TELOMIR™
Pharmaceuticals, Inc.

NASDAQ: TELO

Oral Epigenetic Therapy for Triple-Negative Breast Cancer (TNBC)

FDA IND CLEARED

Phase 1/2 Ready

Forward Looking Statements

This presentation contains "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995. These forward-looking statements generally can be identified by the use of words such as "anticipate," "expect," "plan," "can," "could," "would," "may," "will," "believe," "estimate," "forecast," "goal," "project," "guidance," "potential," "intend," "seek," "target" and other words of similar meaning, although not all forward-looking statements include these words. Forward-looking statements may include, but are not limited to, statements regarding the therapeutic potential, mechanism of action, development plans, regulatory pathway, safety profile, clinical utility, market opportunity, and future development of Telomir-Zn, and the Company's other product candidates. These forward-looking statements are based on current expectations, estimates, forecasts, and projections, as well as management's beliefs and assumptions, and are subject to significant risks and uncertainties that could cause actual results to differ materially from those expressed or implied by such statements. These risks and uncertainties include, among others, risks related to preclinical and clinical development, the ability to obtain regulatory approvals, the outcome of future studies, reliance on third parties, intellectual property protection, financing needs, market conditions, and the other risks identified under the "Risk Factors" section contained in the Company's Annual Report on Form 10-K and the Company's other filings with the SEC. Forward-looking statements contained in this presentation speak only as of the date hereof, and the Company undertakes no obligation to update or revise such statements, whether as a result of new information, future events, or otherwise, except as required by applicable law.

We caution investors not to place undue reliance on the forward-looking statements contained in this presentation. You are encouraged to read our filings with the SEC, available at SEC website and in the "Investors" section of our website, for a discussion of these and other risks and uncertainties.

Preclinical and early-stage data, including cell line, xenograft, and animal model results referenced in this presentation, may not predict clinical safety or efficacy outcomes in humans.

Why Telomir-Zn

A differentiated oncology program entering the clinic

NOVEL MECHANISM | **CLINICAL CATALYST** | **UNMET NEED** | **BROAD ONCOLOGY POTENTIAL**

Oral Epigenetic Reprogramming via Metal Modulation

Telomir-Zn is an oral metal-modulating therapy designed to restore normal gene regulation by targeting iron-dependent epigenetic pathways implicated in tumor growth and treatment resistance.

IND Cleared by FDA for Telomir-Zn in TNBC

Complete GLP safety package. No significant treatment-related toxicity across cardiovascular, respiratory, and repeat-dose tox studies.

TNBC Remains a High Unmet Need Setting

Median OS ~11–13 months for patients with metastatic TNBC[1]. Recent approvals are gated by biomarker status and immunotherapy eligibility. Most patients still rely on chemotherapy.

Beyond TNBC

A focused TNBC program supported by broad preclinical activity across multiple tumor models.

1 Kesireddy M, et al. *Cancers*. 2024.

Triple-Negative Breast Cancer

No estrogen receptor. No HER2 overexpression. ***Limited targeted treatment options for most patients.***

~10-15%

of all breast cancer diagnoses[1]

Median OS

11–13 months[2]

(metastatic)

TNBC Represents a Significant Unmet Medical Need

Aggressive Disease Biology

Characterized by early recurrence, rapid disease progression, and limited durable treatment responses.

Genomically Complex

TNBC lacks a single dominant oncogenic driver, limiting the applicability of targeted therapies across the broader patient population.

Multi-Billion Global TNBC Market Opportunity[3]

Why Current Options Fall Short

Recent therapeutic advances have expanded treatment options for selected patients. However, treatment remains dependent on disease stage, biomarker status, and prior therapy. Chemotherapy continues to play an important role, and recurrence, progression, and treatment resistance remain major clinical challenges.

The Epigenetic Angle

Epigenetic dysregulation contributes to tumor progression and treatment resistance in TNBC. Telomir-Zn is designed to reprogram iron-dependent epigenetic pathways through metal modulation.

1. American Cancer Society. *Breast Cancer Facts & Figures 2026.*
2. Kesireddy M, et al. *Cancers*. 2024.
3. DelveInsight. *Triple-Negative Breast Cancer Market Insights*. 2025.

Telomir-Zn: Epigenetic Reprogramming via Metal Homeostasis

MECHANISM

Telomir-Zn modulates intracellular metal homeostasis, including iron and copper, to influence iron-dependent epigenetic pathways associated with tumor growth and treatment resistance.

WHAT MAKES IT DIFFERENT

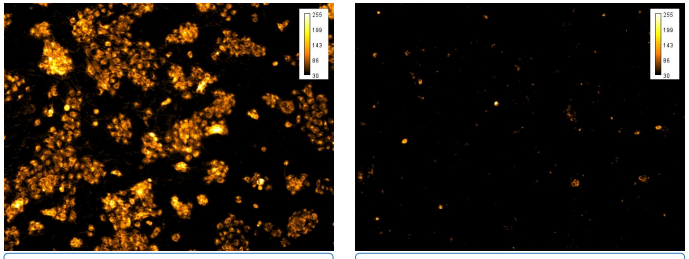
Rather than targeting a single epigenetic enzyme, Telomir-Zn is designed to influence multiple iron-dependent epigenetic pathways through a differentiated upstream mechanism.

