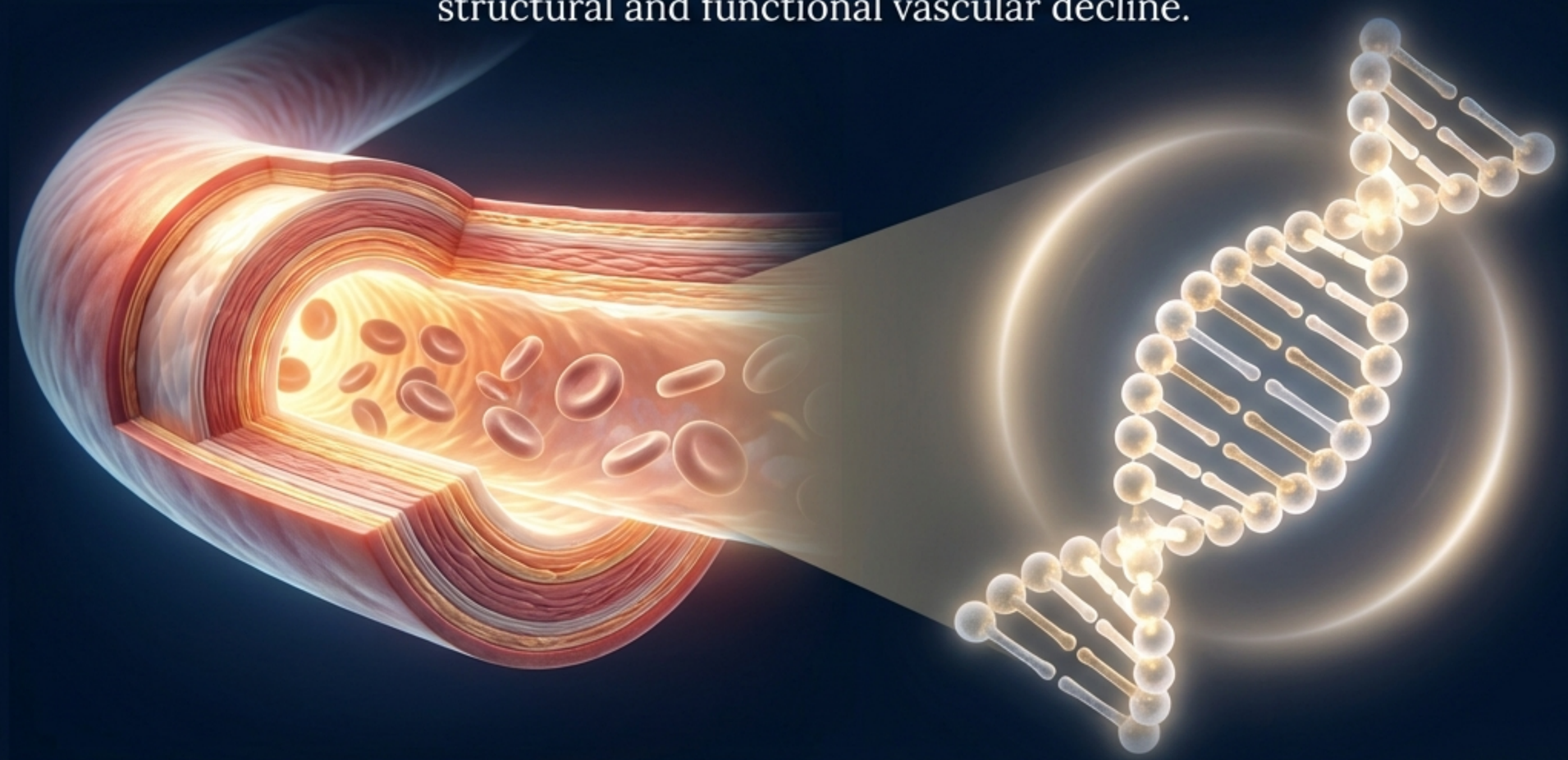
Ginsenoside Rb1 Restores Arterial Youth at the Nanoscale

Targeting DNA damage to reverse age-related structural and functional vascular decline.



