Leukemia insights

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In this month's Leukemia Insights newsletter, written by Ghayas Issa, M.D., and sponsored in part by the Charif Souki Cancer Research Fund, we discuss Menin Inhibitors: Monotherapy and Combination Studies at MD Anderson Cancer Center. Learn more about our Leukemia program.

Menin Inhibitors: Monotherapy and Combination Studies
A New Class of Targeted Therapy for Acute Leukemia

Introduction

Menin inhibitors are novel targeted agents currently in clinical development for the treatment of genetically defined subsets of acute leukemia. Menin has a tumor suppressor function in endocrine glands. Germline mutations in the gene encoding menin cause the multiple endocrine neoplasia type 1 (MEN1) syndrome, a hereditary condition associated with tumors of the endocrine glands. However, menin is also critical for leukemogenesis in subsets driven by rearrangement of the *lysine methyltransferase 2A* (*KMT2A*) gene, previously known as *mixed-lineage leukemia* (*MLL*), which encodes an epigenetic modifier. These seemingly opposing functions of menin can be explained by its various roles in gene regulation.

Leukemias with rearrangements of *KMT2A (KMT2Ar)* are susceptible to menin inhibition. These leukemias affect infants, children and adults, and lead to adverse outcomes with current standard therapies. Recent studies have identified novel targets in acute leukemia that are susceptible to menin inhibition, such as mutated *nucleophosmin 1* (*NPM1mt*), t he most common genetic alteration in adult acute myeloid leukemia (AML). In addition, other leukemia subsets with similar transcriptional dependency could be targeted. This led to rationally designed clinical studies investigating small-molecule oral menin inhibitors in relapsed acute leukemias with promising early results.

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Leukemia with KMT2Ar

Acute leukemia with *KMT2Ar* have been recognized for decades as disease-defining abnormalities associated with an adverse prognosis. They are driven by an oncogenic fusion of the *KMT2A* gene, located on 11q23, with more than 80 different partners. These rearrangements occur in 5%-10% of acute leukemias and are the most common cause of infant leukemia (70%-80%). Fusion partners likely influence the leukemia phenotype, where t(9;11) (p21;q23) or *KMT2A-MLLT3* (also known as *AF9*) is most common in AML, whereas t(4;11) (q21;q23) or *KMT2A-MLLT2* (also known as *AFF1* or *AF4*) is most common in acute lymphoblastic leukemia (ALL). *KMT2Ar* are detected in up to 70% of therapy-related AML following treatment with a topoisomerase II inhibitor, with a typical latency to clinical presentation from 6 months to 2 years.

KMT2Ar leukemias are associated with resistance to standard therapies and higher rates of relapse. They are frequently characterized by hyperleukocytosis, hepatosplenomegaly, and CNS involvement. *KMT2Ar* can be detected by conventional cytogenetics where translocations involving 11q23 are seen or by fluorescence in situ hybridization (FISH), with some assays developed for detection of rearrangements regardless of the fusion partner (split-signal FISH concept).

KMT2Ar leukemias have a unique, highly distinct gene expression profile characterized by overexpression of the *HOX* genes along with their co-factor *MEIS1*. The menin binding site is preserved throughout all KMT2A fusion proteins and is an essential co-factor for binding to *HOX* gene promoters. In mouse models of *KMT2Ar* leukemia, genetic ablation of menin reversed aberrant *HOX* gene expression, leading to abrogation of the differentiation arrest and the oncogenic properties of KMT2A.

NPM1mt mutated AML

NPM1mt mutations are the most common genetic alterations in adult AML, detected in 20%-30% of cases at diagnosis. These mutations are considered leukemia-initiating. They consist of 4 base-pair frameshift insertions or duplications in exon 12, leading to truncation of the protein and disruption of the nuclear shuttling of NPM1. Therefore, mutated *NPM1mt* persists in the cytoplasm, which explains why it is exclusively cytoplasmic when mutated. Mutated *NPM1mt*[W2] [IC3] is associated with upregulation of *HOX* genes, specifically *HOXA* and *MEIS1*. The similarity in gene expression profiles between *NPM1c* and *KMT2Ar* led to the hypothesis that menin is implicated in this aberrant transcription, and that targeting menin could be a therapeutic strategy in AML with mutated *NPM1mt*. Genetic editing studies confirmed dependency of mutated NPM1[W4] [IC5] on menin and MEIS1[W6] [IC7] to exert a leukemogenic function.

