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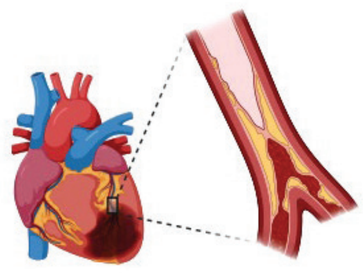
A Molecular Switch That Could Protect the Heart After a Heart Attack

Restoring blood flow after a heart attack is lifesaving, but it can also trigger additional damage inside mitochondria, the cell's power plants. My research focuses on Cyclophilin. Understanding how Cyclophilin D is regulated could reveal more precise ways to protect heart cells when blood flow is restored.

When Saving the Heart Can Also Damage It

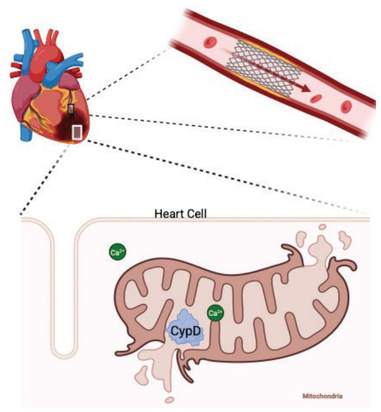
- During a heart attack, blocked blood flow deprives heart cells of oxygen.
- Restoring blood flow is lifesaving.
- But the sudden return of oxygen can trigger additional heart-cell damage.



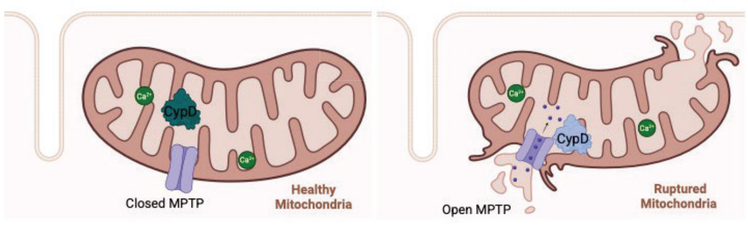
Can we protect the heart when blood flow returns?

The Damage Starts Inside the Cell's Power Plants

- When blood flow returns, mitochondria experience a sudden surge of stress and calcium.
- This can open a destructive gateway in the mitochondria.

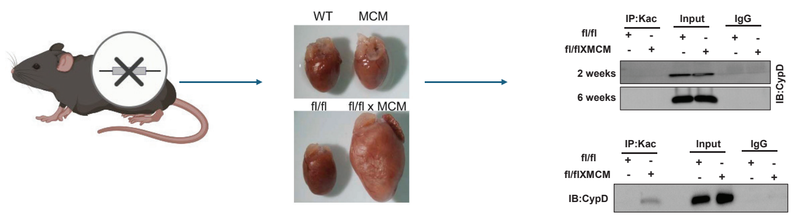


Cyclophilin D Is a Potential Target for Heart Protection



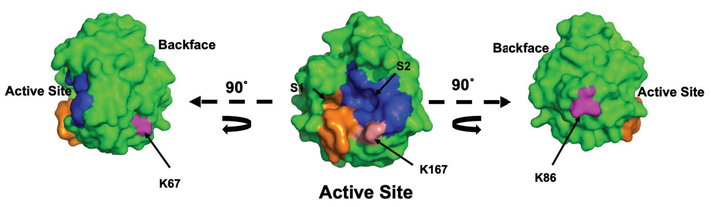
Because Cyclophilin D helps determine whether this gateway opens, controlling it could help protect mitochondria during severe cardiac stress.

The Stressed Heart Naturally Modifies Cyclophilin D



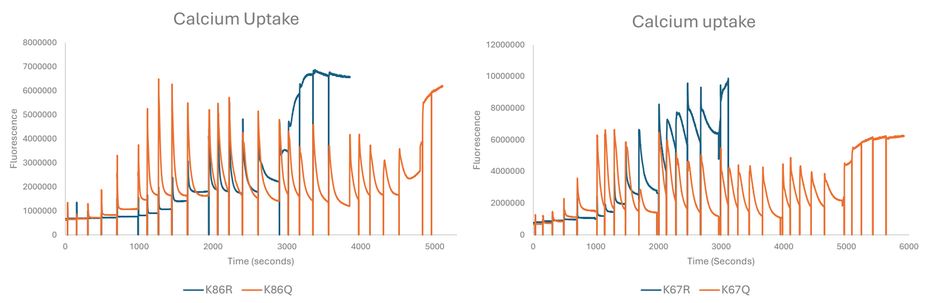
Could this be the heart's way of adjusting its mitochondrial damage switch?

Recreating the Heart's Molecular Changes



- We recreated acetylation at two specific locations on Cyclophilin D
- We measured MPTP opening with and without the modifications

Small Molecular Changes Can Make Mitochondria More Resilient



CypD modification helps keep the mitochondrial damage gate closed longer.

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