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): September 13, 2023

ZENTALIS PHARMACEUTICALS, INC.
(Exact name of registrant as specified in its charter)

Delaware	**001-39263**	**82-3607803**
(State or other jurisdiction of incorporation or organization)	**(Commission File Number)**	**(I.R.S. Employer Identification No.)**

1359 Broadway, Suite 801
New York, New York 10018
(Address of principal executive offices) (Zip Code)

(212) 433-3791
(Registrant's telephone number, include area code)

N/A
(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 Stock, $0.001 par value per share	ZNTL	The Nasdaq Global 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 7.01 Regulation FD Disclosure.

Beginning on September 13, 2023, spokespersons of Zentalis Pharmaceuticals, Inc. (the “Company”) plan to present the information in the Corporate Presentation furnished as Exhibit 99.1 to this Current Report on Form 8-K (this "Current Report") and incorporated herein by reference at conferences and in meetings with investors and analysts.

The information contained in Item 7.01 of this Current Report (including Exhibit 99.1 attached hereto) shall not be deemed “filed” for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the “Exchange Act”), or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, whether made before or after the date hereof, except as expressly provided by specific reference in such a filing.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

The following Exhibit 99.1 relating to Item 7.01 shall be deemed to be furnished, and not filed:

ExhibitNo.	Description
99.1	Corporate Presentation, dated September 2023.
104	Cover Page Interactive Data File (embedded within the inline XBRL document)

SIGNATURES

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.

ZENTALIS PHARMACEUTICALS, INC.

Date: September 13, 2023

By: /s/ Kimberly Blackwell, M.D.
Kimberly Blackwell, M.D.
Chief Executive Officer

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Corporate Presentation

September 2023

Nasdaq: ZNTL

Forward Looking Statements and Disclaimer

Zentalis Pharmaceuticals, Inc. ("we," "us," "our," "Zentalis" or the "Company") cautions that this presentation (including oral commentary that accompanies this presentation) contains forward-looking statements within the meaning of the U.S. Private Securities Litigation Reform Act of 1995. All statements contained in this presentation that do not relate to matters of historical fact should be considered forward-looking statements, including without limitation statements regarding potential for our product candidates to be first-in-class and/or best-in-class; the franchise potential of azenosertib (ZN-c3); potential for rapid registrational paths; the indication opportunities for ZN-d5; our positioning to execute and deliver; our cash runway; the size of the commercial opportunities for our product candidates; timing of initiation of clinical trials; timing of disclosure of clinical data; our development approach for our product candidates; timing of declaring a monotherapy RP2D for ZN-d5; timing of providing updates on azenosertib program timelines and potential paths to registration; the potential that we are generating registrational data; the timing of preclinical and clinical program updates; the potential of azenosertib to address large unmet need across an array of cancers; the suitability of azenosertib to address tumors with high genomic instability; the potential for azenosertib to transform the treatment paradigm for patients and capture significant market share in ovarian cancer; the potential role for azenosertib at every state of metastatic therapy; the potential unmet need in a particular indication and/or patient population; potential for combinations including our product candidates and the potential benefits thereof; the potential to advance azenosertib into multiple difficult-to-treat tumor types; the potential to advance azenosertib into Phase 3 in ovarian cancer; the plan to initiate a Phase 3 study of azenosertib in combination with paclitaxel or with carboplatin in platinum sensitive ovarian cancer that will evaluate Cyclin E1 status as a potential patient enrichment strategy; the potential benefits of the designs of our product candidates; the target profiles and potential benefits of our product candidates and their mechanisms of action, including as a monotherapy and/or in combination; the market opportunities for and market potential of our product candidates; our anticipated milestones, as well as statements that include the words "design," "estimate," "expect," "may," "milestone," "opportunity," "plan," "potential," "predicts," "strategy," "will" and similar statements of a future or forward-looking nature. These statements are neither promises nor guarantees, but involve known and unknown risks, uncertainties and other important factors that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements, including, but not limited to, the following: our limited operating history, which may make it difficult to evaluate our current business and predict our future success and viability; we have and expect to continue to incur significant losses; our need for additional funding, which may not be available; our plans, including the costs thereof, of development of any diagnostic tools; the outcome of preclinical testing and early trials may not be predictive of the success of later clinical trials; failure to identify additional product candidates and develop or commercialize marketable products; potential unforeseen events during clinical trials could cause delays or other adverse consequences; risks relating to the regulatory approval process or ongoing regulatory obligations; failure to obtain U.S. or international marketing approval; our product candidates may cause serious adverse side effects; inability to maintain our collaborations, or the failure of these collaborations; our reliance on third parties; effects of significant competition; the possibility of system failures or security breaches; risks relating to intellectual property; our ability to attract, retain and motivate qualified personnel, and risks relating to management transitions; and significant costs as a result of operating as a public company. Other risks and uncertainties include those identified under the caption "Risk Factors" in our most recently filed periodic reports on Forms 10-K and 10-Q and subsequent filings with the U.S. Securities and Exchange Commission in the future could cause actual results to differ materially from those indicated by the forward-looking statements made in this presentation. Any such forward-looking statements represent management's estimates as of the date of this presentation. New risks and uncertainties may emerge from time to time, and it is not possible to predict all risks and uncertainties. While we may elect to update these forward-looking statements at some point in the future, we assume no obligation to update or revise any forward-looking statements except to the extent required by applicable law. Although we believe the expectations reflected in such forward-looking statements are reasonable, we can give no assurance that such expectations will prove to be correct. Accordingly, readers are cautioned not to place undue reliance on these forward-looking statements. No representations or warranties (expressed or implied) are made about the accuracy of any such forward-looking statements.

This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and other data about our industry. These data involve a number of assumptions and limitations, and you are cautioned not to give undue weight to such data and estimates. In addition, projections, assumptions and estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk. Neither we nor our affiliates, advisors or representatives makes any representation as to the accuracy or completeness of that data or undertake to update such data after the date of this presentation.

Zentalis' product candidates are investigational drugs and have not yet been approved by the U.S. Food and Drug Administration or any other regulatory authority.

Transforming the Treatment Paradigm in Gynecologic Cancers and Beyond

Azenosertib: First-in-Class WEE1i Candidate with Broad Franchise Potential

ACCELERATING DEVELOPMENT

- Compelling clinical monotherapy activity
- Synergistic anti-tumor activity with chemotherapy and molecularly targeted agents
- Best-in-class safety and tolerability to date
- Enriched activity in tumors with high genomic instability:
  - Cyclin E1+ and HRD+ cancers
- 10 trials; large indications; 400+ patients dosed

BLOCKBUSTER OPPORTUNITY

- Potential for rapid registrational paths for monotherapy and chemotherapy combos
- Ovarian + USC treatable population of ~58K patients / year across the US and EU5
- Potential to expand to address ~140K patients / year across broad set of tumors

Highly Selective BCL-2i

- Multiple opportunities in solid tumors and heme malignancies, including combination with azenosertib in AML

Positioned to Execute and Deliver

- Deep oncology experience
- Veteran scientific, clinical advisors
- Partnerships with Pfizer, GSK
- Cash runway into 2026

Pipeline Addresses Difficult to Treat Cancers with Large Commercial Opportunities

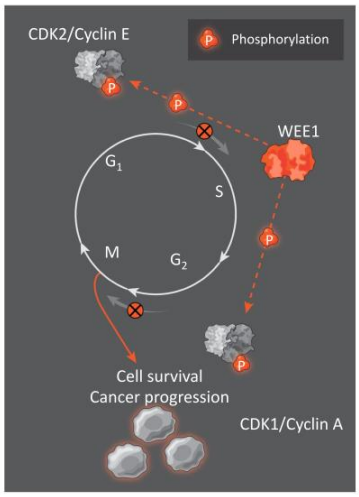
COMPOUND	INDICATION + DEVELOPMENT APPROACH	PRECLINICAL	Phase 1	Phase 1b	Phase 2	Phase 3	STATUS / EXPECTED MILESTONES
Azenosertib WEE1 Inhibitor	**Platinum Sensitive Ovarian Cancer** + Paclitaxel or Carboplatin						Initiate Q1 2024
	DENALI: Platinum Resistant Ovarian Cancer Monotherapy						Enrolling
	TETON: Uterine Serous Carcinoma Monotherapy						Enrolling; FDA Fast Track Designation
	MAMMOTH: PARP Resistant Ovarian Cancer Azenosertib monotherapy, alternating with niraparib or concurrent with niraparib				GSK		Enrolling
	Monotherapy in Solid Tumors						Enrolling; Update efficacy clinical data 2H23
	MUIR: Platinum Resistant Ovarian Cancer + multiple chemotherapy backbones						Enrolling
	Osteosarcoma + gemcitabine						Phase 1 enrollment completed
	BRAF Mutant Colorectal Cancer + encorafenib and cetuximab				Pfizer		Enrolling
	Pancreatic Cancer + gemcitabine						Dana Farber Cancer Institute, funded by SU2C/Lustgarten
ZN-d5 BCL-2 Inhibitor	**Light Chain (AL) Amyloidosis** Monotherapy						Enrolling; Provide interim clinical data and declare RP2D for monotherapy 2H23
	Non-Hodgkins Lymphoma (NHL) Monotherapy						Enrolling
	Acute Myeloid Leukemia (AML) + azenosertib						Enrolling; Provide preliminary data from clinical trial 2H23

