

Designed to reverse cellular aging...

TELOMIRTM

Pharmaceuticals, Inc.

November 2025

Nasdaq: **TELO**

CORPORATE OVERVIEW

Telomir Pharmaceuticals Inc. (Nasdaq: TELO) is a pre-clinical-stage pharmaceutical company leading the development of age-reversal science by focusing on addressing the root causes of age-related conditions, rather than just managing the Symptoms.

“Telomir-1 is a novel oral small molecule that regulates metal ions, extends telomere caps, affects epigenetics features, normalize mitochondrial functions and combat oxidative stress—key drivers of aging, cancer and disease. By balancing essential metals like iron and copper, it may protect against cancer and age-related conditions”



Cancer



Wilson's Disease



Alzheimer's Disease, Spasmodic dysphonia and other neuronal and muscular diseases



Progeria and Werner Syndrome Research



Metabolic syndrome and Type 2 Diabetes

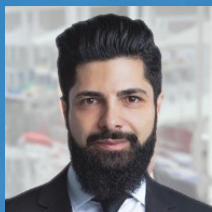


Age-related Macular Degeneration (AMD)



Bactria and Virus

TELOMIR MANAGEMENT



Erez Aminov | Chairman & CEO

- A biotechnology leader driving innovation in drug development and strategic growth.
- Current Chairman and CEO of Mira Pharmaceuticals (Nasdaq: MIRA); Led the preclinical development of multiple drug candidates and successfully submitted an IND while securing funding and meeting critical deadlines.
- Collaborated with major universities like University of Miami, Bascom Palmer Eye Institute, and helped form strategic partnerships.



Alan Weichselbaum, CPA, MBA | CFO

- Seasoned Financial Executive with 30+ years of experience in corporate finance, capital markets, and strategic advisory; currently CFO of both Telomir and Mira Pharmaceuticals.
- Board and Advisory Leadership as Director of FinWise Bancorp (Nasdaq: FINW) and founder of The Wexus Group, advising growth-stage companies on capital structuring and exit strategies.
- Capital Markets Expertise gained through senior Wall Street roles, hedge fund management, and leadership in institutional transactions across public and private markets.



Itzhak Angel, PhD | Chief Scientific Advisor

- Over 40 years of experience in guiding medical, pharmaceutical, drug, and business development in both large and emerging companies.
- Expertise in small molecules, botanical drugs, biotechnology products, delivery systems, medical devices, and drug-device combinations.
- Former Head of Pharmacology at Synthelabo (Sanofi-Aventis) where he participated in research and development of drugs such as Xatral (alfuzosin), Ambien (zolpidem) and Mizollen (mizolastine).



Alex Weisman, PhD | Scientific Advisor

- Occupied executive positions of VP R&D and Chief Scientist at numerous Israeli and international pharmaceutical companies. Currently serve as an advisor and management team member for companies developing new products for the chemicals, pharmaceuticals, and food industries.
- More than 30 years of experience in the development, characterization, scale-up, technology transfer, troubleshooting, production and registration of novel and generic drugs, and other pharmaceutical and chemical products.

THERAPEUTIC RESEARCH APPROACHES FOR TELOMIR-1

Key ongoing approaches include:



Identify potential molecular and epigenetic targets for selective modification and control by Telomir-1



Investigating Telomir-1's effects on copper and iron regulation at the cellular level and in preclinical models.



Explore available disease and functional models to better understand the therapeutic potential of the drug



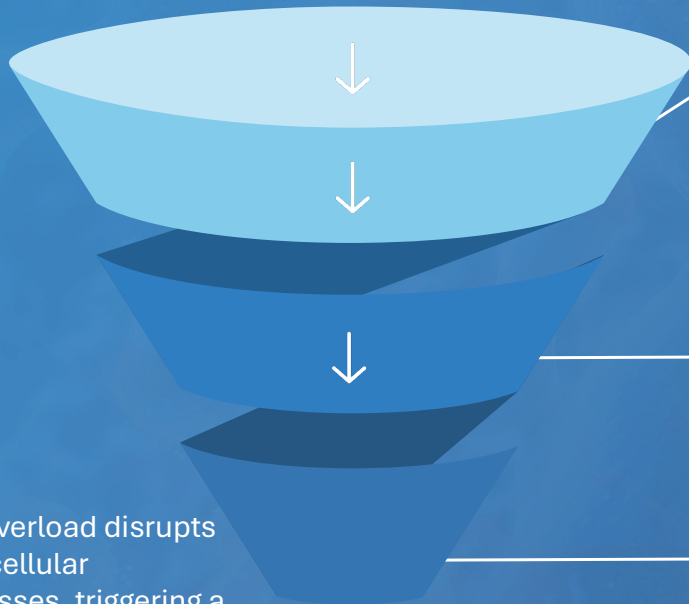
Explore the interactions between iron, copper and zinc on variable cellular functions focusing on cancer, cellular aging and its regulation



Exploring Telomir-1's role in addressing metal toxicity, which occurs when metals like copper, iron, or lead accumulate to harmful levels in the body.

IMPACT OF IRON OVERLOAD

IRON OVERLOAD



Iron overload disrupts core cellular processes, triggering a cascade of functional decline and disease development.

Cellular

- Oxidative stress
- Mitochondrial dysfunction
- DNA dysfunction
- Epigenetics
- Ferroptosis
- Cellular senescence

Pathology

- Cancer
- Progeria and Werner syndromes
- Metabolic Syndrome
- Age-related Macular Degeneration (AMD)
- Spasmodic Dysphonia
- Alzheimer's Disease

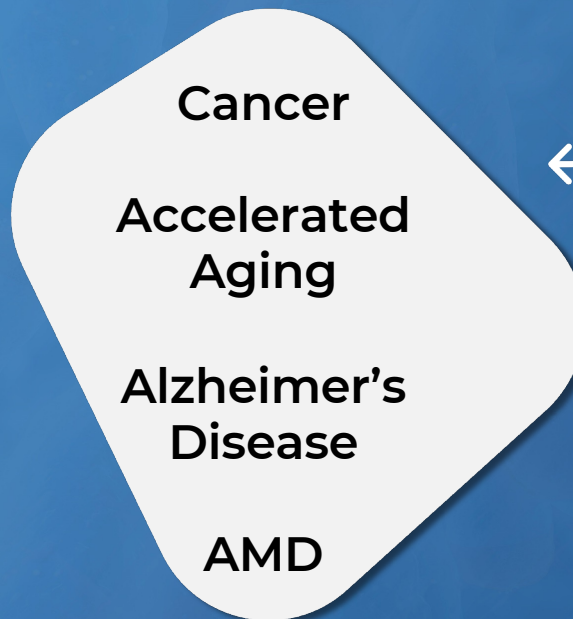
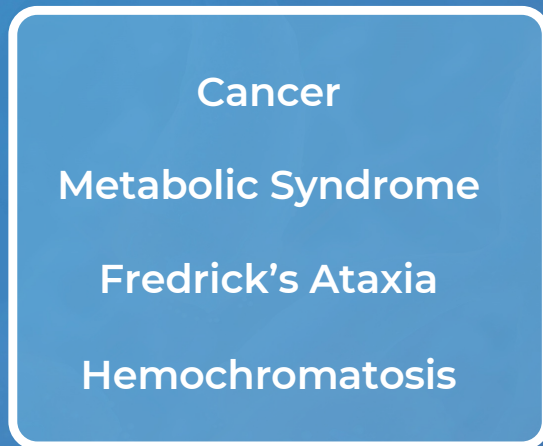
Functional

- Cancer
- Accelerated aging
- Senescence
- Telomer shortening
- Insulin resistance
- Inflammation
- Neurodegeneration

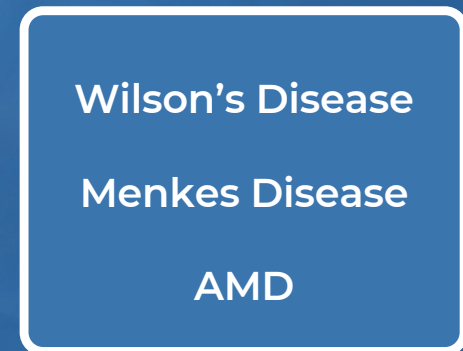
IMPACT OF IRON AND COPPER OVERLOAD

Iron and copper overload contribute to AMD and retinal aging, while copper overload is also central to Wilson's disease and broader age-related neurodegenerative processes.

IRON OVERLOAD



COPPER OVERLOAD



THERAPEUTIC AREAS

Key ongoing initiatives include:



Alzheimer's Disease, Spasmodic Dysphonia and other neuronal and muscle diseases

Investigating Telomir-1 for its potential to improve neuronal and muscle functions and mitigate cognitive decline and neurodegeneration associated with Alzheimer's and Dysphonia.



Type 2 Diabetes Studies

Building on zebrafish success, Telomir is testing a rat diabetic model to confirm Telomir-1's efficacy in reversing metabolic parameters, including reduced insulin resistance (HOMA-IR).



Cancer Models

Exploring epigenetic mechanisms and anti-cancer applications using in vitro and xenograft studies.



Progeria and Werner syndrome Research

Following promising *C. elegans* and zebra fish results, Telomir-1 restored lifespan and normalized aging in wrn-1 mutant nematodes and zebra fish (models for accelerated aging), showing enhanced longevity, DNA functions and physiology.



Macular Degeneration (AMD)

Exploring Telomir-1's role in addressing retinal cell degeneration and Drusen formation, which occurs when metals like copper or iron accumulate to harmful levels.



Wilson's Disease Study

Investigating Telomir-1's effects on copper regulation in preclinical models.

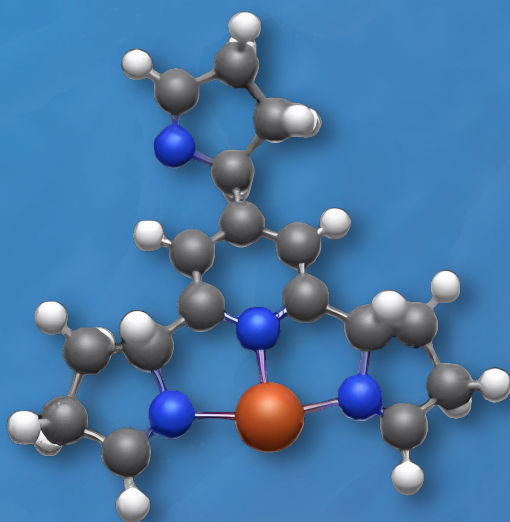


Bacterial and viral infection

Exploring Telomir-1 or Telomir-Ag2 effects on processes associated with bacterial and viral infections.

PRE-CLINICAL RESEARCH

Age Reversal and Increased Longevity



White: Hydrogen

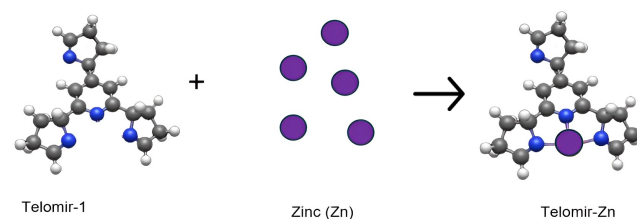
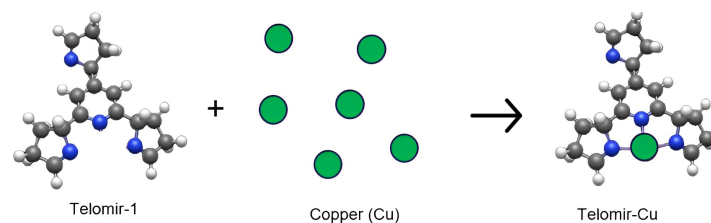
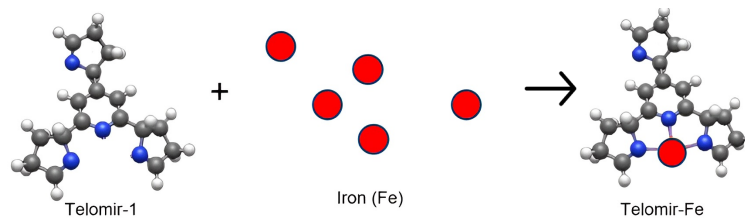
Gray: Carbon

Blue: Nitrogen

Orange: Metal



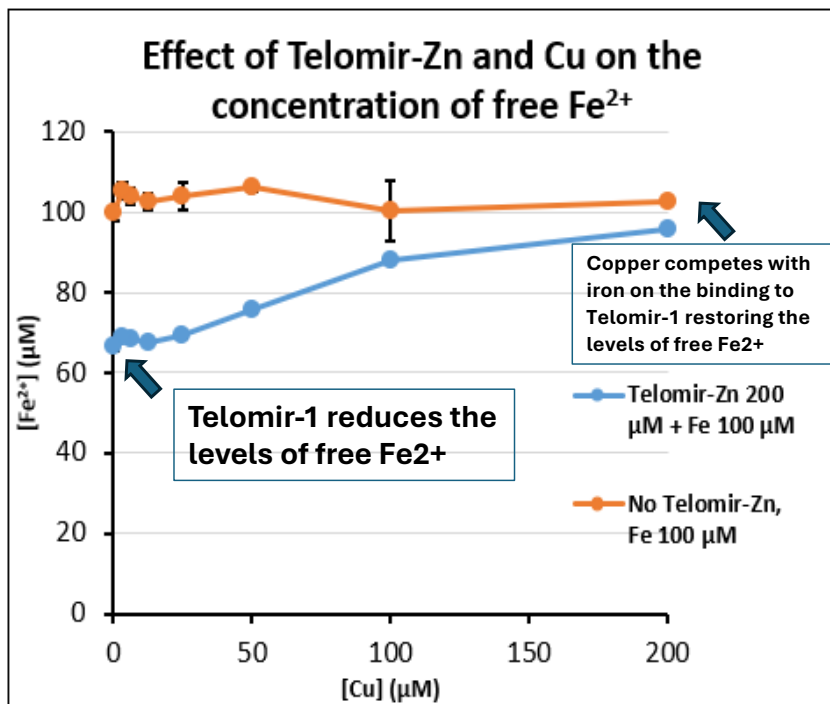
Our molecule, Telomir-1 can effectively bind and chelate several ions. It has high affinity for iron and copper and lower affinity for zinc.



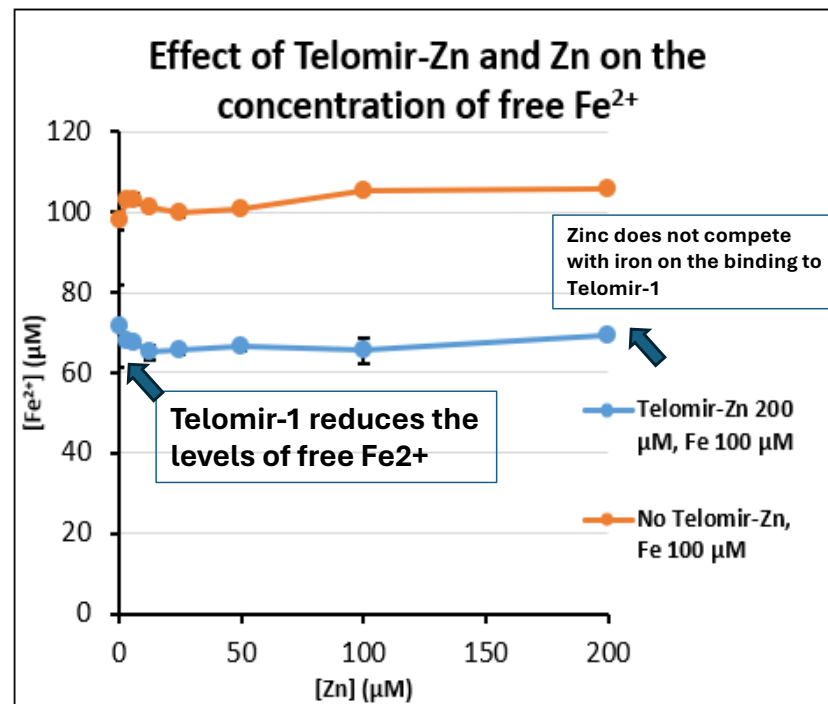
PRE-CLINICAL RESEARCH

Effective iron and copper chelation

Our molecule, Telomir-1 can effectively bind and chelate several ions. It has high affinity for iron and copper and lower affinity for zinc.



