in silico)	Interpretation
Tumor apoptosis (IC50)	~11.5 µM	Potent induction of programmed tumor-cell death
CD8+ immune reactivation	~4.1×	Strong cytotoxic T-cell re-engagement
Long-term non-relapse rate	~72%	Durable control across modeled follow-up
Mutation-escape suppression	<4%	Low probability of resistance emergence
Chronic-use safety	180 days	No modeled toxicity over extended dosing
Oral bioavailability	~87%	Enteric-coated delivery simulation

Note — all figures are computationally derived. Wet-lab and preclinical confirmation are the immediate next milestones.

TECHNOLOGY STACK

An unprecedented in-silico validation engine.

Quantum binding simulations
Molecular binding modeled with Qiskit + PennyLane for pre-synthesis precision.

MOISL — immune ecosystem
Models how the therapy reshapes the tumor–immune microenvironment.

TEHM — exhaustion horizon
T-cell exhaustion modeling to predict durability of immune response.

WB-MEM — microdosing map
Whole-body map projecting distribution and exposure across tissues.

Virtual clinical trial
1,000-avatar simulated cohort stress-testing response and variability.

3D organoid + delivery
Tumor-organoid modeling plus enteric-coated delivery (~87% bioavailability).

PIPELINE

One platform. Many resistant tumors.

Indication	Biology	Stage
Colorectal cancer	KRAS / NRAS Q61H — lead indication	Lead / modeled
Lung adenocarcinoma	KRAS+	Evaluation
Multiple myeloma	Hematologic	Evaluation
Glioblastoma · DIPG	CNS / pediatric brain tumor	Exploratory
Pancreatic · Melanoma	Solid tumors	Exploratory
TNBC · NSCLC	Breast / lung	Exploratory

WHY THIS MATTERS

The strengths of three modalities, in one.

Attribute	Chemotherapy	Checkpoint / biologics	MilanoQ
Tumor-killing efficacy	High	Variable	**Chemo-comparable**
Systemic toxicity	Severe	Moderate	**Non-toxic (natural)**
Immune activation	Suppresses	Strong	**~4.1× CD8+**
Resistant-mutation reach	Limited	Limited	**Multi-tumor**
Combination-ready	Limited	Yes	**DCV / checkpoint combo**

NEXT STEPS & THE ASK

From simulation to preclinical evidence.

MilanoQ is entering partner conversations and seed-stage investment to advance toward IND-enabling studies. Near-term priorities:

- **CRO-led preclinical validation** — converting the in-silico package into wet-lab and in-vivo evidence.
- **DCV integration strategy** — dendritic-cell-vaccine and immune-checkpoint combination pathways.

- **Seed-stage investment** — to fund IND-enabling studies and build the founding scientific team.

Explore a partnership or investment

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