UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): July 26, 2024

APTOSE BIOSCIENCES INC.
(Exact name of registrant as specified in its charter)

Canada	**001-32001**	**98-1136802**
(State or Other Jurisdiction of Incorporation)	(Commission File Number)	(I.R.S. Employer Identification No.)

66 Wellington Street West, Suite 5300
TD Bank Tower, Box 48
Toronto, Ontario M5K 1E6
Canada
(Address of Principal Executive Offices) (Zip Code)

(647) 479-9828
(Registrant's telephone number, including area code)

251 Consumers Road, Suite 1105
Toronto, Ontario M2J 4R3
Canada
(Former name or former address, if changed since last report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)
☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)
☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))
☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Shares, no par value	APTO	NASDAQ Capital Market

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 9.01. Financial Statements and Exhibits.

99.1	Aptose Corporate Presentation - July 2024
104	Cover Page Interactive Data File (embedded within the Inline XBRL document)

SIGNATURE

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Aptose Biosciences Inc.

Date: July 26, 2024

By: /s/ William G. Rice, Ph.D.
William G. Rice, Ph.D.
Chairman, President, and Chief Executive Officer

Aptose

Precision oncology company developing oral targeted agents to treat hematologic malignancies

Corporate Presentation
July 26, 2024

APTOSE BIOSCIENCES

Tuspetinib to Treat Newly Diagnosed AML

Aptose Disclosure

This presentation does not, and is not intended to, constitute or form part of, and should not be construed as, an offer or invitation for the sale or purchase of, or a solicitation of an offer to purchase, subscribe for or otherwise acquire, any securities, businesses and/or assets of any entity, nor shall it or any part of it be relied upon in connection with or act as any inducement to enter into any contract or commitment or investment decision whatsoever.

This presentation contains forward-looking statements, which reflect APTOSE Biosciences Inc.'s (the "Company") current expectations, estimates and projections regarding future events, including statements relating to our business strategy, our clinical development plans, our ability to obtain the substantial capital we require, our plans to secure strategic partnerships and to build our pipeline, our clinical trials and their projected timelines and milestones, the efficacy and toxicity of our product candidates, potential new intellectual property, our plans, objectives, expectations and intentions; and other statements including words such as "anticipate", "contemplate", "continue", "believe", "plan", "estimate", "expect", "intend", "will", "should", "may", and other similar expressions. Such statements constitute forward-looking statements within the meaning of securities laws.

Although the Company believes that the views reflected in these forward-looking statements are reasonable, such statements involve significant risks and uncertainties, and undue reliance should not be placed on such statements. Certain material factors or assumptions are applied in making these forward-looking statements, and actual results may differ materially from those statements. Those factors and risks include, but are not limited to, our ability to raise the funds necessary to continue our operations, changing market conditions, the successful and timely completion of our clinical studies including delays, the demonstration of safety and efficacy of our drug candidates, our ability to recruit patients, the establishment and maintenance of corporate alliances, the market potential of our product candidates, the impact of competitive products and pricing, new product development, changes in laws and regulations, uncertainties related to the regulatory approval process and other risks detailed from time to time in the Company's ongoing quarterly filings and annual reports.

Forward-looking statements contained in this document represent views only as of the date hereof and are presented for the purpose of assisting potential investors in understanding the Company's business and may not be appropriate for other purposes. The Company does not undertake to update any forward-looking statements, whether written or oral, that may be made from time to time by or on its behalf, except as required under applicable securities legislation. Investors should read the Company's continuous disclosure documents available at EDGAR at www.sec.gov/edgar.shtml and SEDAR+ at www.sedarplus.com, especially the risk factors detailed therein.

APTOSE BIOSCIENCES

Aptose Management Team

William G. Rice, PhD
Chairman, President
& Chief Executive Officer

Fletcher Payne
Sr. VP, Chief Financial Officer
& Chief Business Officer

Rafael Bejar, MD, PhD
Sr. VP & Chief Medical Officer
KOL, Hematologic Malignancies

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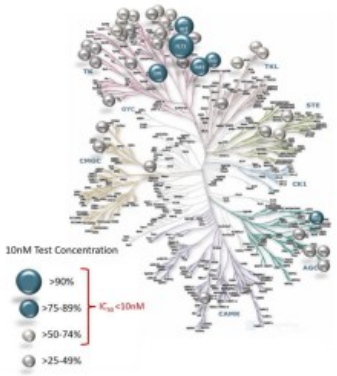
Tuspetinib Lead Clinical Asset

TUS+VEN+HMA triplet is being developed as frontline therapy to treat newly diagnosed AML