Other Leukemia Subtypes Susceptible to Menin Inhibition

The current prevailing model of dependency on menin in acute leukemias is linked to overexpression of *HOX* genes their co-factor *MEIS1*, therefore this gene expression profile can be used as a biomarker of response to menin inhibition. However, given the current lack of validated assays, leukemia genotypes previously shown to have this gene expression signature could be used as surrogate markers, and assessed for response. This expression signature is also shared by other genotypes or recurrent cytogenetic abnormalities in AML in addition to mutated *NPM1mt* and *KMT2Ar* (Table 1). To highlight one example, leukemias with rearrangements involving the *nuclear pore complexes 98* (*NUP98*), which are rare but associated with an adverse prognosis, have overexpression of *HOXA9* in preclinical models and patient samples, with preclinical data demonstrating susceptibility to menin inhibition.

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Table 1: Genetic alterations with overexpression of HOXA genes predicted to potentially respond to menin inhibitors (Adapted from Issa et al. Leukemia 2021).[1]

Alteration/Mutation	Cytogenetics	Phenotype				References
KMT2Ar	11q23 rearrangements	AML, ALL, MPAL	✓	✓	✓	2-4
KMT2A-PTD	Normal karyotype	AML	✓	✓		2,5
NPM1c	Normal karyotype	AML	✓	✓	✓	2,6
NPM1-MLF1	t(3;5)(q25;q34)	MDS, AML	✓			7,8
NUP98r	11p15 rearrangements	AML, T-ALL, MDS	✓	✓	✓	9-11
SET-NUP214	t(9;9)(q34;q34)	AML, T-ALL, AUL	✓		✓	12
RUNX1-EVI1	t(3;21)(q26;q22)	AML	✓		✓	13
MYST3-CREBBP	t(8;16)(p11;p13)	AML	✓			14
CDX2-ETV6	t(12;13)(p13;q12)	AML		✓		15
CALM-AF10	t(10;11)(p13;q14-21)	T-ALL, AML, MPAL	✓	✓	✓	16-18
MN1-ETV6	t(12;22)(p13;q12)	AML, MDS		✓	✓	19
EZH2	-	MDS, AML	✓			20
IDH1/IDH2	-	MNs			✓	21,22
ASXL1	-	MNs		✓		23
CEBPA	-	AML			✓	24
	Trisomy 8	MNs	✓			25

denotes direct examination of patient samples with the corresponding genotype showing upregulation of *HOXA* genes.

denotes mouse models of the corresponding genotype leading to upregulation of *Hox* genes.

denotes examination of cells lines or other *in vitro* investigations demonstration a role of *HOX* genes or menin inhibition in the corresponding genotype.

AML, acute myeloid leukemia; ALL, acute lymphoblastic leukemia, MPAL, mixed-phenotype acute leukemia; MDS, myelodysplastic syndrome; AUL, acute leukemia of undifferentiated lineage.

Clinical Results with Menin Inhibitors

Identification of the KMT2A binding pocket on menin led to development of potent small-molecule, orally available inhibitors of the menin-KMT2A interaction. Therefore, menin inhibitors disrupt binding of KMT2A to menin, an essential co-factor necessary for binding of the KMT2A complex to promoters of target genes. Pharmacologic inhibition of menin-KMT2A proved to be an effective antileukemic strategy in preclinical models of susceptible leukemias without affecting normal hematopoiesis by downregulating the aberrant gene expression profile. This led to release of the differentiation block in these leukemias, with a pronounced increase in markers of myeloid differentiation and apoptosis following treatment.

Given the strong preclinical rationale justifying use of menin inhibitors as a novel class of targeted therapy in acute leukemias, multiple clinical trials with these agents have been started with positive early results. Results of the phase 1 clinical trial of the menin inhibitor revumenib (previously SNDX-5613) have been published in Nature. In this phase 1, multicenter, open-label, dose-escalation study, revumenib was shown to be safe and with promising preliminary efficacy in patients with relapsed/refractory *KMT2Ar* or *NPM1mt* leukemia[W8] [IC9] (Issa GC Nature 2023)[26]. Revumenib was administered orally either in capsule or liquid formulations every 12 hours in 28-day continuous cycles. Patients were enrolled in two parallel arms using a rolling-6 design (an algorithm-based extension of the 3+3 design) to determine the recommended phase 2 dose, with and without concomitant CYP3A4 inhibitors (i.e. antifungal prophylaxis). A total of 37 and 31 patients were enrolled in Arm A (without a concomitant strong CYP3A4 inhibitor) and Arm B (with a concomitant strong CYP3A4 inhibitor), respectively. Fifty-six patients (82%) had AML, 11 (16%) had ALL, and one (2%) had mixed-phenotype acute leukemia. Forty-six patients (68%) had *KMT2Ar* and 14 (21%) had *NPM1mt*.