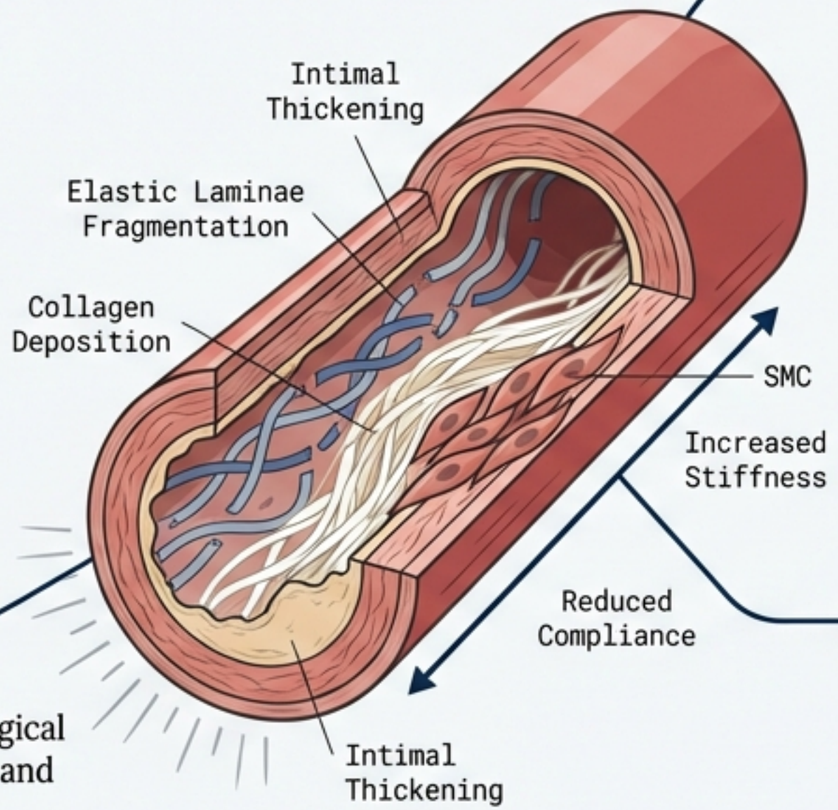
Human Aging is Determined by the Youthfulness of Our Blood Vessels

1 The Macro Threat

CVDs are the leading cause of death worldwide, driven fundamentally by population aging.

2 The Physical Failure

Arterial aging is the pathological foundation of hypertension and atherosclerosis.



3 The Structural Symptoms

Intimal thickening, fragmentation of elastic fibers, collagen deposition, and smooth muscle cell (SMC) disorganization.

4 The Functional Consequence

Increased arterial stiffness and reduced compliance (the loss of youthful elasticity).

The Compounding Drivers of Arterial Degeneration

SASP & Inflammation

Senescent cells produce chronic inflammation.

Oxidative Stress

Mitochondrial dysfunction dysregulates vascular tone.

Unrepaired DNA Damage

The core trigger for irreversible vascular inflammation, calcification, and cellular senescence.

Lipid Disorders

Metabolic dysfunction exacerbates cellular stress.

Epigenetic Loss

Gradual loss of youthful epigenetic information.

Ginsenoside Rb1: A Natural Molecule Put to a 6-Week Test

Ginsenoside Rb1 (Rb1), the primary active component of Panax ginseng, known for broad anti-aging properties.

Group 1: Young
12-week-old mice + PBS

Group 2: Old
78-week-old naturally aged mice + PBS

Group 3: Old + Rb1
78-week-old mice + 20 mg/kg Rb1 injected for 6 weeks

6-WEEK STUDY DURATION

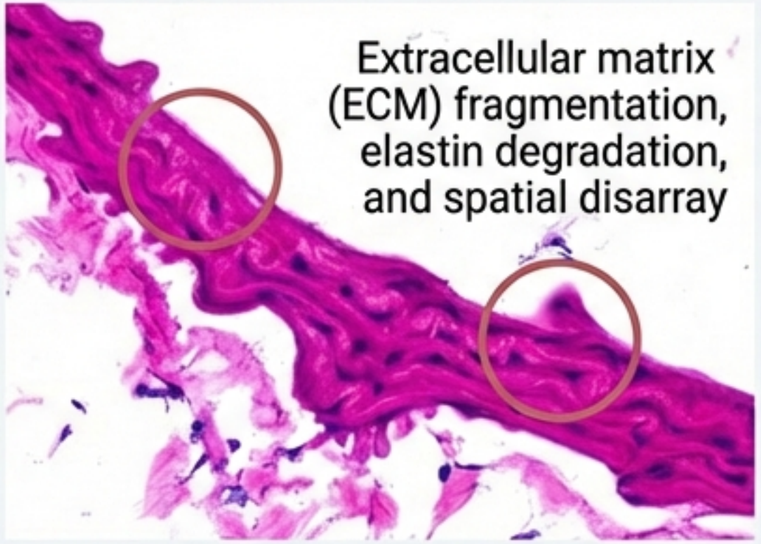
The study evaluates the aorta across 4 descending dimensions:
Tissue Morphology → Nanoscale Biomechanics → Cellular Senescence → DNA Integrity.