Frontline triplet clinical data expected 2H 2024

Potently targets SYK, FLT3, KIT^{MUT}, JAK1/2, RSK2 kinases



TUS : Tuspetinib ; **VEN** : Venetoclax ; **HMA** : Hypomethylating agent
AML : Acute Myeloid Leukemia ; **SOC** : Standard of Care

Unmet Need for Superior Frontline (1L) Therapy in AML

- Progress made with **VEN+HMA (SOC)** but unmet needs still exists
  - **Response rates** too low and **survival** too short in 1L therapy
  - ***Resistance to VEN*** compromises subsequent R/R therapies
- **A 3rd agent** is needed to boost responses with VEN+HMA SOC
- Current 3rd agents inadequate – only address specific genetic subtypes and are limited by toxicities

Tuspetinib Ideal 3rd Agent for Addition to VEN and HMA

- **TUS** has **excellent safety** in combination with VEN and HMA
- **TUS increases efficacy** in combination with VEN and HMA
- **TUS** has **broad scope** of activity across AML genetic subgroups
- **TUS may minimize VEN resistance** by targeting VEN-resistance events

TUS+VEN+HMA

- **Bring new SOC** addressing needs of newly diagnosed AML patients
- **$1Bn+** market potential in frontline AML

Tuspetinib Enhancing Venetoclax Efficacy in Frontline Therapy

TUS and VEN mechanistically cooperate to prevent drug resistance

Tuspetinib Compensates for Venetoclax Resistance

VEN BCL-2i resistance involves mutations in multiple pathways to evade BCL-2 blockade

TUS targets VEN resistance pathways

- TUS suppresses SYK, KIT^{MUT}, FLT3, JAK/STAT, RAS/MAPK pro-survival signaling and indirectly suppresses MCL-1 anti-apoptotic signaling

By shutting down escape pathways, TUS may re-sensitize prior-VEN failures to venetoclax

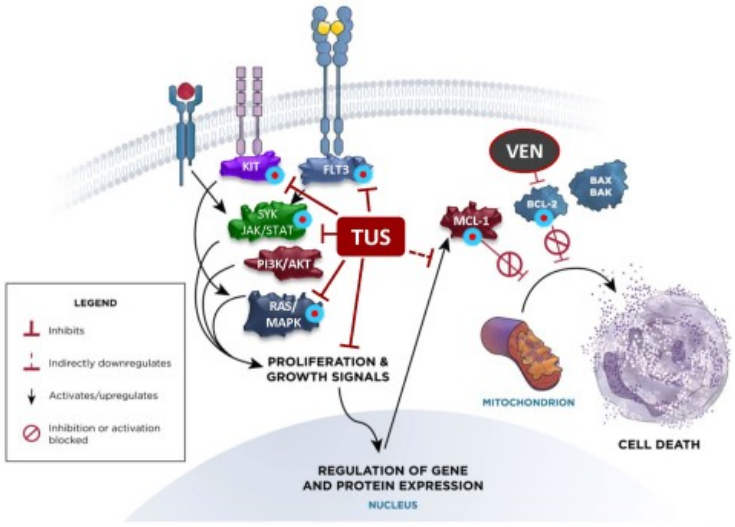
ESH EUROPEAN SCHOOL OF HAEMATOLOGY

Tuspetinib Oral Myeloid Kinase Inhibitor Creates Synthetic Lethal Vulnerability to Venetoclax

American Society of Hematology

Tuspetinib Oral Myeloid Kinase Inhibitor Safety and Efficacy As Monotherapy and Combined with Venetoclax in Phase 1/2 Trial of Patients with Relapsed or Refractory (R/R) Acute Myeloid Leukemia (AML)

RATIONALE FOR THE COMBINATION OF TUSPETINIB AND VENETOCLAX



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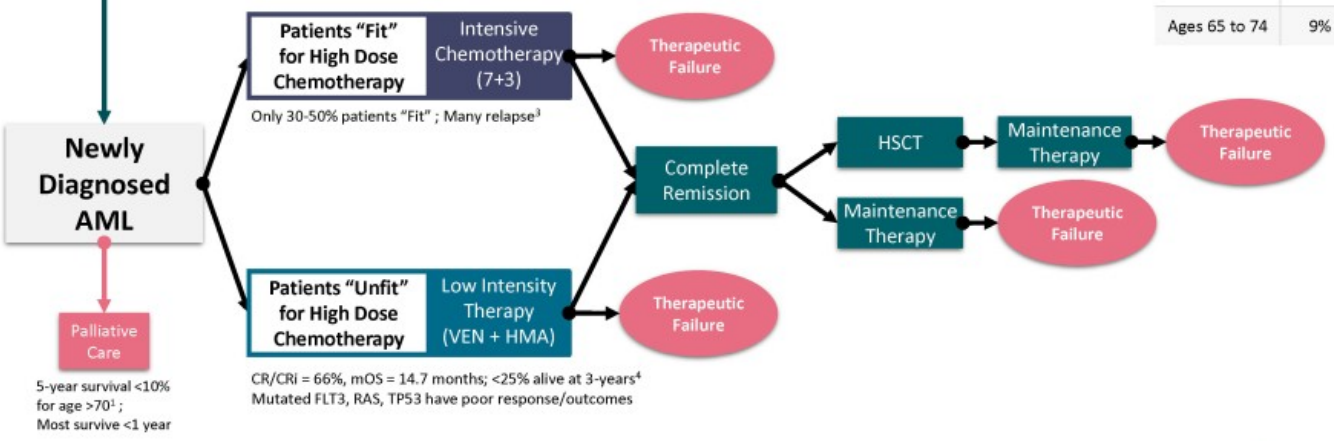
AML Patient Journey | 1L Therapy | Current Standard-of-Care (SOC) lead to therapeutic failure.....