REGULATORY STATUS

FDA IND
CLEARED ✓

Phase 1/2
READY

FerroOrange Assay Demonstrates Reduced Intracellular Labile Iron



No Treatment | Telomir-Zn at 1 µM

Oral Metal-Modulating Epigenetic Therapy

Completed GLP Nonclinical Safety Package

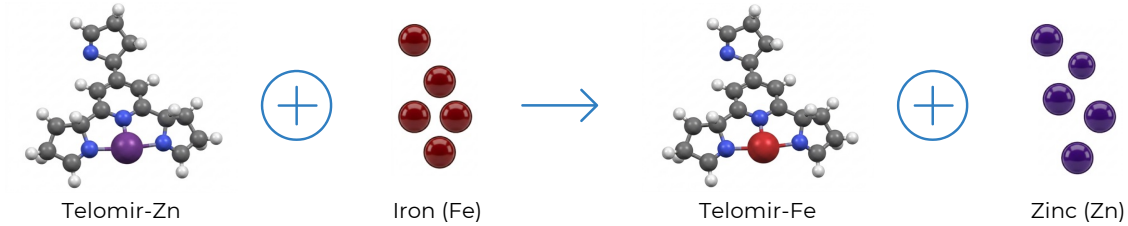
No treatment-related AEs

IC_{50} = 0.87 µM

Anti-tumor activity in HCC1806 TNBC cell line

Direct measurements in HaCat cells of the effects of Telomir-Zn on intracellular iron, as stained with FerroOrange at 24 hours.

Mechanism of Action: Epigenetic Modulation via Metal Homeostasis



Telomir-Zn chelates excess intracellular iron (Fe^{2+}), releasing zinc ions

Inside the Cell

Telomir-Zn enters the cell and exits as Telomir-Fe following intracellular iron exchange.

Outside the Cell

Telomir-Zn is designed to modulate intracellular metal homeostasis, influencing iron-dependent epigenetic pathways involved in tumor growth and treatment resistance.

Metal Homeostasis → Epigenetic Modulation → Restoration of Gene Regulation → Anti-Tumor Activity

Broad Preclinical Anti-Tumor Activity Across Multiple Tumor Types

Triple-Negative Breast Cancer

Demonstrated anti-tumor activity across multiple TNBC models, including paclitaxel-resistant HCC1806 xenografts.

IC_{50}: 0.87 µM in HCC1806 cells and 1.4 µM in MDA-MB-468 cells.

Prostate Cancer

Single-agent anti-tumor activity demonstrated in PC-3 prostate cancer xenografts.

~50% tumor volume reduction with oral Telomir-Zn.

Leukemia (HL-60)

Concentration-dependent anti-proliferative activity demonstrated in HL-60 leukemia cells

Pancreatic (PANC-1)

Concentration-dependent anti-tumor activity in treatment-resistant PANC-1 cells.

Outperformed DFO

Reduced intracellular labile iron more rapidly than deferoxamine (DFO) at the 6-hour time point.

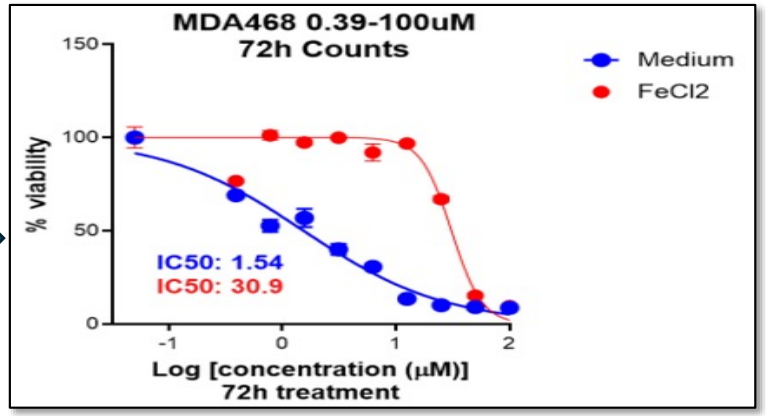
Source: AACR Annual Meeting 2026; Data on file, Telomir Pharmaceuticals.

Iron Rescue Demonstrates Iron-Dependent Activity

Key Takeaways

- $FeCl_2$ partially rescued Telomir-Zn-induced loss of cell viability.
- $FeCl_2$ shifted the IC_{50} from ~1.5 µM to ~31 µM **(~20-fold).**
- These findings support an iron-dependent mechanism of action.