Copper replaces bound Iron to Telomir-1

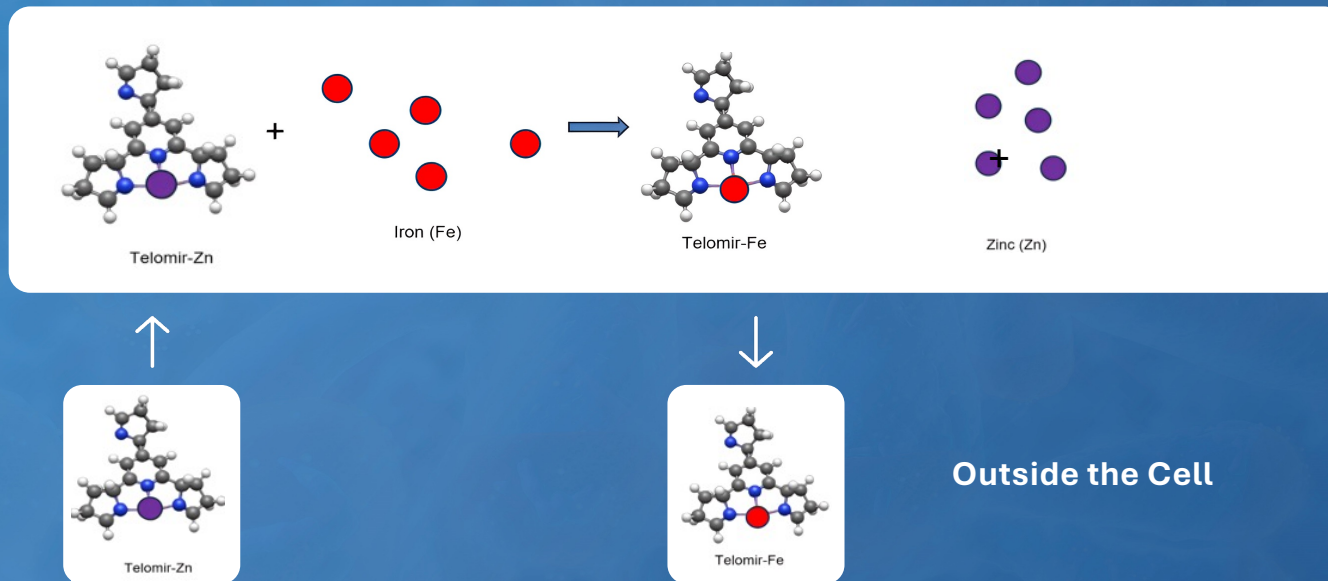


Zinc does not replace bound Iron to Telomir-1

PRE-CLINICAL RESEARCH

Mechanisms of action

Telomir-Zn, a lipophilic form of Telomir-1, readily enters cells and leverages its higher affinity for iron and copper over zinc to exchange harmful iron or copper ions with beneficial zinc. This exchange mechanism allows Telomir-Zn to chelate and remove excess iron or copper from cells while delivering protective zinc, aiding in cellular detoxification and restoring metal balance.



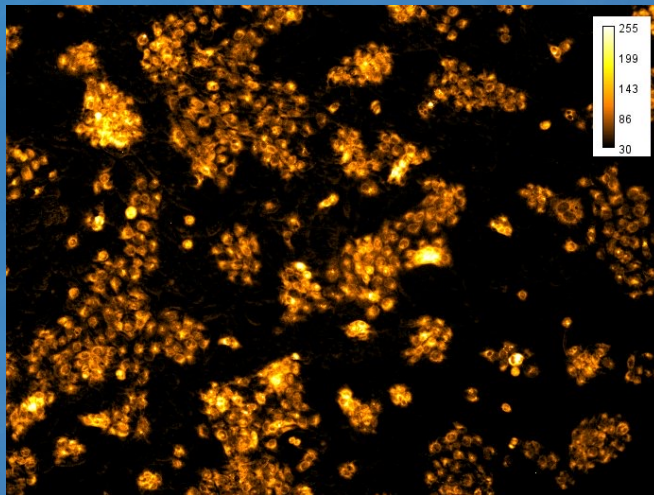
PRE-CLINICAL RESEARCH

Mechanisms of action –Reduction of intracellular Iron levels. Measurements of intracellular iron in HaCat cells

Direct measurements of the effects of Telomir-1 on intracellular iron, as stained with FerroOrange at 24 hours.

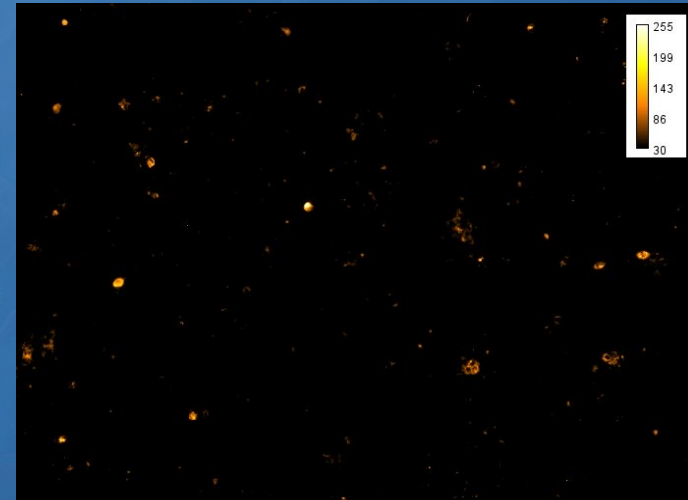
TELOMIRPHARMA.COM

High intracellular iron (yellow color)



No treatment

Reduced intracellular iron (yellow color)

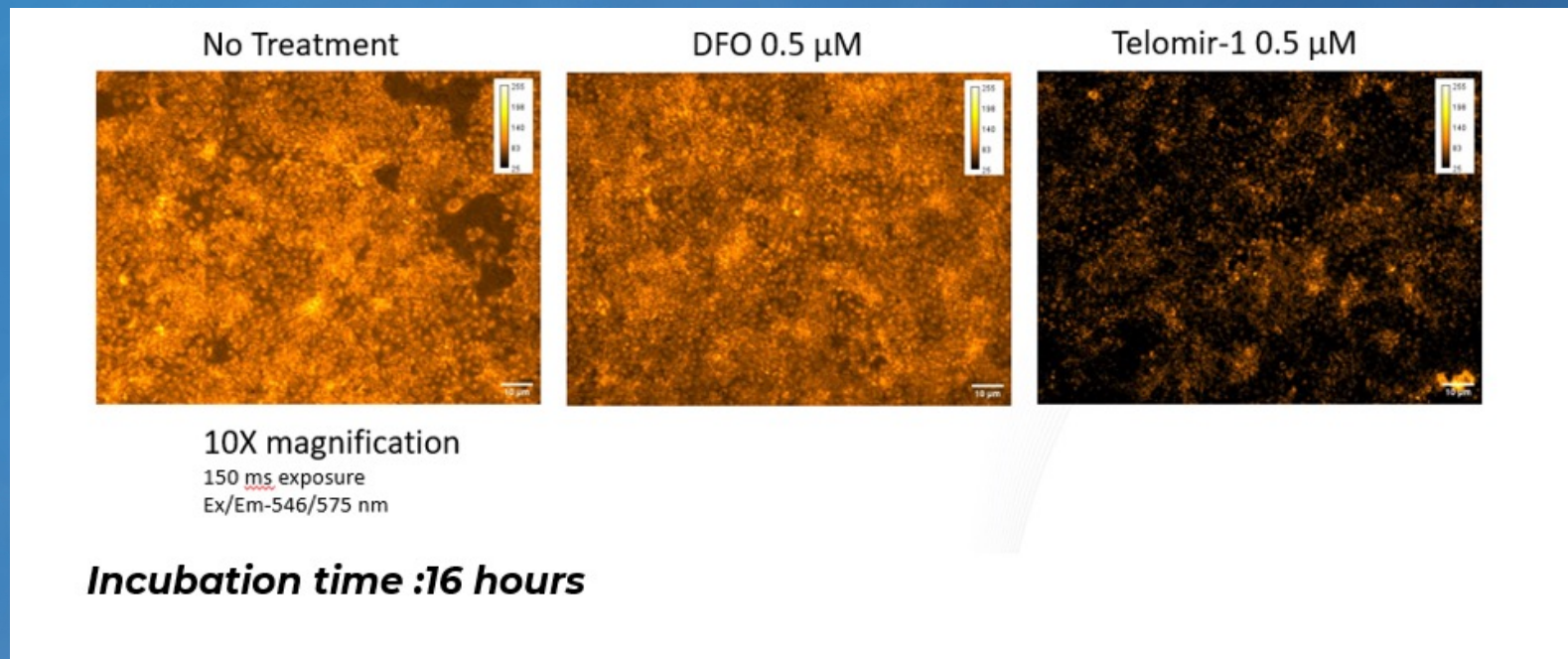


Telomir-1 at 1 µM

PRE-CLINICAL RESEARCH

Mechanisms of action –Reduction of intracellular Iron levels. Measurements of intracellular iron in HaCat cells

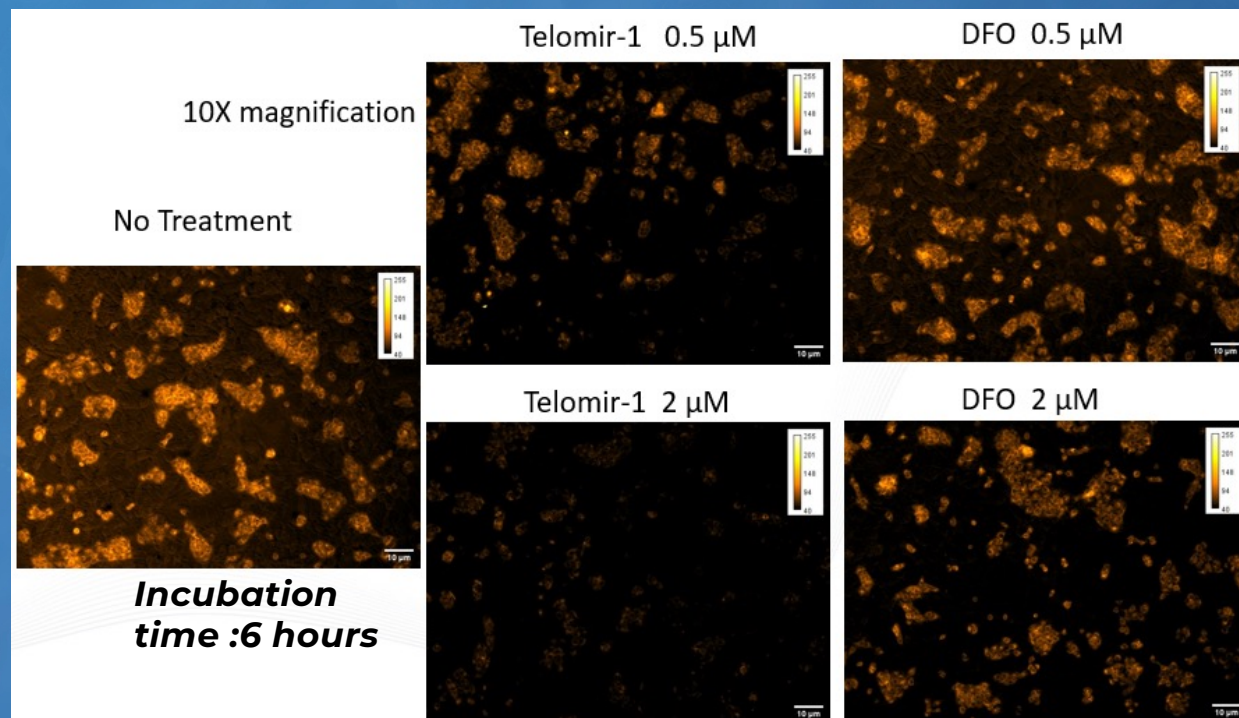
Direct measurements of the effects of Telomir-1 and DFO on intracellular iron, as stained with FerroOrange at 16 hours. While Deferoxamine (DFO) is more potent chelator in vitro, it is less effective than Telomir-1 in reducing intracellular iron levels



PRE-CLINICAL RESEARCH

Mechanisms of action –Reduction of intracellular Iron levels. Measurements of intracellular iron in HaCat cells

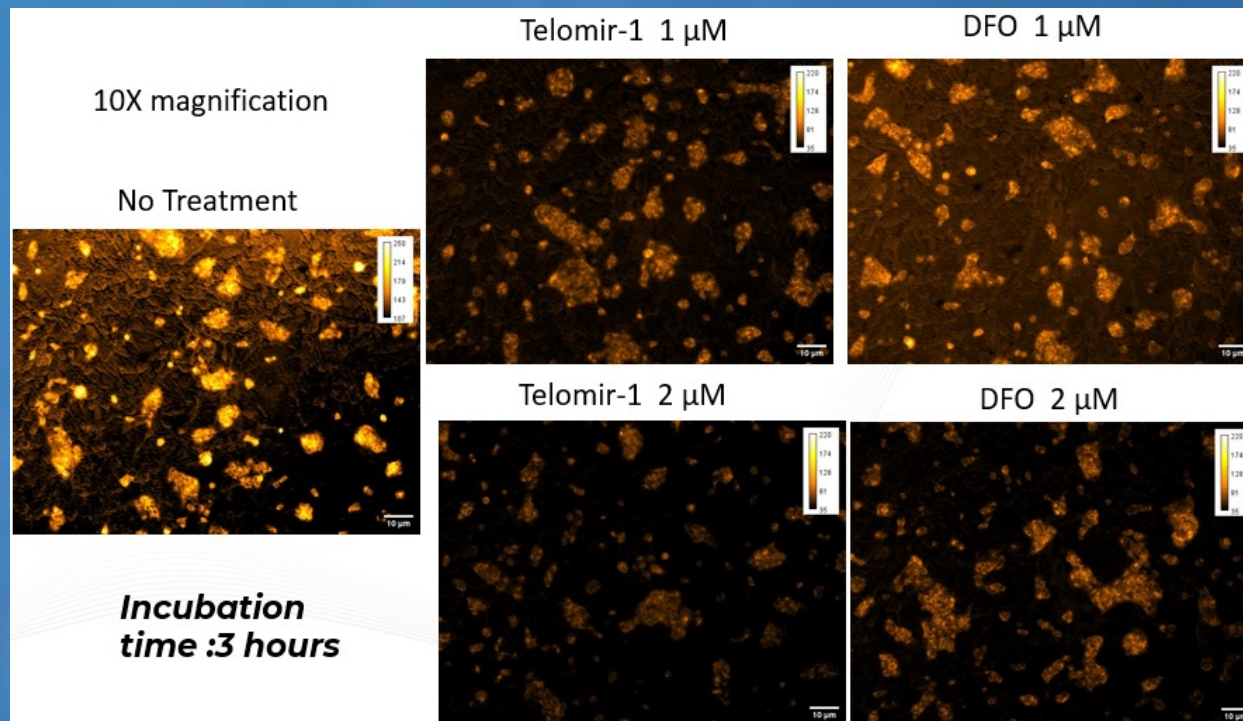
Direct measurements of the effects of Telomir-1 and DFO on intracellular iron, as stained with FerroOrange at 6 hours. While Deferoxamine (DFO) is more potent chelator in vitro, it is less effective than Telomir-1 in reducing intracellular iron levels



PRE-CLINICAL RESEARCH

Mechanisms of action –Reduction of intracellular Iron levels. Measurements of intracellular iron in HaCat cells

Direct measurements of the effects of Telomir-1 and DFO on intracellular iron, as stained with FerroOrange at 3 hours. While Deferoxamine (DFO) is more potent chelator in vitro, it is less effective than Telomir-1 in reducing intracellular iron levels



PRE-CLINICAL RESEARCH

Aging and longevity research

- *Telomers*
- *Life-span and activity*
- *Mechanisms of action*

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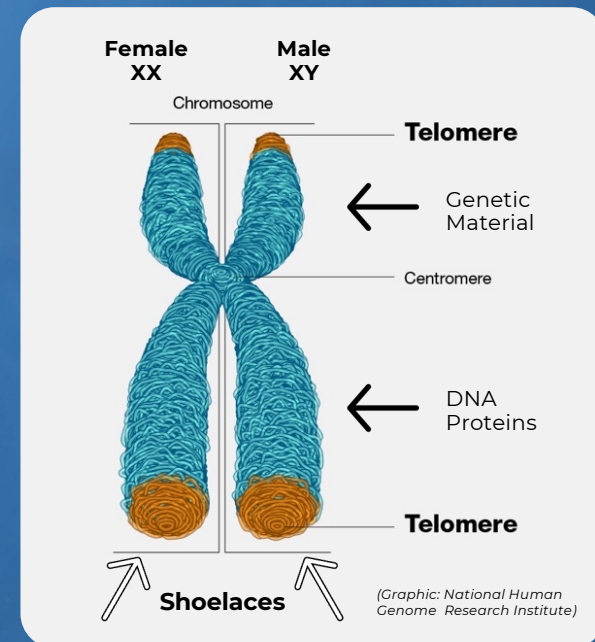


WHAT ARE TELOMERES?