Azenosertib

WEE1 Inhibitor with Potential to Address Large Unmet Need Across Array of Cancers

Azenosertib Targets WEE1, a Critical Protein for Cancer Cell Survival

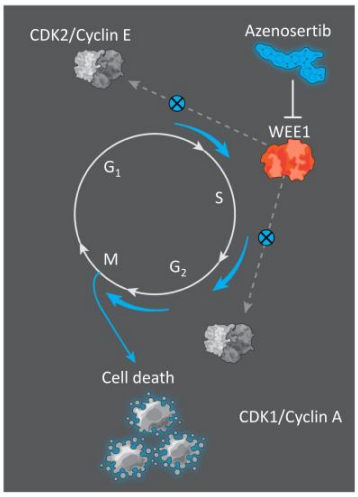
WEE1 activity in untreated cancer cell



- **WEE1 phosphorylates CDK/Cyclin complexes to engage cell cycle checkpoints, allowing DNA repair to occur**
- **Azenosertib inhibits WEE1:**
  - Inactivates CDK1 and 2
  - WEE1 has important roles during S-phase and at G2/M checkpoint
  - Cell cycle progresses without sufficient DNA repair leading to mitotic catastrophe

Azenosertib's MOA well suited to addressing tumors with high genomic instability

Azenosertib blocks WEE1 resulting in cancer cell death



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Luserna di Rora, et al. *J Hem Oncl* (2020), Elbaek *et al. Cell Reports* (2022) 38:110261. Abbreviation: MOA, mechanism of action

Tumors with High Genomic Instability are Sensitive to Azenosertib

High genomic instability can be caused by:

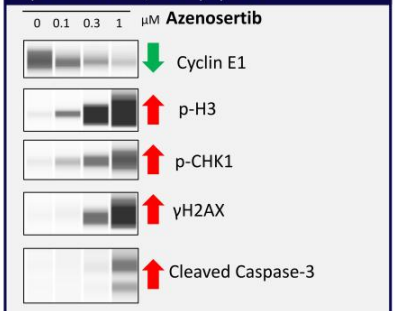
Cyclin E1+ Tumors

- Cyclin E1 overexpression can occur with or without CCNE1 gene amplification
- Cyclin E1+ drives accelerated entry into S-phase through its partnership with CDK2
- Replication machinery is overloaded, resulting in genomic instability

Homologous Recombination Repair Deficiency (HRD) Tumors

- Results in genomic instability through tumors inability to repair double stranded DNA breaks.

Azenosertib induces markers of DNA damage, replicative stress, and apoptosis*



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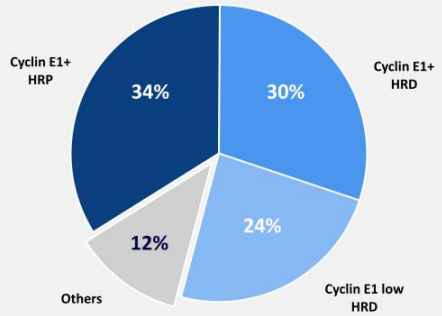
*OVCAR3 Cyclin E1 positive cells, 16-hour treatment

Potential to Transform Treatment Paradigm for Patients and Capture Significant Market Share in Ovarian Cancer

Azenosertib Monotherapy Potentially Addresses 88% Of High Grade Serous Ovarian Cancer

- Ongoing clinical programs will evaluate Cyclin E1+ and HRD+ as patient enrichment strategies
  - Opportunity is much larger segment of ovarian cancers than recently approved therapies
- Data support potential role for azenosertib at every stage of metastatic therapy:
  - **Platinum sensitive**: combination with chemotherapy
  - **Platinum resistant**: monotherapy and combination with chemotherapy

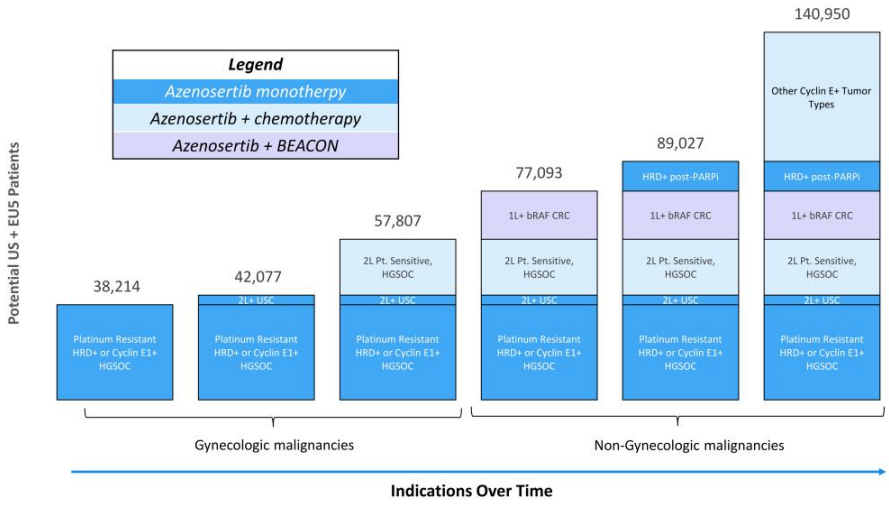
High Grade Serous Ovarian Cancer Patient Segments



HRD: Homologous recombination deficient
HRP: Homologous recombination proficient

Sources:
1. HRD prevalence derived from Konstantinopoulos, et al *Cancer Discov* (2015)
2. CCNE1 amplification prevalence of ~20% reported in Aziz et al *Gynecol Oncol* (2018) and TGCA Network *Nature* volume 474 (2011)
3. Cyclin E1 expression and copy number extracted from the digital analysis of Aziz et al Figure 3B to infer full distribution of Cyclin E1 H-scores and overlap with CCNE1 amplification based on Cyclin E1 high definition of H-score >50
4. HRD prevalence and proportion of overlap with CCNE1 amplification from Konstantinopoulos et al, Figure 2
5. Total HGSOC incidence estimates (US, EU5) sourced from SEER and ECIS are 35, 388 individuals/year

Addressable Patient Population More than Doubles as Franchise Expands to Non-Gynecological Malignancies



Source: Used 'drug-treatable' estimates from DRG Clarivate for all Ovarian, USC, CRC, Breast, Prostate and Pancreatic. For 'Other Cyclin-E1 driven solid tumors' used incidence reported by SEER and ECIS.
Cyclin E1 prevalence in platinum sensitive ovarian cancer derived from Petersen, et al CCNE1 and BRD4 co-amplification in high-grade serous ovarian cancer is associated with poor clinical outcomes, Gynecologic Oncology,Volume 157, Issue 2, 2020
Abbreviations: bRAF+ CRC: bRAF mutant Colorectal Cancer; HRD+ : Homologous Recombinant Repair Deficiency; HGSOC: High Grade Serous Ovarian Cancer; 2L: Second Line
HRD+ Post PARPi tumor types: Prostate, Pancreas and Breast; Other Cyclin E+ Tumor Types include bladder, stomach, esophageal, lung squamous, lung adenocarcinoma, and breast cancer

Azenosertib

Clinical Data Shows Efficacy as Monotherapy in Gynecologic Malignancies with Favorable Safety and Tolerability Profile

Azenosertib Monotherapy Activity Supports Advancement into Multiple Difficult-to-Treat Tumor Types

- **37% Objective Response Rate** with durable responses using intermittent dosing in ovarian and USC patients
- Monotherapy **RP2D** established: 400 mg 5:2
- **Doubled steady state drug exposure** compared to continuous dosing
- **Maintained safety and improved tolerability** compared to continuous dosing
- **No treatment-related discontinuations** in patients who were administered intermittent dosing

Three ongoing Phase 2 monotherapy trials have the potential to support rapid paths to registration in ovarian cancer and USC

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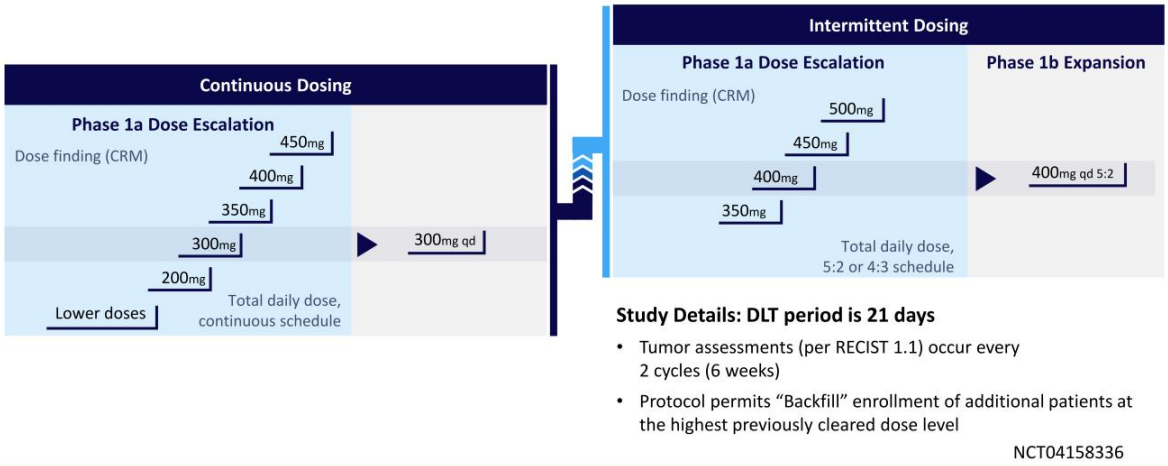
Abbreviations: RP2D: recommended phase 2 dose; 5:2 refers to administration schedule of five days on therapy and two days off; USC, uterine serous carcinoma