Annual new cases in U.S. ≈ 21,000[2]
Annual deaths in U.S. ≈ 11,200[2] | 5-yr survival ≈ 30% in adults[2]
Roughly equivalent in Europe | Median age at diagnosis 68[2] | 5-yr survival 9% for age >65[2]

Age[2]	5-year survival rate
Children < 14	65-70%
Ages 15 to 34	52%
Ages 35 to 54	37%
Ages 55 to 64	20%
Ages 65 to 74	9%

- Need to Drive more Newly Diagnosed AML Patients into Complete Remissions and Keep Them There
- Want to Prevent from Becoming a VEN Failure R/R Patient



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New Paradigm in Frontline Therapy to Treat Newly Diagnosed AML

Deploying Triplet Combinations of Targeted Drugs | Building on VEN + HMA Backbone for 1L Therapy

Proof for Triplets : Addition of a 3rd Targeted Agent Boosts VEN+HMA Responses in 1L AML

Addition of gilteritinib (Gilt) FLT3i to VEN+HMA boosts CR rate to 90% in newly diagnosed FLT3+ AML patients[1]

So, What's the Problem: Current 3rd Agents for Triplets have Limitations

*Gilt is **not active in FLT3-Wildtype** AML (70% of patients) and **toxicities** of Gilt with VEN+HMA require SOC **dose reductions***

Solution: TUS Fulfills Ideal Profile as 3rd Agent for 1L Triplet

TUS clean safety is ideal for addition to VEN+HMA backbone

- TUS shows no QTc prolongation, muscle damage, differentiation syndrome, or prolonged myelosuppression in remission
- TUS is not expected to require dose reductions or interruptions to SOC drugs

TUS clinical efficacy broader than Gilt and achieves CR in high-risk AML

- TUS achieves clinical responses in patients who failed prior therapy with Gilt
- TUS achieves clinical responses at lower and better-tolerated doses than Gilt
- TUS achieves clinical responses in $FLT3^{WT}$ patients (70% of AML population), a population not addressable by Gilt FLT3i

TUS preclinical safety, antitumor, mechanistic findings superior to Gilt

- TUS MOA targets VEN-resistance mechanisms and re-sensitizes cells to VEN
- TUS suppresses more oncogenic signaling pathways than Gilt and at lower doses
- TUS potent antitumor activity in animal models of human AML resistant to Gilt
- TUS+VEN & TUS+HMA safe and effective in animal models of human AML

1 Short et al. J Clin Oncol. 2024 Jan 26:JCO2301911. Epub ahead of print. PMID: 38277619.

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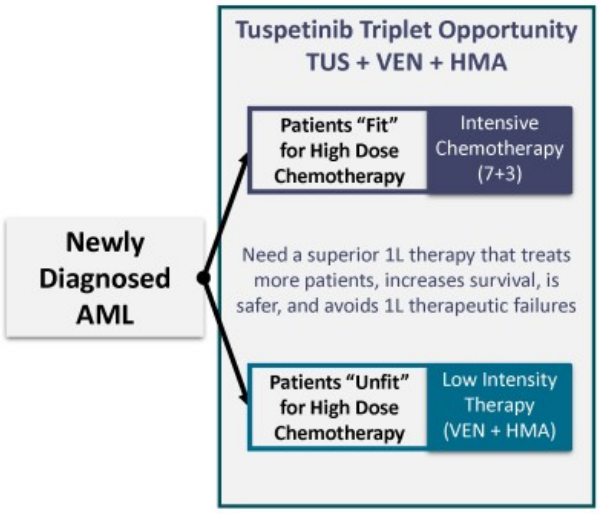
AML Patient Journey | 1L Therapy High-Level Overview