Age Reversal and Increased Longevity

Telomeres are the protective end caps of a chromosome made up of DNA sequences and proteins (TTAGGG).

- Telomeres protect chromosome ends during cell division, preventing gene loss, like shoelace caps.
- This could alter genes, causing cell death, cancer, or diseases.
- Telomeres shorten with age, and metal reactivity speeds this, raising the risk of age-related diseases.



Telomir-1 Restores Telomere Length in Accelerated Aging Models

Telomir-1 reverses telomere shortening and restores length beyond wild-type levels in models mimicking Progeria and Werner syndromes.



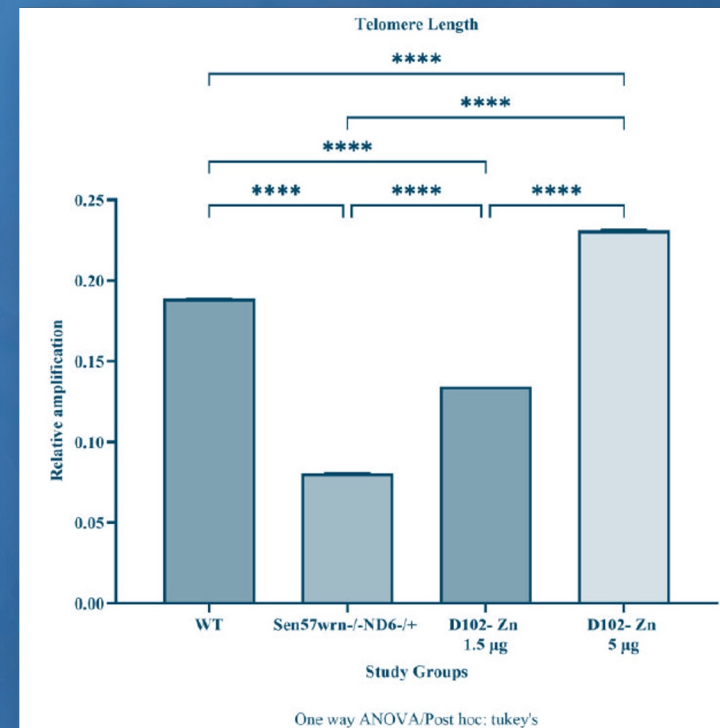
Reversal by Telomir-1 of shortened telomere length in an accelerated aging model in zebra fish

Study Model & Design

- Zebrafish model exhibiting rapid telomere loss and aging, mimicking biology of both Progeria and Werner syndromes
- **Dosing:** Oral Telomir-1 for 14 days

Key Results

- Untreated fish showed severe telomere shortening
- Telomir-1 significantly and dose-dependently reversed this, surpassing wild-type telomere length



Telomir-1 treatment dose-dependently reversed telomere shortening in aged zebrafish, restoring levels beyond wild-type controls.

PRE-CLINICAL RESEARCH

Age Reversal and Increased Longevity



Study Overview

This preclinical study, in collaboration with Nagi Bioscience, used an in vivo microfluidic-based assay to evaluate Telomir-1 in *Caenorhabditis elegans*, a well-established aging model. The platform enabled real-time tracking of lifespan, healthspan, and mobility decline, allowing precise measurement of Telomir-1's effects.

Telomir-1 significantly enhanced lifespan and health metrics in aged organisms.

Key Findings Included:

Increased Lifespan:

Telomir-1 extended survival by ~2.5 days at high dose

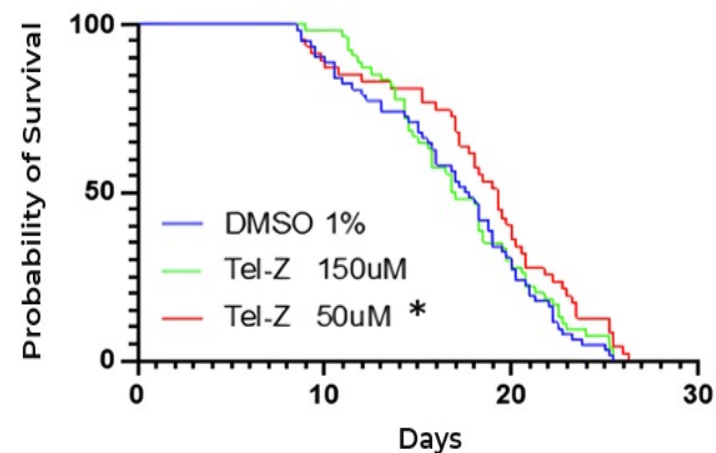
Enhanced Motility:

Improved motility in aged organisms indicates a slowdown in biological decline

Reduced Biological Aging:

Reversal of key age-related markers supports Telomir-1's potential as a longevity therapy

High-dose Telomir-1 significantly extended nematode lifespan by ~2.5 days, demonstrating a strong longevity effect.



PRE-CLINICAL RESEARCH

Age Reversal and Increased Longevity



Study in Adult Progeria - Summary and Methodology

The preclinical study with Nagi Bioscience SA used *C. elegans* with a *wrn-1* gene mutation, the human equivalent of which is linked to Werner Syndrome (Adult Progeria), showing reduced life expectancy compared to wild-type nematodes.

Key Findings Included:

Increased Lifespan:

The study demonstrates significant age-reversal effects in *wrn-1* mutated nematodes treated with Telomir-1. It was demonstrated that this treatment was capable to effectively bring the longevity level back to levels which are not significantly different from normal animals

Enhanced Mobility in Older Organisms:

These effects include an extended healthy lifespan and normalization of several other physiological parameters such as movement velocity and tail amplitude.

Telomir-1 normalizes reduced viability induced by copper and iron in progeria cells

Protective and regulatory effect observed at low Telomir-1 concentrations

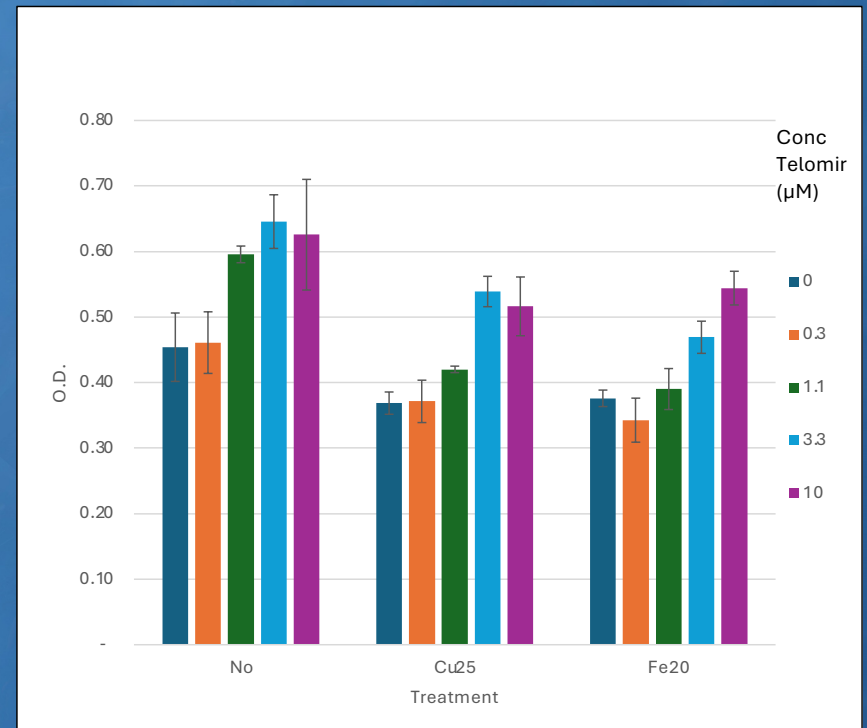


Study Summary:

- Progeria fibroblasts were treated with increasing concentrations of Telomir-1 following exposure to copper (Cu) and iron (Fe).
- Viability/metabolic activity was measured after 24 hours to assess the compound's protective effects.

Key Findings:

- In untreated cells, viability increased dose-dependently with Telomir-1 exposure.
- Copper and iron exposure reduced viability by ~20%, indicating cellular stress.
- At 1.1 μM and above, Telomir-1 reversed this reduction, restoring viability to levels above the untreated control.
- The effective concentrations of Telomir-1 were significantly lower than those of the metal ions, suggesting a regulatory mode of action rather than simple chelation.



Telomir-1 restores cell viability suppressed by copper and iron, with a reversal threshold beginning at 1.1 μM . These effects are consistent with a regulatory action rather than chelation.

Telomir-1 Reduces ROS and Reverses Oxidative Stress Induced by Copper and Iron

ROS modulation observed under both basal and metal-induced stress conditions

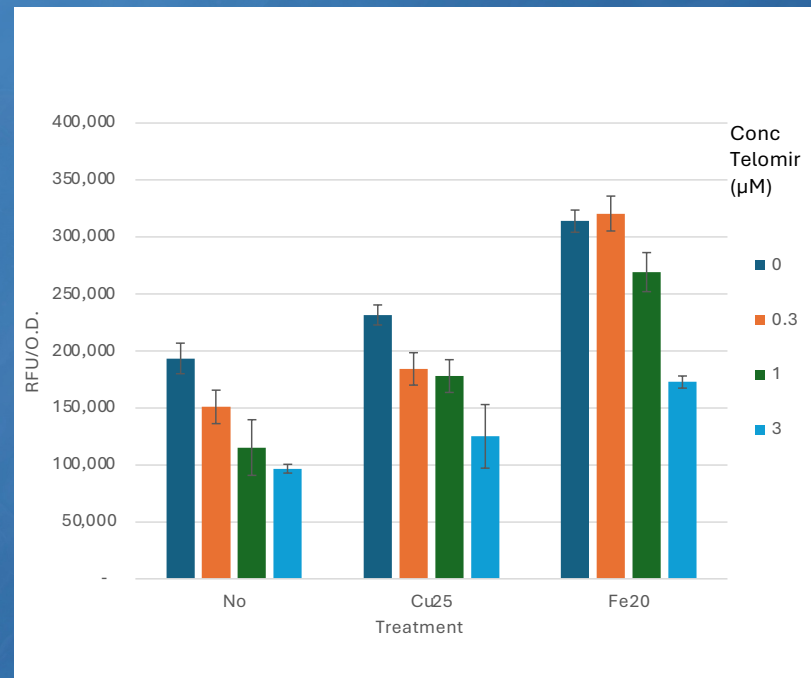


Study Summary:

- Progeria fibroblasts were evaluated for reactive oxygen species (ROS) levels 24 hours after treatment with varying concentrations of Telomir-1.
- Cells were also challenged with copper (Cu) and iron (Fe) to assess oxidative stress induction and Telomir-1's protective response.

Key Findings:

- Under basal (unchallenged) conditions, Telomir-1 caused a marked, dose-dependent reduction in ROS, indicating strong intrinsic antioxidant properties.
- Both copper and iron exposure led to significant ROS elevation, with iron showing a more pronounced effect.
- Telomir-1 reversed this ROS increase in a concentration-dependent manner, restoring levels close to or below untreated baseline.



Telomir-1 reduces ROS in both unchallenged and Cu/Fe-challenged progeria cells. ROS elevation induced by metals is reversed at 1 μM and 3 μM doses, supporting a potent protective antioxidant response.

Telomir-1 Protects Progeria Cells from Multiple Mitochondrial Stressors

Dose-dependent normalization of intracellular calcium linked to mitochondrial protection



Study Summary:

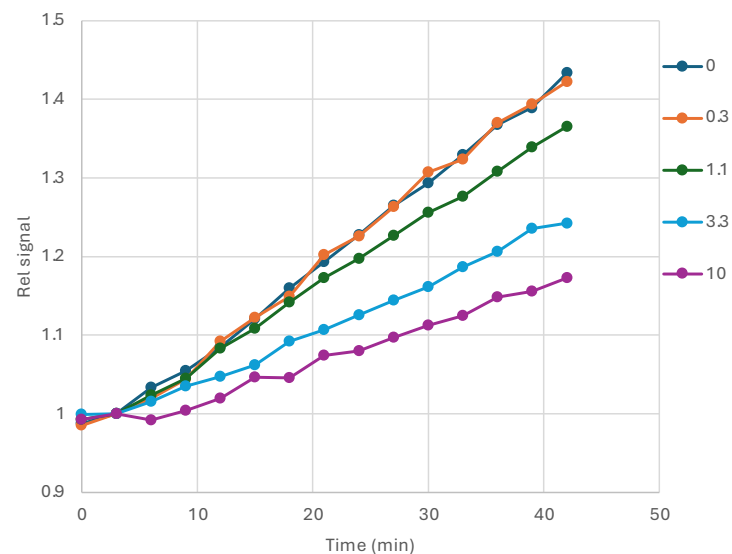
- We studied the effects of Telomir-1 on Progeria fibroblasts, evaluating key parameters of mitochondrial health and cellular viability, including: Reactive Oxygen Species (ROS), Intracellular calcium levels, and Cell viability/metabolic activity
- To model cellular stress, fibroblasts were challenged with copper (Cu) and iron (Fe) — two metals known to disrupt redox balance and mitochondrial function.

Key Results:

- Copper and iron exposure: Increased ROS and intracellular calcium levels and Decreased cell viability

Conclusion:

- Telomir-1 acts as a multi-target protective agent in Progeria cells**, countering the noxious effects of copper and iron. These results support its potential to **normalize mitochondrial function, reduce oxidative and calcium stress, and restore cell health** in models of accelerated aging.



Telomir-1 reduces intracellular calcium levels induced by iron exposure. The calcium rise is progressively blunted at increasing Telomir-1 concentrations, indicating dose-dependent mitochondrial protection.

Telomir-1 activates Mitochondrial activity in Progeria Cells

Dose-dependent normalization of intracellular calcium linked to mitochondrial protection



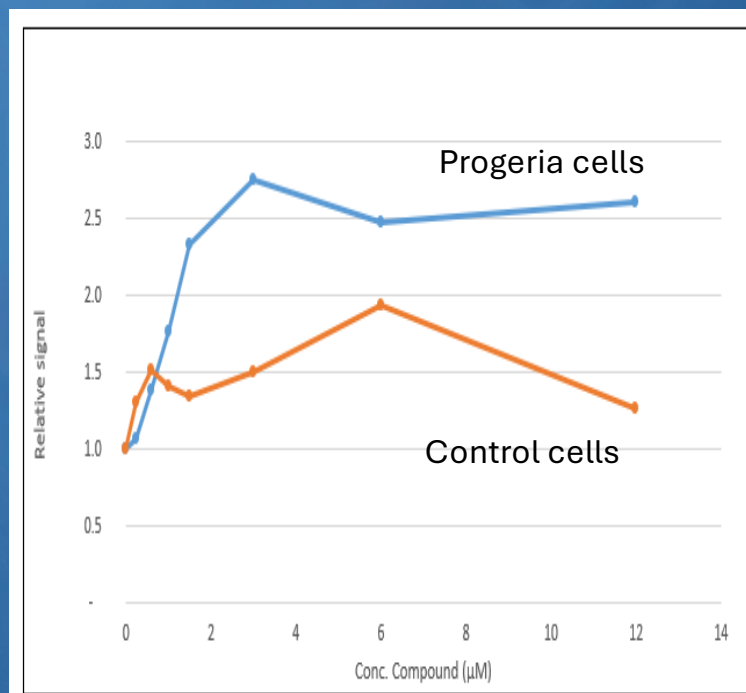
Study Summary:

- We studied the effects of Telomir-1 on Progeria fibroblasts, evaluating key parameters mitochondrial metabolic activity compared to cell proliferation

Key Results:

- Under the experimental conditions, Progeria cells and control fibroblast do not proliferate under Telomir-1 (Calcein study)
- However, progeria cells selectively show a marked, but saturated increase in mitochondrial activity

Augmentation of mitochondrial activity



Telomir-1 selectively increase mitochondrial activity in Progeria cells, with only limited effect in control fibroblasts

Telomir-1 activates Mitochondrial activity in Progeria Cells

Dose-dependent normalization of intracellular calcium linked to mitochondrial protection



Study Summary:

- We studied the effects of Telomir-1 on Progeria fibroblasts, evaluating key parameters mitochondrial metabolic activity compared to cell proliferation

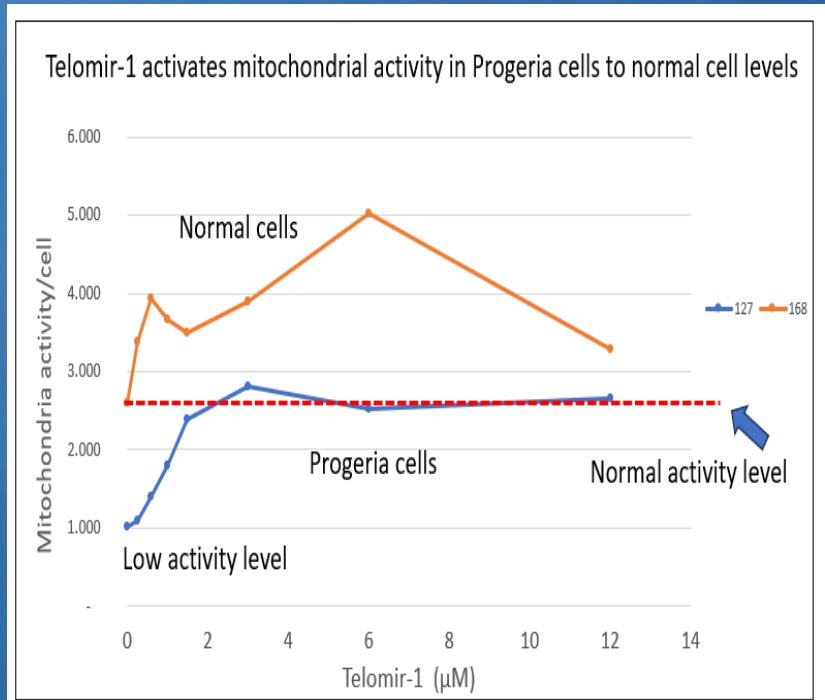
Key Results:

- Under the experimental conditions, Progeria cells and control fibroblast do not proliferate under Telomir-1 (Calcein study)
- The lower basal mitochondrial activity in Progeria cells was increase to levels as in normal cells

Conclusion:

Telomir-1 acts as a multi-target protective agent in Progeria cells, countering the noxious effects of copper and iron. This unique cellular activity may help restore energy balance and improve essential cell functions such as protein synthesis and membrane stability in disease cells, where mitochondrial failure and oxidative stress drive progression.