Zentalis 001 Study Enabled Rapid and Efficient Approach to Dose Optimization



Study Details: DLT period is 21 days

- Tumor assessments (per RECIST 1.1) occur every 2 cycles (6 weeks)
- Protocol permits "Backfill" enrollment of additional patients at the highest previously cleared dose level

NCT04158336

Primary objectives: Safety, PK (Steady State Exposure (AUC_{0-24}) & Concentration Maximum (C_{max}))

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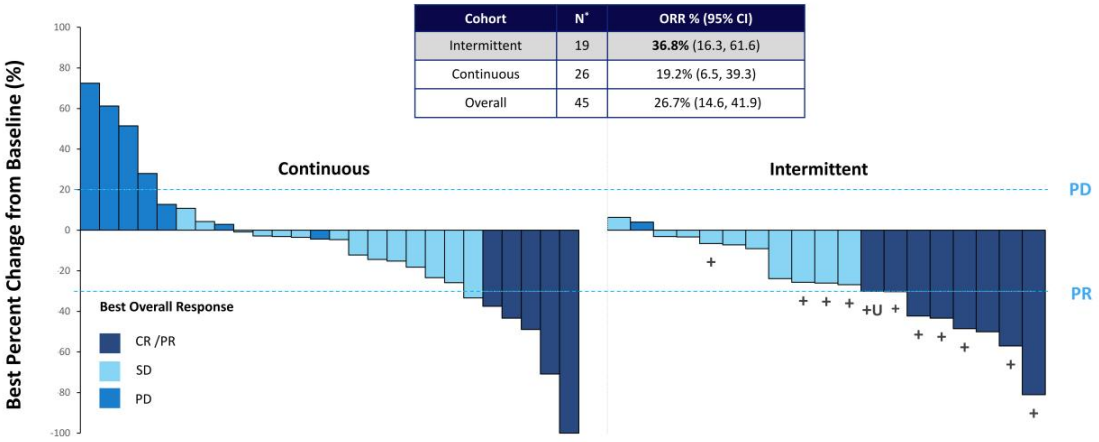
Abbreviations: CRM, continual reassessment method; qd, once daily; 5:2, 5-days of treatment followed by 2-days off treatment; 4:3, 4-days of treatment followed by 3-days off treatment; DLT, dose limiting toxicity; RECIST, response evaluation criteria in solid tumors; PK, pharmacokinetics; AUC, area under the curve

Azenosertib Intermittent Dosing Schedule Doubles Objective Response Rate In Ovarian/USC Populations

Cohort	N*	ORR % (95% CI)
Intermittent	19	**36.8%** (16.3, 61.6)
Continuous	26	19.2% (6.5, 39.3)
Overall	45	26.7% (14.6, 41.9)

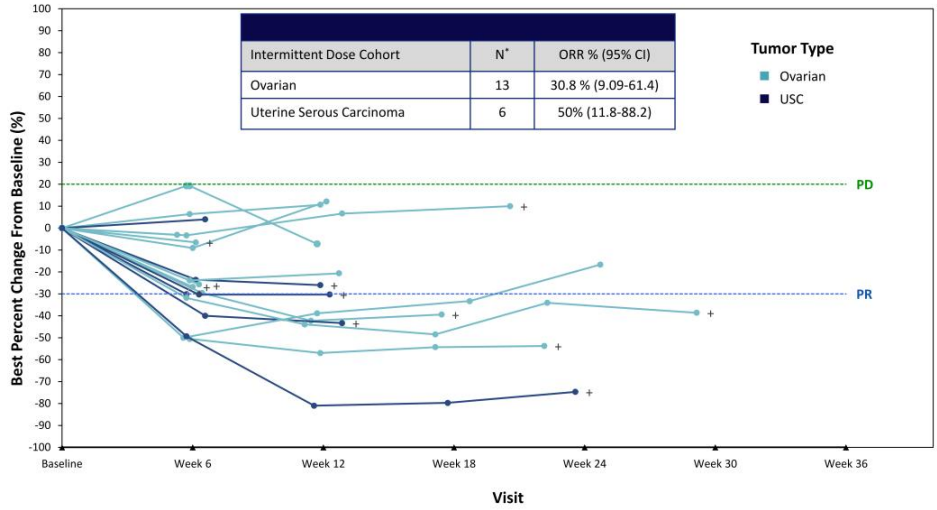


*Response evaluable: have baseline measurable disease by RECIST 1.1, at least one post baseline scan, and received at least one dose of drug
Patients who received ≥300 mg. Abbreviations: USC, uterine serous carcinoma; CR, complete response; PR, partial response;
SD, stable disease; PD, progressive disease; ORR, objective response rate; CI, confidence interval; +: Patients remain on therapy at the time of data cut-off

Data cut-off: June 2, 2023

Azenosertib Monotherapy Intermittent Dosing: 89% of Ovarian and USC Patients Had Target Lesion Reductions from their Baseline Scans

Intermittent Dose Cohort	N*	ORR % (95% CI)
Ovarian	13	30.8 % (9.09-61.4)
Uterine Serous Carcinoma	6	50% (11.8-88.2)



- 12/19 (63%) patients remain on therapy
- Median follow up of 4.4 months
- mPFS of 5.68 months (2.79, NR)
- 10/13 (77%) of ovarian cancer patients had received a prior PARP inhibitor

*Response evaluable: have baseline measurable disease by RECIST 1.1, at least one post baseline scan, and received at least one dose of drug; Patients who received ≥300 mg.
Abbreviations: USC, uterine serous carcinoma; HGSOC, high-grade serous ovarian cancer SD, stable disease; PR, partial response; ORR, confirmed objective response rate; mPFS, median progression free survival; complete response; NR, Not reached, +: Patients remain on therapy at the time of data cut-off

Azenosertib Monotherapy Demonstrates Favorable Safety Profile

	Continuous (n=67)		Intermittent (n=27)		Total* (n=94)	
Treatment Related AEs, N (%)	**All Grade**	**Grade 3/4**	**All Grade**	**Grade 3/4**	**All Grade**	**Grade 3/4**
Gastrointestinal						
Nausea	46 (68.7)	2 (3.0)	9 (33.3)	-	55 (58.5)	2 (2.1)
Diarrhea	31 (46.3)	4 (6.0)	11 (40.7)	3 (11.1)	42 (44.7)	7 (7.4)
Vomiting	28 (41.8)	-	3 (11.1)	-	31 (33.0)	-
Decreased appetite	20 (29.9)	1 (1.5)	4 (14.8)	1 (3.7)	24 (25.5)	2 (2.1)
Dehydration	6 (9.0)	-	3 (11.1)	-	9 (9.6)	-
Fatigue	30 (44.8)	8 (11.9)	11 (40.7)	2 (7.4)	41 (43.6)	10 (10.6)
Hematologic						
Anemia	6 (9.0)	2 (3.0)	6 (22.2)	3 (11.1)	12 (12.8)	5 (5.3)
Thrombocytopenia	4 (6.0)	3 (4.5)	2 (7.4)	-	6 (6.4)	3 (3.2)
Neutropenia**	1 (1.5)	1 (1.5)	4 (14.8)	3 (11.1)	5 (5.3)	4 (4.3)

*Safety Evaluable Population: Received at least one dose of drug;
**No incidence of febrile neutropenia in either dosing group
Continuous 300, 350, 400; Intermittent 350 5:2 and 400 5:2
Treatment Related AEs > 10% and treatment related AEs of interest: All Tumor Types
Abbreviations: AE, adverse event

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Azenosertib At Intermittent Schedules Reduces Dose Modifications And Serious Adverse Events

	Continuous N = 67	Intermittent N = 27	Total* N =94
Treatment Related AEs leading to, N (%):			
Dose reduction	19 (28.4)	4 (14.8)	23 (24.5)
Dose interruption	17 (25.4)	9 (33.3)	26 (27.7)
Discontinuation	4 (6.0)	-	4 (4.3)
Death	-	-	-
Treatment Related SAEs	5 (7.5)	-	5 (5.3)

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*Safety Evaluable Population: Received at least one dose of drug; Continuous 300, 350, 400; Intermittent 350 5:2 and 400 5:2
Abbreviations: SAEs, serious adverse events

Azenosertib Monotherapy

Multiple Ongoing Studies Generating Potentially Registrational Data

Zentalis 004 (TETON): Azenosertib Monotherapy In Women With ≥2L Advanced Uterine Serous Carcinoma

CURRENTLY ACCRUING- FDA Fast track designation

Key Eligibility: Recurrent or persistent USC; ≥1 prior platinum-based chemotherapy regimen; Prior HER-2 directed therapy for known HER-2+; Prior anti-PDL-1; Measurable disease; ECOG PS 0-1; No prior WEE1 inhibitor; No prior cell cycle checkpoint inhibitor.