Tuspetinib-containing triplet can become a new 1L SOC to increase survival



Tuspetinib Frontline Triplet Opportunities

- TUS has broad activity across AML genetic subgroups: Including those with TP53, RAS/MAPK, FLT3 mutations
- TUS is the only agent being developed in combination with VEN+HMA for high-risk AML subtypes with highly adverse *TP53 and RAS mutations*
- Potential to increase CR rates and survival of *$FLT3^{MUT}$* patients without the need to dose reduce SOC drugs
- TUS is the only agent being developed in combination with VEN+HMA for *$FLT3^{WT}$* AML patients (70% of AML)
- TUS+VEN+HMA expected to be a *safer* and *broader* therapy for "unfit" patients than any other triplet
- TUS expected to *minimize VEN resistance*

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FDA Requirements for TUS to Enter Frontline Therapy in Newly Diagnosed AML

Tuspetinib has Met the FDA Requirements to Perform the Triplet Pilot Study

What Does the FDA Want? Begin in R/R AML with TUS and TUS+VEN	Aptose Completed
TUS Single Agent Study in R/R AML	
Thorough Single Agent **Dose Exploration**	√
Demonstrate Single Agent **Responses**	√
Demonstrate Single Agent **Safety**	√
Orphan Drug Designation and **Fast Track Status**	√
Tus+Ven Doublet Study in R/R AML	
Characterize **Safety** of TUS+VEN Doublet	√
Characterize **PK** of TUS and VEN in Doublet	√

Next Step: TUS+VEN+AZA Triplet Study

Initiate dosing and collect data from Triplet Pilot Study in Newly Diagnosed AML Patients

- FDA allowed triplet initiation with 40mg TUS (typical one dose level below single agent RP2D) and then escalate
- Protocol implemented and clinical sites being prepared
  - Select optimal dose of TUS that allows for SOC dosing
  - Characterize safety and mitigate myelosuppression
  - Characterize activity in $TP53^{MUT}$ and $N/KRAS^{MUT}$
  - Characterize activity in $FLT3^{MUT}$ and $FLT3^{UNMUT}$
  - Characterize PK of TUS and VEN in triplet
  - Determine CR, CRh, CRc, MRD rates
  - Characterize duration of dosing
  - Characterize mOS

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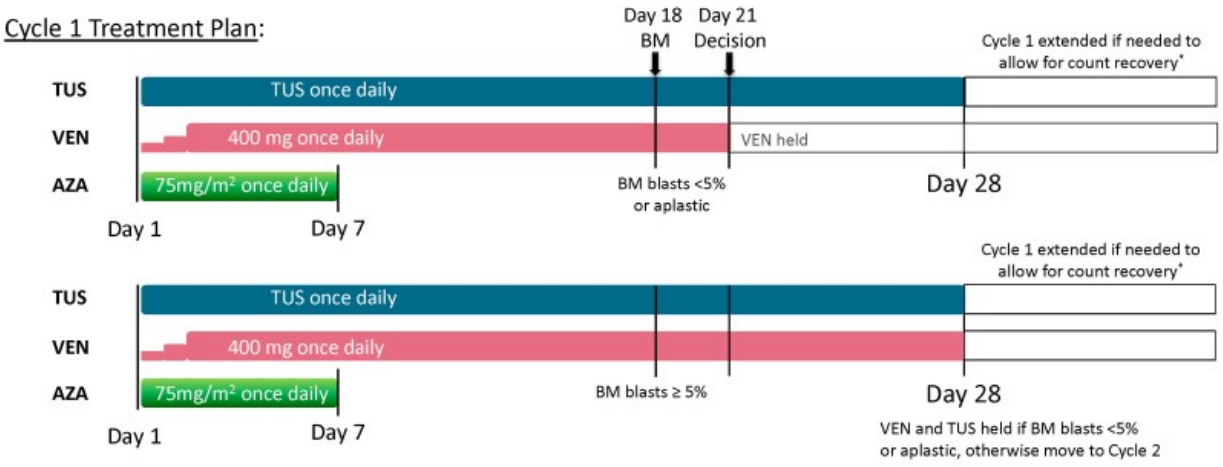
TUS+VEN+AZA TRIPLET Ph 1/2 Study: Design, Patient Populations, Dose Selection, Goals

Patient Populations | Approx. 18-26 Pts Total | 50% FLT3-MUT | <20% TP53+/CK

Trial Goals | Safety, CR rate, MRD negativity and OS across AML subtypes ($FLT3^{MUT/WT}$, $TP53^{MUT}$, RAS^{MUT})

Dose Characterization | Demonstrate TUS is Highly Active and May Avoid SOC Dose Reductions