Level 1: Rb1 Rescues Structural Remodeling in the Aorta

Young

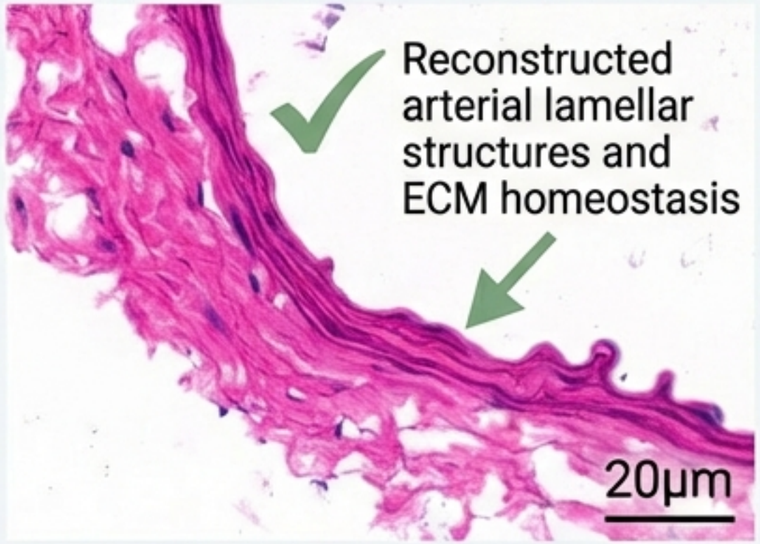
Perfectly aligned smooth muscle cells (SMCs) and smooth intima (Roboto Mono)

Old



Extracellular matrix (ECM) fragmentation, elastin degradation, and spatial disarray

