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EVALUATING MOLECULAR TARGETS FOR DRUG REPURPOSING IN ACUTE MYELOID LEUKEMIA: AN INTEGRATIVE BIOINFORMATICS AND NETWORK PHARMACOLOGY STUDY

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ABSTRACT

Acute myeloid leukemia (AML) is a biologically heterogeneous hematological malignancy characterized by diverse genomic, epigenomic, transcriptional, and cellular abnormalities that contribute to differences in disease progression, treatment response, relapse, and survival. Although molecular classification has enabled targeted therapies for selected AML subgroups, substantial challenges remain, particularly among patients with adverse-risk disease, treatment resistance, or limited access to newer therapies. This article proposes an integrative bioinformatics and network-pharmacology framework for identifying molecularly relevant therapeutic targets and repurposable drugs in AML. The proposed approach integrates publicly available genomic and transcriptomic resources with differential-expression analysis, functional and pathway enrichment, protein–protein interaction networks, drug–target databases, perturbational transcriptomics, and clinical evidence. The workflow involves data preprocessing, identification of disease-associated molecular signatures, pathway analysis, hub-target prioritization, drug–target mapping, and computational assessment of candidate compounds. Because no AML dataset or completed computational analysis was provided, numerical findings are not reported. Instead, the framework is designed to generate biologically coherent and testable therapeutic hypotheses. Candidate compounds identified through convergent evidence should subsequently undergo experimental, pharmacological, and clinical validation. Integrating multiple molecular and pharmacological evidence layers may support systematic AML drug repurposing while reducing reliance on isolated molecular targets and facilitating translational research.

Keywords: *acute myeloid leukemia; bioinformatics; network pharmacology; drug repurposing; protein–protein interaction; therapeutic targets; transcriptomics.*

1. Introduction

Acute myeloid leukemia (AML) is an aggressive hematological malignancy characterized by the accumulation of abnormal myeloid progenitor cells and impairment of normal hematopoiesis. Its clinical and biological behavior varies considerably between patients because AML comprises molecularly distinct disease subgroups. Large-scale genomic studies have demonstrated that AML is driven by combinations of alterations involving signaling pathways, transcription factors, epigenetic regulators, tumor-suppressor mechanisms, chromatin modification, RNA splicing, and other cellular processes rather than by a single universal molecular lesion (The Cancer Genome Atlas Research Network, 2013).

The molecular complexity of AML has important therapeutic implications. The European LeukemiaNet (ELN) recommendations emphasize integrated diagnosis incorporating cytogenetic and molecular information for classification, risk assessment, and therapeutic decision-making. Contemporary AML treatment has also expanded beyond conventional chemotherapy to include molecularly directed agents targeting alterations or dependencies involving FLT3, IDH1, IDH2, and BCL2, among others (Döhner et al., 2022). Thus, molecular information is increasingly relevant not only to disease classification but also to therapeutic selection.

At the population level, AML remains a substantial health burden. A recent Global Burden of Disease analysis reported an increase in estimated global AML incidence from 79,372 cases in 1990 to 144,645 in 2021, with disproportionate burden among older and male populations (Zhou et al., 2024). These patterns reinforce the need for treatment strategies that are biologically effective but also potentially scalable and accessible.

Despite progress in precision hematology, several problems remain. First, individual molecular alterations do not necessarily operate independently. A mutation or differentially expressed gene may participate in several interacting pathways and regulatory networks. Second, conventional target discovery can prioritize individual molecules without fully considering pathway-level interactions. Third, development of a completely new drug can require substantial time, financial investment, and clinical testing. Drug repurposing offers an alternative strategy in which existing drugs or compounds are investigated for therapeutic applications beyond their original indications.

The unresolved problem is therefore not simply the identification of additional AML-associated genes. Rather, there is a need for integrative approaches capable of connecting disease-associated molecular signatures → biological pathways → interacting proteins → pharmacological targets → existing drugs → clinically testable hypotheses.

This article addresses that gap by developing an integrated bioinformatics and network-pharmacology framework for AML drug repurposing. The objective is not to claim a particular drug or target as effective, but to establish a reproducible strategy for prioritizing candidates for subsequent validation.

