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Q-BRM: The Quantum Biomedical Reasoning Model

How GNQ Insilico's Proprietary Reasoning Model Redefines Drug Development and Clinical Decision Support

Confidential | For Authorized Use Only | **April 2026**
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Executive Summary

The dominant paradigm in AI-driven drug development and clinical decision support rests on a fundamental limitation: it is **correlational**. Systems trained on population data learn statistical patterns — associations between features and outcomes — without understanding the causal mechanisms that actually drive disease, treatment response, and resistance. This is not a data problem. It is an **architectural problem.**

To solve this at the architectural level[3,4], GNQ Insilico has developed the first precision medicine engine, the **Quantum Biomedical Reasoning Model** (Q-BRM) that operates simultaneously across all three rungs of Pearl's Causal Ladder: association, intervention, and counterfactual inference. It is grounded in quantum-accurate molecular physics and a relational dynamic biological knowledge graph. Q-BRM powers GNQ's three integrated platforms: the Drug Assessment Platform (DAP), Drug Simulation Platform (DSP), and Digital Twin Platform (DTP).[1]

Critically, GNQ does not **only** rely on real-world clinical data as its ***primary source of insight***. GNQ platform applies causal reasoning from first principles — biology, chemistry, and quantum physics, to infuse mechanistic precision into actionable medical insights. Real-world evidence, such as the MMRF dataset (1,100+ multiple myeloma patients), serves as **validation** of computationally derived predictions, not the origin of those predictions. This inverts the traditional paradigm and fundamentally changes what is possible with limited or novel patient populations.

> **Investment Thesis in One Sentence**
>
> Q-BRM is a proprietary platform technology at the convergence of quantum-enhanced causal AI and precision medicine — addressing two multibillion-dollar markets (drug development efficiency and clinical decision support) with a single defensible architectural moat that can only be replicated by building from the ground up.

1. The Market Problem: Why Correlation AI Is Failing

The pharmaceutical industry spends over $2.5 billion to bring a single drug to market, with Phase II and Phase III failure rates exceeding 60%. A dominant driver of late-stage failure is misidentification of the right patient population and underestimation of resistance mechanisms — problems that correlation-based AI cannot solve because they require **causal** reasoning about biological mechanisms, not statistical pattern recognition.

In clinical decision support, the picture is similarly concerning. Independent benchmarking studies demonstrate that current LLMs perform significantly worse than physicians on realistic clinical decision tasks, fail to follow diagnostic or treatment guidelines, and cannot reliably interpret laboratory results.[3] A comprehensive taxonomy of LLM reasoning failures confirms these are **architectural limitations** — not data or model size problems.[4]

GNQ addresses both markets with the same underlying architectural solution: replace correlation-based pattern matching with mechanistic causal reasoning powered by quantum physics.

2. Q-BRM: Architecture and Components

Q-BRM — the **Quantum Biomedical Reasoning Model** — is GNQ's proprietary AI reasoning engine. It is composed of three integrated architectural layers, each of which represents a distinct technical advance over the current state of the art.

2.1 Layer 1: The Relational Dynamic Graph (RDG)

The foundation of Q-BRM is the **Relational Dynamic Graph (RDG)** — GNQ's proprietary biological knowledge graph encoding 681+ pathway relationships, 20,000+ genes, and 2M+ causal links with typed, weighted, directional semantics. Unlike flat knowledge graphs or standard DAG-based SCMs, the RDG encodes **relational biological heterogeneity**: the type of each relationship (encodes, activates, inhibits, phosphorylates, modulates) is part of the causal model.[5]

Critically, the RDG supports biological feedback loops — BCL2 anti-apoptotic cycling, NF-κB inflammatory signaling, MAPK crosstalk — that are structurally incompatible with the acyclicity requirement of standard SCM DAGs. This is not a workaround; it is the correct representational choice for biological reality.[1,5]

2.2 Layer 2: Quantum Relational Causal Model (Quantum RCM)

Q-BRM's causal inference framework extends Pearl's Structural Causal Models to the relational domain and adds quantum-computed molecular priors. Two key innovations define this layer:

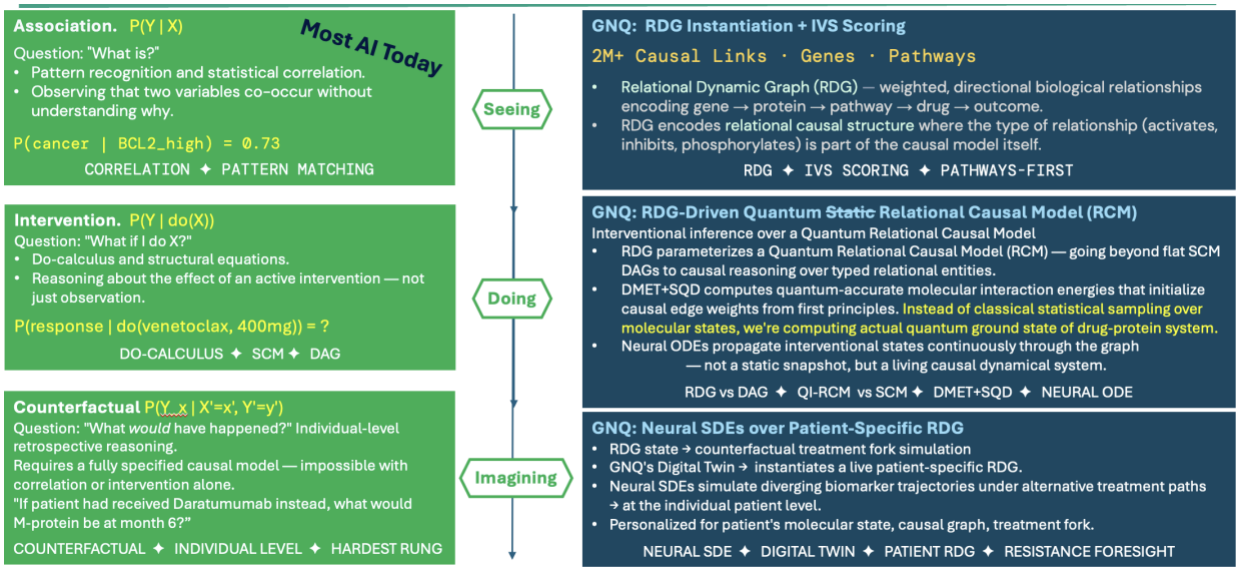
- **DMET+SQD Quantum Edge Weights:** IBM's Density Matrix Embedding Theory + Sample-based Quantum Diagonalization framework computes binding free energies (ΔG) at quantum accuracy on real near-term hardware. These become the causal edge weights in the RDG — grounding GNQ's causal model in physical reality rather than statistical estimation.[2]
- **Neural ODE Interventional Propagation:** Graph Neural Ordinary Differential Equations propagate interventional causal states continuously over time through the RDG, implementing do-calculus as dynamic trajectory simulation rather than static snapshot inference.[6]

2.3 Layer 3: BRM Agentic Orchestration

The BRM (Biomedical Reasoning Model) is Q-BRM's agentic interface layer, built on LangGraph and Model Context Protocol (MCP). The BRM handles natural language intent parsing and orchestrates calls to DAP, DSP, and DTP — but critically, it does not perform causal reasoning.

Causal inference, pathway scoring, and quantum simulation are handled by deterministic mechanistic engines. MCP-based structured access to the RDG — rather than RAG-based vector search — ensures every biological query traverses verified causal graph structure.

2.4 Pearl's Causal Ladder: Q-BRM Implementation



3. Two Markets, One Platform

Q-BRM's architecture addresses two distinct, large markets through the same underlying causal intelligence engine. This is the core investment efficiency argument: GNQ is not building two products — it is applying one architectural platform across two monetizable domains.

3.1 Drug Development: Shifting the rNPV Curve

For pharmaceutical and biotech clients, Q-BRM's value proposition is expressed in **rNPV terms** — the risk-adjusted net present value framework that governs every capital allocation decision in drug development. Q-BRM improves rNPV by directly improving the input parameters that drive it:

- **Probability of Success (PoS):** Q-BRM's causal pathway analysis identifies responder subpopulations from first principles — not from historical trial data — raising PoS estimates by grounding them in mechanism rather than observation.
- **Go/No-Go Timing:** Resistance prediction via Neural SDE simulation identifies likely failure modes before Phase II enrollment, enabling earlier go/no-go decisions and reducing sunk cost on mechanistically doomed programs.