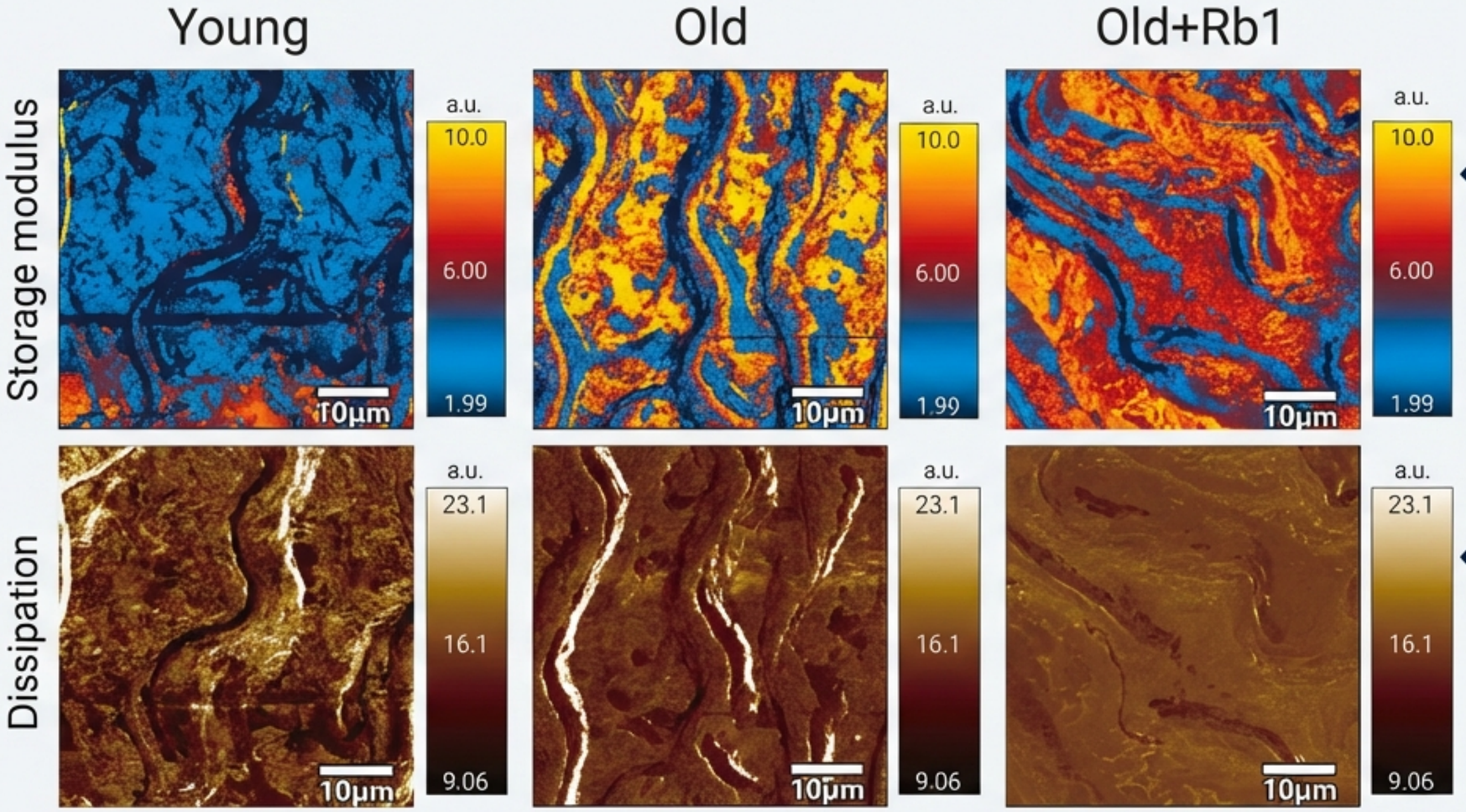
Old+Rb1



Reconstructed arterial lamellar structures and reorganized ECM homeostasis

6 weeks of Rb1 intervention restores smooth muscle cell organization and clears abnormal extracellular matrix deposition in aged aortas.

Level 2: Reversing Arterial Stiffness at the Nanoscale



Warmer colors = Higher Stiffness.

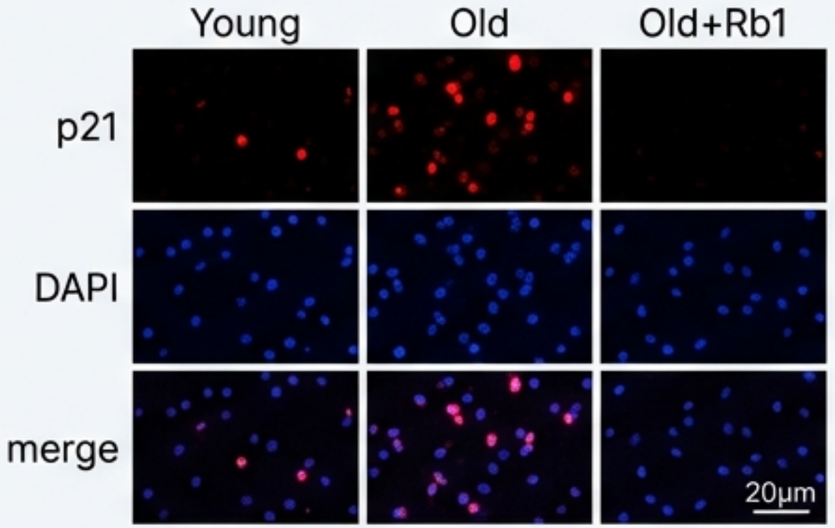
Notice the intense yellow/red in the Old group, and how the Old+Rb1 group cools down to mimic the Young group.

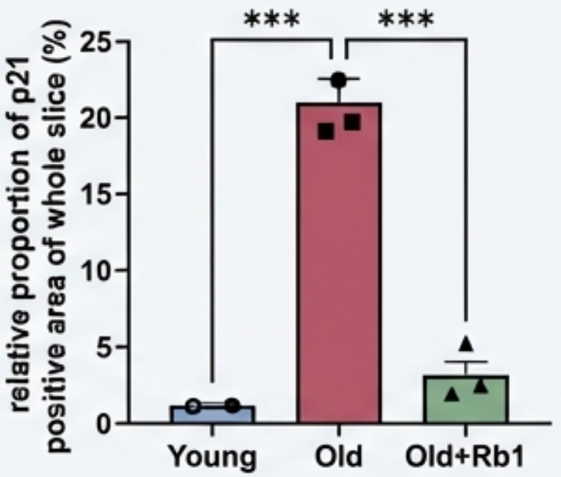
Brighter signals = Higher Viscosity.