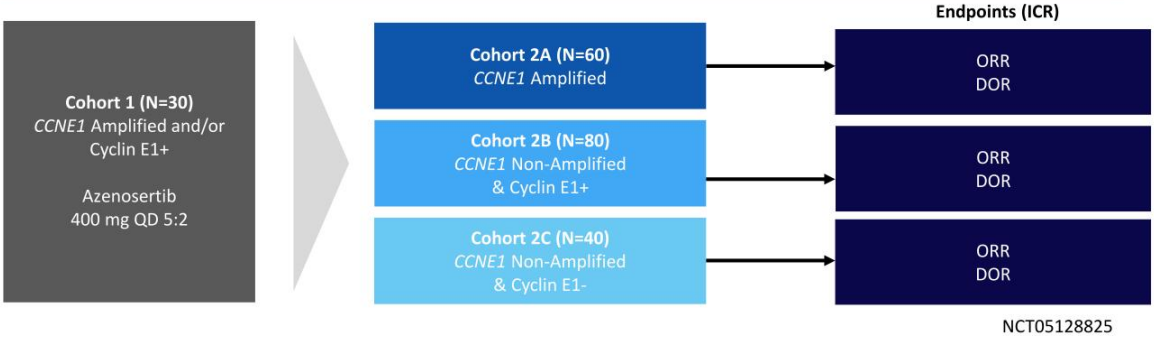
NCT04814108

Abbreviations: 2L, two lines; USC, uterine serous carcinoma; ECOG PS, Eastern Cooperative Oncology Group performance score; QD, once daily; 5:2, 5-days of treatment followed by 2-days off treatment; ORR, objective response rate; DOR, duration of response
The FDA granted Fast Track designation in November 2021 to azenosertib in patients with advanced or metastatic USC who have received at least one prior platinum-based chemotherapy regimen for management of advanced or metastatic disease.

Zentalis 005 (DENALI): Evaluating Impact of *CCNE1* Amplification and Cyclin E1+ in Platinum-Resistant High-Grade Serous Ovarian Cancer

CURRENTLY ACCRUING

Key Eligibility: High-Grade Serous Ovarian Cancer; ECOG PS 0-1; Platinum-resistant (excluding Platinum-refractory); 1-3 prior lines of chemotherapy; Measurable disease per RECIST v 1.1



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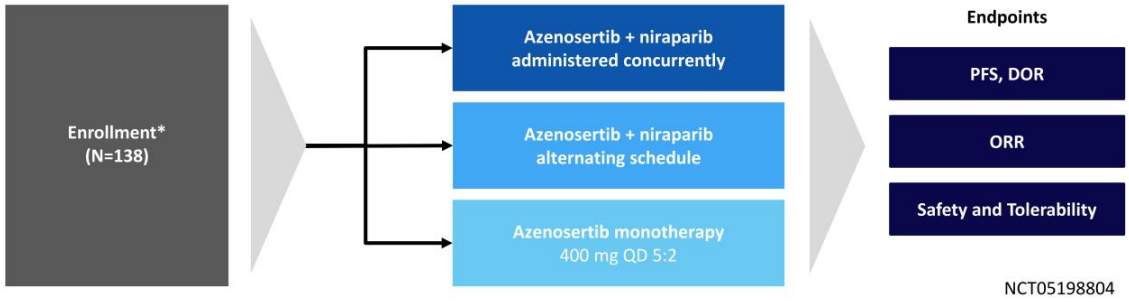
Abbreviations: ECOG PS, Eastern Cooperative Oncology Group performance score; RECIST, response evaluation criteria in solid tumors; QD, once daily; 5:2, 5-days of treatment followed by 2-days off treatment; ORR, objective response rate; DOR, Duration of Response; ICR, Independent Central Review

Zentalis 006 (MAMMOTH): Revised Phase 1/2 Study Of Azenosertib In Combination With Niraparib Or Alternating With Niraparib Or As A Monotherapy in Patients With PARP-resistant Ovarian Cancer

CURRENTLY ACCRUING

Key Eligibility: Recurrent high-grade epithelial ovarian, primary peritoneal, or fallopian tube cancer (serous, clear cell or endometrioid); 1 – 5 prior lines for advanced/metastatic disease; Relapsed within 6 months of platinum therapy (platinum resistant), progressed after taking at least 3 months of PARPi as maintenance treatment.



NCT05198804

* Enrollment Based on Slot Availability

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Abbreviations: PARPi, poly-ADP ribose polymerase inhibitor; QD, once daily; 5:2, 5-days of treatment followed by 2-days off treatment; PFS, progression free survival; ORR, objective response rate

Azenosertib Combination with Chemotherapy

Clinical Data Shows Strong Efficacy and Favorable Safety Profile Across Several Chemotherapy Backbones Enable Advancement into Phase 3 in Ovarian Cancer

Addition of Azenosertib to Chemotherapies Increases Response Rates and Durability of Response in Ovarian Cancer Compared to Chemotherapy Alone

- **50% Objective Response Rate** with 7.4-month Progression Free Survival in paclitaxel combination
- Superior durability in carboplatin combination with **10.4-month Progression Free Survival** and 36% Objective Response Rate
- Overall tolerability of paclitaxel and carboplatin combinations **compares favorably to SOC** chemotherapy doublets paclitaxel-carboplatin or PLD-carboplatin
- **Cyclin E1+ status** associated with **superior Objective Response Rate and longer Progression Free Survival** across response-evaluable patient population

Registrational Phase 3 Trial Announced in Platinum Sensitive Ovarian Cancer

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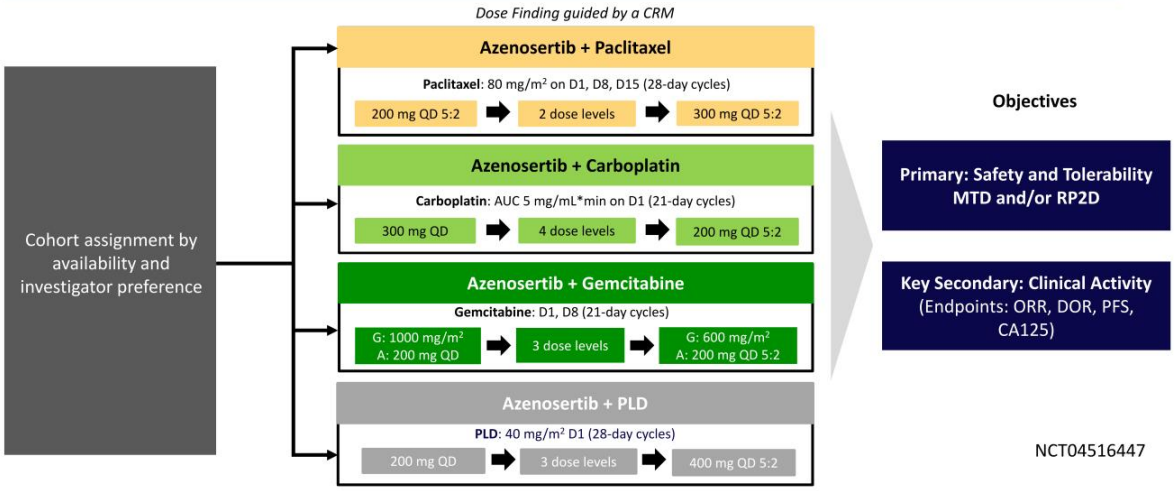
SOC, standard of care; PLD, pegylated liposomal doxorubicin

Zentalis 002: Phase 1b Combination Study To Define RP2D Dosing

Key Eligibility: High-Grade Serous Ovarian Cancer; ECOG Performance Status 0-2; Platinum-resistant/refractory; Up to 3 prior lines of chemotherapy; Measurable disease per RECIST v 1.1



NCT04516447

Abbreviations: ; ECOG, Eastern Cooperative Oncology Group; RECIST, response evaluation criteria in solid tumors; 5:2, 5-days of treatment followed by 2-days off treatment; CRM, continuous reassessment model; QD, once daily; D, day; AUC, area under the curve; G, gemcitabine; A, azenosertib; PLD, pegylated liposomal doxorubicin; MTD, maximum tolerated dose; RP2D, recommended Phase 2 dose; ORR, objective response rate; DOR, duration of response; PFS, progression-free survival;

Liu, J., et al. "Correlation of Cyclin E1 expression and clinical outcomes in a Phase 1b dose-escalation study of Azenosertib (ZN-c3), a WEE1 inhibitor, in combination with chemotherapy (CT) in patients (pts) with platinum-resistant or refractory (R/R) epithelial ovarian, peritoneal, or fallopian tube cancer (EOC)." Poster presented ASCO 2023

Encouraging Efficacy and Durability in Azenosertib + Full Dose Chemotherapy Doublets