- **Compound Rescue:** Q-BRM's zero-shot combination prediction identifies previously unexplored patient subpopulations or combination partners for shelved compounds using causal pathway complementarity — not population-level correlations.
- **Trial Design Biomarkers:** Novel predictive signatures emerge from Q-BRM's latent causal graph — surfacing enrollment criteria that improve trial efficiency and patient stratification beyond standard genomic markers.

3.2 Clinical Decision Support: Patient-Level Causal Intelligence

For clinical providers — oncology centers, neurodegenerative clinics, functional health networks — Q-BRM delivers individual-patient treatment optimization grounded in causal biology, not population statistics. Q-BRM's Digital Twin Platform builds a live patient-specific RDG from multi-omics, FHIR R4/R5 clinical records, and longitudinal biomarker streams. Neural SDEs simulate diverging biomarker trajectories under alternative treatment paths with calibrated uncertainty bounds — operationalizing Pearl's Rung 3 counterfactual inference at the bedside.

Clinicians receive not just a recommendation but a mechanistic rationale: not 'this therapy is recommended' but 'because this patient's BCL2 pathway is overactive due to t(11;14) translocation, venetoclax will restore apoptotic sensitivity, while the PSMB5 A49T variant predicts bortezomib resistance.'

3.3 Real-World Evidence: Validation, Not Source

Q-BRM generates insight from quantum physics, systems biology, and causal inference — not only from historical patient data. Real-world evidence, such as the **MMRF CoMMpass dataset (1,100+ multiple myeloma patients)**, validates Q-BRM's computationally derived predictions. This inverted epistemology enables reliable prediction for novel drug combinations, rare genotypes, and newly approved therapies — scenarios where data-driven platforms are silent.

4. Competitive Differentiation

Capability	Correlation AI (Most Competitors)	SCM / Causal AI (Emerging)	GNQ Q-BRM
Causal Reasoning	✗ Association only	⚠ Static SCM	✓ Full RCM — all 3 rungs
Biological Fidelity	✗ Flat feature vectors	⚠ Simplified DAG	✓ RDG: typed relational graph
Feedback Loops	✗ Not modeled	✗ DAG acyclic only	✓ RDG supports cycles
Quantum Physics Priors	✗ None	✗ None	✓ DMET+SQD edge weights
Temporal Dynamics	✗ Static snapshot	⚠ Discrete time steps	✓ Continuous Neural ODE/SDE

Capability	Correlation AI (Most Competitors)	SCM / Causal AI (Emerging)	GNQ Q-BRM
Patient Counterfactuals	✗ Population averages	⚠ Theoretical only	✅ Digital Twin Neural SDEs
Drug Dev: rNPV Inputs	⚠ Empirical / indirect	⚠ Limited	✅ Causal PoS + timing signals
Resistance Prediction	✗ Empirical flags	⚠ Limited	✅ Emergent from pathway state
Combo Therapy Design	✗ Empirical / trial	⚠ Manual	✅ Zero-shot quantum prediction
Interpretability	✗ Black box	⚠ Graph visible	✅ Mechanistic pathway rationale
Real-World Evidence Role	⚠ Primary source	⚠ Primary source	✅ Validation layer (MMRF 1,100+)

The competitive moat is architectural and cumulative. Each of Q-BRM's four differentiating components — RDG relational structure, DMET+SQD quantum priors, Neural ODE/SDE dynamics, and patient-specific digital twin — individually represents a significant technical advance. Their integration creates a system that cannot be replicated by bolting quantum modules onto existing correlation platforms or by adding a knowledge graph to a standard SCM framework.

5. Validated Commercial Traction

GNQ's platform is not pre-revenue. The following partnerships represent contracted revenue, active deployments, and strategic institutional relationships that validate both market demand and platform capability.

- **MMRF CoMMpass Collaboration:** 1,100+ patient multiple myeloma genomic dataset; primary validation substrate for Q-BRM drug development capabilities.
- **KAIRA Health (Multi-Year Agreement):** Digital Twin Platform deployed across clinics in Toronto, New York, Miami, Chicago — first at-scale clinical validation of patient-specific RDG and counterfactual simulation.
- **IBM Joint Marketing Agreement:** Active co-sell arrangement positioning Q-BRM based products within IBM's enterprise life sciences and quantum computing ecosystem. June 2026 IBM Silicon Valley Labs innovation session with Pfizer R&D represents a flagship near-term pharma engagement.