Iron-addicted tumors are selectively vulnerable, while normal cells with lower labile iron remain comparatively spared.



Cell Line Used	Cancer Type	Telomir-Zn IC_{50} (µM)
HCC 1806	TNBC	0.87
MDA-MB-468		1.5
HCC 70		3.6
HL 60	Leukemia	1.6
PC3	Prostate (androgen-independent)	14.3
LNCap	Prostate (androgen-dependent)	21.3
PANC-1	Pancreatic	38.6
HaCat	Normal keratinocytes	>50

Source: AACR Annual Meeting 2026 (TNBC data); Data on file, Telomir Pharmaceuticals.

Oral Telomir-Zn Reduces Tumor Growth in PC-3 Xenografts

STUDY DESIGN

- PC-3 androgen-independent prostate xenograft
- Daily oral dosing for 21 days
- Vehicle and active comparator controls

KEY RESULTS

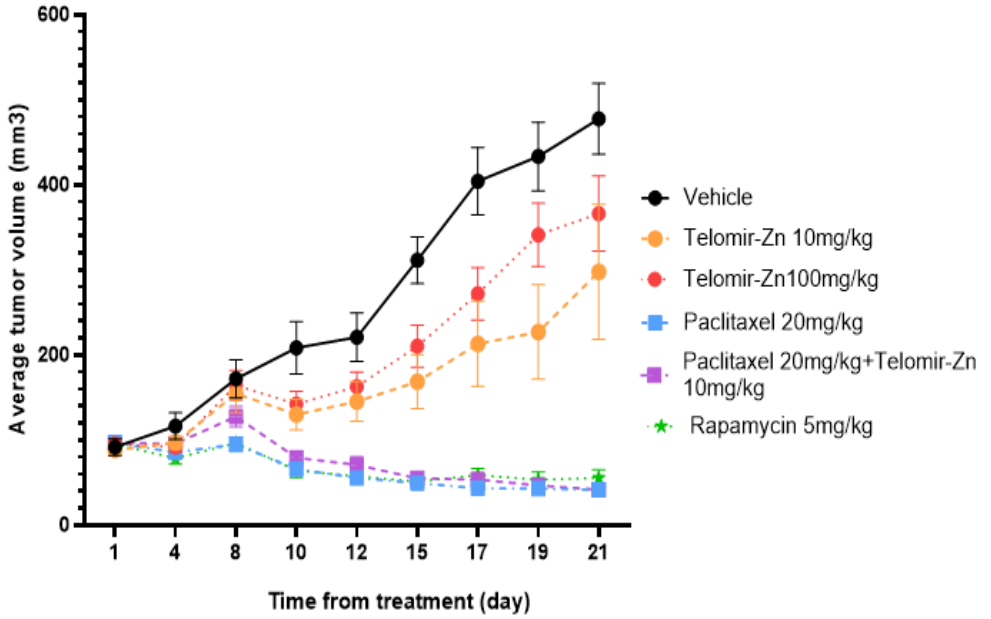
10 mg/kg
Statistically significant tumor suppression from Day 10 ($p<0.05$) — earlier onset, sustained throughout study

100 mg/kg
Statistically significant tumor suppression from Day 15 ($p<0.05$)

Combo+PTX
Marked tumor suppression

SIGNIFICANCE

Demonstrates in vivo proof-of-concept for oral metal-modulating epigenetic therapy



~50% Tumor Volume Reduction vs. Vehicle

Source: Data on file, Telomir Pharmaceuticals.

Telomir-Zn Reverses DNA Hypermethylation of Key Tumor-Suppressor Genes In Vivo

WHY THIS MATTERS

Untreated tumors progressively silence tumor-suppressor genes through promoter hypermethylation.

Telomir-Zn reversed this silencing in vivo in a dose-dependent manner.

These findings provide mechanistic evidence that Telomir-Zn acts to restore tumor-suppressor pathways within growing tumors.

Inhibition of DNA methylation— Day 21 vs. Vehicle

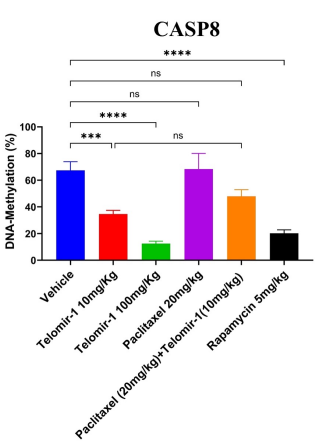
CASP8
Mediates death-receptor-dependent apoptosis. Reduced methylation observed with Telomir-Zn.

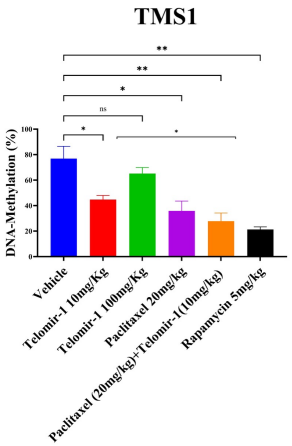
TMS1
Regulates programmed cell death and immune-mediated tumor destruction. Significantly reduced methylation by Telomir-Zn + PTX.

