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Erratum

PROTECTIVE EFFECTS OF A - CRYSTALLIN ON B - AMYLOID (Aβ) INDUCED TOXICITY

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ABSTRACT

Objectives: To investigate the protective role of α-crystallin against β-amyloid aggregation.

Methods: *In vitro* spectroscopic methods and cell culture studies were done to validate our objective.

Results: The molecular basis of alzhemiers disease has been proposed to be accumulation and aggregation of β-amyloid (Aβ). However, prevention of β-amyloid aggregation is still a promising means to reduce its neurotoxicity. In this work, we show that α-crystallin was able to inhibit cellular toxicity of Aβ on astrocytes and lymphocytes. Theα-crystallin (αA and αB): the two vertebrate eye lens proteins that ar e related to the small heat shock protein family, was able to reverse the oxidative stress induced by Aβ1-42. Treatment of α-crystallin enhances the activity of proteasome and it also induces the expression of Hsp70 which is known to inhibit the intramolecular misfolding. We also demonstrate that Aβ1-42 suppresses the expression of TriC chaperonin subunits TCPβ and TCPε, which are known to play a role in folding of misfolded proteins.α-crystallin reverses this effect and enhances the expression of TCPβ and TCPε.

Conclusions: Research findings in this study provide the basis for the development of novel pharmacotherapy for Alzhemier's disease.

Keywords: Alzhemiers disease, α-crystallin, Proteasome, HSP70, TCPβ and TCPε.

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