* GCSF permitted after D28 per protocol

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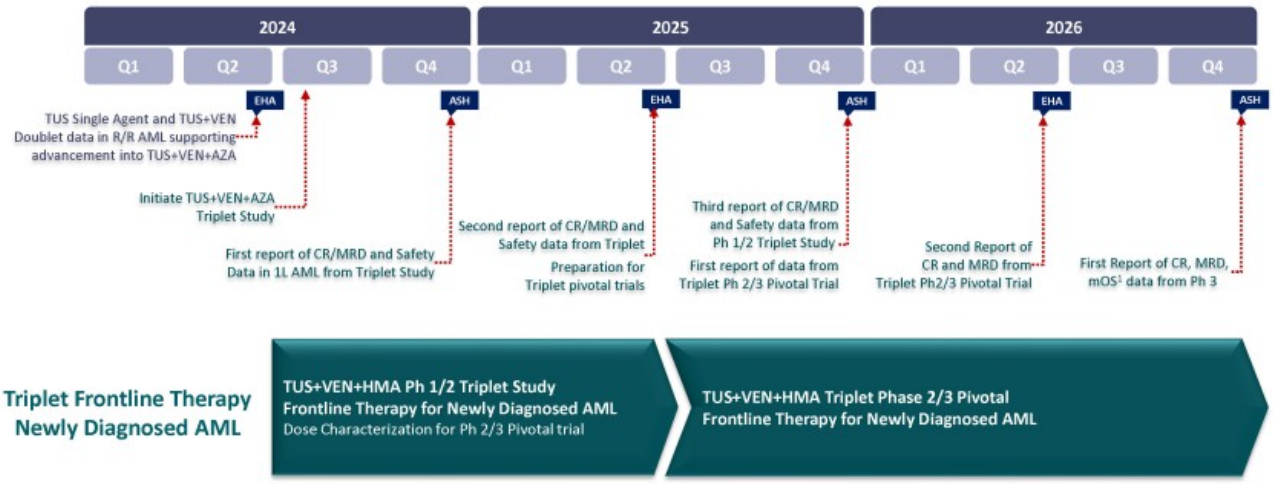
TUS+VEN+HMA Planned Clinical Development Plan, Timelines and Milestones



Triplet Frontline Therapy Newly Diagnosed AML

TUS+VEN+HMA Ph 1/2 Triplet Study
Frontline Therapy for Newly Diagnosed AML
Dose Characterization for Ph 2/3 Pivotal trial

TUS+VEN+HMA Triplet Phase 2/3 Pivotal
Frontline Therapy for Newly Diagnosed AML

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Tuspetinib (TUS) Single Agent and TUS+VEN Doublet Clinical Findings Support TUS+VEN+HMA Triplet

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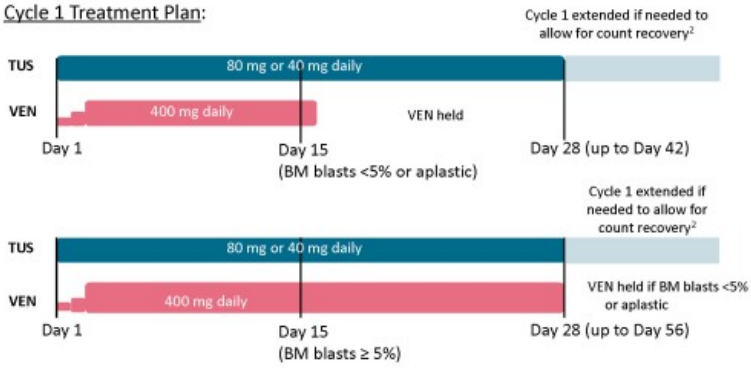
TUS Single Agent and TUS+VEN Doublet Study Designs in R/R AML Patients
Positions TUS for Combination 1L Therapy to Treat Newly Diagnosed AML

TUS Single Agent Dose Escalation and Exploration[1]	Total n=91
Cohort 1: 20 mg QD	2
Cohort 2: 40 mg QD	17
Cohort 3: 80 mg QD	20
Cohort 4: 120 mg QD	32
Cohort 5: 160 mg QD	16
Cohort 6: 200 mg QD	4

Doses with CR & no DLT

TUS+VEN Doublet Study[1]
Extensive dose exploration (TUS 80mg or 40mg + VEN 400mg)
n=**77** dosed | n=65 in 80 mg/400 mg | n=12 in 40 mg/400 mg

Cycle 1 Treatment Plan:



[1] Data cut Feb 09, 2024
[2] GCSF permitted anytime per protocol

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TUS and TUS+VEN : Patient Characteristics of Highly Treatment Experienced R/R AML Patients

TUS Single Agent Study Baseline Patient Characteristics[1]
- **91 patients** dosed as of Feb 09, 2024
- Median **age > 60 years**: Older population
- Over 36% **failed Prior-transplant**
- 50% of FLT3^{MUT} **failed Prior-FLT3i**
- Population included **FLT3^{WT}** and **FLT3^{MUT}**
- Population included 16% **TP53^{MUT}**/CK and 15% **NKRAS^{MUT}**
- Over 56% **failed Prior-VEN** : Correlates with poor outcome

