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First in class covalent inhibitor of Fis1 prevents mitochondrial fragmentation and dysfunction

Researchers at Stanford University have discovered a first-in-class covalent inhibitor that binds to activated Fis1 and prevents mitochondrial fission and dysfunction.

Mitochondria, essential for cellular function, undergo dynamic processes of fusion and fission and balancing the two is critical to retain optimal function. Fis1 protein responds to cellular stress by recruiting Drp1 to mitochondria and driving excessive fission. This process is known to cause mitochondrial fragmentation and dysfunction and contribute to pathogenesis of variety of health conditions (such as neurodegeneration, inflammatory and cardiovascular diseases). Previously, the molecular mechanism of activation of Fis1 during stress was unknown and thus hard to target.

Now, researchers have identified the mechanism of Fis1 activation and discovered a first-in-class covalent inhibitor of activated Fis1. The small molecule covalently binds to activated Fis1, preventing pathological fission and protecting against oxidative stress-mediated mitochondrial fragmentation and dysfunction. These compounds have potential to be developed as therapeutics for a number of disease conditions with underlying mitochondrial pathologies that currently have no approved therapies.

Stage of Development

In vivo

Applications

- Treating diseases impacting mitochondrial fission

    - Neurodegeneration
    - Inflammatory conditions
    - Cardiovascular diseases

Advantages

- First-of-its-kind molecule to covalently bind to activated Fis1
- Prevents pathological mitochondrial fission without affecting physiological mitochondrial fission

Innovators

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