

STUDY SHOWS ARG-007 DOES NOT DEGRADE WHEN CO-ADMINISTERED WITH ISCHEMIC STROKE THERAPEUTICS

Highlights:

- *Preliminary study shows ARG-007 has the potential to continue offering neuroprotection when co-administered with the clot dissolving drug Tissue Plasminogen Activator.*
- *Clot dissolving drugs are administered to stroke patients following diagnosis of an ischemic stroke, but do not prevent or reverse neuronal damage or cell death.*
- *The results indicate ARG-007 could potentially continue to offer stroke victims neuroprotection prior to formal diagnosis (imaging) while in-the field, or when clot dissolving drugs are administered in the hospital.*
- *The in vitro study is currently being prepared as a publication for submission to a peer reviewed scientific journal, and Argenica aims to confirm these results in an animal model stroke study.*

Perth, Australia; 12 JULY 2021 - Argenica Therapeutics Limited (ASX: AGN) (“Argenica” or the “Company”), a biotechnology company developing novel therapeutics to reduce brain tissue death after stroke, is pleased to provide positive preliminary results showing ARG-007 has the potential to continue offering neuroprotection when co-administered with Tissue Plasminogen Activator (tPA).

tPA, or alteplase, is an intravenous medicine given to ischemic stroke victims that can dissolve the stroke-causing clot. tPA is administered after imaging diagnosis confirms an ischemic stroke, which can occur many hours after the initial stroke and does not prevent or reverse neuronal damage and cell death, but rather dissolves the clot causing the stroke.

Results from studies with other potential competitive neuroprotective peptides have suggested that the active peptide can get degraded following administration of tPA, which reduces the efficacy after tPA administration. This limitation would significantly impact the applicability of the neuroprotective agent in cases where tPA is expected to be administered as an acute ischemic stroke treatment.

Argenica can now confirm, that in an *in vitro* neuronal excitotoxicity model, the ARG-007 peptide is resistant to proteolytic degradation when incubated with tPA and maintains its neuroprotective properties post tPA exposure. A previous study also confirmed the neuroprotective stability of ARG-007 when incubated with plasmin, an enzyme activated by tPA that dissolves the clot causing the stroke. These results suggest that ARG-007 could potentially continue to provide neuroprotective benefits after clot dissolving drugs are administered to stroke patients.

To further confirm proteolytic stability, ARG-007 also remained stable, maintaining its neuroprotective properties when incubated with trypsin, an enzyme found in the body which has broad peptide proteolytic degradation properties.

In previous animal studies, ARG-007 has already shown a reduction in brain tissue death up to 28 days after a stroke. Combined with today's promising preliminary data, ARG-007 shows increased potential to continue neuroprotection both prior to formal diagnosis (imaging) and after treatment with tPA. This could lead to improved outcomes and faster recovery for all stroke patients, regardless of tPA treatment.

NEURONAL GLUTAMATE EXCITOTOXICITY MODEL

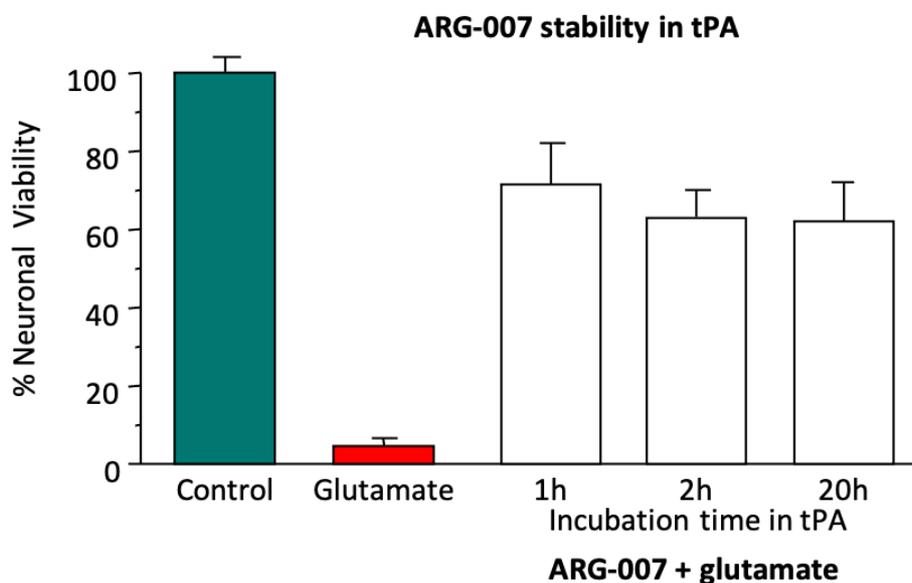


Figure 1. Brain cell (neuronal) viability in an *in vitro* excitotoxicity model which uses glutamate to induce cell death. Brain cells exposed to glutamate only results in significant cells death, however by adding ARG-007 and incubating with TPA, the neuroprotective effect of ARG-007 is maintained, even after incubation with tPA for various lengths of time.

Argenica's CEO, Dr Liz Dallimore said: "These preliminary results confirming the stability of ARG-007 when administered with tPA *in vitro* are hugely encouraging. We know from other companies' clinical trials that neuroprotective peptides have been shown to degrade following administration of tPA, nullifying the neuroprotective effects of the peptide following tPA treatment. Whilst tPA degradation does not impact neuroprotection up until that time point, by showing that ARG-007 is resistant to proteolytic degradation whilst maintaining its neuroprotective properties, we have great confidence as we progress to clinical trials."

Next steps

The study was led by Argenica's Chief Scientific Officer, Prof Bruno Meloni, at the Perron Institute for Neurological and Translational Sciences (Perron) using an *in vitro* neuronal excitotoxicity model which exposes neurons to glutamate to induce cell death. *In vitro* testing is a straightforward research methodology and occurs in a laboratory without using animals or human subjects.

This study is currently being prepared as a publication for submission to a peer reviewed scientific journal.

The Company is currently undertaking additional further pre-clinical trials to confirm these results in an animal model of stroke. These subsequent trials, to be undertaken by Prof Meloni, will examine the *in vivo* neuroprotective efficacy of ARG-007 when administered with tPA following stroke in animals. Argenica anticipates the preliminary results from this animal study will be available in October.

This announcement has been approved for release by the Board of Argenica

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ABOUT ARGENICA

Argenica (ASX: AGN) is developing novel therapeutics to reduce brain tissue death after stroke and improve patient outcomes. Our lead neuroprotective peptide candidate, ARG-007 has been successfully demonstrated to improve outcomes in pre-clinical stroke models and is in the process of being verified for its safety and toxicity before commencing Phase 1 clinical trials in humans. The aim is for our therapeutic to be administered by first responders to protect brain tissue against damage during a stroke with further potential to enhance recovery once a stroke has taken place.