TUS+VEN Doublet Study Baseline Patient Characteristics[1]
- **77 patients** dosed as of Feb 09, 2024
- Median **age > 68 years**: Older than TUS single agent trial
- Over 25% **failed Prior-transplant**
- Over 77% of FLT3^{MUT} **failed Prior-FLT3i**
- Population included **FLT3^{WT}** and **FLT3^{MUT}**
- Population included **30% TP53^{MUT}**/CK and 15% **NKRAS^{MUT}**
- Over 74% **failed Prior-VEN** : Correlates with poor outcome

Patient Characteristics (n=91)	FLT3^{MUT}	FLT3^{WT}
Patient number n (%)[2]	34	56
Median Age Years (Range)	60 (21-84)	65.5 (18-83)
Female n (%)	14 (41.2%)	24 (42.9%)
Lines prior therapy Mean (Range)	3.3 (1-11)	2.4 (1-6)
Prior-VEN	19 (55.9%)	33 (58.9%)
Prior FLT3 Inhibitor	17 (50.0%)	3 (5.4%)
Prior Cytotoxic chemotherapy	26 (76.5%)	36 (64.3%)
Prior HMAs	22 (64.7%)	37 (66.1%)
Prior HSCT	14 (41.2%)	19 (33.9%)

Patient Characteristics (n=77)	FLT3^{MUT}	FLT3^{WT}
Patient number n (%)[3]	18	57
Median Age Years (Range)	68.5 (39-84)	69 (31-86)
Female n (%)	10 (55.6%)	28 (49.1%)
Prior lines of therapy Mean (Range)	2.9 (1-5)	2.2 (1-7)
Prior-VEN	14 (77.8%)	41 (71.9%)
Prior FLT3 Inhibitor	14 (77.8%)	7 (12.3%)
Prior Cytotoxic chemotherapy	10 (55.6%)	29 (50.9%)
Prior HMAs	13 (72.2%)	44 (77.2%)
Prior HSCT	7 (38.9%)	13 (22.8%)

[1] Data cut Feb 09, 2024
[2] Number of patients in the Single Agent Study as of Feb 09, 2024 . One patient had an indeterminant status for FLT3.
[3] Four patients had an indeterminant status for FLT3 as of Feb 09, 2024 in the TUS+VEN Doublet Study.

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TUS and TUS+VEN Safe and Well Tolerated in Highly Treatment Experienced R/R AML

TUS Single Agent Study Excellent Safety and Tolerability

- No drug-related myelosuppression in remission
- No treatment related QTc prolongation or CPK elevations
- No drug-related discontinuations or deaths
- No drug-related non-hematologic SAEs
- No differentiation syndrome

TUS+VEN Doublet Study Excellent Safety and Tolerability

- No new or unexpected safety signals with TUS+VEN
- No drug related AE of QTc prolongation
- No differentiation syndrome observed
- No drug related deaths