High viscosity promotes thrombus formation. Rb1 visibly reduces these viscous characteristics.

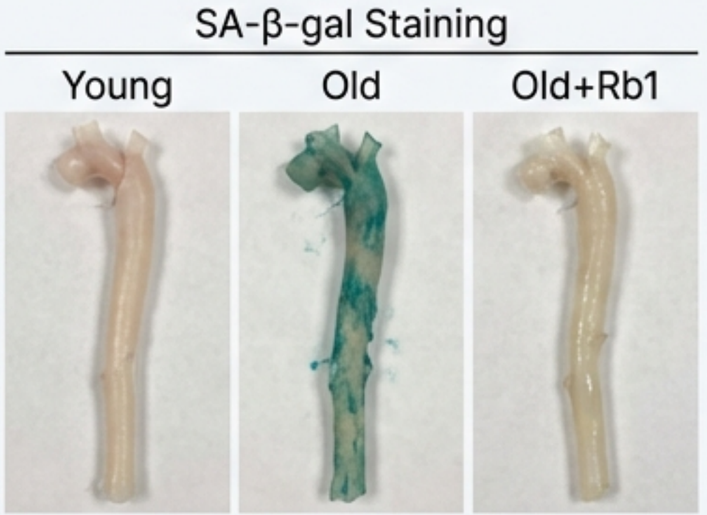
AFM mapping proves Rb1 physically restores the elastic and viscous parameters of the aging aorta, directly improving vascular compliance and reducing cardiac afterload.

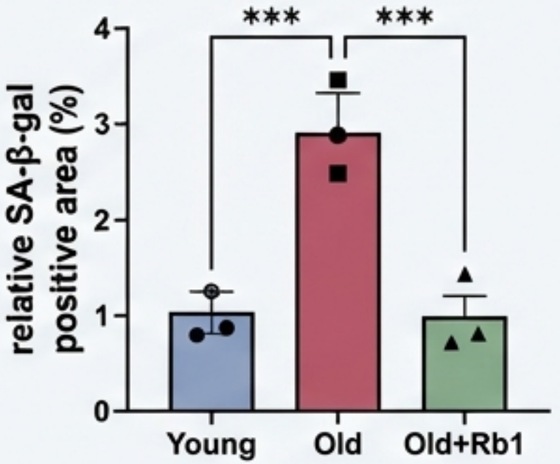
Level 3: Rb1 Halts the Accumulation of Senescent Cells





p21 expression forces cells into an irreversible state of arrest. **Notice the severe accumulation in the Old group and near-total suppression in the Old+Rb1 group.**