Sixty patients were adults (age 18 or older) and eight were children or adolescents (< age 18). The median age overall was 42.5 years (range 0.8-79). In the pediatric population, the median age was 2.5 years (range, 0.8-16). In patients with *KMT2Ar* or *NPM1mt* R/R acute leukemia, revumenib led to overall response rates (ORR) of 59% (27/46 patients) and 36% (5/14 patients) in patients with relapsed or refractory *KMT2A*r and *NPM1mt* leukemias, respectively. In the *KMT2Ar* population, 9 (20%) had a complete remission (CR), 6 (13%) had a complete remission with partial hematologic recovery (CRh), 5 (11%) had a complete remission with incomplete platelet recovery (CRp), and 7 (15%) had a morphologic leukemia-free state (MLFS). The MRD-negative rate in patients who attained a CR or CRh was 78% (14/18 patients). Of those with *KMT2Ar* who achieved a morphologic clearance of their myeloblasts, the rate of complete cytogenetic response was 64% (16 of 25 patients). In the *NPM1mt* AML population, 3 (21%) had a CR and 2 (14%) had MLFS. In the pediatric population, morphologic remission was noted in four of eight patients (50%; 95% CI 15.7-84.3).

Results of the KOMET-001 trial (NCT04067336) were presented at the Late Breaking Session of the 2023 European Hematology Association meeting showing clinical activity of the oral menin inhibitor ziftomenib (previously KO-539). As of the data cutoff on April 12, 2023, seven of the 20 patients (35%) with *NPM1*-mutant AML[W10] [IC11] treated at the recommended phase 2 dose (RP2D) of 600 mg achieved a CR with count recovery.

Clinical Trials Investigating Menin Inhibition at MD Anderson

Given the emerging importance of this new class of drugs and the unmet need for patients with these resistant leukemias, there are currently multiple ongoing clinical trials at MD Anderson with menin inhibitors, as single agents or in combination.

Below are the menin inhibitors and the latest stage in clinical development:

- Revumenib (previously SNDX-5613) (Syndax), phase II
- Ziftomenib (previously KO-539) (Kura), phase II
- JNJ-75276617 (Janssen), phase 1
- BMF-219 (Biomea Fusion), phase 1
- DSP-5336 (Sumitomo), phase 1

A. Menin Inhibition Monotherapy

- Revumenib (SNDX-5613) (AUGMENT-101, NCT04065399), Phase II, Age > 30d, in R/R AML, ALL, MPAL, *KMT2Ar* or *NPM1mt*
- Revumenib (SNDX-5613) (NCT05406817), ADME study (Absorption, Distribution, Metabolism and Excretion), broader eligibility including CNS disease, MRD disease only or extramedullary disease, R/R leukemia of any genotype
- Ziftomenib (KO-539) in R/R AML, phase II, Age > 18y, AML, *NPM1mt*
- JNJ-75276617 (NCT04811560), phase I, Age > 18y, in R/R AML, ALL, MPAL, *KMT2Ar* or *NPM1mt*
- DSP-5336 (NCT04988555), phase 1, Age > 18y, in R/R AML, ALL, *KMT2Ar* or *NPM1mt*
- BMF-219 (NCT05153330), phase 1, Age >18y, in R/R AML, ALL, *KMT2Ar* or *NPM1mt*

B. Menin Inhibition in Combination

- **S**NDX-5613 (revumenib)**,** oral decitabine (**A**STX727) and **V**enetoclax **(SAVE) (**NCT05360160**),** phase I/II, Age >12y, R/R AML or myeloid MPAL, *KMT2Ar* or *NPM1mt or NUP98r*
- SNDX-5613 (revumenib) with fludarabine and cytarabine (AUGMENT-102, NCT05326516), Phase I, Age > 30d, in R/R AML, ALL, MPAL, *KMT2Ar* or *NPM1mt or NUP98r*
- JNJ-72576617 with venetoclax or azacitidine or azacitidine and venetoclax, Phase Ib, R/R AML *KMT2Ar* or *NPM1mt*
- KO-539 (Ziftomenib) with venetoclax/azacitidine or venetoclax or standard induction cytarabine/daunorubicin (7+3), newly diagnosed on R/R AML with *KMT2Ar* or *NPM1mt.*

C. Menin Inhibition for Targeting MRD

There are two large ongoing efforts to characterize and target MRD in AML, the Break Through Cancer project and the INTERCEPT trial [Investigating Novel Therapy to Target Early Relapse and Clonal Evolution as Pre-emptive Therapy in AML]. Both efforts seek to improve our understanding of the mechanism underlying MRD, specific biology and characteristics with the goals of improving detection and eradication of MRD. There are two trials investigating menin inhibition for MRD eradication in patients with morphologic remission about to open:

