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Method and Compound for Inhibition of Cell Death

Background

Many of today's medical diseases can be attributed directly or indirectly to problems with apoptosis- a programmed cell suicide mechanism. Programmed cell death is vital to existence of virtually all organisms. Typically, one thinks of cell death as being a pathological phenomenon, but in fact each second nearly one million cells of the body undergo apoptosis. Programmed cell death generally occurs by apoptosis, and defects in the physiological pathways for apoptosis have a role in many diseases. Consequently, great interest has emerged in devising therapeutic strategies for modulating key molecules that regulate apoptosis. Apoptosis is generally mediated by proteases known as caspases. Recently, a novel serine protease, Omi/HtrA2, has been shown to disrupt the inhibition of apoptosis protein-caspase interaction. The current invention describes a novel inhibitor of Omi/HtrA2, which counteracts its action and increases cell life.

Invention

A chemical compound (UCF 101) that inhibits the apoptotic activity of the protease Omi/HtrA2 resulting in increased cell lifetime.

Application

UCF 101 is a potential therapeutic intervention for apoptosis related disease. UCF 101 can be used to develop drugs capable of targeting cells with excessive apoptosis properties.

Advantages

- Omi/HtrA2 inhibitors could be used to promote increased cell life in various apoptosis related disorders such as ischemia, heart failure, inflammation, and bacterial infection.
- UCF 101 could be a pharmaceutical option of treatment.

Lead Inventor

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Selected References

Althaus J, Siegelin MD, Dehghani F, Cilenti L, Zervos AS, Rami A.. The serine protease Omi/HtrA2 is involved in XIAP cleavage and in neuronal cell death following focal cerebral ischemia/reperfusion. *Neurochem Int.* 2007 Jan;50(1):172-80.

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