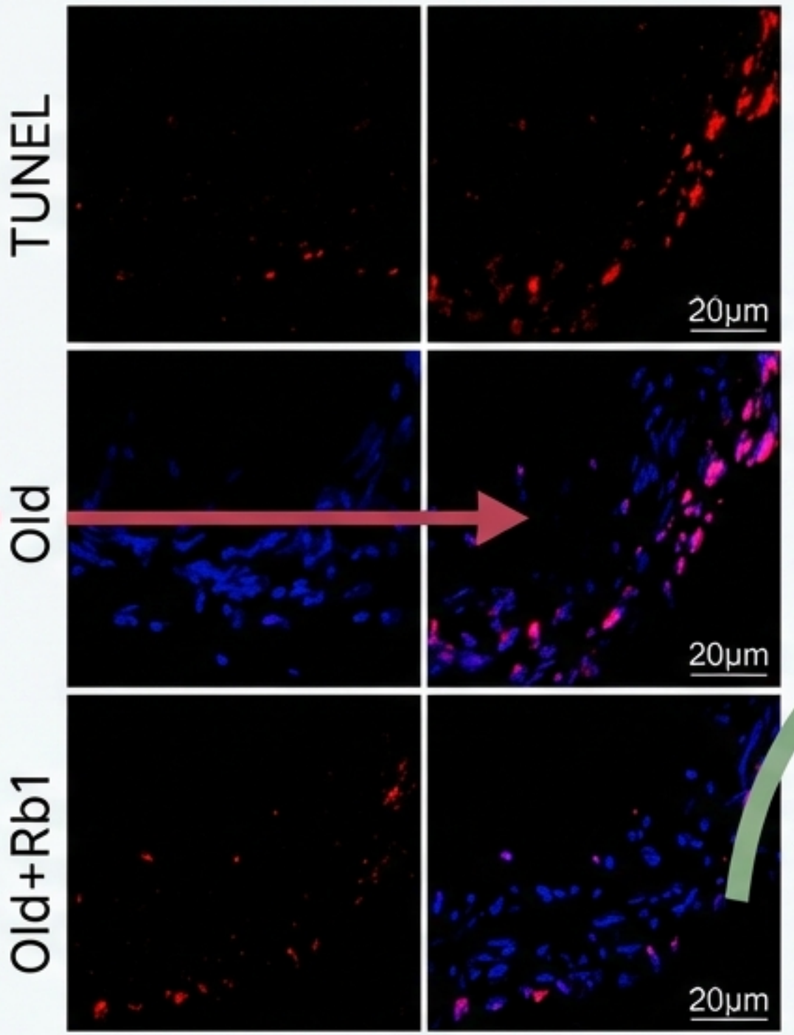




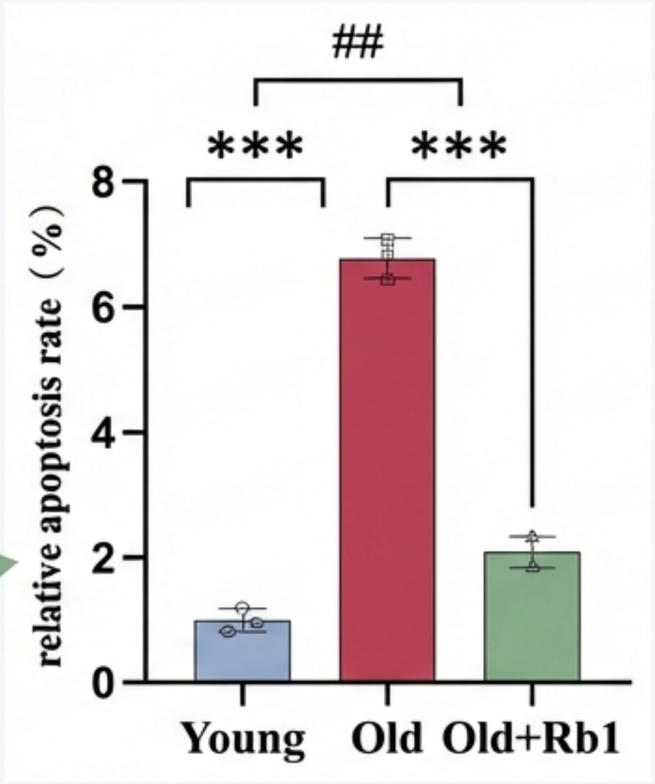
Macroscopic view showing heavy blue-green senescence staining in the Old aorta, which is visually cleared after Rb1 intervention.

Rb1 effectively downregulates cell cycle inhibitors and frees the arterial wall from senescent cell accumulation.

Level 4: Preventing Irreversible Cellular Death (Apoptosis)



Active cell death destroying arterial tissue integrity.



When DNA damage exceeds repair capacity, the protective pause of senescence turns into irreversible cell death. Rb1 aggressively suppresses this terminal cascade.

The Root Cause: Rb1 Alleviates DNA Double-Strand Breaks

Brown-yellow staining reveals severe, persistent DNA damage.