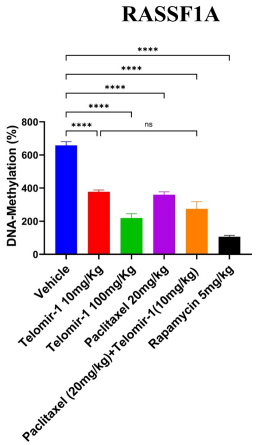
RASSF1A
Most potent effect ($p<0.0001$). Dose-dependent. Regulates apoptosis & mitosis; frequently silenced in TNBC & prostate cancer.

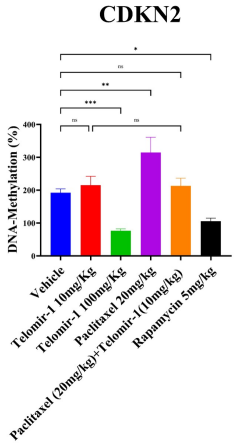
CDKN2A
Dose-dependent. Encodes p16INK4A cell-cycle regulator; loss drives uncontrolled proliferation.

Telomere length: NOT affected at either dose — demonstrating epigenetic specificity









Source: Data on file, Telomir Pharmaceuticals. PC-3 prostate cancer xenograft study.

Intellectual Property: Strong Global IP Position With Exclusive Worldwide Rights

PATENT ESTATE

- Published PCT application (WO2024/191676 A1).
- Patent estate includes composition-of-matter, formulation, and method-of-use claims.
- National phase filings submitted across major commercial territories.

EXCLUSIVITY

- Exclusive worldwide rights to develop and commercialize Telomir-Zn across all therapeutic indications.

DEVELOPMENT READINESS

- IND-cleared oncology program.
- Completed GLP safety package with Phase 1/2 readiness.

Published PCT · Worldwide Exclusivity · FDA IND CLEARED

Source: WO 2024/191676 A1; National Phase Filings; Telomir Pharmaceuticals.

FDA IND Cleared. Phase 1/2 Ready.

3+3

Dose Escalation Design (Ph 1) for Oral, once-daily monotherapy

Simon 2-Stage

ORR Primary Endpoint (Ph 2)

Top Centers

Lead Clinical Site Established with Top Cancer Center and Expansion Discussions Ongoing

Secondary Endpoints

Duration of Response (DoR), Progression-Free Survival (PFS), and continued safety monitoring

GLP Safety Package: Complete

Cardiovascular, respiratory, phototoxicity, and repeat-dose toxicology studies complete — no treatment-related adverse events, no dose-limiting toxicities. Package supports IND clearance and Phase 1 initiation.

Focused Clinical Strategy

Asset	Indication	Preclinical	IND Filed	Phase 1/2	Phase 3	WW Rights
Telomir-Zn	Triple-Negative Breast Cancer (TNBC)	✓	✓	**IND Cleared**		TELO

Telomir-Zn is a focused clinical-stage oncology program for Triple-Negative Breast Cancer (TNBC).

Completed GLP safety package, FDA IND clearance, and exclusive worldwide rights position the program for Phase 1/2 clinical development.

Focused Clinical Strategy subject to regulatory developments and capital raise milestones.

Clinical Development Milestones

FDA IND Submission Completed

FDA IND submission completed following completion of the GLP safety package

FDA Clearance

Completed

IND clearance received. The program is eligible to proceed to Phase 1/2 clinical evaluation.

Phase 1/2 Initiation

Subject to operational readiness and site activation

Planned Phase 1 dose escalation followed by Simon two-stage Phase 2 design in TNBC.