Augmentation of mitochondrial activity



Telomir-1 selectively increase mitochondrial activity in Progeria cells, to normal cell levels

PRE-CLINICAL RESEARCH



Significant Anti-Cancer Activity in Prostate Cancer Model

Telomir-1 Demonstrates Promising Efficacy in Aggressive Human Prostate Cancer Model (PC3 cells)

Key Study Highlights:

Model Used: Preclinical in vitro study using human prostate cancer PC3 cells

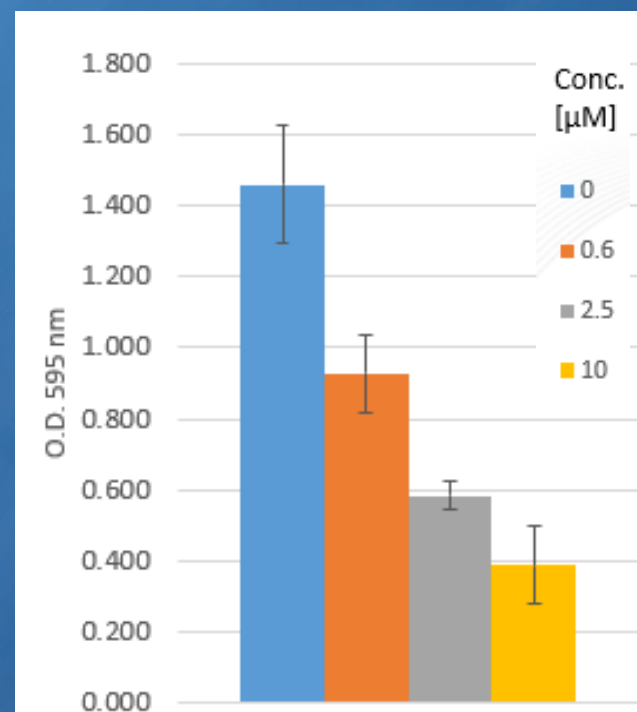
Results:

- Telomir-1 significantly inhibited tumor growth in highly aggressive prostate cancer (PC3 cells)

Significance:

- Suggests Telomir-1 may hold potential as a novel therapeutic candidate for hormone-independent, advanced prostate cancer
- Supports further investigation into Telomir-1 as a multi-indication oncology asset

PC3 tumor growth in vitro



PRE-CLINICAL RESEARCH

Cancer Research

- *Anti-cancer activity*
- *Epi-genetics*
- *Mechanisms of action*

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PRE-CLINICAL RESEARCH

Significant Anti-Cancer Activity in **Triple negative breast cancer (TNBC)** cells



Telomir-1 kills TNBC cells and this effect is protected by iron

Key Study Highlights:

Model Used: In vitro studies in human TNBC cells (MDA MB 231). Viable cells were counted with incubation for 72 hours with increasing concentrations of Telomir-1, with and without FeCl_2 (10 μM)

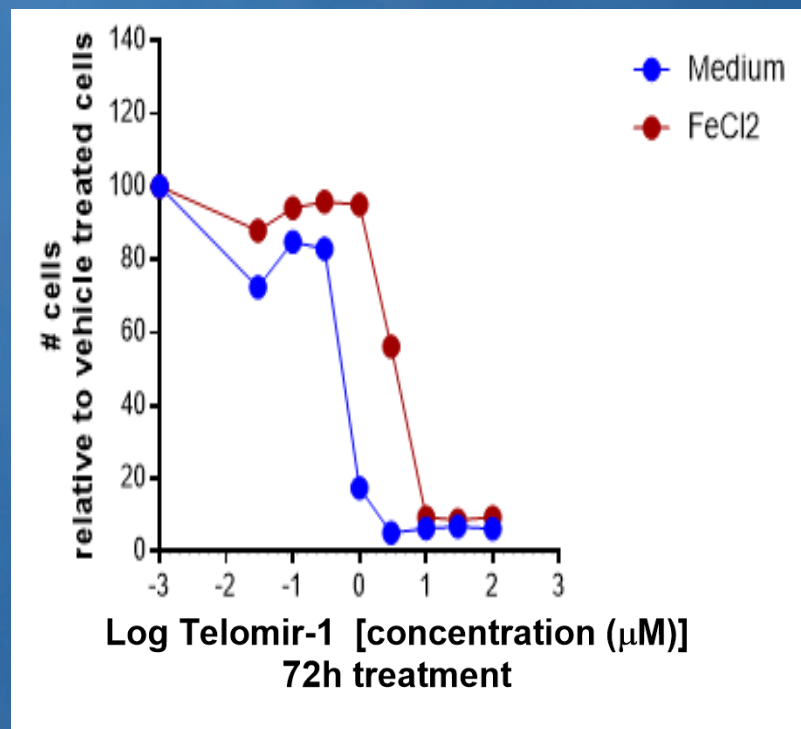
Results:

- Telomir-1 reduced cell viability as from 1 μM . This effect was protected (partially) by the presence of iron ions.

Significance:

- Suggests Telomir-1 may hold potential as a novel therapeutic candidate for TMBC tumors
- The iron dependency confirmed that iron metabolism contributes significantly to this phenotype. Telomir-1 appears to exploit a fundamental metabolic vulnerability unique to these tumor cells

MDA MB 231 cell viability



PRE-CLINICAL RESEARCH

Significant Anti-Cancer Activity in Prostate Cancer Model

Telomir-1 Demonstrates Promising Efficacy in Aggressive Human Prostate Cancer Model

Key Study Highlights:

Model Used: Preclinical in vivo study using human prostate cancer PC3 cells in murine xenograft model
Telomir-1 was administered orally daily for 21 days to mice implanted with PC3 cells

Results:

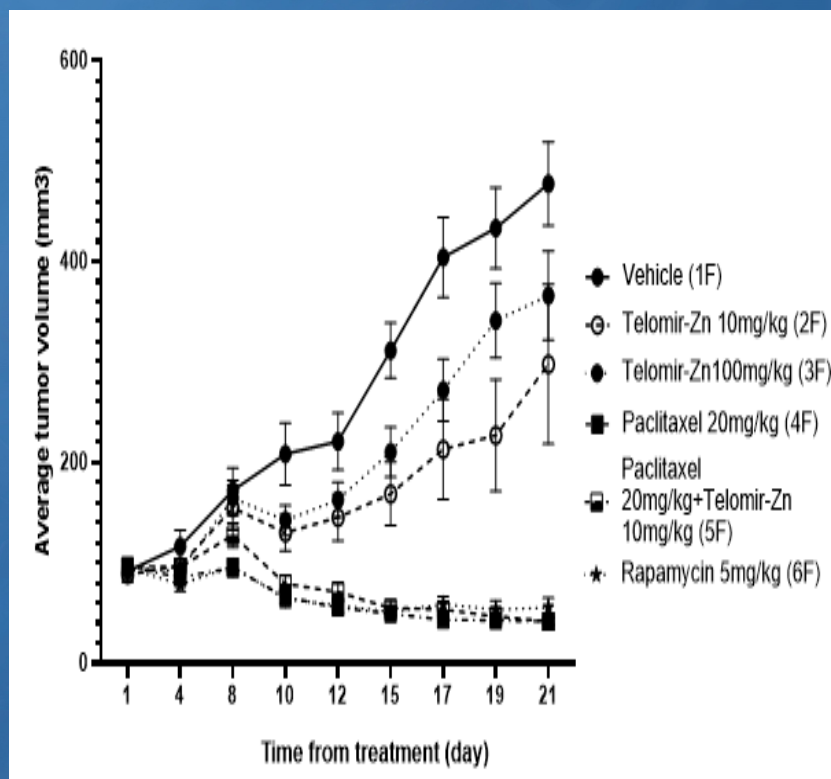
- Telomir-1 showed tumor volume suppression, lower but comparable to standard chemotherapeutics (Paclitaxel)

Significance:

- Suggests Telomir-1 may hold potential as a novel therapeutic candidate for hormone-independent, advanced prostate cancer
- Supports further investigation into Telomir-1 as a multi-indication oncology asset



PC3 tumor growth in mice



PRE-CLINICAL RESEARCH

Significant Anti-Cancer Activity in Prostate Cancer Model

Exploring the role of DNA-methylation in Aggressive Human Prostate Cancer Model

Key Highlights:

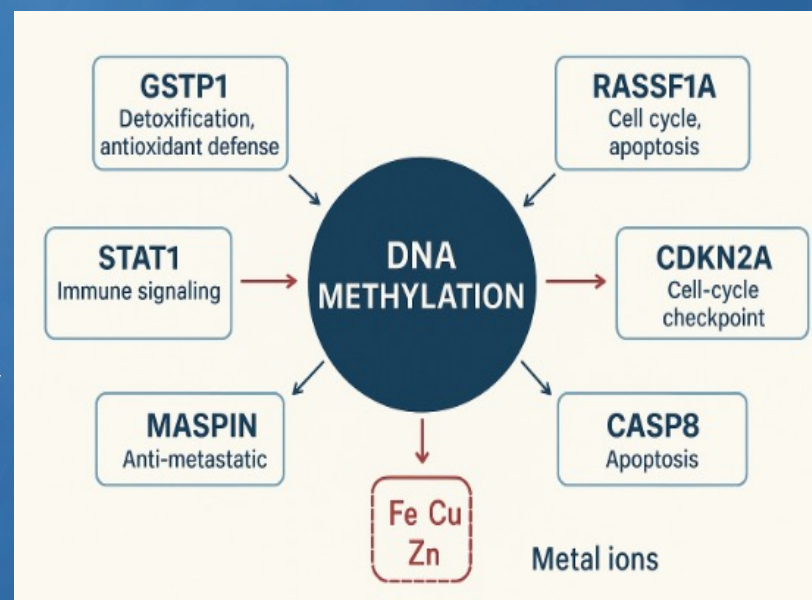
In prostate cancer, aberrant DNA methylation is a central epigenetic event that inactivates tumor suppressor genes and alters cellular detoxification, apoptosis, and metastasis control. Several key genes—including SMTA1, GSTP1, CDKN2A, RASSF1A, CASP8, and MASPIN—These proteins/genes are predominantly tumor suppressors whose expression is silenced by promoter hypermethylation which contributes substantially to malignancy progression

Model Used: Preclinical in vivo study using human prostate cancer PC3 cells in murine xenograft model. Measuring DNA methylation at day 10 and 21 of tumor progression and treatment

Significance: Inhibition of these DNA-methylation releases their function suppression and enables their functions and cellular protection



DNA methylation of key proteins and their respective functions



Copper enhance methylation through oxidative stress and activation of DNA methyltransferases (DNMTs), while **zinc** plays a stabilizing or regulatory role

PRE-CLINICAL RESEARCH

Significant Anti-Cancer Activity in Prostate Cancer Model



Telomir-1 reduces Stat-1
DNA methylation at day 21

Telomir-1 Demonstrates Promising reduction of STAT1 hypermethylation in Aggressive Human Prostate Cancer Model

Key Study Highlights:

STAT1 (Signal Transducer and Activator of Transcription 1)

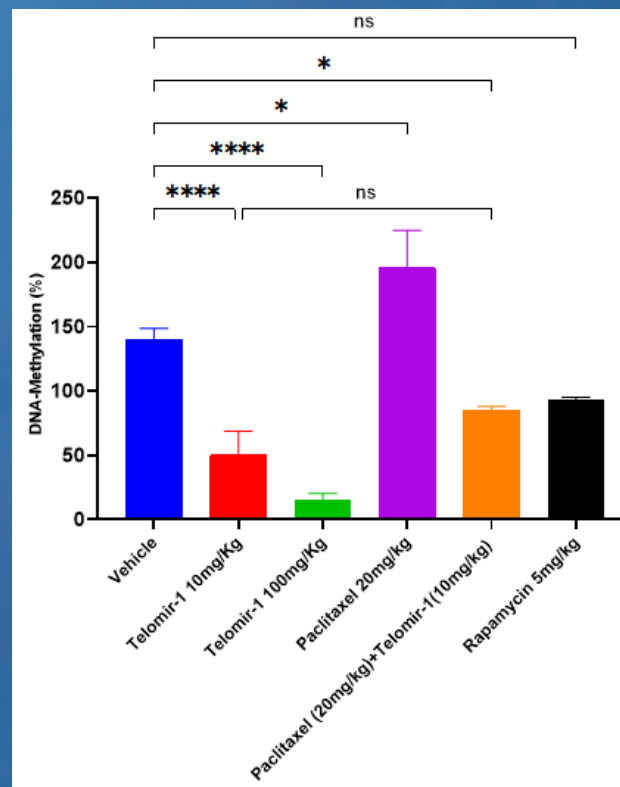
- **Function:** Mediates interferon (IFN)-dependent **anti-proliferative** and **immune surveillance** pathways.
- **Effect of inhibition:** Reduced immune response and lower tumor immunogenicity.
- **Metal ion link:** Copper and iron influence JAK/STAT pathway activation via oxidative or redox-sensitive mechanisms. Excess iron can suppress interferon signaling and STAT1 activation indirectly.

Results:

- Telomir-1 showed a marked suppression of STAT1 DNA methylation in PC3 cells
- Paclitaxel and Rapamycin had no or a lower effect on STAT1 DNA methylation

Significance:

- Telomir-1 can restore STAT1 transcription and resensitize tumor cells to chemo- and immunotherapies.



PRE-CLINICAL RESEARCH

Significant Anti-Cancer Activity in Prostate Cancer Model



Telomir-1 Demonstrates reduction of CDKN2A hypermethylation in Aggressive Human Prostate Cancer Model

Key Study Highlights:

The CDKN2A gene encodes the tumor suppressor proteins, which regulate cell cycle progression and prevent uncontrolled proliferation. Promoter hypermethylation in ~30-50% of Prostate cancer cases, more frequent in aggressive subtypes.