Endpoint	Azenosertib + Paclitaxel (N=26)	Azenosertib + Carboplatin (N=36)	Azenosertib + Gemcitabine (N=18)	Azenosertib + PLD (N=35)	Total (N=115)
Response-Evaluable* (N)	22	28	13	31	94
ORR (confirmed), N (%)	11 (50.0)	10 (35.7)	5 (38.5)	6 (19.4)	32 (34.0)
Median DOR (95% CI) in months	5.6 (3.8-NE)	11.4 (8.3-NE)	6.2 (NE)	7.3 (1.5-NE)	8.3 (5.6-12.4)
Clinical Benefit Rate (CR + PR + SD for ≥ 16 weeks), N (%)	18 (81.8)	16 (57.1)	6 (46.2)	24 (77.4)	64 (68.1)
Median PFS (95% CI) in months	7.4 (5.5-NE)	10.4 (3.3-14.5)	8.3 (3.3-NE)	6.3 (3.7-11.0)	9.0 (5.8-13.7)

*Response evaluable subjects are treated subjects with baseline measurable disease per RECIST version 1.1 and at least one post-baseline assessment.
All objective responses were confirmed per RECIST v 1.1.
Abbreviations: ; PLD, pegylated liposomal doxorubicin; ORR, objective response rate; DOR, duration of response; CI, confidence interval; NE, not estimable; CR, complete response; PR, partial response; SD, stable disease; PFS, progression-free survival; RECIST, response evaluation criteria in solid tumors
Liu, J., et al. "Correlation of Cyclin E1 expression and clinical outcomes in a Phase 1b dose-escalation study of Azenosertib (ZN-c3), a WEE1 inhibitor, in combination with chemotherapy (CT) in patients (pts) with platinum-resistant or refractory (R/R) epithelial ovarian, peritoneal, or fallopian tube cancer (EOC)." Poster presented ASCO 2023

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Data cut-off: April 10, 2023

Azenosertib Combo Safety Profile Across Chemotherapy Backbones Consistent with Monotherapy or Chemo Alone

Treatment-Related Adverse Event ≥20% N (%)			Azenosertib + Paclitaxel (Continuous, N=7; Intermittent, N=19)		Azenosertib + Carboplatin (Continuous, N=22; Intermittent, N=14)		Azenosertib + Carboplatin (Continuous, N=14; Intermittent, N=8)		Azenosertib + Gemcitabine (Continuous N=8; Intermittent, N=10)		Azenosertib + PLD (Continuous N=27; Intermittent, N=8)		Total (Continuous, N=64; Intermittent, N=51)	
			All Doses*		All Doses		Doses ≤ MTD		All Doses**		All Doses*			
Grade			All Gr	Gr ≥3	All Gr	Gr ≥3	All Gr	Gr ≥3	All Gr	Gr ≥3	All Gr	Gr ≥3	All Gr	Gr ≥3
Hematologic	Neutropenia	C	5 (71.4)	5 (71.4)	9 (40.9)	7 (31.8)	4 (28.6)	3 (21.4)	7 (87.5)	6 (75.0)	19 (70.4)	17 (63.0)	40 (62.5)	35 (54.7)
		I	11 (57.9)	5 (26.3)	7 (50.0)	1 (7.1)	4 (50.0)	-	7 (70.0)	4 (40.0)	3 (37.5)	3 (37.5)	28 (54.9)	13 (25.5)
	Thrombo-cytopenia	C	4 (57.1)	2 (28.6)	16 (72.7)	11 (50.0)	11 (78.6)	6 (42.9)	8 (100.0)	5 (62.5)	9 (33.3)	2 (7.4)	37 (57.8)	20 (31.3)
		I	4 (21.1)	-	9 (64.3)	5 (35.7)	4 (50.0)	2 (25.0)	8 (80.0)	6 (60.0)*	3 (37.5)	3 (37.5)	24 (47.1)	14 (27.5)
	Anemia	C	5 (71.4)	-	10 (45.5)	3 (13.6)	5 (35.7)	1 (7.1)	6 (75.0)	2 (25.0)	11 (40.7)	4 (14.8)	32 (50.0)	9 (14.1)
		I	8 (42.1)	1 (5.3)	10 (71.4)	4 (28.6)	4 (50.0)	1 (12.5)	5 (50.0)	2 (20.0)	2 (25.0)	1 (12.5)	25 (49.0)	8 (15.7)
Gastro-intestinal	Nausea	C	4 (57.1)	-	15 (68.2)	1 (4.5)	10 (71.4)	1 (7.1)	5 (62.5)	-	16 (59.3)	2 (7.4)	40 (62.5)	3 (4.7)
		I	7 (36.8)	1 (5.3)	6 (42.9)	-	3 (37.5)	-	5 (50.0)	-	4 (50.0)	1 (12.5)	22 (43.1)	2 (3.9)
	Vomiting	C	3 (42.9)	1 (14.3)	8 (36.4)	-	6 (42.9)	-	1 (12.5)	-	11 (40.7)	2 (7.4)	23 (35.9)	3 (4.7)
		I	2 (10.5)	1 (5.3)	2 (14.3)	-	2 (25.0)	-	1 (10.0)	-	4 (50.0)	1 (12.5)	9 (17.6)	2 (3.9)
	Diarrhea	C	4 (57.1)	1 (14.3)	4 (18.2)	-	1 (7.1)	-	1 (12.5)	-	8 (29.6)	-	17 (26.6)	1 (1.6)
		I	6 (31.6)	1 (5.3)	5 (35.7)	-	3 (37.5)	-	6 (60.0)	-	2 (25.0)	-	19 (37.3)	1 (2.0)
Other	Fatigue	C	6 (85.7)	1 (14.3)	8 (36.4)	-	3 (21.4)	-	3 (37.5)	1 (12.5)	8 (29.6)	3 (11.1)	25 (39.1)	5 (7.8)
		I	8 (42.1)	2 (10.5)	5 (35.7)	1 (7.1)	4 (50.0)	-	6 (60.0)	2 (20.0)	2 (25.0)	-	21 (41.2)	5 (9.8)

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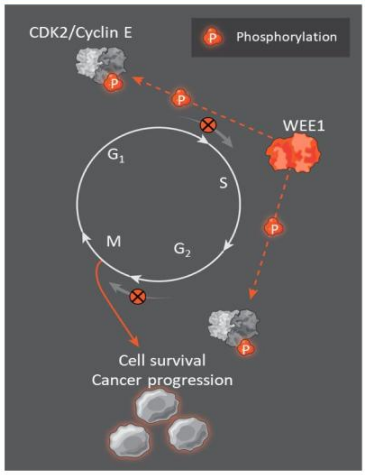
Abbreviations: C, Continuous azenosertib dosing; I, Intermittent azenosertib dosing; MTD, maximum tolerated dose; PLD, pegylated liposomal doxorubicin
*All doses were at or below the MTD
**A MTD for Gemcitabine + Azenosertib has not been determined, further dose cohorts are ongoing.

Liu, J., et al. "Correlation of Cyclin E1 expression and clinical outcomes in a Phase 1b dose-escalation study of Azenosertib (ZN-c3), a WEE1 inhibitor, in combination with chemotherapy (CT) in patients (pts) with platinum-resistant or refractory (R/R) epithelial ovarian, peritoneal, or fallopian tube cancer (EOC)." Poster presented ASCO 2023

Data cut-off: April 10, 2023

Cyclin E1 is a Hallmark of Certain Cancers and Associated with Poor Outcomes

Zentalis evaluating CCNE1 amplification and / or Cyclin E1 over-expression as a potential marker for the enrichment of patient populations for treatment with azenosertib



Cyclin E1 is encoded by the CCNE1 gene and forms a complex with CDK2[1]

- Cyclin E1/CDK2 complex plays a key role in regulating cell cycle progression and the G1/S transition[2]
- Oncogenic activation of Cyclin E/CDK2 complex impairs normal DNA replication, causing replication stress and DNA damage, leading to genomic instability[4]
- WEE1 inhibition exacerbates Cyclin E1 induced replication stress drives cancer cells into mitotic catastrophe and cell death[5]

CCNE1 gene amplification

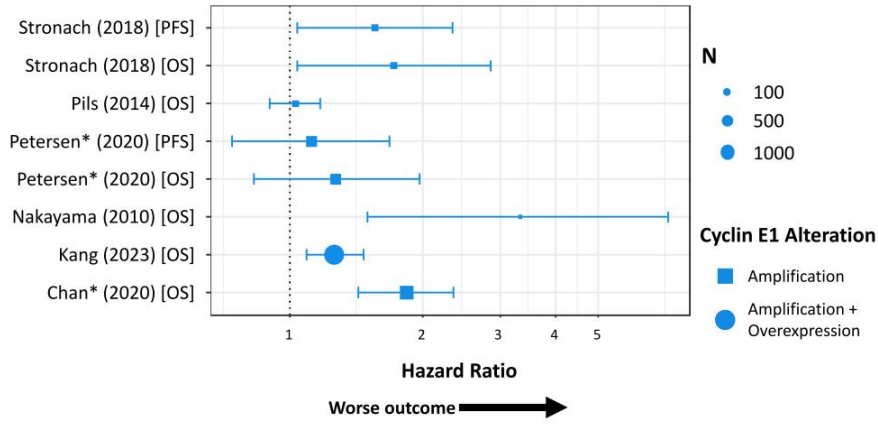
- Associated with poor prognosis and chemotherapy resistance in ovarian cancer[3]
- Genomic alterations of CCNE1 can be detected by Fluorescent/Chromogenic In Situ Hybridization (FISH/CISH) or Next Generation Sequencing (NGS)

