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'Chaperones' ward off Parkinson's



The fly experiments point to possible approaches in humans

A particular body chemical may be able to halt the destructive progress of Parkinson's disease, according to researchers.

While their experiments have focused primarily on fruit flies, they are convinced that a similar mechanism exists in humans.

Parkinson's disease is one of the most common disorders causing brain degeneration in humans.

Over time, the cells in the brain which produce a vital brain signalling chemical called dopamine die off - which leads to progressively worse movement problems.

There are no treatments which can actually halt the degeneration of the neurons.

Another protein, called alpha-synuclein, is considered a candidate for contributing to this damage.

Misfolded proteins

It is suspected that it either is toxic to the neurons, or somehow hampers the work of other chemicals - dubbed "chaperones" - whose job it is to make sure that the production and configuration of yet more proteins is proceeding correctly.

In humans, it is thought that a particular chemical, called Hsp-70, may perform a "chaperone" role.

Alpha-synuclein has been implicated in other degenerative diseases, such as Alzheimer's

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and Lewy body disease, which cause dementia.

The latest research, at the University of Pennsylvania at Philadelphia, looked at what happened in fruit flies when alpha-synuclein was present, with or without the "chaperone".

“
These data suggest that altered chaperone activity may be involved in the progression of Parkinson's disease
”

**Dr Pavan Auluck,
University of
Pennsylvania**

Flies with alpha-synuclein heavily present lost approximately half their dopamine-producing neurons in certain brain areas by the time they reached 20 days old.

When they were genetically modified to remove the "chaperone" protein, this marked neuronal degeneration took just one day to happen.

'Neuronal arsenal'

However, when they genetically altered the flies so that the "chaperone" protein was present in greater quantities, the reverse happened.

The modified flies had just as many of these brain cells at 20 days as on their first day.

This, say the researchers, suggest that treatments which look to boost the quantity of "chaperone" proteins may be worth looking at for humans.

Dr Pavan Auluck, lead author of the project, said: "These data suggest that altered chaperone activity may be involved in the progression of Parkinson's disease, and that chaperones such as Hsp-70 may be a critical part of the neuronal arsenal that fights neurodegenerative disease."

Dr Nancy Bonini, another of the researchers, said: "If Hsp-70's role of protecting against alpha-synuclein toxicity is confirmed in mammals, an important step would be to pursue drugs that upregulate the stress response that produces these chaperones, and determine whether they will protect neurons."

The study was published in the journal Science.

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