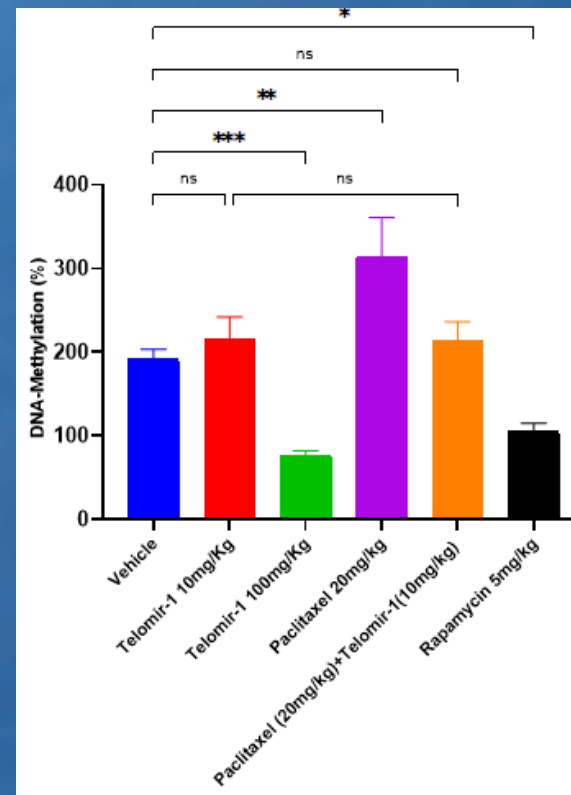
Results:

- Telomir-1 at the high dose showed a marked suppression of CDKN2A DNA methylation in PC3 cells
- Paclitaxel and Rapamycin had no or a lower effect on STAT1 DNA methylation

Significance:

- Telomir-1 can restore CDKN2A transcription which can reactivate its tumor suppressive functions, induce cell cycle arrest, and sensitize PC3 cells to apoptosis or senescence

Telomir-1 reduces CDKN2A DNA methylation at day 21



PRE-CLINICAL RESEARCH

Significant Anti-Cancer Activity in Prostate Cancer Model



Telomir-1 Demonstrates reduction of TSM1 hypermethylation in Aggressive Human Prostate Cancer Model

Key Study Highlights:

TSM1 (also known as ASC) undergoes frequent methylation-induced silencing in human breast cancers, contributing to the pathogenesis of invasive disease by interrupting programmed cell death mechanisms. Aberrant methylation of the TSM1/ASC CpG island is found in a significant fraction (24–40%) of breast cancer cases, but is not present in normal breast tissue, suggesting its role as an early epigenetic marker for transformation and tumor progression.

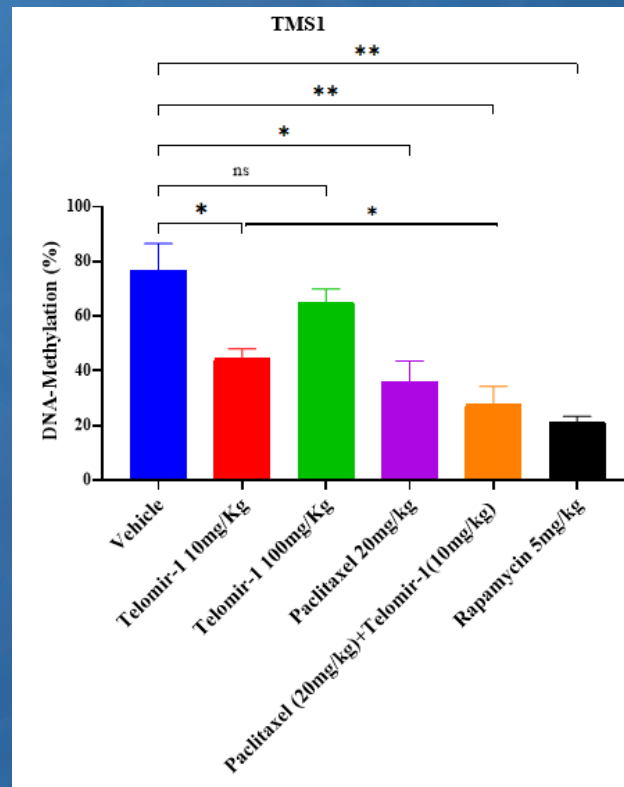
Results:

- Telomir-1 at the low dose showed a significant suppression of TSM1 DNA methylation in PC3 cells

Significance:

- Telomir-1 can restore TSM1 transcription which can reactivate its tumor suppressive functions

Telomir-1 reduces TSM1 DNA methylation at day 21



PRE-CLINICAL RESEARCH

Significant Anti-Cancer Activity in Prostate Cancer Model



Telomir-1 Demonstrates reduction of GSTP1 hypermethylation in Aggressive Human Prostate Cancer Model

Key Study Highlights:

GSTP1 (Glutathione S-Transferase Pi 1)

- **Function:** Detoxification enzyme that conjugates glutathione to electrophilic compounds and protects DNA from oxidative damage.
- **Methylation in PCa:** One of the **most frequently hypermethylated genes** in prostate cancer (seen in >90% of cases).
- **Effect of methylation:** Silencing of GSTP1 → accumulation of reactive oxygen species (ROS) → DNA damage → further genomic instability.
- **Metal ion link:** Iron and copper enhance ROS, which increase oxidative stress and may drive methylation of GSTP1 promoter.

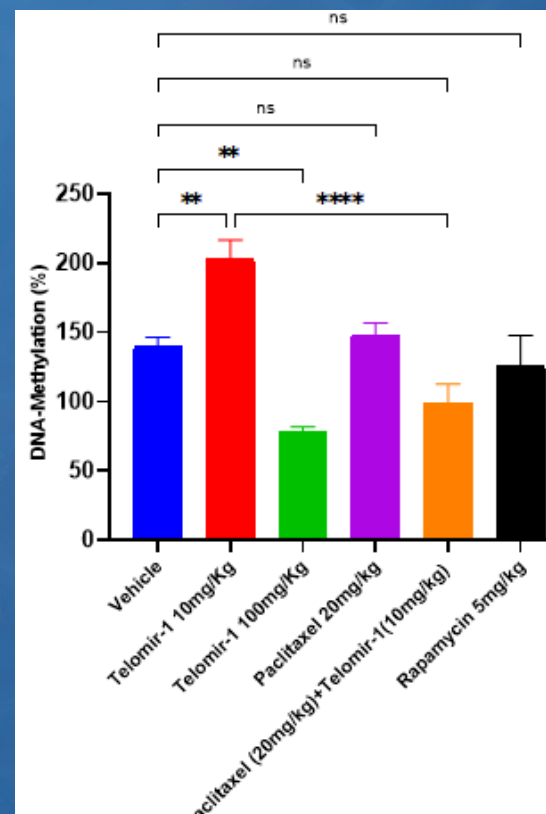
Results:

- Telomir-1 at the high dose showed a marked suppression of GSTP1 DNA methylation in PC3 cells
- Paclitaxel and Rapamycin had no or a lower effect on GSTP1 DNA methylation

Significance:

- Telomir-1 can restore GSTP1 transcription which can reverse genomic instability

Telomir-1 reduces GSTP1 DNA methylation at day 21



PRE-CLINICAL RESEARCH

Significant Anti-Cancer Activity in Prostate Cancer Model



Telomir-1 Demonstrates reduction of RASSF1A hypermethylation in Aggressive Human Prostate Cancer Model

Key Study Highlights:

RASSF1A (Ras Association Domain Family Member 1A)

- **Function:** Tumor suppressor involved in **microtubule stabilization, cell cycle arrest, and apoptosis.**
- **Methylation in PCa:** Frequently hypermethylated, leading to loss of expression and **escape from apoptosis.**
- **Effect of inhibition:** Promotes proliferation, chromosomal instability, and resistance to apoptosis.
- **Metal ion link:** Iron and copper influence oxidative DNA damage and signaling via MAPK and Ras pathways, indirectly promoting RASSF1A silencing. Chelation (e.g., by Telomir-1) can restore RASSF1A expression.

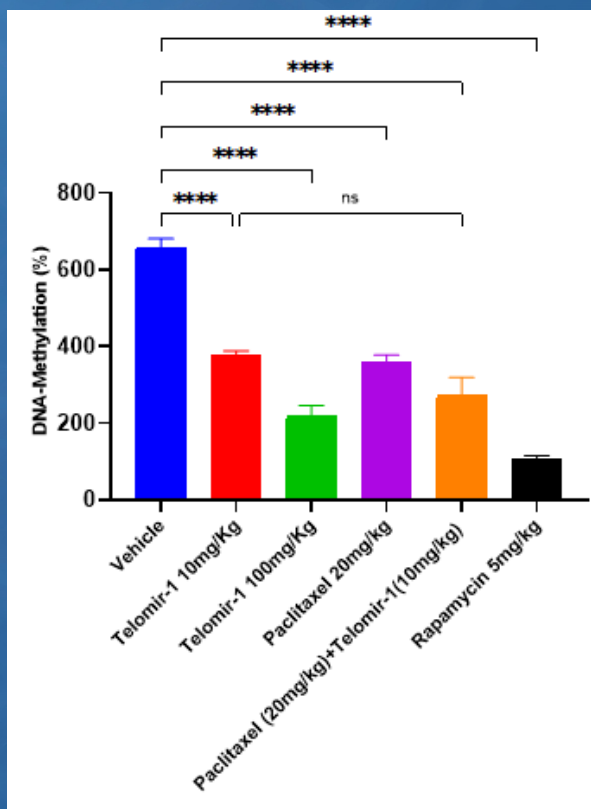
Results:

- Telomir-1 at the high dose showed a marked and dose-dependent suppression of RASSF1A DNA methylation in PC3 cells

Significance:

- Telomir-1 can restore RASSF1A transcription which can reactivate its tumor suppressive functions.

Telomir-1 reduces RASSF1A DNA methylation at day 21



PRE-CLINICAL RESEARCH

Significant Anti-Cancer Activity in Prostate Cancer Model



Telomir-1 Demonstrates reduction of MASPIN hypermethylation in Aggressive Human Prostate Cancer Model

Key Study Highlights:

MASPIN (Serpine B5)

- **Function:** Tumor suppressor **serpin** regulating cell adhesion, invasion, and angiogenesis.
- **Methylation in PCa:** Often **hypermethylated and silenced** in aggressive and metastatic prostate tumors.
- **Effect of inhibition:** Loss of MASPIN facilitates invasion and metastasis.
- **Metal ion link:** Iron may influence MASPIN promoter methylation via oxidative stress. Chelators or demethylating agents can restore expression.

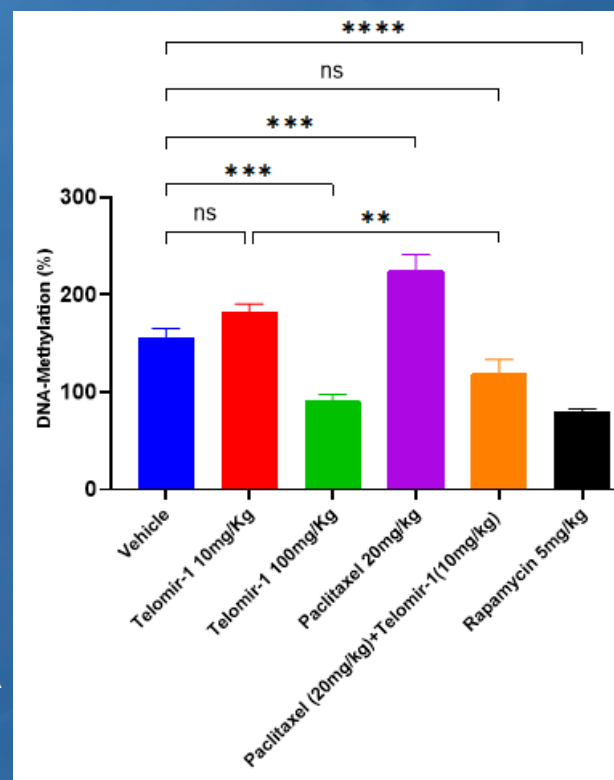
Results:

- Telomir-1 at the high dose showed a marked suppression of MASPIN DNA methylation in PC3 cells
- Paclitaxel and Rapamycin had no or similar effect on MASPIN DNA methylation
- Telomir-1 synergize with Paclitaxel on the inhibition

Significance:

- Telomir-1 can restore MASPIN transcription which can reactivate its metastatic suppressive functions.

Telomir-1 reduces MASPIN DNA methylation at day 21



PRE-CLINICAL RESEARCH

Significant Anti-Cancer Activity in Prostate Cancer Model



Telomir-1 Demonstrates reduction of CASP8 hypermethylation in Aggressive Human Prostate Cancer Model

Key Study Highlights:

CASP8 (Caspase-8)

- **Function:** Initiator caspase of the **extrinsic apoptotic pathway**, activated by death receptors.
- **Effect of inhibition:** Resistance to immune-mediated apoptosis and therapy-induced cell death.
- **Metal ion link:**
- Iron and copper excess induce oxidative stress and may inhibit caspase activity indirectly.

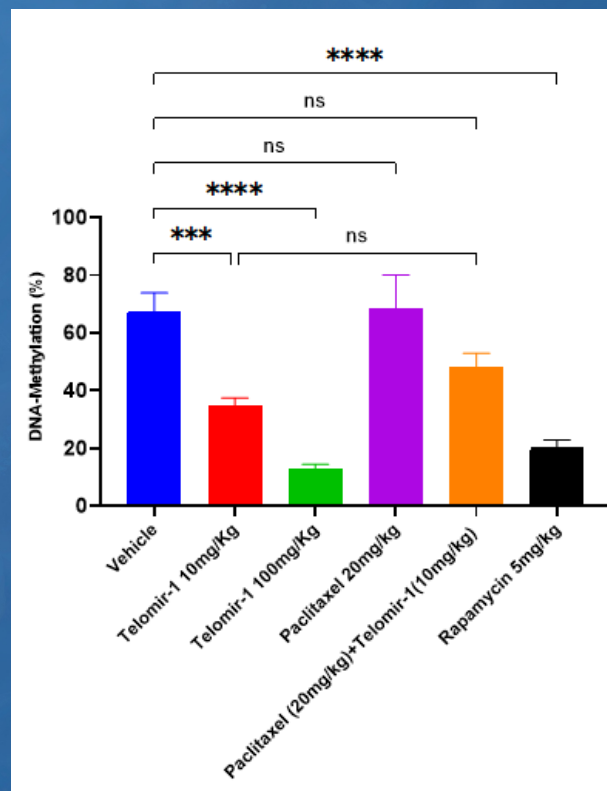
Results:

- Telomir-1 at the high dose showed a dose-dependent suppression of CASP8 DNA methylation in PC3 cells
- Paclitaxel and Rapamycin had no or similar effect on STAT1 DNA methylation
- Telomir-1 synergize with Paclitaxel on the inhibition

Significance:

- Telomir-1 can restore CASP8 transcription which can reactivate its apoptotic functions.

Telomir-1 reduces CASP8 DNA methylation at day 21



PRE-CLINICAL RESEARCH

Interaction with key epigenetic enzymes

Exploring the role of Histone demethylases

Key Highlights:

Histone demethylases (KDMs) remove methyl groups from lysine residues on histone tails, reversing the repressive or activating marks set by histone methyltransferases. Aberrant KDM activity leads to epigenetic deregulation, affecting oncogenes and tumor suppressor genes (TSGs). All three families discussed below are Fe^{2+} , meaning metal ions (iron) are essential cofactors for their activity — linking them directly to cellular redox and metabolic states.

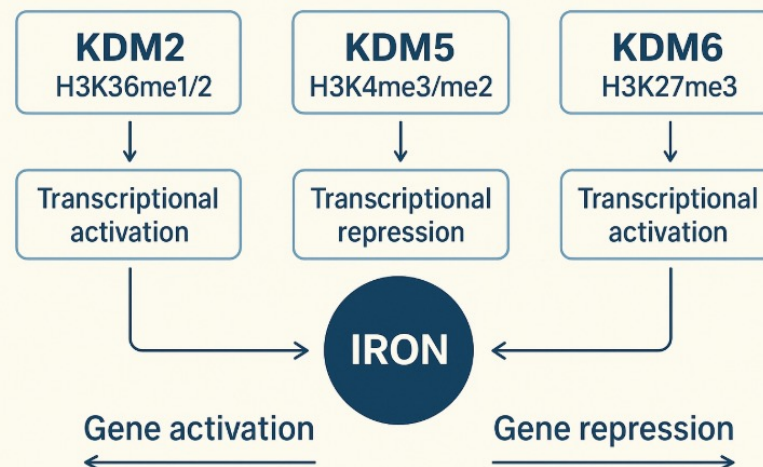
Model Used: Evaluating the effects of Telomir-1 on isolated enzymes of several KDMs families (Eurofins Research)

Significance: All three families reflect a convergence between metal ion metabolism, epigenetic control, and tumor evolution — making them attractive therapeutic targets



Key Histon Demethylase families and their respective functions

KDM2/KDM5/KDM6 IN CANCER



All these enzymes depend on iron for their activity

PRE-CLINICAL RESEARCH

Significant epigenetic Activity



Telomir-1 was shown to inhibit histone demethylases. These are epigenetic enzymes that play critical roles in DNA methylation, gene regulation, cancer, neurodegeneration, metabolic dysfunction, inflammation, and aging

Key Study Highlights:

- **FBXL10 (KDM2B)** – Frequently overactive in aggressive cancers where it allows tumors to maintain “stem-like” properties that drive relapse and treatment resistance.
- **FBXL11 (KDM2A)** – Elevated in several cancers, where it helps tumors grow and evade immune detection.
- **UTX (KDM6A)**, an enzyme that acts like an “eraser” of chemical tags on DNA packaging proteins.
- **JMJD3 (KDM6B)** – A major regulator of inflammation and tumor progression.. By altering histone marks that interact with DNA methylation, JMJD3 fuels metastasis and helps cancers escape immune attack.