- Break Through Cancer: SNDX-5613 (revumenib) with venetoclax for MRD+ only, AML with *KMT2Ar* or *NPM1mt* or *NUP98r,* Age > 12y
- INTERCEPT: SNDX-5613 (revumenib) for MRD+ only, AML with *KMT2Ar* or *NPM1mt,* Age >18y

D. Menin Inhibition for Leukemia Associated with Upregulation of *HOX* Genes

The pivotal phase 2 clinical trial evaluating the efficacy of revumenib in *KMT2A*-rearranged or *NPM1*-mutant leukemia is ongoing (AUGMENT-101 above). However, in addition to these alterations, other leukemia subsets with a similar transcriptional dependency could be targeted through menin inhibition. In this clinical trial, “A Phase II Study of the Menin Inhibitor Revumenib in Leukemia Associated with Upregulation of *HOX* Genes” we are testing the efficacy of revumenib in various leukemia subsets associated with *HOX* gene upregulation and this trial is about to launch.

Patients, age 12 and older with any leukemia associated with the genetic alterations below would be eligible:

Alteration/Mutation	Cytogenetics
KMT2A-PTD	Normal karyotype
NPM1-MLF1	t(3;5)(q25;q34)
NUP98r	11p15 rearrangements
SET-NUP214	t(9;9)(q34;q34)
RUNX1-EVI1	t(3;21)(q26;q22)
MYST3-CREBBP	t(8;16)(p11;p13)
CDX2-ETV6	t(12;13)(p13;q12)
CALM-AF10	t(10;11)(p13;q14-21)
MN1-ETV6	t(12;22)(p13;q12)

Summary

Extraordinary efforts by numerous scientists over the years allowed menin inhibitors to reach investigation in clinic. Though this is only the beginning of clinical investigations of these molecules, early results are highly encouraging. This new class of drugs has allowed remission in patients with highly resistant leukemias with no other available or effective therapy.

The immediate next steps that follow investigation of safety and efficacy of menin inhibitors are optimal combination strategies with other effective agents to treat various subtypes of acute leukemia, getting us closer to what ultimately matters, curing more patients with acute leukemia.

1. Issa GC, Ravandi F, DiNardo CD, Jabbour E, Kantarjian HM, Andreeff M. Therapeutic implications of menin inhibition in acute leukemias. Leukemia 2021.
2. Spencer DH, Young MA, Lamprecht TL, et al. Epigenomic analysis of the HOX gene loci reveals mechanisms that may control canonical expression patterns in AML and normal hematopoietic cells. Leukemia 2015;29:1279-89.
3. Kawagoe H, Humphries RK, Blair A, Sutherland HJ, Hogge DE. Expression of HOX genes, HOX cofactors, and MLL in phenotypically and functionally defined subpopulations of leukemic and normal human hematopoietic cells. Leukemia 1999;13:687-98.
4. Ferrando AA, Armstrong SA, Neuberg DS, et al. Gene expression signatures in MLL-rearranged T-lineage and B-precursor acute leukemias: dominance of HOX dysregulation. Blood 2003;102:262-8.
5. Zorko NA, Bernot KM, Whitman SP, et al. Mll partial tandem duplication and Flt3 internal tandem duplication in a double knock-in mouse recapitulates features of counterpart human acute myeloid leukemias. Blood 2012;120:1130-6.
6. Alcalay M, Tiacci E, Bergomas R, et al. Acute myeloid leukemia bearing cytoplasmic nucleophosmin (NPMc+ AML) shows a distinct gene expression profile characterized by up-regulation of genes involved in stem-cell maintenance. Blood 2005;106:899-902.
7. Florent D, Aline R, Caroline M-R, Olivier N, Elise L, Claude P. Acute myeloid leukemia with translocation t(3;5): new molecular insights. Haematologica 2013;98:e52-e4.
8. Lim G, Choi JR, Kim MJ, et al. Detection of t(3;5) and NPM1/MLF1 rearrangement in an elderly patient with acute myeloid leukemia: clinical and laboratory study with review of the literature. Cancer Genet Cytogenet 2010;199:101-9.
9. Gough SM, Slape CI, Aplan PD. NUP98 gene fusions and hematopoietic malignancies: common themes and new biologic insights. Blood 2011;118:6247-57.
10. Borrow J, Shearman AM, Stanton VP, Jr., et al. The t(7;11)(p15;p15) translocation in acute myeloid leukaemia fuses the genes for nucleoporin NUP98 and class I homeoprotein HOXA9. Nature genetics 1996;12:159-67.
11. Wang GG, Cai L, Pasillas MP, Kamps MP. NUP98–NSD1 links H3K36 methylation to Hox-A gene activation and leukaemogenesis. Nature Cell Biology 2007;9:804-12.