Cyclin E1 over-expression

- Associated with worse survival in ovarian cancer patients treated with platinum-based chemotherapy[6]
- Results from multiple mechanisms including gene amplification and transcriptional upregulation
- Protein expression detected by immunohistochemistry (IHC)

1. Koff A., Cross F., Fisher A., Schumacher J., Leguellec K., Philippe M., Roberts J.M. Human cyclin E, a new cyclin that interacts with two members of the *CDC2* gene family. *Cell.* 1991;66:1217–1228
2. Fisher D. Control of DNA replication by cyclin-dependent kinases in development. Results Probl. Cell Differ. 2011;53:201–217
3. Etemadmoghadam D., DeFazio A., Beroukhim R., Mermel C., George J., Getz G., Tothill R., Okamoto A., Raeder M.B., Harnett P., et al. Integrated genome-wide DNA copy number and expression analysis identifies distinct mechanisms of primary chemoresistance in ovarian carcinomas. Clin. Cancer Res. 2009;15:1417–1427
4. Spruck C.H., Won K.A., Reed S.I. Deregulated cyclin E induces chromosome instability. Nature. 1999;401:297–300
5. Kok, Y.P., Guerrero Llobet, S., Schoonen, P.M. et al. Overexpression of Cyclin E1 or Cdc25A leads to replication stress, mitotic aberrancies, and increased sensitivity to replication checkpoint inhibitors. Oncogenesis 9, 88 (2020)
6. Kang, E, et. Al, Cancer. 2023 Mar 1; 129(5): 697–713

Ovarian Cancer Patients with CCNE1 Amplified and/or Cyclin E1 Positive Cancers have a Worse Outcome Following Platinum-Based Chemotherapy Treatment Independent of Platinum-Sensitivity Status



- 6 Studies; n=5404
- 4 Studies where timing of tissue collection was available-all were platinum sensitive tissue collected after ≤ 1 course of chemotherapy); 3533/5404 (65%)
- Other 2 Studies did not disclose timing of tissue collection

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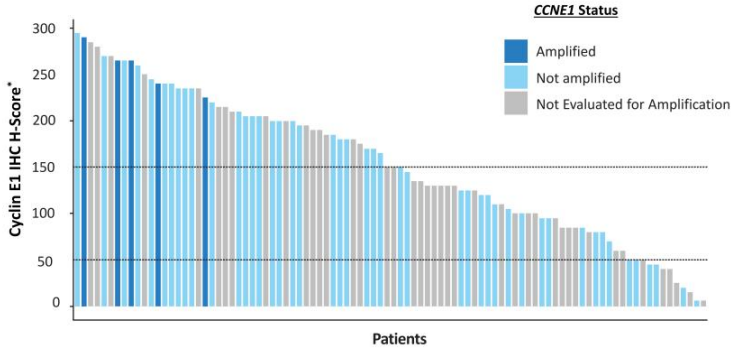
*Timing of tissue collection was not disclosed. Abbreviations: PFS: progression free survival, **OS; overall survival**

Zentalis 002: Majority of Ovarian Cancers are Cyclin E1+

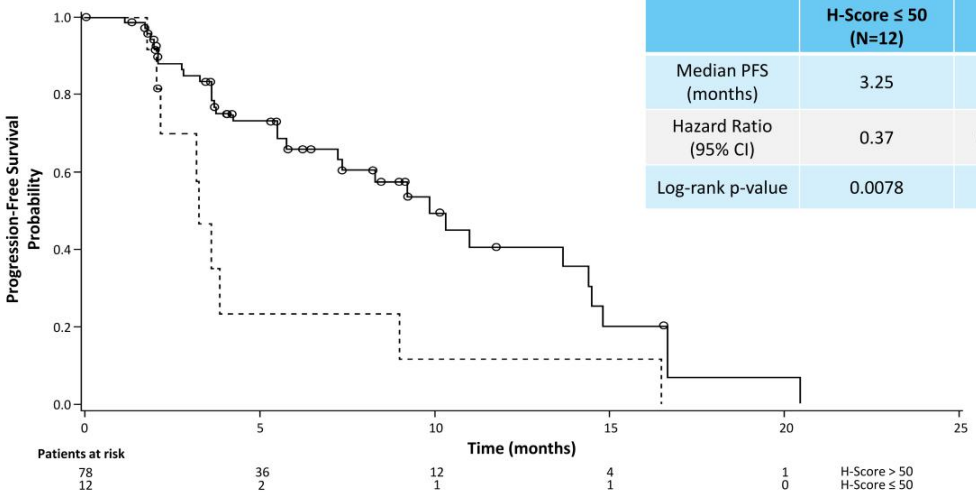


IHC H-Score*	>150	≤ 150 to > 50	≤ 50
CCNE1 Amplified	5	0	0
CCNE1 Not Amplified	25	15	6
Tissue Not Evaluated for Amplification	16	21	6

- H-score > 50 includes all *CCNE1* amplified tumors
- Prevalence of Cyclin E1-IHC+, H-score > 50 of all safety evaluable patients with tissue is 82/94 (**87**%);
- Prevalence of Cyclin E1+ in the response evaluable patients with tissue is 70/82 **(85%).**

*H-scores calculated by multiplying the percentage of cells (0 to 100%) with intensity of Cyclin E1 expression (0 to 3); IHC: Immunohistochemistry
Safety evaluable: received at least one dose of drug; Response evaluable: have baseline measurable disease by RECIST 1.1, at least one post baseline scan, and received at least one dose of drug
Liu, J., et al. "Correlation of Cyclin E1 expression and clinical outcomes in a Phase 1b dose-escalation study of Azenosertib (ZN-c3), a WEE1 inhibitor, in combination with chemotherapy (CT) in patients (pts) with platinum-resistant or refractory (R/R) epithelial ovarian, peritoneal, or fallopian tube cancer (EOC)." Poster presented ASCO 2023

Durability Triples in Patients with Cyclin E1+ Tumors Independent of Chemotherapy Backbone



	H-Score ≤ 50 (N=12)	H-Score > 50 (N=78)
Median PFS (months)	3.25	9.86
Hazard Ratio (95% CI)	0.37	(0.18 – 0.79)
Log-rank p-value	0.0078	

*Response evaluable patients (having received at least one scan)
Abbreviations: IHC, immunohistochemistry ; CI, confidence interval
Liu, J., et al. "Correlation of Cyclin E1 expression and clinical outcomes in a Phase 1b dose-escalation study of Azenosertib (ZN-c3), a WEE1 inhibitor, in combination with chemotherapy (CT) in patients (pts) with platinum-resistant or refractory (R/R) epithelial ovarian, peritoneal, or fallopian tube cancer (EOC)." Poster presented ASCO 2023

Data cut-off: April 10, 2023

Data Supports Advancement of Azenosertib-Chemotherapy Combination into Platinum-Sensitive Ovarian Cancer & Earlier Line Therapy

RP2D established for paclitaxel, carboplatin and PLD combinations

RP2D		
	Azenosertib	Chemotherapy
Paclitaxel	300 mg QD 5:2	80 mg/m^2 on D1, D8, D15 (28-day cycles)
Carboplatin	200 mg QD 5:2	AUC=5 on D1 (21-day cycles)
Gemcitabine	TBD*	TBD*
PLD	400 mg QD 5:2	40 mg/m^2 D1 (28-day cycles)

Main Takeaways

- Strong and durable efficacy signal across chemotherapy backbones
- Cyclin E1 status predicts benefit of azenosertib addition to chemotherapy
  - Suggests azenosertib restores chemotherapy sensitivity in heavily pre-treated platinum-resistant ovarian cancer
- **Plans to initiate Phase 3 study of azenosertib in combination with paclitaxel or with carboplatin in Cyclin E1+ platinum sensitive ovarian cancer**

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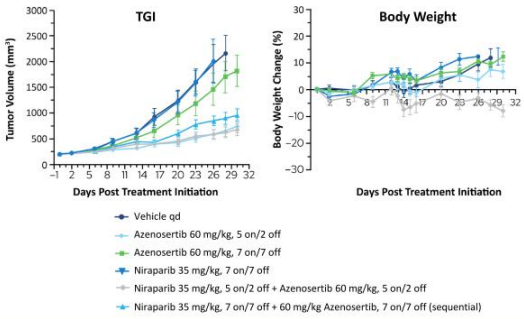
*Gemcitabine + Azenosertib has exciting and durable activity-a MTD has not been determined, further dose cohorts are ongoing.
Abbreviations: RP2D, recommended phase 2 dose; PLD pegylated liposomal doxorubicin; QD, once daily; 5:2, 5-days of treatment followed by 2-days off treatment; D, day; AUC, area under the curve; mg/mL*min

Azenosertib

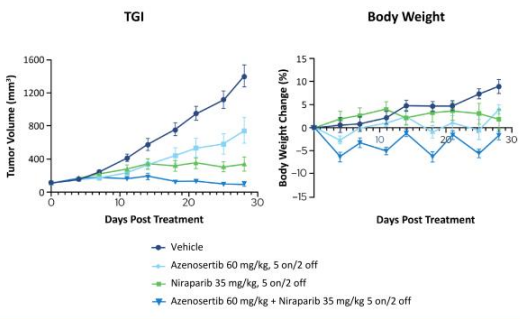
Advancing Programs Investigating Post-PARPi Treatment and Post-BEACON BRAF mCRC, Supported by Strong Body of Preclinical Data

