

# THE ROLE OF ASTROCYTES IN TAU-RELATED NEURONAL CELL DEATH

<https://neurodegenerationresearch.eu/survey/the-role-of-astrocytes-in-tau-related-neuronal-cell-death/>

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### Country

United Kingdom

## Title of project or programme

THE ROLE OF ASTROCYTES IN TAU-RELATED NEURONAL CELL DEATH

## Source of funding information

Alzheimer's Research UK

## Total sum awarded (Euro)

€ 67,739

## Start date of award

01/07/2015

## Total duration of award in years

1.2

## Keywords

### Research Abstract

In Alzheimer's disease, the neuronal protein tau becomes misfolded and assembled into neurofibrillary tangles, a leading cause of neuronal cell death. A central aim of our recent ARUK grant was to identify how transplanted astrocytes – key support cells in the brain – protected neurons from death in a P301S tau transgenic mouse that develops progressive tau pathology. Interestingly, whilst studying how transplanted astrocytes elicit neuroprotection, we have unexpectedly discovered that the local astrocytes in P301S mice actually elicit neurotoxicity. Significantly, the protective effects of normal astrocytes, and toxic effects of P301S tau mouse-derived astrocytes, were reproduced in cell culture, the toxic element being released into the culture medium. This extension will be used to confirm that the astrocytes' neurotoxic effect is

general and not specific to our mouse line by testing the effects of astrocytes from other transgenic mouse lines with or without tau pathology. Time permitting, we hope to extend these observations using medium from human astrocytes derived from stem cells of patients with a similar tau mutation. These validations, together with ongoing attempts to identify the toxic element, could point to new ways to intervene in development of Alzheimer's disease and to identify therapies for its treatment.

**Further information available at:**

**Types:**

Investments < €500k

**Member States:**

United Kingdom

**Diseases:**

N/A

**Years:**

2016

**Database Categories:**

N/A

**Database Tags:**

N/A