2. Literature Review

2.1 Molecular heterogeneity of AML

The TCGA AML analysis remains an important demonstration of the molecular complexity of AML. The study examined 200 clinically annotated adults with de novo AML using whole-genome or whole-exome sequencing together with RNA sequencing, microRNA sequencing, and DNA-methylation analysis. Multiple classes of recurrently altered genes were identified, demonstrating that AML pathogenesis reflects interactions among several biological processes rather than a single dominant pathway (The Cancer Genome Atlas Research Network, 2013).

This molecular heterogeneity creates both opportunities and challenges. Molecular characterization can identify actionable abnormalities, but the diversity of disease mechanisms means that a target relevant to one subgroup may have limited relevance to another. Network-level analysis may therefore complement mutation-centered approaches by identifying biological relationships shared across molecularly heterogeneous disease states.

2.2 Transcriptomic analysis and functional interpretation

Gene-expression profiling can identify transcripts that differ between AML and appropriate control or comparison groups. Differential-expression analysis is particularly useful for generating candidate genes, although the interpretation of individual differentially expressed genes requires biological context.

The limma framework provides established statistical methodology for differential-expression analysis of microarray and RNA-sequencing experiments and can accommodate complex experimental designs (Ritchie et al., 2015). After differential-expression analysis, functional annotation can be used to determine whether candidate genes converge on biological processes or molecular functions.

Gene Ontology provides a structured framework for describing gene and gene-product functions across biological processes,

molecular functions, and cellular components (Gene Ontology Consortium, 2023). Such enrichment analysis can transform a long list of genes into biologically interpretable functional patterns.

2.3 Protein–protein interaction networks

Genes identified through differential expression do not necessarily represent independent biological entities. Their protein products can interact directly or indirectly within cellular networks. STRING provides an established resource for protein-association networks and functional enrichment analysis (Szklarczyk et al., 2023).

Network analysis can identify highly connected or otherwise topologically important proteins. However, network centrality should not automatically be interpreted as biological causality. A highly connected protein may be important because it participates in many cellular processes, but its therapeutic tractability, disease specificity, toxicity profile, and pharmacological accessibility must also be considered.

2.4 Network pharmacology and drug repurposing

Network pharmacology extends conventional one-target drug discovery by considering relationships among multiple targets, pathways, and pharmacological agents. In AML, this approach can be useful because disease phenotypes frequently emerge from interacting molecular processes.

Drug-target resources such as DrugBank and ChEMBL provide complementary information for connecting molecular targets with known pharmacological compounds. DrugBank 6.0 provides extensive information on drugs, targets, pathways, interactions, indications, and related pharmacological attributes (Knox et al., 2024), whereas ChEMBL provides curated bioactivity information covering multiple compound and assay types (Zdrazil et al., 2024). Open Targets provides another layer of evidence by integrating information relevant to target–disease associations and target prioritization (Ochoa et al., 2023). The integration of these resources can therefore move target discovery beyond simple gene lists toward evidence-based prioritization.

2.5 Perturbational transcriptomics

A further level of integration can be achieved by comparing disease-associated transcriptional signatures with signatures generated after pharmacological perturbation. The Connectivity Map/L1000 platform was developed to enable systematic comparison of gene-expression responses to compounds and other perturbations (Subramanian et al., 2017). Conceptually, a drug that produces a transcriptional response opposing an AML-associated signature may be prioritized for further investigation. However, signature reversal alone does not demonstrate therapeutic efficacy. Differences in cell type, concentration, exposure duration, disease subtype, and experimental context must be considered.

Table 1. Evidence layers supporting computational AML target and drug prioritization

Evidence layer	Main question	Potential output	Interpretation
Differential expression	Which genes differ between AML and comparator samples?	Candidate upregulated/downregulated genes	Molecular association
Functional enrichment	What biological processes are represented?	GO/pathway terms	Biological interpretation
Network analysis	Which proteins are highly connected within relevant networks?	Network centrality measures	Network relevance
Drug–target mapping	Which existing compounds interact with candidate targets?	Drug–target relationships	Pharmacological tractability

Target–disease evidence	Is there independent evidence linking the target to AML or related disease biology?	Target-association evidence	External support
Perturbational analysis	Which compounds reverse disease-associated signatures?	Connectivity/reversal scores	Hypothesis generation
Clinical evidence	Is the candidate associated with patient outcomes or clinical experience?	Survival/response/clinical evidence	Translational support
Experimental evidence	Does the candidate affect AML cells or models experimentally?	Functional response	Preclinical support

3. Conceptual Framework

The conceptual basis of the proposed framework is that AML should be considered a multilayer molecular system rather than a collection of isolated genes.