Monotherapy Azenosertib +/- PARP Inhibitor Combinations are Active in Both Ovarian CDX and TNBC PDX Models

CDX Ovarian Cancer Model (OVCAR3; *CCNE1* Amp)



PDX TNBC Model (HBCx-10; *BRCA2*mt)



- Combination of PARP and WEE1 inhibitors in TNBC results in synergistic cell killing in preclinical models with either BRCA mutations or high levels of Cyclin E1 [1]

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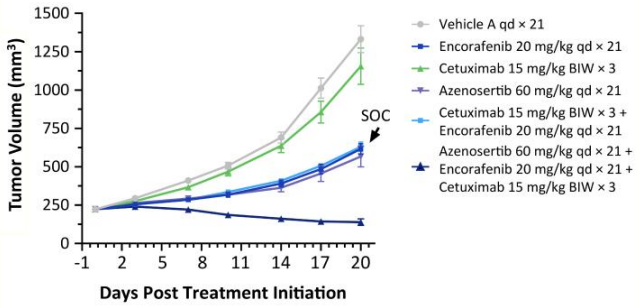
1. Chen X Cancers (Basel). 2021 Apr 1;13(7). Abbreviations: PARP, poly (ADP-ribose) polymerase; CDX, cell line derived xenograft; TNBC, triple-negative breast cancer; PDX, patient derived xenograft; TGI, tumor growth inhibition

Preclinical Data Supports the Combination of Azenosertib with Encorafenib and Cetuximab (BEACON Regimen)

LS411N (BRAF mutant CRC model)



- Oncogene-induced replication stress in mutationally driven cancers such as BRAF mutant colorectal cancer leads to DNA damage and genomic instability
- Oncogene activation disrupts replication regulation leading to slow and stalled replication forks and other defects and leads to DNA damage
- Dependency of cancers with replication stress on WEE1 signaling provides a mechanistic basis for synergy with EGFR/BRAF inhibition
- Addition of azenosertib to the BEACON regimen is well tolerated and provides superior efficacy in an *in vivo* model of BRAF mutant CRC

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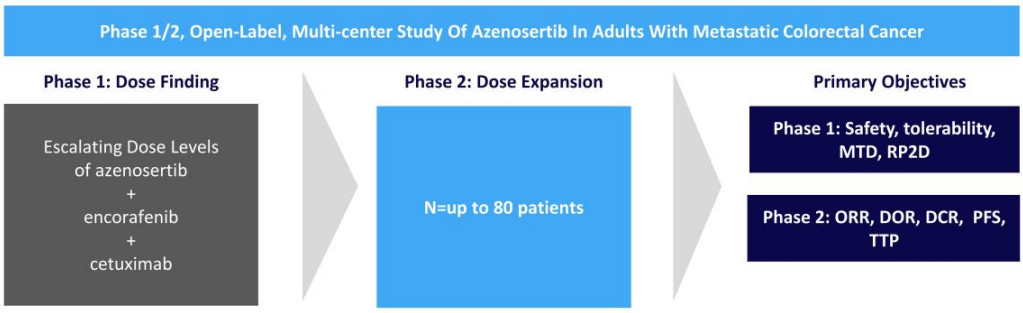
Source: Zentalis Data on File

Kotsantis, et al. Cancer Discov. 2018 May; 8(5): 537–555.

BRAF mCRC Study in Collaboration with Pfizer

Key Eligibility: Patients with mCRC and documented BRAFV600E mutation; Disease progression after 1 or 2 previous regimens for metastatic disease; Prior therapy may include BRAF and/or EGFR directed therapy (e.g., may have progressed after BEACON regimen)



Triplet Combination to be Investigated in Patients With Significant Unmet Need

- Median OS in BRAF mutant CRC patients <1 year, vs. BRAF WT >2 years2
- While targeted BRAF inhibition (e.g., vemurafenib) has been successful in melanoma with response rates >80%, this strategy has failed in CRC (OR ~5%) due to innate resistance3
- Encorafenib in combination with cetuximab (BEACON) was approved for BRAF V600E mCRC in April 2020 and is now the standard of care

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1.Sorbye H, Dragomir A, Sundström M, et al. High BRAF Mutation Frequency and Marked Survival Differences in Subgroups According to KRAS/BRAF Mutation Status and Tumor Tissue Availability in a Prospective Population-Based Metastatic Colorectal Cancer Cohort. PLoS One. 2015;10(6):e0131046. 2 Corcoran et al. Combined BRAF and MEK Inhibition With Dabrafenib and Trametinib in BRAF V600–Mutant Colorectal Cancer. J Clin Oncol (2015) Dec 1; 33(34): 4023-4031 3 Kopetz et al. Encorafenib, Binimetinib, and Cetuximab in BRAF V600E–Mutated Colorectal Cancer. NEJM (2019) 381: 1632-1643 Zentalis maintains full economic ownership and control of azenosertib, apart from Greater China rights (Zentara).

Abbreviations: mCRC, metastatic colorectal cancer; MTD, maximum tolerated dose; RP2D, recommended phase 2 dose; ORR, objective response rate; DOR, duration of response; DCR, disease control rate; PFS, progression free survival; TTP, time to progression; OS, overall survival

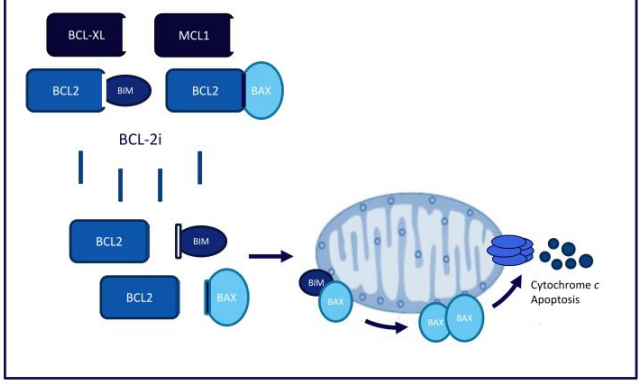
ZN-d5

BCL-2 Inhibitor with Potential Best-in-Class Profile

BCL-2: A Clinically Validated Oncology Target

- BCL-2 is an anti-apoptotic protein involved in tumor survival and resistance to chemotherapy [1]
- The intrinsic apoptotic pathway is controlled by the BCL-2 protein family on outer mitochondrial membrane [2, 3]
- BCL-2 inhibitors may restore the normal apoptosis process, making it an important cancer therapeutic target

Mechanism of Action of BCL-2 Inhibitors [1]



1. Konopleva M et al. Cancer Discov. 2016 Oct;6(10):1106-1117 2. Konopleva M and Letai A. Blood. 2018 Sep 6;132(10):1007-1012 3. Bhola PD and Letai A. Mol Cell. 2016;61(5):695-704

ZN-d5: A Potent BCL-2 Inhibitor Designed with Improved Selectivity for BCL-2

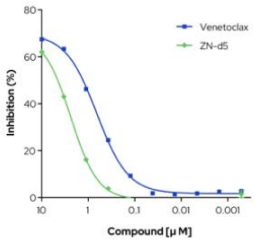
ZN-d5 has 10x Improved Selectivity for BCL-2 vs BCL-xL and Binds With Higher Affinity to BCL-2 Mutants than Venetoclax

Compound ID	Affinity (Kd, nM)			IC_{50} (nM) BCL-2 Type			
	BCL-2	BCL-xL	MCL-1	WT	G101V	F104L	D103Y
Venetoclax	0.41	28	>30000	1.3	7.3	8.4	18.3
ZN-d5	0.29	190	>30000	1.4	3.7	1.4	5.0

ZN-d5 Exhibits Potent In Vitro Activity Across Multiple Tumor Cell Lines

Compound ID	CTG IC_{50} (nM)							
	ALL	MCL		DLBCL		AML		
	RS4;11	Mino-1	Granta-519	DOHH-2	Toledo	HL-60	Molm-13	MV4-11
Venetoclax	2.9	1.1	161	43	191	26	18	3.8
ZN-d5	5.1	0.1	89	50	92	21	39	5.1

ZN-d5: Less Human Platelet Toxicity Compared to Venetoclax in an *in vitro* Assay



Compound ID	CTG (24 h) IC_{50} (mM)
Venetoclax	0.6
ZN-d5	2.4

ZN-d5 shows activity in preclinical models of ALL, NHL and AML

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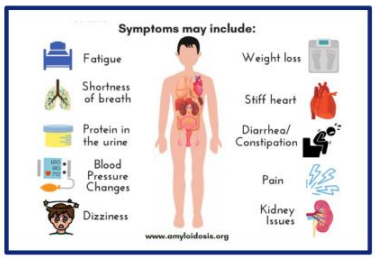
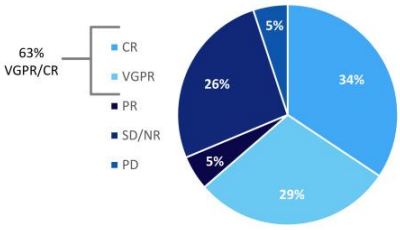
*Venetoclax data based on evaluation of comparable proxy chemical compound purchased from commercial sources rather than obtained from the pharmaceutical company developing the compound