First Patient Enrollment

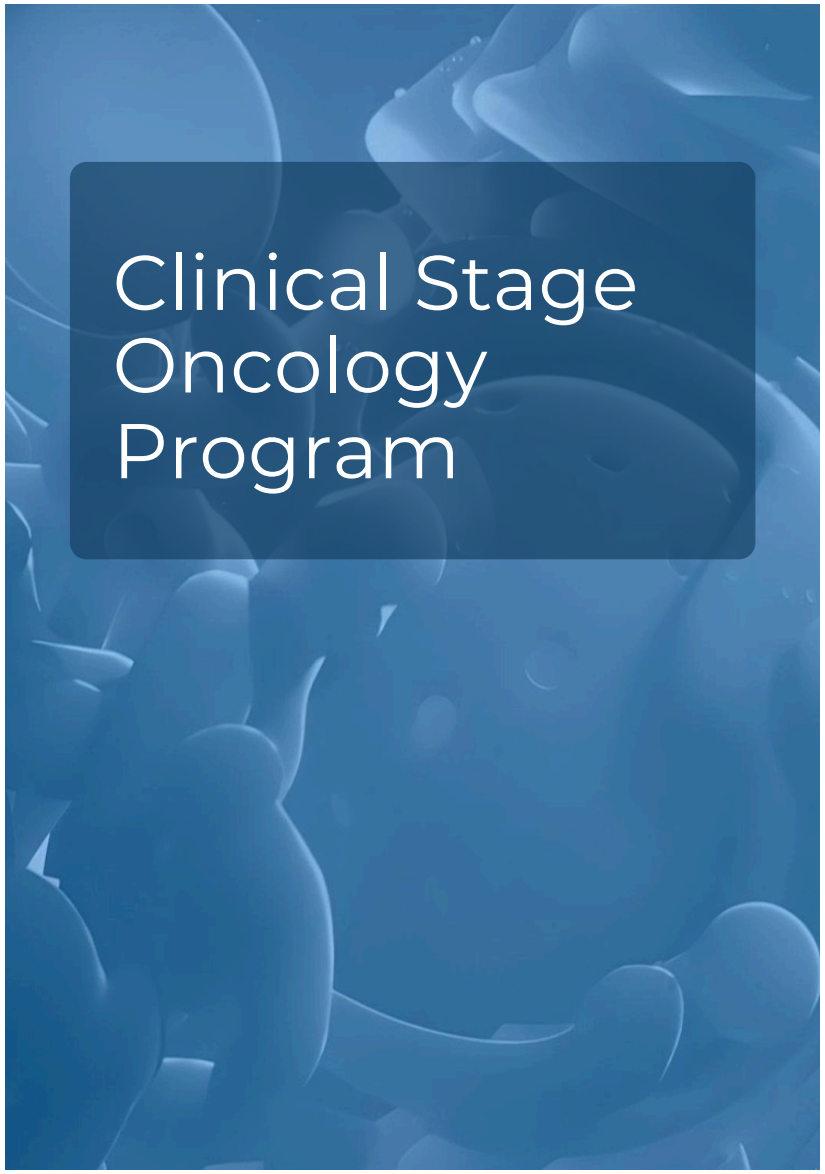
Following site activation

Enrollment will begin after site activation and study initiation.

A Mechanistically Distinct Approach to Epigenetic Oncology

Attribute	Telomir-Zn	Direct KDM Inhibitors	Iron Chelators (DFO)	PD-L1 Inhibitors
Mechanism	**Metal-mediated epigenetic modulation**	Direct enzyme inhibition	Systemic iron depletion	Immune checkpoint
Selectivity	**Broad KDM modulation with minimal GCN5L2 activity**	Generally target-specific	Non-selective	T-cell dependent
TNBC Efficacy	**IC_{50} 0.87 µM; demonstrated in vivo xenograft activity**	No clinical data	Systemic iron depletion	Primarily indicated in PD-L1–selected populations
Systemic Toxicity	**Completed GLP safety studies; no DLTs observed**	Class-related risks	Chronic systemic iron depletion	Immune-related adverse events (irAEs)
Oral Administration	**Yes**	Varies	Mainly IV/SC	IV
Clinical Stage	**FDA IND Cleared**	Preclinical/Early Ph1	Not oncology-focused	FDA Approved (subset)

Comparisons are based on published literature, regulatory status, and class characteristics and are not based on head-to-head studies.



Positioned for Clinical Execution

Clinical Outlook

FDA IND clearance establishes Telomir-Zn as a clinical-stage oncology program. The completed GLP safety package and Phase 1/2 protocol provide the foundation for advancing into clinical evaluation in triple-negative breast cancer, subject to operational readiness.

Forward-looking statements and risk factors are described in the Company's SEC filings.

Leadership Team & Scientific Advisors

Chief Executive Officer & Executive Chairman
Leads corporate strategy, financing, business development, and execution of Telomir's clinical programs.

Erez Aminov

Chief Scientific Advisor
40+ years of drug discovery and translational pharmacology. Former Head of Pharmacology at Sanofi/Synthelabo. Scientific architect of the Telomir-Zn program.

Itzchak Angel, PhD

Chief Financial Officer
20+ years of SEC reporting, public company finance, compliance, and capital markets experience.

Andriy Mushak, CPA

Scientific Advisor
30+ years leading CMC, regulatory strategy, and clinical development across multiple therapeutic areas.

Alex Weisman, PhD

Scientific Advisory Board

Francis O'Donnell, MD
Drug Development · 40 Patents

Distinguished clinical development advisor.

Peter McDonnell, MD
Wilmer Eye Institute, Johns Hopkins

Strategic advisor

Michael Roizen, MD
Cleveland Clinic

Chief Wellness Officer Emeritus. Strategic visibility and medical communications.

1. Source: Telomir Pharmaceuticals Corporate Profile

Telomir Pharmaceuticals, Inc.