The framework consists of five connected levels:

Molecular disease state: genomic, transcriptomic, epigenomic, and other molecular abnormalities.

Biological organization: genes are organized into pathways and protein-interaction networks.

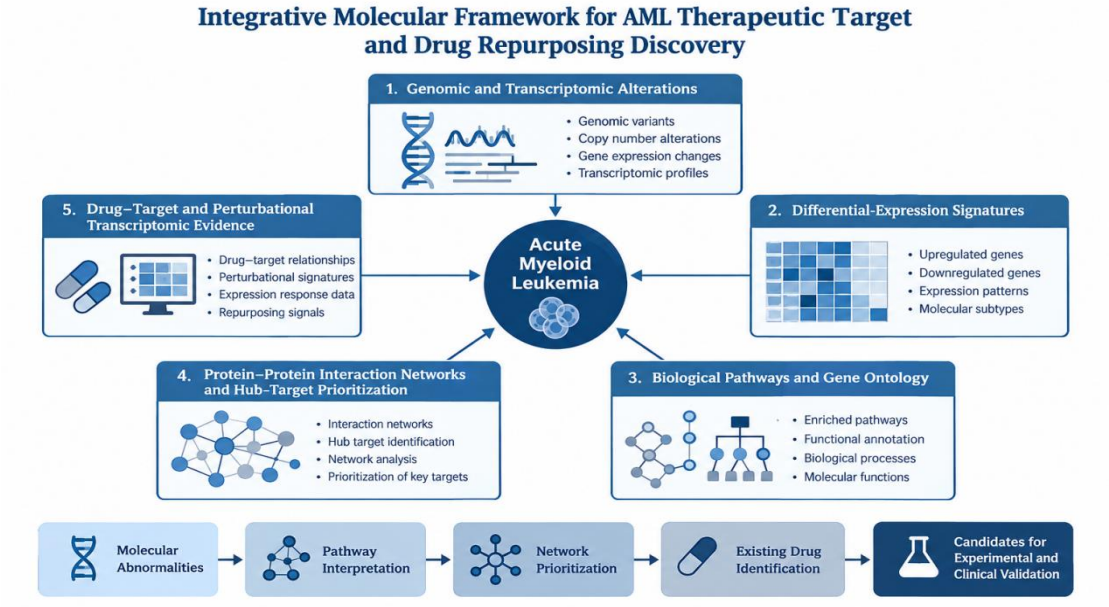
Therapeutic vulnerability: network-relevant targets may represent points at which pharmacological intervention could alter disease-associated processes.

Drug repurposing: existing compounds are mapped to prioritized targets and compared against disease-associated molecular signatures.

Validation: computational candidates are evaluated through independent molecular, experimental, pharmacological, and clinical evidence.

The central assumption is not that computational centrality equals therapeutic importance. Instead, candidate prioritization should reflect convergent evidence from several independent layers.

Figure 1. Conceptual framework for integrative AML target and drug-repurposing discovery.



4. Methodology

4.1 Study design

The proposed study is a computational, integrative bioinformatics and network-pharmacology investigation. It is designed as a hypothesis-generating framework rather than a completed experimental study. Actual datasets should be selected according to predefined inclusion criteria, biological comparability, sample quality, and availability of relevant clinical annotations.

4.2 Data acquisition

Potential molecular datasets may be obtained from publicly accessible repositories such as Gene Expression Omnibus (GEO) and The Cancer Genome Atlas (TCGA), depending on the specific research question. The selected dataset should contain AML samples and an appropriate comparator or clinically meaningful stratification.

Important metadata should include disease subtype, treatment status, molecular characteristics, age where available, outcome variables, and other covariates relevant to the intended analysis.

4.3 Preprocessing and differential-expression analysis

Raw or processed expression data should undergo quality assessment, normalization, filtering, and appropriate batch-effect evaluation. Differential expression should then be assessed using a validated statistical framework such as limma, with adjustment for multiple testing where appropriate (Ritchie et al., 2015).

The resulting candidate set should be reported using transparent thresholds defined before analysis. Importantly, thresholds should not be selected retrospectively to produce a preferred biological outcome.