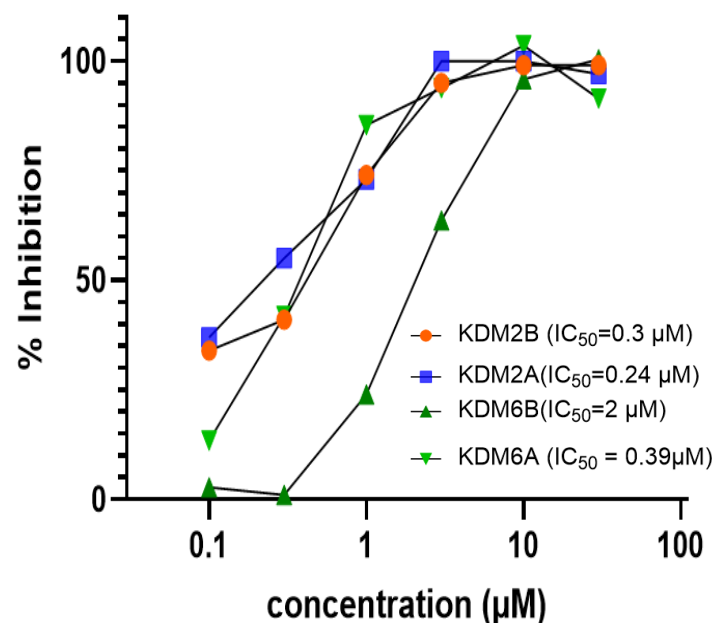
Significance:

these findings support its emerging profile as a broad epigenetic reset therapy that may:

- Help reawaken tumor suppressor genes (STAT1, TMS1).
- Block cancer's growth enablers (FBXL10/11).
- Dial down inflammation (JMJD3/UTX).
- Support more youthful patterns of gene regulation across multiple disease pathways

Telomir-1 potently inhibits Histone demethylases (KDMs) KDM2 and KDM6

Histone demethylases



PRE-CLINICAL RESEARCH

Significant epigenetic Activity



Telomir-1 was shown to also inhibit histone demethylases 5 (JARID1). JARID1A, JARID1B, and JARID1C (also known as KDM5A, KDM5B, and KDM5C) are members of the KDM5 (lysine demethylase 5) family of histone demethylases. They specifically demethylate H3K4me3 and H3K4me2, histone marks that are usually associated with active transcription

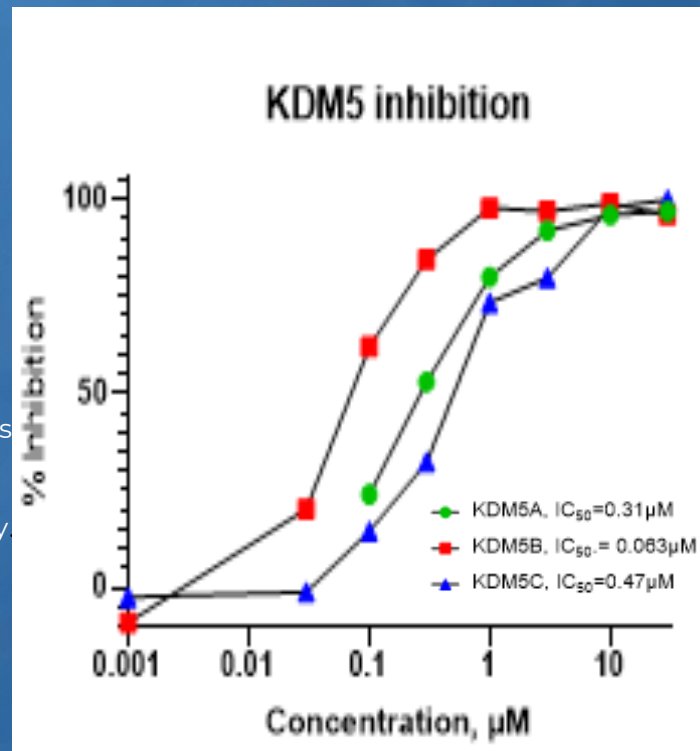
Key Study Highlights:

- **JARID1A (KDM5A)** – Is implicated in tumorigenesis and stem-like cancer cell phenotypes. Its inhibition leads to increased levels of H3K4me3, a marker of transcriptionally active chromatin, at promoters of tumor suppressor genes, enhancing their expression and potentially increasing sensitivity to DNA-damaging agents and ionizing radiation
- **JARID1B (KDM5B)** – Is overexpressed in various cancers and is crucial for cancer stem cell maintenance and drug resistance. Inhibiting JARID1B lowers cell viability, induces cell cycle arrest, upregulates tumor, and impairs DNA damage repair, all contributing to reduced tumor proliferation and suppressors and enhanced sensitivity to chemotherapy and radiotherapy
- **JARID1C (KDM5C)** – Influences genomic stability by regulating heterochromatin structure and silencing non-coding RNAs in these regions. Inhibition or knockdown of JARID1C results in genomic instability, aberrant expression of satellite repeats, and impaired heterochromatin formation, thereby contributing to cancer susceptibility

Significance:

These findings support its emerging profile as a broad epigenetic reset therapy that promotes cancer death and enhanced sensitivity to treatments.

Telomir-1 potently inhibits Histone demethylases 5 (KDM5s, JARID1)



PRE-CLINICAL RESEARCH

Interaction with key epigenetic enzymes

Telomir-1 demonstrated low-level inhibitory activity against Tankyrases (PARP5A and PARP5B). Tankyrases regulate the Wnt/ β -catenin pathway — one of the body's key growth-control circuits that cancers often hijack as a “fuel line” for unchecked growth and treatment resistance.

Key Study Highlights:

Results:

Telomir-1 demonstrated low-level inhibitory activity against Tankyrases (PARP5A and PARP5B). In the low micromolar range

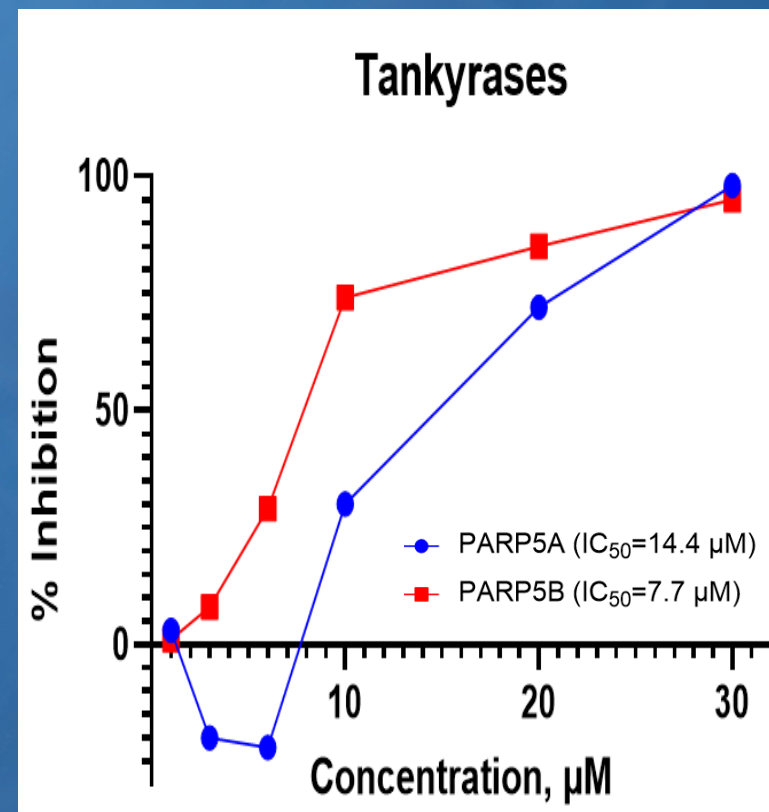
Significance:

Unlike potent Tankyrase inhibitors, which can cause excessive telomere shortening and systemic toxicity, Telomir-1's modest Tankyrase inhibition may provide just enough activity to cut off cancer's fuel line without disrupting healthy telomere biology.

Importantly, previously reported results in a validated Werner Syndrome accelerated-aging model showed that Telomir-1 significantly elongated telomeres beyond healthy levels while also reversing abnormal DNA methylation, restoring youthful gene regulation, and resetting the body's epigenetic clock. This distinction reinforces Telomir-1's differentiated safety profile — demonstrating that, unlike other Tankyrase-targeting drugs, it may protect and lengthen telomeres rather than shorten them.



Telomir-1 only mildly inhibits Tankyrases (PARP5)



PRE-CLINICAL RESEARCH

Interaction with key epigenetic enzymes



Telomir-1 did not show any activity against GCN5L2 (KAT2A), a broad acetyltransferase enzyme, whose inhibition is associated with widespread toxicity. This selective profile may allow Telomir-1 to achieve its effects with a cleaner safety margin than many existing epigenetic drugs. It also does not interact with Cyclin kinase p21.

Key Study Highlights:

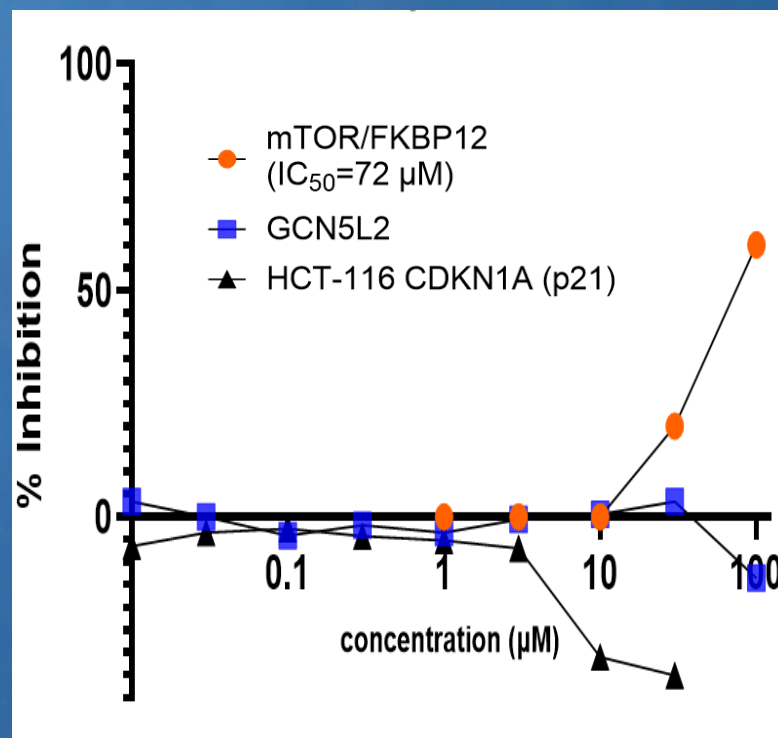
Results:

- Telomir-1 showed a limited inhibition of mTOR and no inhibition of acetyltransferase or cyclin kinase.

Significance:

mTOR inhibitors (e.g., rapamycin, which binds FKBP12 to inhibit mTORC1) show dual benefits in cancer (reducing growth) and aging (extending lifespan), but side effects like immunosuppression limit use; combination with other agents could enhance efficacy. The low affinity towards mTOR and the lack of affinity towards GCN5L2, confers a good safety profile to the product

Telomir-1 has low affinity for mTOR and does not affect GCN5L2 or HCT-116 CDKN1A (p21)



PRE-CLINICAL RESEARCH

Interaction with key epigenetic enzymes



Telomir-1 did not show any direct agonist or antagonist activity against Keap1-Nrf2. The Keap1-Nrf2 pathway is a crucial regulator of cellular defense against oxidative stress

Key Study Highlights:

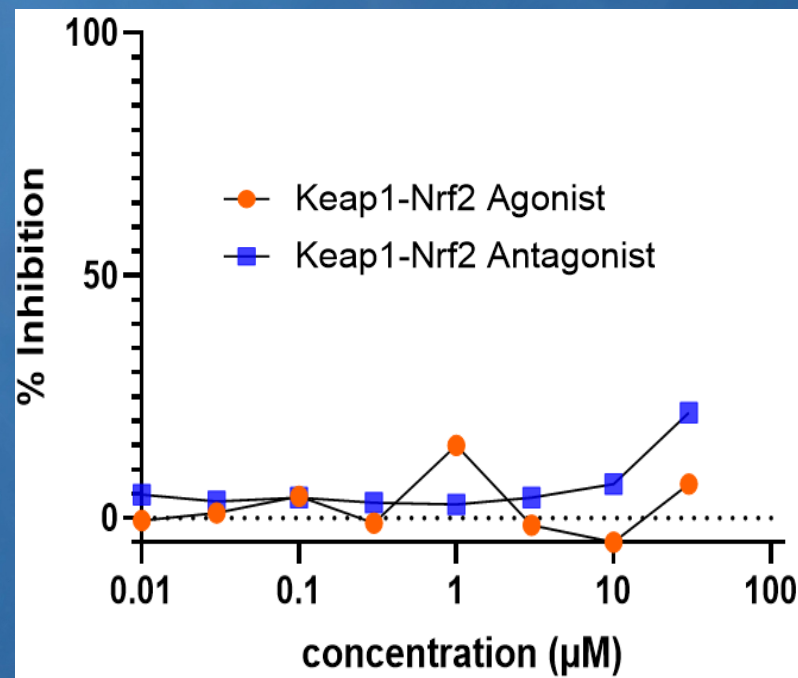
Results:

- Telomir-1 showed no direct agonist or antagonist activity against Keap1-Nrf2.

Significance:

The Keap1-Nrf2 pathway is a crucial regulator of cellular defense against oxidative stress. While Iron and copper are central to redox biology and oxidative stress; deregulated iron status can increase production of reactive oxygen species (ROS), which is counteracted by the antioxidant defenses regulated by Nrf2. All the effects on ROS by Telomir-1 are therefore a result of regulatory effect and no direct interaction at the level of Nrf2

Telomir-1 has no agonist or antagonist activities on Keap1-Nrf2



PRE-CLINICAL RESEARCH

Multifactorial interaction of DNA-methylation, KDMs and Tankyrases on key processes

CANCER

Oncogenic
Tumor promoting
Tumor plasticity
Immune evasion

GSTP1

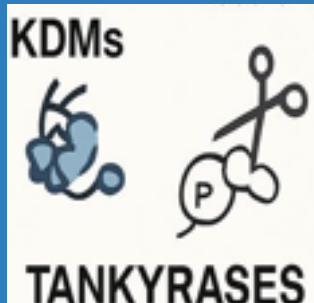
CASP8

RASSF1A

MASPIN

STAT1

CDKN2A



mTOR

AGING

Epigenetic drift
Activation of senescence genes
Accelerated aging
Modulation of telomere length

METABOLISM

Nutrient sensing
Activation of metabolic genes
Interaction with mTOR

INFLAMMATION

Proinflammatory transcription
Affect chronic inflammation

PRE-CLINICAL RESEARCH

Additional Potential Indications

- *Type two diabetes*
- *Age related macular degeneration (AMD)*
- *Wilson's disease*
- *Progeria and Werner's syndrome*



PRE-CLINICAL RESEARCH

Reversal of Key Type 2 Diabetes Parameters



Study Overview and Results

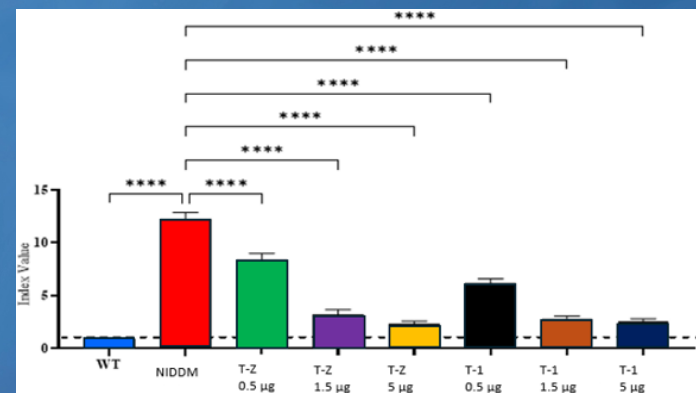
In collaboration with Pentagrit, Telomir evaluated two forms of Telomir-1, administered orally at three different doses, in zebrafish models of Type 2 diabetes mellitus induced by a high-calorie diet. The study assessed key metabolic indicators, including fasting glucose levels, Oral Glucose Tolerance Test (OGTT), insulin concentrations and HOMA-IR.