NASDAQ: TELO

Oral Epigenetic Therapy for Triple-Negative Breast Cancer

FDA IND Cleared

telomirpharma.com

Appendix

Supporting Preclinical Data

Telomir Pharmaceuticals, Inc.

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Preclinical Research: Cell Viability Across Cancer Types

Iron-dependent selectivity confirms mechanistic anti-cancer activity

Preclinical Research

Significant anti-cancer activity across multiple cancer cell lines with iron-dependent selectivity.

Study Summary

- Telomir-Zn tested on 6 cancer cell lines and 1 normal control cell line
- Activity expressed by IC_{50} (concentration for 50% viability reduction)
- Normal keratinocyte (HaCaT) IC_{50} > 50 μM — no meaningful toxicity

Key Finding

- Greater than 100-fold therapeutic window between most active cancer line and normal cells
- Drug sensitivity mirrors cellular iron dependence — mechanistically consistent

Cell Line	Cancer Type	Iron Dependency	IC_{50} (μM)
HCC1806	TNBC (breast)	Highest	**0.87**
HL-60	Leukemia	Extreme	**1.6**
PC3	Prostate (AI)	Moderate	**14.3**
LNCaP	Prostate (AD)	Low	**21.3**
PANC-1	Pancreatic	Weak	**38.6**
HaCaT	Normal keratinocytes	Minimal	**>50**

>100x

Therapeutic window between TNBC and normal cells

GLP-Cleared

No dose-limiting toxicities in preclinical studies

Source: Data on file, Telomir Pharmaceuticals.

Significant Anti-Cancer Activity in BT 549 Breast Cancer Model

Zebrafish xenograft | Triple-negative breast cancer | Oral administration daily x3 days

Study Design

- **BT549 human triple-negative breast cancer** cells in zebrafish xenograft
- Telomir-Zn administered daily for 3 days to zebrafish embryos
- Comparator: Paclitaxel (standard-of-care chemotherapy)

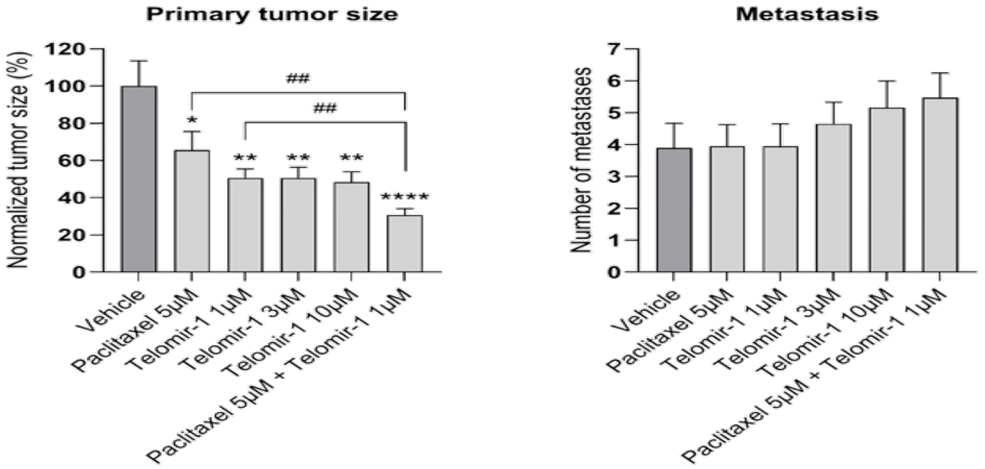
Key Results

- Tumor volume suppression comparable to Paclitaxel at equimolar doses
- Additive anti-tumor effect when combined with Paclitaxel
- No dose-limiting toxicity or off-target effects observed

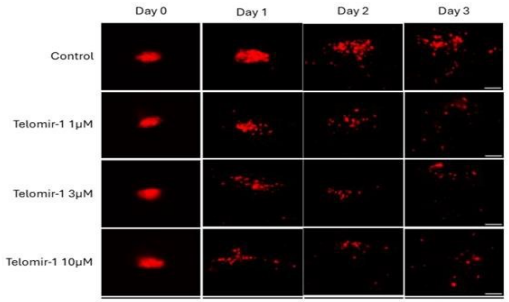
Significance

- First oral small molecule with comparable efficacy to IV chemotherapy in TNBC
- Additive effects enable potential combination therapy strategies

Primary Tumor Size & Metastasis Data (% vs Control)



Fluorescence Microscopy: BT549 Tumor Growth



~70% Reduction

Tumor volume vs. vehicle in combination group

Source: Data on file, Telomir Pharmaceuticals.