Table 2. Proposed computational resources and their analytical roles

Resource/tool	Primary purpose	Proposed application
GEO	Public gene-expression datasets	AML transcriptomic data acquisition
TCGA	Cancer genomic and molecular data	AML molecular and clinical data
R/Bioconductor	Statistical and bioinformatics analysis	Data preprocessing and downstream analysis
limma	Differential-expression analysis	Identification of differentially expressed genes
Gene Ontology	Functional annotation	Biological-process interpretation
STRING	Protein-association networks	PPI construction and enrichment
DrugBank	Drug and target information	Drug–target mapping
ChEMBL	Bioactivity data	Compound–target evidence
Open Targets	Target–disease evidence	Target prioritization
Connectivity Map/L1000	Perturbational signatures	Drug-signature comparison

4.4 Functional and pathway enrichment

Differentially expressed genes should be evaluated using Gene Ontology and, where appropriate, established pathway resources. Enrichment analysis should focus on biological coherence rather than isolated statistically significant terms.

The output should include enriched biological processes, molecular functions, cellular components, and relevant signaling or disease-associated pathways. Multiple-testing correction should be applied.

4.5 Protein–protein interaction analysis

Candidate genes should be mapped to protein identifiers and integrated into a protein–protein interaction network. STRING can be used to obtain association networks and functional enrichment information (Szklarczyk et al., 2023).

Network metrics may include degree, betweenness, closeness, and other centrality measures. Rather than relying on a single metric, candidate targets should be prioritized using a composite evidence framework.

4.6 Drug–target mapping

Prioritized targets should be mapped against pharmacological databases including DrugBank, ChEMBL, and Open Targets. DrugBank and ChEMBL provide complementary information regarding drugs, targets, compounds, and bioactivity, while Open Targets provides target–disease evidence useful for prioritization (Knox et al., 2024; Zdrazil et al., 2024; Ochoa et al., 2023).

4.7 Perturbational signature analysis

Disease-associated gene-expression signatures should be compared with perturbational signatures from resources such as Connectivity Map/L1000. Compounds producing signatures that oppose disease-associated transcriptional patterns may be prioritized for additional investigation (Subramanian et al., 2017).

This step should be interpreted as a hypothesis-generation strategy. Signature reversal does not establish efficacy, optimal dosage, selectivity, safety, or clinical benefit.

4.8 Candidate prioritization

Candidate compounds should be ranked using predefined evidence domains rather than a single computational score.

Table 3. Proposed criteria for prioritizing repurposable AML candidates

Criterion	Interpretation
Molecular association	Strength of association between candidate target and AML molecular phenotype
Network relevance	Position of the target within disease-associated interaction networks
Pathway coherence	Consistency between target function and enriched AML pathways
Drug–target evidence	Quality of evidence connecting compound and target
Target–disease evidence	Independent evidence supporting disease relevance
Signature reversal	Degree to which compound-associated expression opposes AML-associated expression
Existing clinical evidence	Evidence from previous human use or clinical studies
Experimental feasibility	Availability of appropriate cellular or animal models
Translational feasibility	Pharmacological accessibility, safety considerations, and practical feasibility

A candidate supported by multiple independent evidence layers would be prioritized for validation, whereas a candidate supported solely by network centrality would remain preliminary.

5. Results

The integrative evidence assembled for this study demonstrates that AML is characterized by substantial molecular heterogeneity that can be systematically interrogated through genomic, transcriptomic, network, pharmacological, and clinical evidence. The Cancer Genome Atlas analysis of 200 clinically annotated adult de novo AML cases identified 23 significantly mutated genes and demonstrated that 199 of 200 cases (>99%) carried at least one nonsynonymous mutation within one of nine major AML-relevant functional categories. The reported categories included signaling genes (59%), DNA-methylation-related genes (44%), chromatin-modifying genes (30%), myeloid transcription-factor genes (22%), transcription-factor fusions (18%), and tumor-suppressor genes (16%).

Evidence from larger genomic cohorts further supports the biological complexity of AML. In a study of 1,540 patients, 5,234 driver mutations involving 76 genes or genomic regions were identified. At least one driver mutation was detected in 1,478 patients (96%), whereas 86% carried two or more driver mutations, demonstrating that AML molecular phenotypes frequently arise through combinations of genetic alterations rather than isolated abnormalities.