6. Why Now: Technology Convergence

Q-BRM's architecture became practically deployable in 2024–2026 due to the convergence of three previously immature technology vectors:

- **Quantum Chemistry at Clinical Scale:** IBM's DMET+SQD, published in *Science Advances* 2025, validates quantum-accurate chemistry on real near-term hardware. The qiskit-addon-sqd production toolkit enables deployment without fault-tolerant qubits.[2]
- **Neural Differential Equations for Biology:** Neural ODEs and SDEs have matured to production-grade stability, with training efficiency and uncertainty quantification suitable for clinical deployment.[6]
- **Relational Causal Modeling:** The RCM framework provides a formally proven basis for extending Pearl's SCMs to heterogeneous relational biological graphs with feedback loops.[5]

The window of competitive advantage is the interval between current Q-BRM platform maturity and broad market recognition of the architectural shift from correlation to causal AI in precision medicine. GNQ holds the leading position at this inflection point.

7. Conclusion

Q-BRM represents a genuine architectural discontinuity in precision medicine AI — not incremental improvement over existing platforms, but a new inferential paradigm that combines quantum physics, relational causal modeling, and continuous-time dynamics in a single integrated engine. It addresses two large, validated markets through a single defensible technology platform, with contracted revenue, institutional partnerships, and peer-reviewed scientific foundations already in place.

Contact GNQ Insilico

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Glossary of Key Terms

Q-BRM — Quantum Biomedical Reasoning Model. GNQ's proprietary intelligence engine powering DAP, DSP, and DTP. Composed of the Relational Dynamic Graph (RDG), Quantum Relational Causal Model (Quantum RCM), and agentic BRM orchestration layer.

RDG — Relational Dynamic Graph. GNQ's proprietary knowledge graph substrate: 681+ pathway relationships, 20,000+ genes, 2M+ causal links. Typed, weighted, directional biological relationships with continuous temporal dynamics.

Quantum RCM — Quantum Relational Causal Model. The causal inference framework within Q-BRM: DMET+SQD quantum edge weights + Neural ODE/SDE propagation over the RDG. Extends Pearl's SCMs to relational heterogeneous graphs with feedback loops.

DAP — Drug Assessment Platform. GNQ's causal pathway scoring and drug evaluation engine using Integral Variant Scoring (IVS).

DSP — Drug Simulation Platform. GNQ's dynamic causal simulation engine using Neural ODEs and Neural SDEs.

DTP — Digital Twin Platform. GNQ's HIPAA-compliant patient-specific causal modeling and clinical decision support layer.

BRM — Biomedical Reasoning Model. GNQ's agentic orchestration layer built on LangGraph and MCP. Handles natural language intent parsing and orchestrates calls to DAP, DSP, and DTP.

DMET+SQD — Density Matrix Embedding Theory + Sample-based Quantum Diagonalization. IBM's near-term quantum chemistry framework for computing molecular ground states on real quantum hardware without fault-tolerant qubits.

IVS — Integral Variant Scoring. GNQ's proprietary methodology for scoring pathway-level causal impact from patient genomic variant profiles.

Neural ODE — Neural Ordinary Differential Equation. Continuous-time neural architecture for modeling causal state evolution (interventional inference — Pearl Rung 2).

Neural SDE — Neural Stochastic Differential Equation. Extends Neural ODEs with principled stochasticity for uncertainty quantification; enables counterfactual simulation (Pearl Rung 3).

SCM — Structural Causal Model. Judea Pearl's formal framework for causal reasoning using DAGs and structural equations.

DAG — Directed Acyclic Graph. Required by standard SCMs. Cannot represent biological feedback loops — a key limitation addressed by GNQ's RDG.

rNPV — Risk-adjusted Net Present Value. Standard pharma valuation metric for drug programs; GNQ improves rNPV inputs by raising probability of success and accelerating go/no-go timing.

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[3] **Hager, P., Jungmann, F., et al.** "Evaluation and mitigation of the limitations of large language models in clinical decision-making." *Nature Medicine* 30(9), 2613–2622, 2024. DOI: 10.1038/s41591-024-03097-1. Across 2,400 real cases, LLMs performed significantly worse than physicians on realistic clinical tasks.

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