Telomir-Zn Suppresses Tumor Growth & Metastasis

Zebrafish xenograft | Triple-negative breast cancer | Oral administration daily x3 days

Study Design

- HCC1806 chemo-resistant human triple-negative breast cancer cells in zebrafish xenograft
- Telomir-Zn administered daily for 3 days to zebrafish embryos
- Comparator: Paclitaxel (standard-of-care chemotherapy)

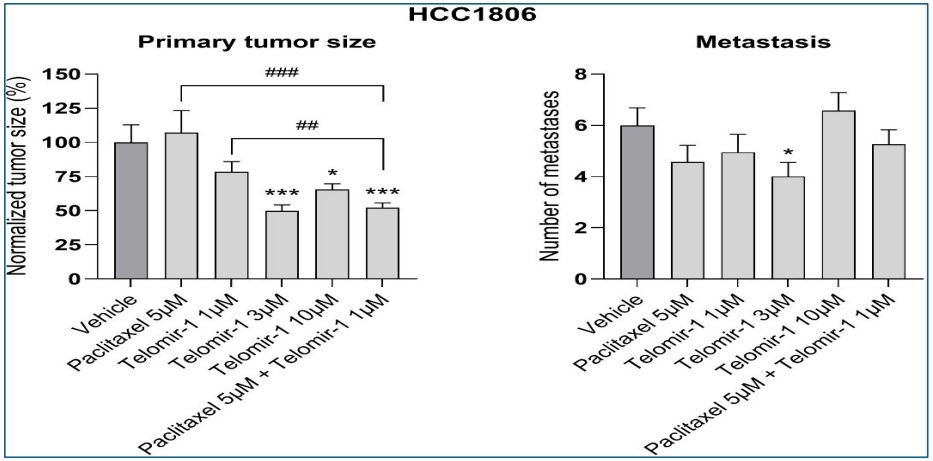
Key Results

- Significant tumor reduction at 3 µM and 10 µM Telomir-Zn ($p<0.05$ and $p<0.001$)
- Additive anti-tumor effect when combined with Paclitaxel vs. either monotherapy alone
- Significant reduction of metastasis
- No dose-limiting toxicity or off-target effects observed

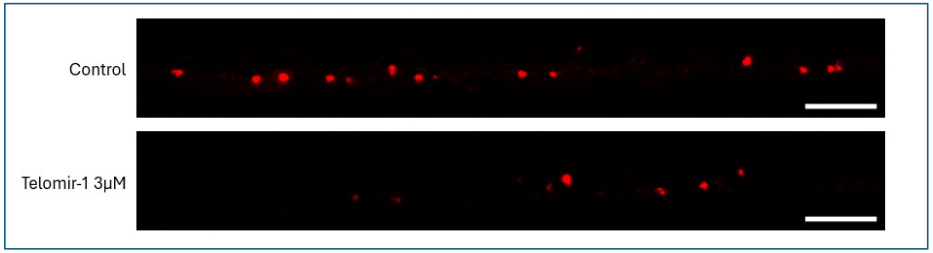
Significance

- Telomir-Zn is the first oral agent to demonstrate both primary tumor suppression and statistically significant reduction of metastatic dissemination in a chemotherapy-resistant TNBC model — a finding not observed with Paclitaxel alone

Primary Tumor Size & Metastasis Data (% vs Control)



Fluorescence Microscopy: HCC1806 Tumor Growth



Source: Data on file, Telomir Pharmaceuticals.

Telomir-Zn Potently Inhibits Histone Demethylases (KDMs) — KDM2 and KDM6

Key epigenetic enzymes driving cancer growth, inflammation, and aging

KDMs in Cancer Biology

- KDMs remove methyl groups from histones — controlling which genes are active or silenced
- Dysregulated KDMs drive oncogene overexpression and tumor-suppressor silencing
- Iron-dependent enzymes — linking metal biology to epigenetic control

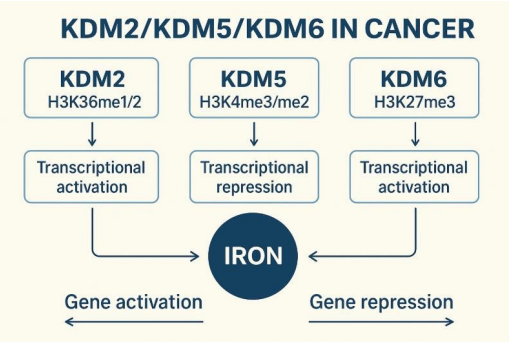
Telomir-Zn IC_{50} Values (Eurofins in vitro)

- **KDM2A (FBXL11):** 0.24 µM — cancer stem-cell and immune evasion driver
- **KDM2B (FBXL10):** 0.30 µM — tumor growth and PRC1 silencing
- **KDM6A (UTX):** 0.39 µM — removes repressive H3K27me3 marks
- **KDM6B (JMJD3):** 2.0 µM — inflammation and metastasis-linked