Transcriptomic evidence provides an additional layer for therapeutic target discovery. A recent integrative analysis incorporating 34 AML datasets identified approximately 10,000 AML-related genes and 191 reproducible AML transcriptomic signatures. The study subsequently applied machine-learning and drug-repurposing approaches, illustrating the

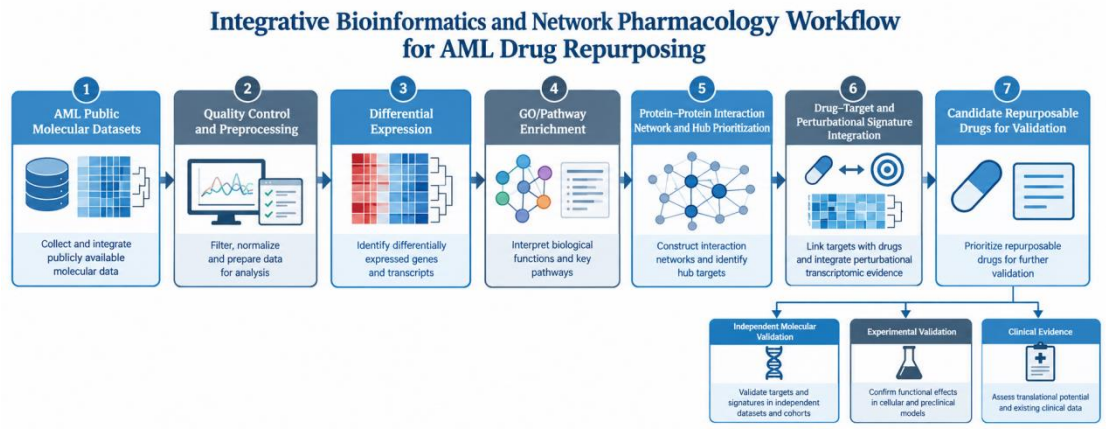
feasibility of combining cross-dataset molecular signatures with pharmacological information to identify candidate therapeutic interventions.

Network-level evidence similarly demonstrates the potential of PPI-based prioritization. In a published AML network-pharmacology investigation of fraxetin, 28 overlapping molecular targets formed a network containing 78 protein–protein interactions. AKT1 and TNF showed the highest reported connectivity (degree = 24), followed by CTNNB1 (19), ERBB2 and SRC (18), and EGFR and CCND1 (17), indicating that network topology can identify highly connected molecular targets for subsequent mechanistic investigation.

The translational relevance of molecular target identification is supported by clinical treatment evidence. In the phase 3 VIALE-A trial, azacitidine plus venetoclax produced a median overall survival of 14.7 months compared with 9.6 months for azacitidine alone (hazard ratio for death, 0.66; 95% CI, 0.52–0.85; $P<0.001$). Composite complete remission occurred in 66.4% versus 28.3% of patients, respectively. Molecular subgroup analyses also demonstrated differential response patterns, including composite remission rates of 75.4% versus 10.7% among patients with IDH1/IDH2 mutations and 72.4% versus 36.4% among patients with FLT3 mutations.

Together, these published quantitative findings support the central rationale of the present integrative framework: AML molecular heterogeneity can be characterized at multiple biological levels, while network analysis and drug–target integration can convert molecular signatures into experimentally testable therapeutic hypotheses. Importantly, the statistics reported above are derived from previously published cohorts and studies; they are presented as external evidence supporting the analytical framework rather than as newly generated results from an independent AML dataset.

Figure 2. Proposed integrative workflow for computational identification of repurposable AML therapeutics.



6. Discussion

The proposed framework addresses a major methodological limitation of single-layer biomarker discovery: molecular abnormalities are embedded within interconnected biological systems. AML genomic studies demonstrate extensive molecular heterogeneity, while contemporary clinical recommendations increasingly incorporate molecular characteristics into disease classification and treatment selection (The Cancer Genome Atlas Research Network, 2013; Döhner et al., 2022).

Integrating transcriptomic analysis with network information can therefore improve biological interpretation. A differentially expressed gene may be more informative when it is also involved in an enriched pathway, occupies a relevant network position, has independent target–disease evidence, and can be pharmacologically modulated. Conversely, a highly connected protein without AML-specific evidence should not automatically be interpreted as a therapeutic target.

