SINGULA · ACUTE MYELOID LEUKEMIA

Singula

Standard-of-Care Therapy Response Index — Per Patient.

PATIENT ID	INDICATION	AGE / SEX	REPORT DATE
CW-2026-001847	AML, IDH2-mutant	67 / M	Jun 18, 2026

TOP RECOMMENDATION

Enasidenib + Azacitidine

Highest predicted Overall Survival (TRI 0–100). Mechanism-aligned to IDH2 driver biology.

84

TRI / 100

WHY

IDH2 hotspot R140Q + DNMT3A loss · enasidenib reverses 2-HG accumulation; azacitidine restores epigenetic reprogramming. Synergy modeled in the CBM at 4.4× ORR uplift.

STANDARD-OF-CARE THERAPIES, RANKED BY TRI

Per-patient prediction across the AML NCCN formulary. TRI integrates predicted OS, PFS, and ORR.

#	Regimen	Mechanism focus	Pred. OS	Pred. ORR	TRI
1	Enasidenib + Azacitidine	IDH2 inhibition + HMA	↑↑	62%	**84**
2	Venetoclax + Azacitidine	BCL-2 + HMA	↑↑	55%	**76**
3	Enasidenib (mono)	IDH2 inhibition	↑	42%	**68**
4	Azacitidine (mono)	HMA	↑	28%	**54**
5	FLAG-IDA induction	Intensive cytotoxic	–	38%	**47**
6	7+3 induction	Standard cytotoxic	–	30%	**39**
7	Gilteritinib	FLT3 inhibition (no FLT3)	↓	9%	**16**

INTERPRETATION

Top 2 regimens both exceed TRI 70 and address the dominant driver biology.
Top regimen TRI gap = 8 points. Consider patient fitness, comorbidity, and access.

PATIENT MOLECULAR PROFILE — DRIVER LANDSCAPE

Mutations, CNVs and pathway impact extracted from the patient sample and modeled in the CBM.

Gene	Variant	VAF	Pathway impact	Therapeutic relevance
IDH2	R140Q	42%	Krebs / α-KG depletion	Driver · enasidenib-sensitive
DNMT3A	R882H	38%	DNA methylation loss	Co-driver · HMA-sensitive
NPM1	W288fs	44%	Nucleolar export defect	Co-driver · favorable risk modifier
TET2	Q1548*	31%	5-hmC depletion	Co-driver · HMA-sensitive
FLT3	WT	—	—	FLT3-i not indicated
TP53	WT	—	—	p53-restoring not required

PATHWAY IMPACT — TOP 3

Epigenetic dysregulation ↑↑↑ · driver via IDH2 + DNMT3A + TET2

Hematopoietic differentiation block ↑↑ · downstream of IDH2 R140Q

BCL-2 / apoptosis ↑ · secondary survival pathway

VALIDATION

90% accuracy

prospective AML/MDS therapy prediction · iCare 1 · p<0.0001

METHODS

Mechanistic biosimulation on the Cellworks Computational Biology Model (CBM):

· 6,000+ genes · 600,000+ molecular interactions · 125 pathways encoded

· Patient-specific tumor model parameterized from genomic + transcriptomic input

· Therapy Response Index (TRI 0–100) integrates predicted OS, PFS, and ORR

Reference data: TCGA · GTEx · 45,000+ peer-reviewed publications · 100K+ patient histories

Delivered as a CLIA-certified service from the Cellworks lab.

DISCLAIMER

This is an example report demonstrating Cellworks Singula deliverable format.
Actual patient reports are derived from real molecular data and reviewed by ordering physicians.
Predictions are decision support — they do not replace clinical judgment, FDA-approved labels, or NCCN guidelines. Sources cited inline within the full deliverable.