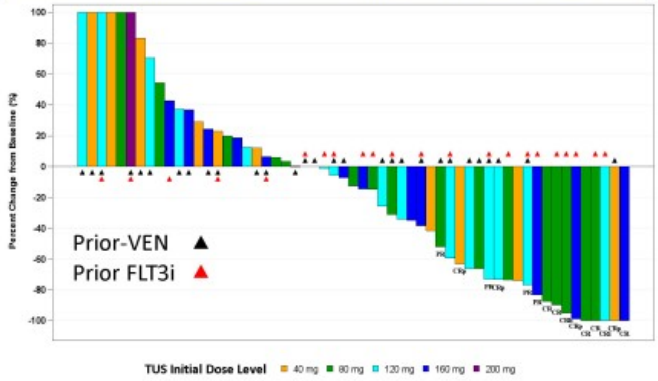
Adverse Events	TUS Single Agent		TUS+VEN Doublet		
			Related to TUS/VEN, n(%) (n=79)		
	Treatment Emergent AEs	Treatment Related AEs	Treatment Emergent AEs	Treatment Emergent AEs Related to TUS	Treatment Emergent AEs Related to VEN
Any	89 (95.7%)	29 (31.2%)	77 (97.5%)	41 (51.9%)	38 (48.1%)
Most Frequent AEs ≥10%					
Pneumonia	31 (33.3%)	0 (0%)	16 (20.3%)	2 (2.5%)	3 (3.8%)
Nausea	20 (21.5%)	9 (9.7%)	21 (26.6%)	14 (17.7%)	10 (12.7%)
Diarrhea	18 (19.4%)	9 (9.7%)	14 (17.7%)	5 (6.3%)	4 (5.1%)
Pyrexia	18 (19.4%)	0 (0%)	11 (13.9%)	1 (1.3%)	1 (1.3%)
Alanine aminotransferase increased	13 (14.0%)	2 (2.2%)	12 (15.2%)	3 (3.8%)	3 (3.8%)
Hypokalaemia	13 (14.0%)	0 (0%)	10 (12.7%)	2 (2.5%)	1 (1.3%)
Epistaxis	12 (12.9%)	0 (0%)	4 (5.1%)	0 (0%)	0 (0%)
Decreased appetite	11 (11.8%)	2 (2.2%)	11 (13.9%)	4 (5.1%)	4 (5.1%)
Hypomagnesaemia	11 (11.8%)	0 (0%)	4 (5.1%)	1 (1.3%)	1 (1.3%)
Febrile neutropenia	11 (11.8%)	1 (1.1%)	20 (25.3%)	3 (3.8%)	4 (5.1%)
Fatigue	10 (10.8%)	2 (2.2%)	15 (19.0%)	6 (7.6%)	5 (6.3%)
Abdominal pain	10 (10.8%)	0 (0%)	4 (5.1%)	1 (1.3%)	1 (1.3%)
Constipation	10 (10.8%)	2 (2.2%)	6 (7.6%)	0 (0%)	0 (0%)
Dyspnoea	10 (10.8%)	0 (0%)	9 (11.4%)	0 (0%)	0 (0%)
Headache	10 (10.8%)	1 (1.1%)	6 (7.6%)	0 (0%)	0 (0%)
Cough	8 (8.6%)	0 (0%)	10 (12.7%)	0 (0%)	0 (0%)
Anaemia	6 (6.5%)	0 (0%)	11 (13.9%)	4 (5.1%)	4 (5.1%)
Neutrophil count decreased	5 (5.4%)	2 (2.2%)	8 (10.1%)	6 (7.6%)	5 (6.3%)
Platelet count decreased	5 (5.4%)	1 (1.1%)	10 (12.7%)	4 (5.1%)	3 (3.8%)
White blood cell count decreased	4 (4.3%)	2 (2.2%)	10 (12.7%)	6 (7.6%)	7 (8.9%)
Aspartate aminotransferase increased	4 (4.3%)	1 (1.1%)	11 (13.9%)	2 (2.5%)	2 (2.5%)
Grade ≥ 3 AEs (≥10%)	66 (71.0%)	9 (9.7%)	67 (84.8%)	23 (29.1%)	23 (29.1%)
Pneumonia	26 (28.0%)	0 (0%)	14 (17.7%)	2 (2.5%)	3 (3.8%)
Febrile neutropenia	10 (10.8%)	1 (1.1%)	19 (24.1%)	2 (2.5%)	3 (3.8%)
Anaemia	5 (5.4%)	0 (0%)	11 (13.9%)	3 (3.8%)	3 (3.8%)
Platelet count decreased	4 (4.3%)	0 (0%)	11 (13.9%)	4 (5.1%)	3 (3.8%)
SAEs					
Leading to treatment termination	12 (12.9%)	1 (1.1%)	11 (11.8%)	1 (1.1%)	3 (3.8%)
Leading to death	18 (19.4%)	0 (0%)	17 (21.5%)	0 (0%)	0 (0%)

Data Cut 26 April 2024

APTOSE BIOSCIENCES

TUS and TUS+VEN : Bone Marrow Blast Reductions and Responses in R/R AML Patients

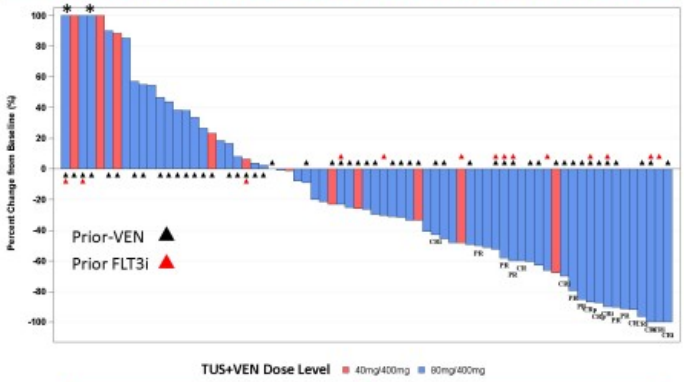
TUS Single Agent Treatment
Bone Marrow Leukemic Blasts | Percent Change from Baseline
Blast Reductions Demonstrate Activity Across 4 Dose Levels
Activity in Patients Who Failed Prior-VEN and Prior-FLT3i



Note: Blast percent change was calculated as 100 X (the lowest post-baseline bone marrow blast - baseline bone marrow blast)/baseline bone marrow blast. Patients with blast percent change >=100% are shown as 100%. Only patients who reported both baseline and any post-baseline bone marrow blast results are included in the figure.