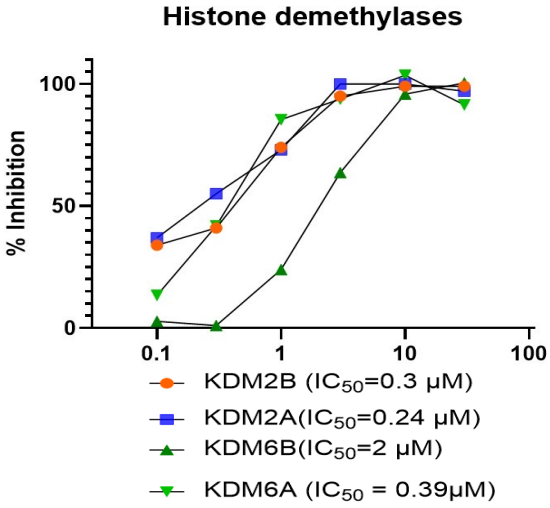
Significance

- Reawakens silenced tumor-suppressor genes (STAT1, TMS1)
- Reduces inflammatory signaling and metastatic potential
- Restores healthy epigenetic patterns disrupted in aging and cancer

KDM2/KDM5/KDM6 Iron-Dependent Mechanism



Dose-Response Curves: % Inhibition vs. Concentration (µM)



Source: Data on file, Telomir Pharmaceuticals. Eurofins (In vitro).

Telomir-Zn Potently Inhibits Histone Demethylases 5 (KDM5s / JARID1)

Epigenetic erasers silencing tumor-suppressor genes and driving cancer stem-cell survival

JARID1 / KDM5 Family in Cancer

- KDM5 enzymes erase activating H3K4me3 marks — silencing tumor-suppressor and differentiation genes
- Overexpressed in breast, prostate, lung, and other solid tumors
- Drive cancer stem-cell identity and resistance to therapy

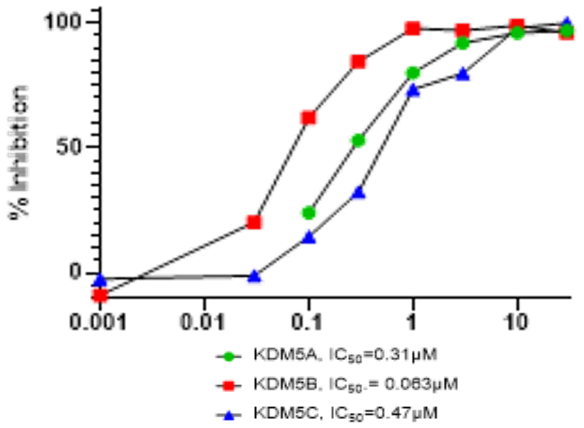
Telomir-Zn IC_{50} Values (Eurofins in vitro)

- **KDM5A (JARID1A):** 0.31 µM — shuts down tumor-suppressor activation marks
- **KDM5B (JARID1B):** 0.063 µM — most potent target; cancer stem-cell survival
- **KDM5C (JARID1C):** 0.47 µM — chromatin stability and gene regulation

Clinical Implications

- Inhibiting KDM5B (IC_{50} = 0.063 µM) may reverse chemo-resistance in breast cancer
- Reactivates tumor-suppressor programs silenced in cancer progression
- Mechanistic synergy with KDM2/KDM6 inhibition for broad epigenetic control

KDM5 Inhibition



0.063 µM

IC_{50} KDM5B — among most potent KDM oncology targets identified for Telomir-Zn

Broad KDM Panel

KDM2, KDM5, KDM6 families all inhibited — unique multi-target epigenetic profile

Source: Data on file, Telomir Pharmaceuticals. Eurofins (In vitro).

Preclinical Research Summary

Complete preclinical package supporting FDA IND clearance

FDA IND Cleared

GLP Safety Package Complete
No DLTs · No treatment-related AEs

Clean Genetic Safety Profile
Non-mutagenic · Non-genotoxic · Non-neurotoxic

Domain	Main Characteristics	Details
Pharmacology	Selective intracellular metal modulation	Affinity order: $Cu^{2+} > Fe^{2+} >> Zn^{2+}$ · Faster and more potent intracellular Fe^{2+} depletion than DFO (gold-standard chelator) at 3h and 6h timepoints · IC_{50} for Fe^{2+} depletion: Telomir-Zn 1.05 μM at 6h vs. DFO 24.6 μM
ADME	Absorption & Metabolism	Rapidly absorbed in rat/dog · Plasma stable up to 6h · 67–99% unbound · Low CYP450 metabolism in dog/human hepatocytes — low drug-drug interaction potential
Selectivity	Target selectivity	No affinity across broad receptor, enzyme, and ion channel panel · Broad KDM modulation with minimal off-target activity · Spares GCN5L2 acetyltransferase · No significant off-target activity identified
Toxicology	Acute + repeated oral (14-day, 2 species)	Established NOAEL in two species supporting clinical dosing.

GLP Safety Package Complete · FDA IND Cleared · Oral Dosing · NOAEL Established in Two Species

Source: FDA IND submission; GLP pharmacology and toxicology studies. Data on file, Telomir Pharmaceuticals.