Key Findings Included:

In a high-fat diet zebrafish model, Telomir-1 (two forms, three doses) normalized fasting blood glucose levels, **restoring glucose balance** in a dose-dependent manner.

Both forms of Telomir-1 **significantly reduced insulin resistance**, restoring HOMA-IR values to near-normal levels—highlighting its therapeutic potential for Type 2 diabetes.

Reversal of insulin resistance (HOMA-IR)



OGTT results confirmed **improved glucose handling**, reinforcing Telomir-1's role in correcting multiple metabolic impairments.

Treated fish also showed **higher survival rates** versus controls, supporting Telomir-1's broad benefits across metabolic health.

Telomir-1™ protects from copper induced oxidative stress in

Retinal cells



Study Overview

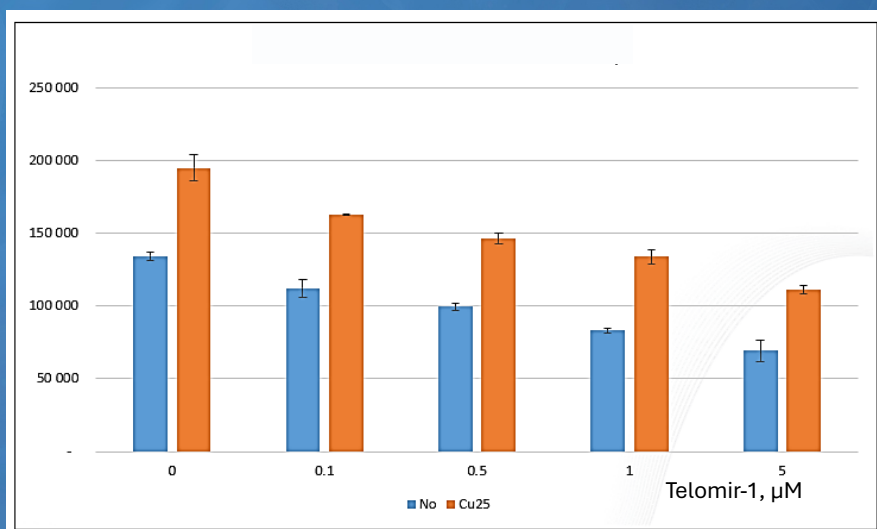
- **Model:** ARPE-19 retinal cells are incubated with varying concentrations of Telomir-1 and without Copper (25 μ M) and levels of reactive oxygen species (ROS) was monitored,

Cellular Recovery

- Telomir-1 by itself reduces ROS level.
- Copper significantly increase ROS levels, and these increase is reversed by Telomir-1

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Oxidative stress in RPE-19 cells



Telomir-1™ protects from copper and iron induced calcium overload in Retinal cells



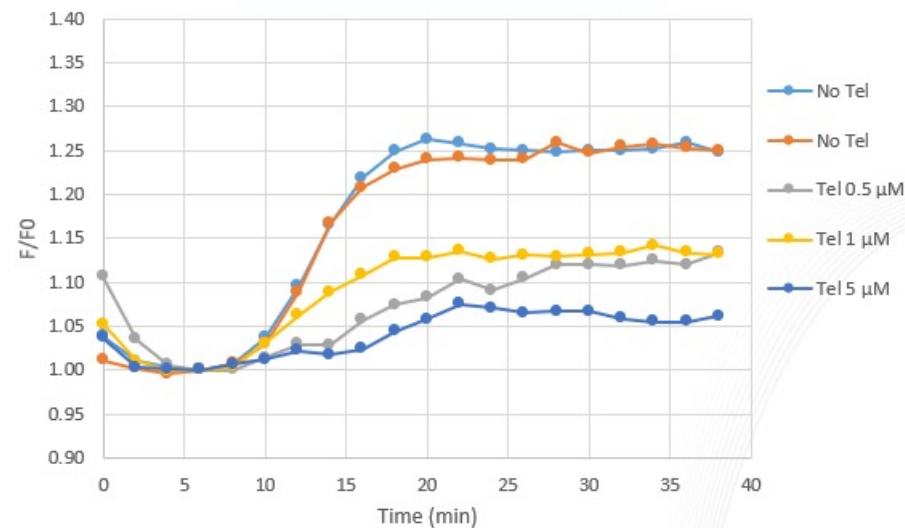
Study Overview

- **Model:** ARPE-19 retinal cells are incubated with varying concentrations of Telomir-1 with and without Copper (25 μ M) and levels of intracellular calcium was monitored,

Cellular Recovery

- Copper induces a rapid rise in intracellular calcium levels, which are released from mitochondria
- Telomir-1 dose dependently reverses this increase in calcium levels.

Copper induced calcium overload in RPE-19 cells



Telomir-1 restores vision, retinal structure, and reduces oxidative damage in AMD model



In a dry AMD model, Telomir-1 achieved functional vision recovery and structural regeneration—marking the first oral regenerative candidate.

- Restored central vision: light and movement responsiveness improved
- ~50% reduction in retinal oxidative stress
- Full restoration of INL (inner nuclear layer) thickness
- Reconstitution of ganglion and plexiform layers
- Oral formulation contrasts injectable ophthalmic therapies



Telomir-1™ Restores Vision in AMD Model

First Oral Therapy Showing Regenerative Effects in FDA-Surrogate AMD Model

Study Overview

- **Model:** 18-month-old *Sen57wrn^{-/-}ND6^{+/-}* zebrafish — recapitulates dry AMD with central vision loss, retinal thinning, oxidative stress and ~15% mortality
- **Dosing:** Oral administration for 14 days

Functional Recovery

- Vision restored: coordinated swimming returned, improved responsiveness to light/movement
- Improved central vision

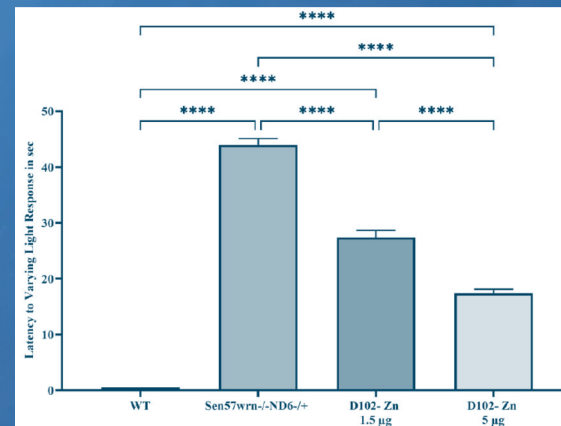
Mechanism Signals

- ~50% reduction in reactive oxygen species — supporting antioxidative/regenerative effect

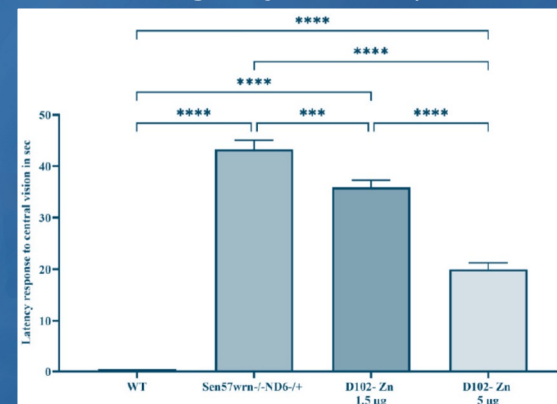
Significance & Difference

- **Oral first-in-class:** contrasts with injectable ophthalmic agents
- Targets **true regenerative endpoints** beyond neuroprotection
- Employs multiple FDA-recognized surrogate markers (function + structure)

Varying light response



Moving objects response



Investor Presentation | Nasdaq: TELO

Telomir-1™ Rebuilds Retinal Layers in AMD Model



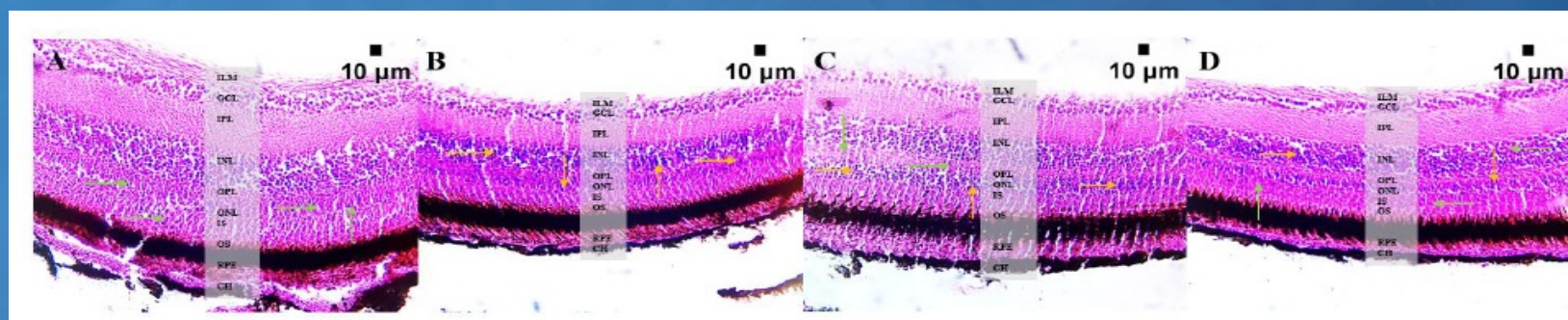
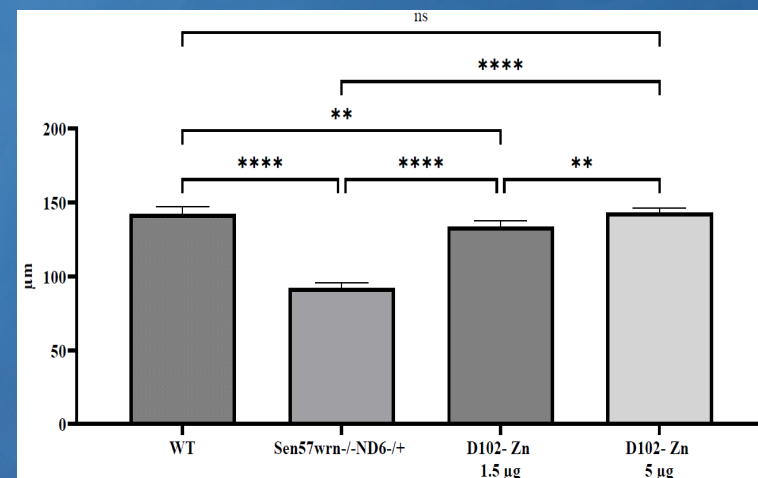
Whole retinal layers

Telomir-1™ Restores Vision & Retinal Structure in AMD Model

First Oral Therapeutic Showing Regenerative Effects in FDA-Surrogate-Driven Proof-of-Concept

Structural Regeneration

- Full restoration of retinal layers thickness



Control
Wilde Type

Control
Sen57wrn-/-ND6-/-

Telomir-1, 1.5 µg
Sen57wrn-/-ND6-/-

Telomir-1, 5 µg
Sen57wrn-/-ND6-/-

Telomir-1™ Rebuilds Retinal Layers in AMD Model

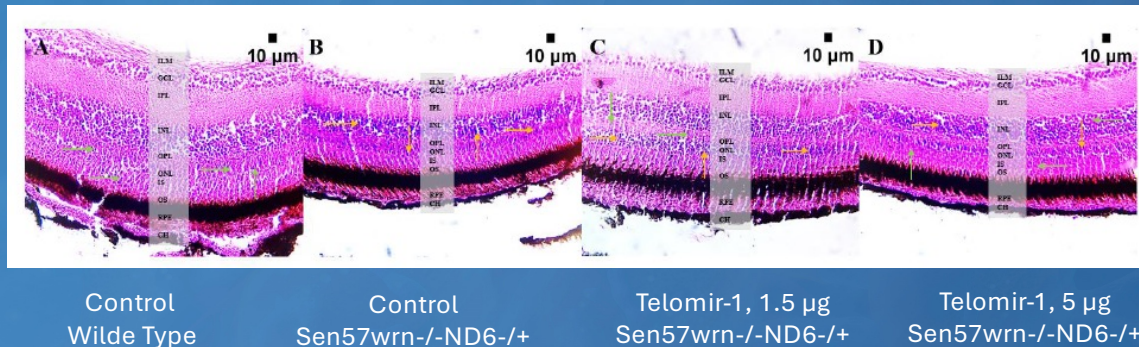


Telomir-1™ Restores Vision & Retinal Structure in AMD Model

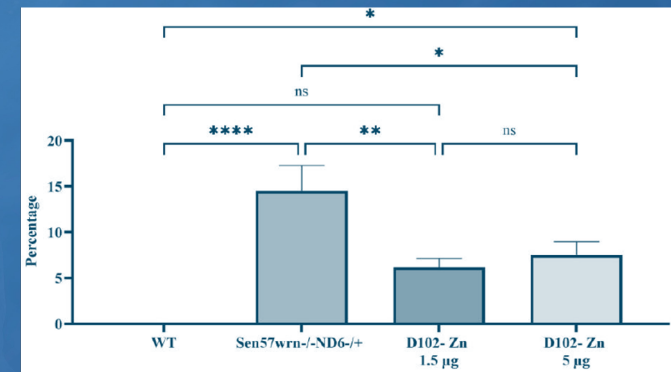
First Oral Therapeutic Showing Regenerative Effects in FDA-Surrogate-Driven Proof-of-Concept

Structural Regeneration

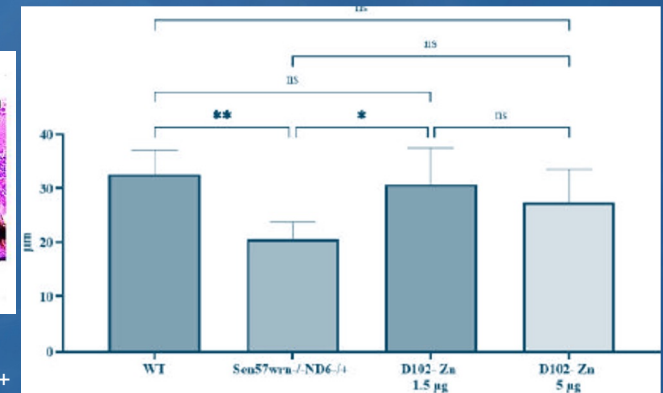
- Marked improvement in the percentage of retinal degeneration
- Full restoration of inner nuclear layer (INL) thickness
- Reconstitution of ganglion cell layer (GCL), inner plexiform layer (IPL), and outer plexiform layer (OPL)



Percent retinal degeneration



Restoration of INL layer thickness



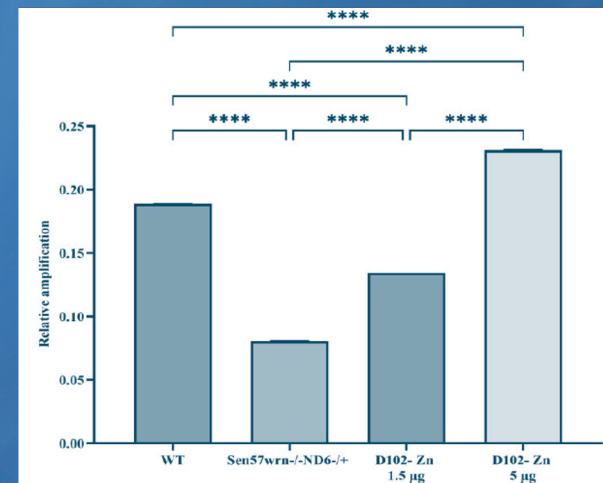
Telomir-1 Reverses Key Aging Markers in Werner Syndrome Model



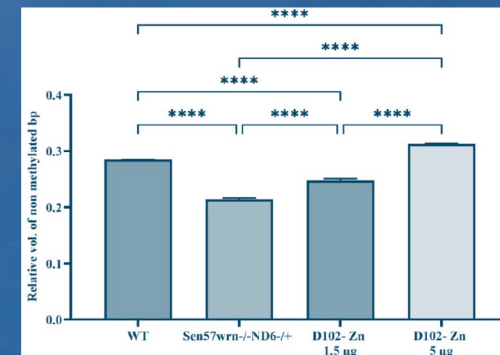
Telomere length and DNA methylation patterns were normalized in Sen57wrn^{-/-} zebrafish—exceeding wild-type levels at higher doses.