Black triangle indicates patients who received prior Ven before starting Tuspetinib.
Red triangle indicates prior FLT3i.
* Black asterisk indicates patients who administered hydoxyurea within 7 days prior to the lowest marrow blast value
Data cut Feb 09, 2024

TUS-VEN Doublet Treatment
Bone Marrow Leukemic Blasts | Percent Change from Baseline
Blast Reductions in VEN-Naïve and Prior-VEN R/R AML
Blast Reduction in R/R AML Who Failed Prior-VEN and Prior-FL3i



40mgTUS+400mgVEN
- 11 Patients evaluable
  - 73% failed prior-VEN
- 55% (n=6/11) achieved blast reductions
  - 67% (4/6) failed prior-VEN

80mgTus+400mgVEN
- 60 Patients evaluable
  - 72% failed prior-VEN
- 67% (n=40/60) achieved blast reductions
  - 65% (26/40) failed prior-VEN

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TUS Single Agent and TUS+VEN Doublet Support Development for 1L Triplet Therapy

TUS Single Agent and TUS+VEN Doublet

- Extensive Dose Exploration to Satisfy Project Optimus
- Highly Favorable Safety Profile
- Highly Active with in VEN-naïve Patients
- Highly Active with in Patients with High-Risk Genetics

TUS+VEN+AZA Protocol is Active

TUS + VEN + HMA Phase 1/2 Triplet Study

Tuspetinib advancing into a triple drug combination (triplet) of target agents

Newly diagnosed AML patients are VEN-naïve, FLT3i-naïve, HMA-naïve and highly responsive to triplets

TUS+VEN+HMA may become a new Standard-of-Care for frontline (1L) therapy of Newly Diagnosed AML Patients

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Key Opinion Leaders (KOLs) highly enthusiastic for TUS+VEN+HMA triplet

- Investigators' enthusiasm enrolled the TUS+VEN doublet trial ahead of schedule
- KOLs now ramping up TUS+VEN HMA triplet trial in newly diagnosed AML

Dr. Naval Daver, MD Anderson Cancer Center

AML Drug Combination KOL and Global Lead Investigator for Tuspetinib Clinical Trials

- "Tuspetinib is clearly an **active and surprisingly well-tolerated agent in one of the most challenging and heterogeneous disease settings in oncology—** relapsed and refractory AML."

Aptose Press Release for ASH 2023 Dec. 09, 2023

- "Tuspetinib has demonstrated **broad activity**, including activity in patients with FLT3 wild-type AML (accounting for more than 70% of the AML population), FLT3 mutated AML, NPM1 mutated AML, as well as in patients with mutations historically associated with resistance to targeted therapy. Most notably, **TUS targets VEN resistance mechanisms**, enabling TUS+VEN uniquely to **treat the very ill prior-VEN AML population**, including both **FLT3 mutant and FLT3 wildtype** disease."
- "From a broader perspective, the growing body of antileukemic activity, and continued favorable safety profile, **support advancement of tuspetinib in a TUS+VEN+HMA triplet for the treatment of frontline newly diagnosed AML patients.**"

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Investment Thesis

- **Highest unmet medical needs in frontline AML**
  - Need to safely increase survival across all subgroups
- **TUS emerging as ideal agent to combine with VEN+HMA**
  - **Excellent safety** profile
  - **Broad activity** on $FLT3^{MUT}$ and $FLT3^{WT}$ AML (70% of cases)
  - **High-risk TP53 and RAS mutated** AML sensitive
  - **May minimize resistance to VEN** (Venetoclax)
  - **KOLs support TUS** as the ideal 3rd agent for 1L triplet
  - **Extended patent life** and **premium pricing**
- **Near-term milestones can create shareholder value**

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Near-Term Milestones

2024: EHA

- Report TUS Single Agent and TUS+VEN Doublet data in R/R AML supporting TUS+VEN+AZA Triplet trial in newly diagnosed AML

2024: Summer (Q3)

- Initiate dosing of TUS+VEN+AZA Triplet in newly diagnosed AML

2024: ASH

- Report of CR/MRD/Safety data from TUS+VEN+AZA Triplet pilot

2025: 1H

- Complete enrollment in TUS+VEN+AZA Triplet pilot and report CR/MRD/Safety data

2025: EHA

- Data readout TUS+VEN+AZA Triplet pilot
- Select TUS dose for TUS+VEN+HMA Triplet PIVOTAL trials

2025: ASH

- Initiate Ph 2 portion of Ph 2 / Ph 3 PIVOTAL program

Thank you

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