ZN-d5 in AL (Primary) Amyloidosis

AL Amyloidosis study is currently enrolling patients

- AL Amyloidosis: Deposition of immunoglobulin light chains
  - Clonal plasma cell population secretes misfolding light chain
  - Progressive systemic amyloid accumulation causes widespread organ damage
  - High morbidity and mortality
- Orphan disease
- Estimated worldwide prevalence is 75,000 [1]
- About 4k new cases/year in the US [2]
- Not a cancer, but treated like one
  - Agents active in multiple myeloma used in first-line and relapsed/refractory settings
  - Daratumumab only approved therapy, for first-line use with CyBorD
- Relapsed/refractory setting is a high unmet medical need





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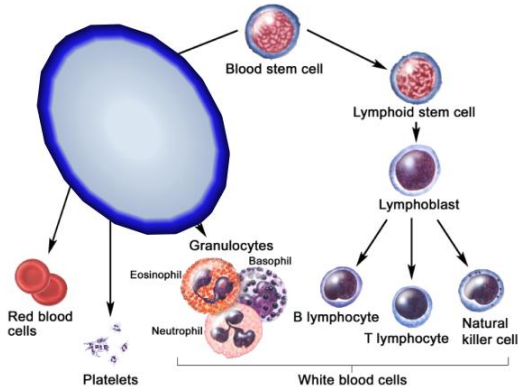
1. Zhang et al. Clin Lymphoma Myeloma Leuk. 2019;19(suppl 10):e339 2. Kyle et al, Mayo Clin Proc. 2019;94:465-471 3. Premkumar et al, Blood Cancer J 2021;11:10; hematologic response rate in 38 evaluable patients.

Acute Myeloid Leukemia (AML) is an Aggressive Heme Malignancy

- AML is an aggressive malignancy of myeloid precursor cells with suppression of normal hematopoiesis & resulting in pancytopenia
- Incidence/mortality in US is approximately 20k/10k per year; 30% 5-year survival[1]
- Venetoclax + low-dose Ara-C or HMAs is approved for newly diagnosed AML in patients ≥75 yrs or in those who cannot tolerate intensive induction chemotherapy[2]
- Relapsed patients, especially those who do not have a FLT3 or IDH1/2 mutation and are not fit to receive intensive chemotherapy, lack tolerable and effective treatment options and therefore require novel treatment options



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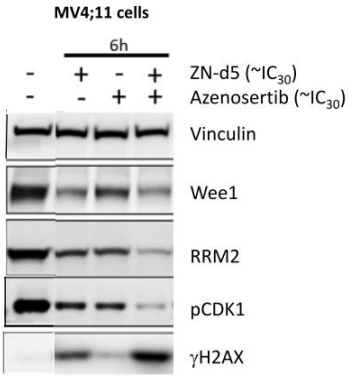
1. NIH NCI Surveillance Cancer Facts Leukemia - AML 2. AbbVie VENCLEXTA (venetoclax) Package Insert

ZN-d5 + Azenosertib Combination Treatment Also Results in Decreased Levels of DDR Proteins

ZN-d5 at subtherapeutic doses activates caspases leading to:

- DNA damage (increased in γH2AX)
- Degradation or decrease of DDR related proteins (Wee1 and RRM2)
- **These effects are increased when combined with azenosertib**

This, in turn, results in inhibition of multiple relevant pathways (e.g., pCDK1) and synergistic anti-tumor activity when combined with azenosertib



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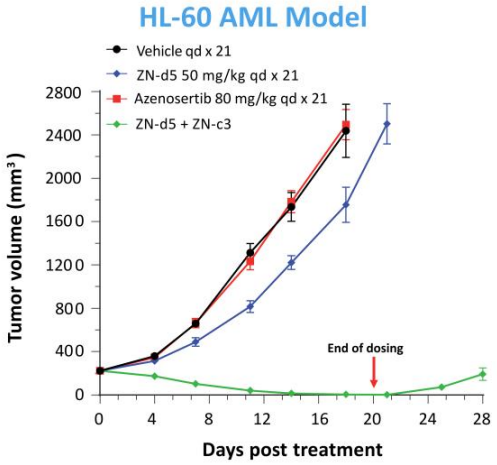
1. Data On File, Zentalis Pharmaceuticals

Combination of ZN-d5 and Azenosertib Results in Synergistic Anti-Tumor Activity in AML



- Significant enhancement of activity vs. use of either agent alone in several indications, including AML
- The effects are seen even at low doses of ZN-d5 leading to regression
- Combination regimen was well-tolerated in mice
- Zentalis is the only company known to have both inhibitors in clinical development

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1. Izadi H et al. AACR 2022 Cancer Res (2022) 82 (12_supplement):2591; Data / Report On File, Zentalis Pharmaceuticals

2023 is a Catalyst Rich Year – Key Milestones

Azenosertib WEE1 Inhibitor

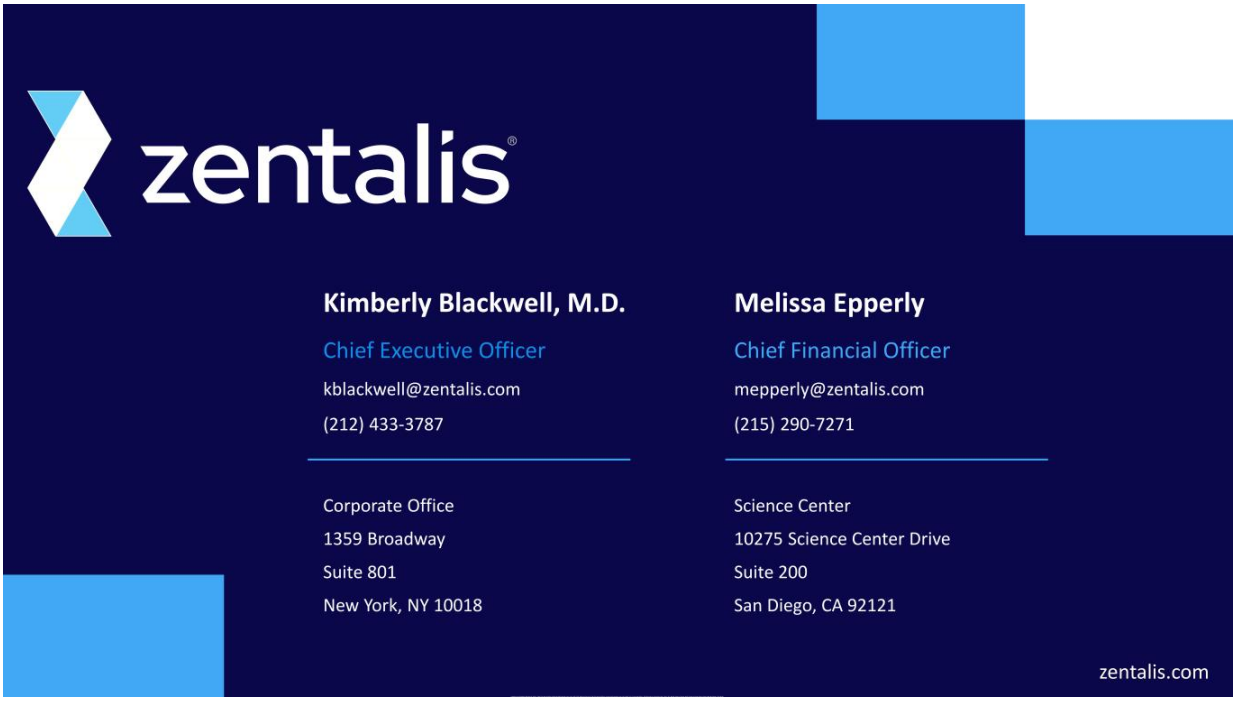
✓ 1Q 2023	Initiate enrollment in the BRAF mutant colorectal cancer BEACON regimen combination clinical trial in collaboration with Pfizer
✓ 1H 2023	Provide preclinical rationale for Cyclin E1 enrichment strategy at a scientific conference
✓ 1H 2023	Declare monotherapy RP2D and provide update on dose optimization activities, program timelines and potential paths to registration
✓ 1H 2023	Results from Phase 1b ovarian chemotherapy combination trial, including clinical translational data on Cyclin E1 amplification / overexpression
2H 2023	Update interim efficacy clinical data from monotherapy dose optimization in solid tumors
2H 2023	Update monotherapy program timelines and potential paths to registration
1Q 2024	Initiate randomized Phase 3 trial of azenosertib + chemotherapy in Cyclin E1+ platinum sensitive ovarian cancer

ZN-d5 BCL-2 Inhibitor

2H 2023	Provide interim clinical data and declare RP2D for Phase 1/2 monotherapy trial in amyloidosis
2H 2023	Provide preliminary data from clinical trial of azenosertib + ZN-d5 in relapsed / refractory acute myeloid leukemia

Discovery

2023	Advance ongoing research on protein degrader programs of undisclosed targets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