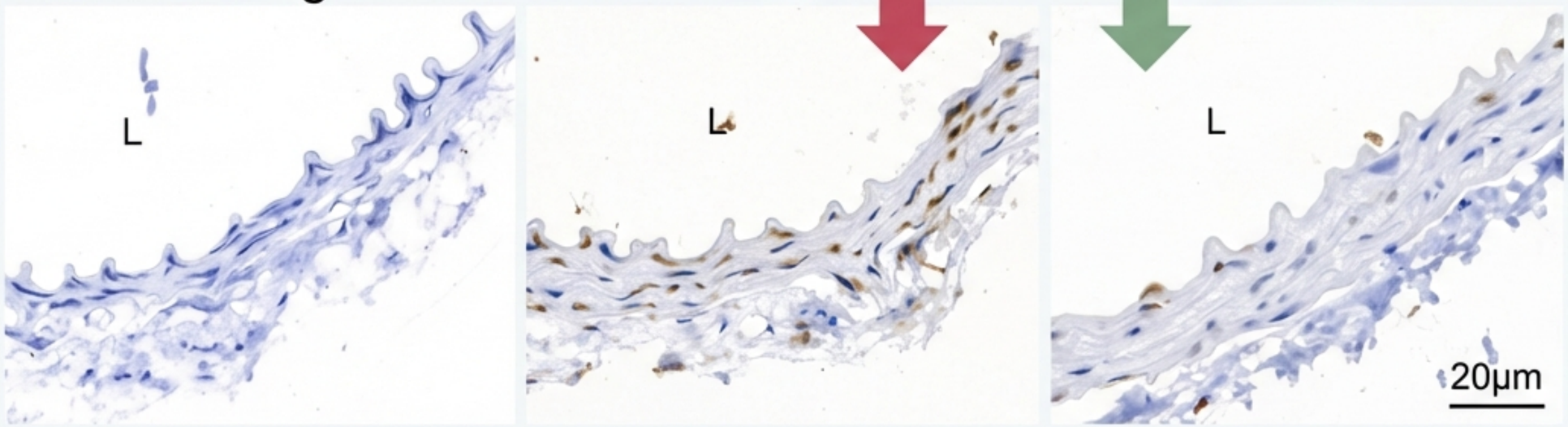
Massive >70% reduction in DNA DNA double-strand breaks.

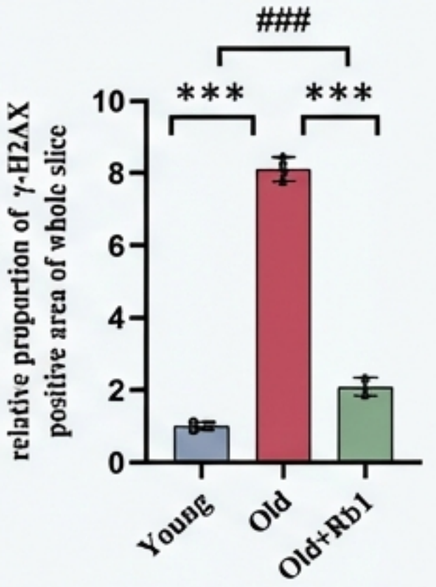
γ-H2AX

Young

Old

Old+Rb1





Persistent DNA damage is the fundamental driver of p21 expression, apoptosis, and structural degeneration. By alleviating this genomic burden, **Rb1 cuts** off vascular aging at its source.

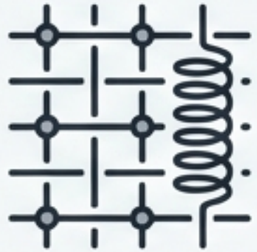
The Cascade of Vascular Rejuvenation

Genomic Rescue

Rb1 reduces DNA double-strand breaks (γ-H2AX).

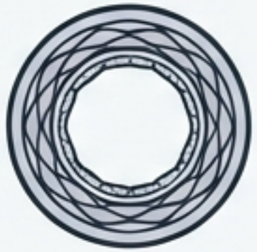
Cellular Survival

Downstream suppression of senescence (p21) and apoptosis (TUNEL).



Nanoscale Restoration

Reversal of chaotic extracellular matrix deposition; elasticity is restored.



Macro Rejuvenation

Reorganized smooth muscle cells, lower arterial stiffness, and enhanced vascular compliance.

Reversing DNA damage physically rebuilds the functional mechanics of the blood vessel.

A New Horizon for Pharmacological Anti-Aging

Natural Therapeutic Potential

Rb1 offers a highly stable, low-side-effect natural intervention capable of targeting both structural and functional vascular degeneration.

Direct Hemodynamic Benefit

By physically reversing arterial stiffness at the nanoscale, Rb1 presents a direct mechanism for reducing cardiac afterload and preventing atherosclerosis.

The Path to Clinical Application

Next steps require evaluating sex-specific effects in female models, correlating AFM nanomechanics with in vivo pulse wave velocity (PWV), and transitioning to human clinical trials.