- Mutant zebrafish showed severe telomere shortening characteristic of Werner pathology
- 14-day oral Telomir-1 treatment significantly reversed telomere loss in a dose-dependent manner—surpassing wild-type levels
- Disrupted DNA methylation was restored by Telomir-1, including in key epigenetic markers like ephb3a
- Findings support Telomir-1's potential to reverse genomic and epigenetic signs of premature aging

Telomer length normalization



Epigenetic Clock restoration- ephb3a- CPG Island



Telomir-1 Restores Physical and Cellular Health in Werner Syndrome Model

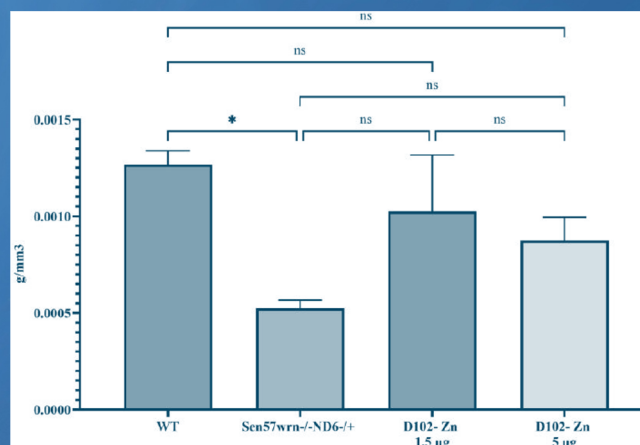
14-day oral treatment reverses muscle loss and oxidative stress in *Sen57wrn*^{-/-} zebrafish



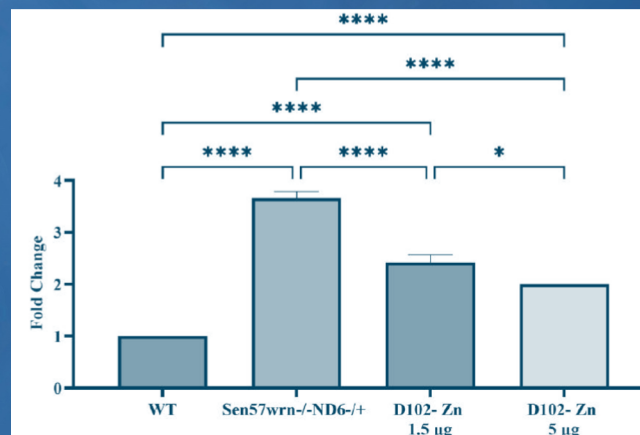
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- Mutant fish displayed significant loss of body and muscle mass
- Telomir-1 fully restored muscle weight—no significant difference from wild-type controls
- Reactive oxygen species (ROS) were highly elevated in mutants
- Telomir-1 reduced ROS levels significantly and dose-dependently—up to 50% reduction

Restoration of lost muscle weight



Reduction of ROS



Telomir-1 Achieves Full Survival and Broad Rejuvenation in Werner Syndrome Model



14-day treatment in Sen57wrn^{-/-} zebrafish reverses aging hallmarks and eliminates mortality

Study Model & Design

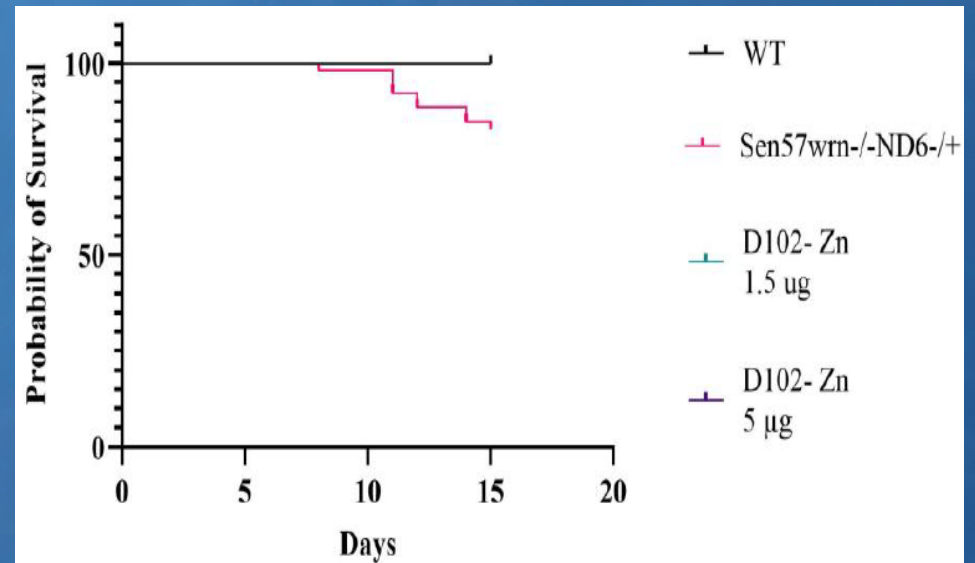
- **Model:** Sen57wrn^{-/-}ND6^{+/+} zebrafish recapitulating Werner Syndrome accelerated aging
- **Dosing:** Oral Telomir-1 for 14 days

Results

Rejuvenation Beyond Aging Biomarkers

- **100% survival** in treated fish vs ~15% mortality in controls
- Suggests Telomir-1 not only halts but partially reverses hallmarks of aging (epigenomic instability, telomere attrition, tissue decline)
- Positions Telomir-1 as a foundational therapy for age-related diseases and ultra-rare progeroid syndromes

Survival analysis – no animals died when treated with telomir-1



Telomir-1™ Restores Neuromotor Function in Wilson's Disease Model

Telomir-1™ Reverses Multi-Organ Dysfunction in **Wilson's Disease Model**

Dose-Dependent Reversal of Neurological, Hepatic & Renal Damage in ATP7B^{-/-} Zebrafish

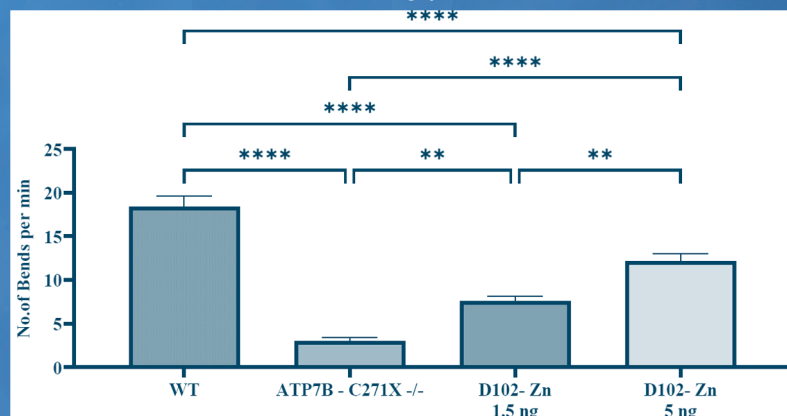
Study Overview

- **Model:** ATP7B C271X^{-/-} zebrafish, replicating human Wilson's disease pathology
- **Dosing:** Multiple Telomir-1 oral doses, demonstrating clear dose-response effects

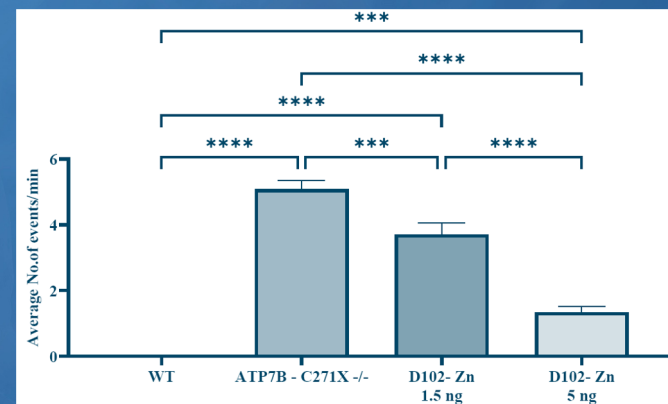
Neurological Recovery

- **4–5× reduction** in tremor frequency
- Restoration of normal swim distance, velocity, and exploratory behavior
- Reversal of ataxia-like behaviors (body bends, turn angles)

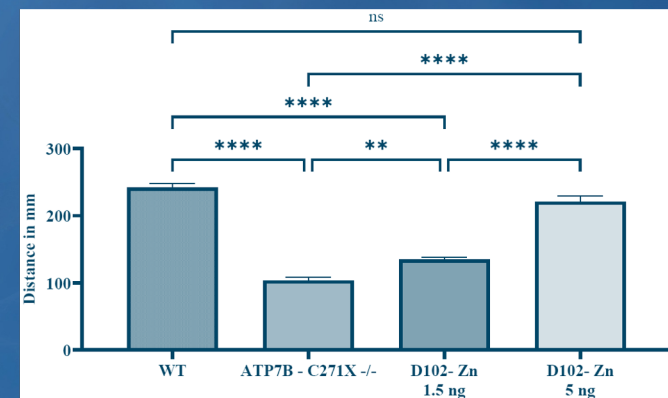
Ataxia stereotype- No. of bends



Episodic tremor events



Swim distance



Telomir-1™ Reverses Multi-Organ Dysfunction in **Wilson's Disease Model**

Dose-Dependent Reversal of Neurological, Hepatic & Renal Damage in ATP7B^{-/-} Zebrafish

Hepatic & Renal Benefit

- ~50% reduction in liver copper accumulation
- Histopathology scores near-normal for liver and kidneys
- ALT, AST, and bilirubin restored to wild-type levels, signaling organ protection

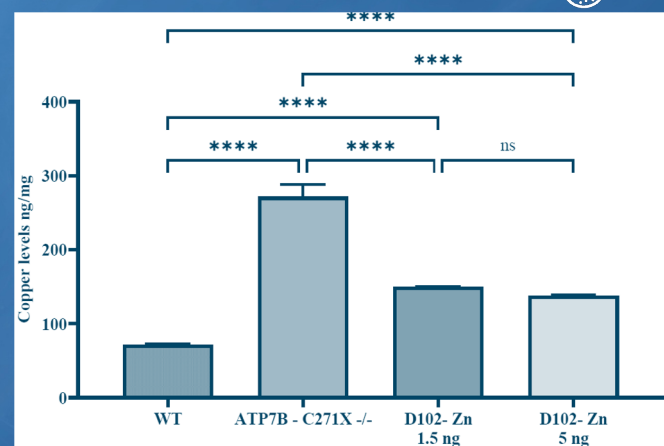
Survival Advantage

- Improved survival under high copper exposure conditions

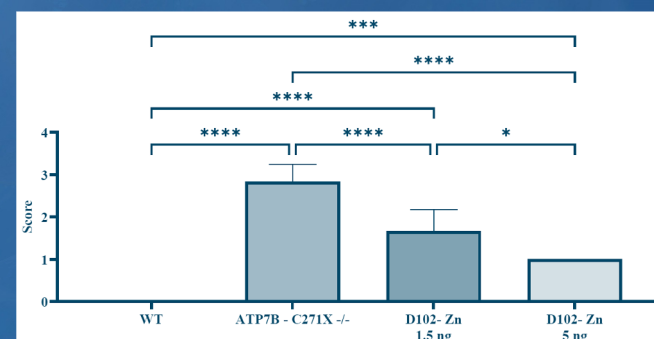
Strategic Importance

- Demonstrates **disease-modifying potential**—beyond chelation, actively reversing established damage
- Reinforces Telomir-1 as a multi-indication platform (complements prior AMD, Werner, and cancer data)
- Supports upcoming IND filing by end-2025 and human studies in first half of 2026

Liver copper levels



Liver Histology



PRE-CLINICAL RESEARCH -SUMMARY

Domain	Main characteristics	details
Pharmacology	Selective metal ion chelator	Fe>Cu>>Zn
ADME	Absorption Metabolism	- Telomir-1 is rapidly absorbed in rat and dog with rapid elimination half-life - Was stable in mouse, rat, dog, monkey, and human plasma for up to 6 hours - Not highly bound to plasma protein for all tested species and the % unbound of Telomir-1 ranged from 67.4% - 98.9% in plasma of tested species - Exhibited extensive turnover in mouse, rat, and monkey, and low turnover in dog and human hepatocyte incubations, generating metabolites resulting from oxidation of pyrrole rings. All the metabolites identified in human hepatocyte incubations were also detected in one or more animal species - Telomir-1 is not subject to metabolism by the common human hepatic drug metabolizing enzymes (Cyp450s)
Selectivity	Target selectivity	No affinity or interaction with a large number of receptors, enzymes and binding sites. Highly selective
Toxicology	Acute oral toxicology Repeated oral toxicology (7 days)	Rats- Well tolerated up to 1000 mg/kg po Dogs – Well tolerated up to 10 mg/kg po Rats – NOAEL – 200 mg/kg/day females; 750 mg/kg/day males Dogs – NOAEL – 7.5 mg/kg/day

ANTICIPATED TIMELINE FOR TELOMIR-1



2025
Q2

- **CMC (Chemistry, Manufacturing, and Controls):** Stability, Pharmacology: Characterization & Consolidation
- **ADME (Absorption, Distribution, Metabolism, and Excretion):** PK/PD (Pharmacokinetics/Pharmacodynamics) and Formal DMPK (Drug Metabolism and Pharmacokinetics)
- **Toxicology:** MTD (Maximum Tolerated Dose) Rat/Dog

2025
Q3

- **GMP CMC (Chemistry, Manufacturing, and Controls):** GMP (Good Drug Substance Manufacturing Practice), Stability, Drug product development and Clinical DP (Drug Product) Development
- **ADME (Absorption, Distribution, Metabolism, and Excretion):** Formal DMPK (Drug Metabolism and Pharmacokinetics)
- **Toxicology:** Formal Toxicology

2025
Q4

- **GMP CMC (Chemistry, Manufacturing, and Controls):** GMP (Good Manufacturing Practice), Drug product and Clinical DP (Drug Product) Manufacturing,
- **Pharmacology:** Consolidation
- **Toxicology:** Formal Toxicology
- **Regulatory:** Prepare IB (Investigator's Brochure) and IND (Investigational New Drug Application)

2026
Q1

- **Toxicology:** Completion Formal Toxicology
- **Regulatory:** Prepare IB (Investigator's Brochure) and IND (Investigational New Drug Application). **IND submission**
- **Clinical:** Initiation of Phase-I/II studies

MARKET OPPORTUNITY

Summary of US Epidemiology

The eligible patient pool analysis for Telomir-1 highlights a potential large patient pool looking for potential treatments to their conditions.

	Total Eligible Population	Diagnosed Prevalence	Treatment Rate	Total Addressable Market
Type 2 Diabetes	34-45M	25-27M	88%	\$57.47B
Cancer	18M	1.9M	Nearly 100%	\$16.7B
Alzheimer's Disease	6.5M	6.5M	50%	\$3.1B
AMD	19.8M	20M	Variable. Around 20%	\$18B

Source: https://oaction.unc.edu/oa-module/oa-prevalence-and-burden/?utm_source=chatgpt.com

MARKET OPPORTUNITY FOR RARE DISEASES

Summary of US Epidemiology

The eligible patient pool analysis for Telomir-1 highlights a potential large patient pool looking for potential treatments to their conditions.

	Total Eligible Population	Diagnosed Prevalence	Treatment Rate	Total Addressable Market
Progeria (Hutchinson-Gilford Progeria Syndrome)	20 children in the U.S.	Most cases	Limited, Lonafarnib is the only approved drug	Minimal
Wilson's Disease	6-10K	Many cases remain undiagnosed.	Includes chelating agents like penicillamine and zinc salts.	\$200-900M
Friedreich's Ataxia	6K	Most cases	SKYCLARYS™ (omaveloxolone) is the first FDA-approved treatment.	\$600.5M, projected to reach 1.71B by 2034
Menkes	16-40 new cases annually	Improved detection through genetic testing	Includes parenteral copper histidinate administration.	Limited

GROWTH STRATEGY

TELOMIRPHARMA.COM



01

Continue pre-clinical development of Telomir-1 across a range of diseases and progress into clinical development

02

Advance Telomir-1 through clinical development and FDA approval

03

Identify additional product candidates and expand current candidates into additional diseases

04

Explore strategic collaborations and partnerships to maximize the value of our product candidates

INVESTMENT HIGHLIGHTS

Telomir-1



TELOMIR-1 is novel small molecule metal ion regulator, with a broad potential to affect several pathologies and age-related diseases such as Progeria, Type 2 Diabetes, AMD, Wilson's disease and cancer.



Based on our pre-clinical studies to date we assemble experimental evidences showing that Telomir-1 may potentially serve as a metal ion regulator of essential metals such as iron, copper and zinc.



The Company's goal is to explore the potential of Telomir-1 starting with ongoing research in animals and then in humans.

A photograph of two people, likely scientists or healthcare professionals, in a laboratory or clinical setting. They are seen from behind, looking at a computer monitor. The image is overlaid with a semi-transparent blue filter. The text "Thank you." is written in large, white, sans-serif font across the center of the image.

Thank you.

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