PARKINSON'S DISEASE NEWS

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CLR01 CLAIMED TO SLOW PARKINSON'S DISEASE

Neurotherapeutics [2012] Feb 29 [Epub ahead of print] (Prabhudesai S, Sinha S, Attar A, Kotagiri A, Fitzmaurice AG, Lakshmanan R, Ivanova MI, Loo JA, Klärner FG, Schrader T, Stahl M, Bitan G, Bronstein JM.)

Aggregation of alpha-synuclein in the cells involved in Parkinson's Disease is claimed as being causative in Parkinson's Disease, multiple system atrophy, and dementia with Lewy bodies. A novel "molecular tweezer" termed CLR01 has been described as a potent inhibitor of alpha-synuclein by preventing it from being formed. In cell cultures CLR01 was shown to greatly lessen alpha-synuclein.

To determine whether CLR01 was also protective in animals, the researchers used a zebrafish, which is a type of fish commonly used in Parkinson's Disease research because it is easily manipulated genetically, and because some of its biochemistry is similar to that in humans. CLR01 significantly improved zebrafish survival, suppressed the aggregation of alpha-synuclein, and also reduced cell death caused by alpha-synuclein. This occurred without evidence of toxicity. The authors consequently claimed that CLR01 stopped the progression of Parkinson's Disease in an animal model, and is therefore a promising therapeutic agent for the treatment of Parkinson's Disease.

However, zebrafish do not have Parkinson's Disease. Parkinson's Disease was not simulated in the zebrafish either. There were therefore no measures of whether or not Parkinson's Disease symptoms altered as a result of CLR01. So it could not reasonably be claimed that CLR01 was shown to stop or slow the progression of Parkinson's Disease, even in animals. A lot of the toxicity that could occur in humans would not be detectable in zebrafish either. Researchers measured the effects in terms of alpha-synuclein, which does not indicate Parkinson's Disease. It occurs in other medical disorders and often fails to occur in Parkinson's Disease.

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