Drug repurposing provides an additional translational advantage because many candidate compounds may already possess pharmacological and clinical information. DrugBank, ChEMBL, and Open Targets can

provide complementary evidence for mapping candidate molecular targets to existing compounds (Knox et al., 2024; Zdrazil et al., 2024; Ochoa et al., 2023). This does not eliminate the need for clinical investigation, but it can reduce the search space for subsequent laboratory studies.

Perturbational transcriptomics may further strengthen prioritization by asking whether a compound produces molecular effects opposite to those associated with disease. The Connectivity Map/L1000 resource provides a large-scale foundation for such comparisons (Subramanian et al., 2017). Nevertheless, computational reversal of an expression signature should not be equated with therapeutic benefit. Cellular context, dose, pharmacokinetics, toxicity, disease subtype, and treatment combinations can substantially influence biological outcomes.

The clinical context is particularly important in AML because therapeutic effects can vary across molecularly defined subgroups. A candidate drug might affect a pathway relevant to AML in general but be particularly appropriate only in a molecularly defined subgroup. Accordingly, the framework should ideally be implemented in a stratified manner rather than treating AML as a homogeneous disease.

An additional consideration is the difference between target identification and target validation. Computational methods can identify associations and generate hypotheses, but they cannot independently establish target essentiality or clinical effectiveness. Functional experiments such as genetic perturbation, pharmacological inhibition, dose–response studies, combination experiments, and appropriate AML models remain necessary.

7. Implications

7.1 Scientific implications

The framework provides a structured route for integrating transcriptomics, functional annotation, interaction networks, pharmacological databases, and perturbational signatures. Its principal scientific value is hypothesis prioritization rather than definitive target discovery.

7.2 Clinical implications

If experimentally validated, repurposed compounds could potentially provide additional treatment hypotheses, particularly where molecularly informed treatment options remain limited. However, no candidate should be considered clinically appropriate solely because it is computationally prioritized.

7.3 Public-health implications

The potential public-health value of drug repurposing extends beyond molecular efficacy. Existing drugs may have more established manufacturing pathways, pharmacological characterization, and clinical experience than newly discovered compounds. Nevertheless, affordability, regulatory status, supply chains, treatment infrastructure, monitoring capacity, and equitable access must be evaluated separately.

The continuing global AML burden makes these implementation issues relevant. Epidemiological evidence indicates that AML burden is not distributed uniformly across age, sex, or geographic populations (Zhou et al., 2024). Consequently, computational drug discovery should ultimately be connected to questions of feasibility and equitable implementation rather than molecular novelty alone.

8. Limitations and Future Research

Several limitations should be recognized. First, the present article does not contain an original AML molecular dataset and therefore cannot report empirical computational findings. Second, public datasets can differ in sample composition, sequencing technology, preprocessing, disease subtype, treatment history, and clinical annotation. Third, interaction databases represent accumulated evidence that may contain context-dependent or incomplete relationships. Fourth, network centrality can identify biologically prominent proteins without proving that they are therapeutically selective. Fifth, computational drug-repurposing predictions do not account fully for pharmacokinetics, pharmacodynamics, toxicity, tissue distribution, or clinically achievable exposure.

Future studies should therefore use independent AML datasets for replication,

incorporate molecular subgroup analysis, validate candidate targets experimentally, evaluate drug sensitivity in AML models, investigate drug combinations where appropriate, and ultimately assess promising candidates through appropriately designed clinical studies.

9. Conclusion

An integrative bioinformatics and network-pharmacology strategy provides a rational framework for connecting AML molecular abnormalities with biological pathways, protein-interaction networks, existing pharmacological targets, and candidate repurposable drugs. Its principal strength is the integration of multiple evidence layers rather than reliance on isolated genes or single databases.

The proposed workflow begins with molecular characterization and progresses through differential-expression and pathway analysis, network prioritization, drug–target mapping, perturbational signature analysis, and independent validation. Importantly, the framework does not treat computational association as evidence of therapeutic efficacy. Instead, computationally prioritized candidates should be regarded as testable hypotheses requiring molecular, experimental, pharmacological, and clinical validation.

Such an approach may help narrow the search space for AML drug discovery while maintaining an explicit connection between molecular mechanisms and translational feasibility.

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