12. Van Vlierberghe P, van Grotel M, Tchinda J, et al. The recurrent SET-NUP214 fusion as a new HOXA activation mechanism in pediatric T-cell acute lymphoblastic leukemia. Blood 2008;111:4668-80.
13. Loke J, Assi SA, Imperato MR, et al. RUNX1-ETO and RUNX1-EVI1 Differentially Reprogram the Chromatin Landscape in t(8;21) and t(3;21) AML. Cell reports 2017;19:1654-68.
14. Camós M, Esteve J, Jares P, et al. Gene expression profiling of acute myeloid leukemia with translocation t(8;16)(p11;p13) and MYST3-CREBBP rearrangement reveals a distinctive signature with a specific pattern of HOX gene expression. Cancer research 2006;66:6947-54.
15. Jin G, Yamazaki Y, Takuwa M, et al. Trib1 and Evi1 cooperate with Hoxa and Meis1 in myeloid leukemogenesis. Blood 2007;109:3998-4005.
16. Novak RL, Harper DP, Caudell D, Slape C, Beachy SH, Aplan PD. Gene expression profiling and candidate gene resequencing identifies pathways and mutations important for malignant transformation caused by leukemogenic fusion genes. Experimental hematology 2012;40:1016-27.
17. Dik WA, Brahim W, Braun C, et al. CALM-AF10+ T-ALL expression profiles are characterized by overexpression of HOXA and BMI1 oncogenes. Leukemia 2005;19:1948-57.
18. 1Caudell D, Aplan PD. The role of CALM-AF10 gene fusion in acute leukemia. Leukemia 2008;22:678-85.
19. Libbrecht C, Xie HM, Kingsley MC, et al. Menin is necessary for long term maintenance of meningioma-1 driven leukemia. Leukemia 2021;35:1405-17.
20. Gao L, Sun J, Liu F, Zhang H, Ma Y. Higher expression levels of the HOXA9 gene, closely associated with MLL-PTD and EZH2 mutations, predict inferior outcome in acute myeloid leukemia. OncoTargets and therapy 2016;9:711-22.
21. Ogawara Y, Katsumoto T, Aikawa Y, et al. IDH2 and NPM1 Mutations Cooperate to Activate Hoxa9/Meis1 and Hypoxia Pathways in Acute Myeloid Leukemia. Cancer research 2015;75:2005-16.
22. Chaturvedi A, Araujo Cruz MM, Jyotsana N, et al. Mutant IDH1 promotes leukemogenesis in vivo and can be specifically targeted in human AML. Blood 2013;122:2877-87.
23. Abdel-Wahab O, Adli M, LaFave LM, et al. ASXL1 mutations promote myeloid transformation through loss of PRC2-mediated gene repression. Cancer cell 2012;22:180-93.
24. Schmidt L, Heyes E, Scheiblecker L, et al. CEBPA-mutated leukemia is sensitive to genetic and pharmacological targeting of the MLL1 complex. Leukemia 2019;33:1608-19.
25. Kok CH, Brown AL, Ekert PG, D'Andrea RJ. Gene expression analysis reveals HOX gene upregulation in trisomy 8 AML. Leukemia 2010;24:1239-43.
26. Issa GC, Aldoss I, DiPersio J, et al. The menin inhibitor revumenib in KMT2A-rearranged or NPM1-mutant leukaemia. Nature 2023;615:920-4.

Announcements

SOHO 2023 Annual Meeting-Hybrid

The Eleventh Annual Meeting of the **Society of Hematologic Oncology** (SOHO 2023) is scheduled for **September 6–9, 2023** at the George R. Brown Convention Center in Houston, Texas. As a hybrid event, SOHO 2023 offers in-person and virtual attendance options. Organized by its founders and world class committees, SOHO is the only worldwide society specific to the field of hematologic malignancies. The 2023 meeting promises to be a dynamic and informative event. SOHO is the premier meeting that focuses specifically on new advances and practical clinical applications in the field of hematologic malignancies. The speakers are a multidisciplinary group of internationally recognized experts that represent the spectrum of these diseases. Click here to register.

Leukemia Faculty Contacts

Completing these studies in a timely manner will allow us to move quickly on positive leads. We appreciate referrals and will make every effort, once a patient is enrolled on a study, to continue as much of the care as possible through the referring oncologist. We also will keep you apprised of the patient's progress. View our faculty roster.

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