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COMPANY SNAPSHOT



Commercialise "best in class" novel neuroprotective therapeutics to reduce brain cell death following stroke and other brain injuries

LEAD DRUG CANDIDATE

ARG-007

Neuroprotective peptide that could offer protection to the brain following stroke and other acute central nervous system injuries











GRANTED PATENTS

Granted in the EU, Japan China, and the US

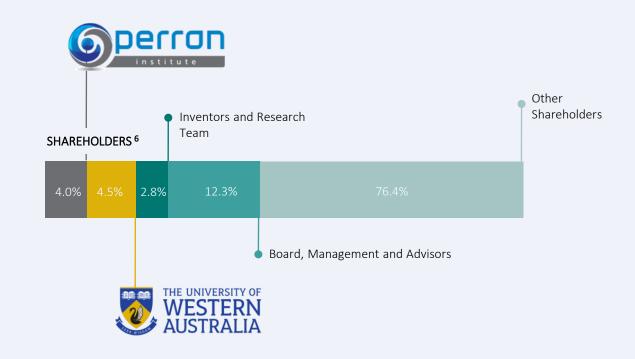
FREE OF ENCUMBRANCES

IP 100% owned by Argenica and free of royalties or other encumbrances



CAPITAL STRUCTURE

Total Shares on issue	86,922,250
Shares subject to escrow for 24 months ¹	22,625,752
Options on issue (escrow for 24 months) 1 & 2	8,300,000
Options on issue (not escrowed) ³	1,700,000
Cash Balance ⁴	Circa \$9.54M
Market Capitalisation @ \$0.40 ⁵	Circa \$34.8M
Enterprise Value (EV) @ \$0.40 ⁵	Circa \$25.2M



- 1. 24 months from the date of commencement of Official Quotation on ASX -11^{th} June 2023
- 2. Option Terms Ex price \$0.30, expiry 30 Sept 2024
- Option Terms 800,000 Ex price \$0.30, expiry 6 Aug 2023; 300,000 Ex price \$1.10, expiry 1 Apr 2025; 600,000 Ex price \$0.65, expiry 10 Jun 2024
- 4. As @ 31 March 2022 adjusted for \$5.5m placement in Jun 2022 net of cash capital raising fees
- Closing price as @ 8 Jun 2022
- 6. Percentages are estimates only and subject to slight variation



HOW ARG-007 WORKS



Cell death in the brain, or infarction, results from inadequate blood supply to the affected area.



Initial infarction sets off a cascade of cell death.



While no drug can stop the initial infarct injury, ARG-007 has **multiple mechanisms** of action to stop the cascade of cell death that happens after the initial injury.



In animal models of stroke, ARG-007 <u>slows</u> the progression of neuronal cell death and preserve still viable brain tissue.



This increases the amount of available salvageable brain tissue.



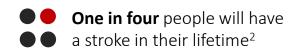


STROKE IS A GLOBAL ISSUE



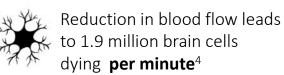


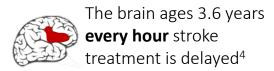












There are **no** universally available drugs that protect brain cells following stroke.

CURRENT STROKE TREATMENT

IN-FIELD TRIAGE

Patient in ambulance and arrives at emergency



Diagnose type of stroke

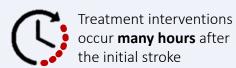


Rapidly increasing level of neural cell death and damage



Current therapeutic treatment

THERAPEUTICS ARE ADMINISTERED AFTER DIAGNOSING THE PATIENT IS HAVING A STROKE





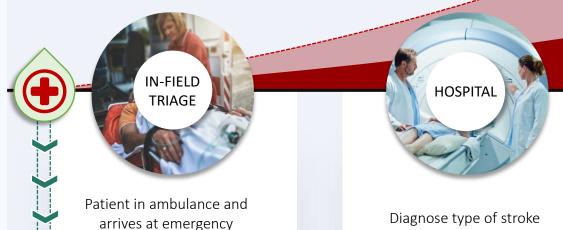
Does not prevent or reverse further neuronal damage and cell death

Long term cell damage impacts patient outcomes and recovery



1-4 weeks acute hospitalisation¹ followed by intensive rehabilitation and potential complications

ARGENICA'S SOLUTION



neural cell death and damage

Reduction in

Current therapeutic treatment

TREATMENT

PLAN

Reduction in patient recovery time



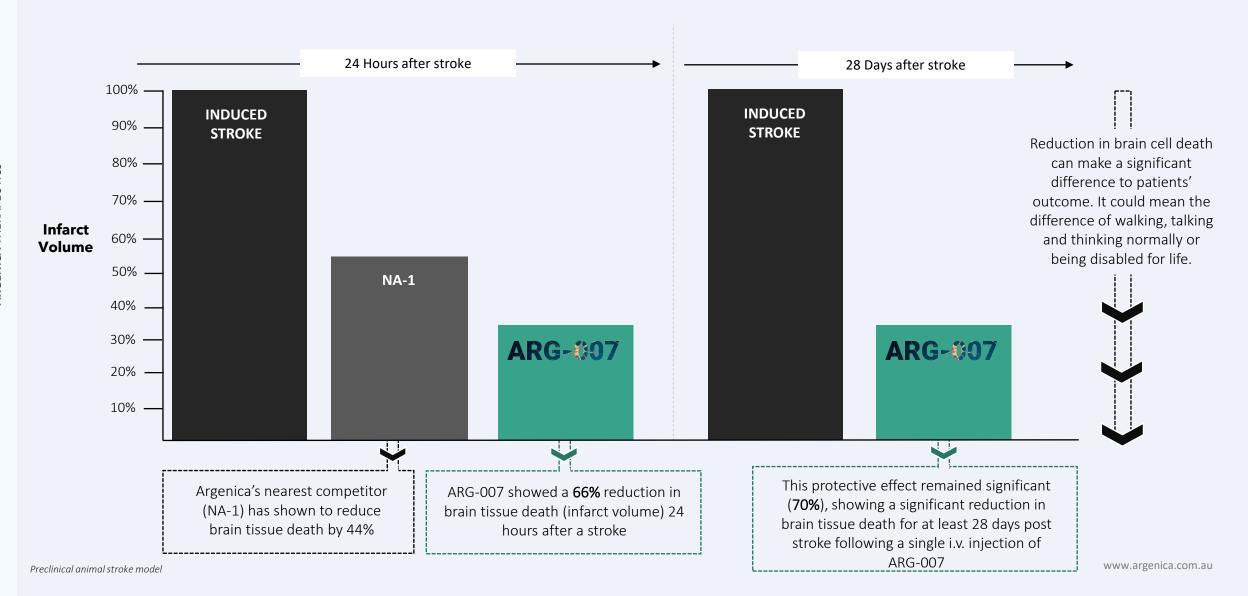
1-4 weeks acute hospitalisation¹ followed by intensive rehabilitation and potential complications

ARG-007 TO BE ADMINISTERED BY FIRST RESPONDERS IN THE FIELD TO MINIMISE A PATIENT'S BRAIN CELL DAMAGE



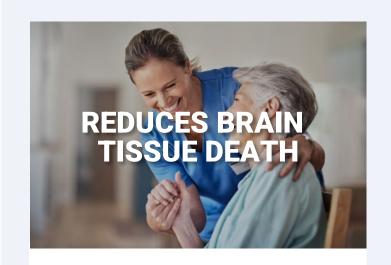
ARG-007 REDUCES BRAIN TISSUE DEATH

Percentage <u>reduction</u> of brain tissue death after stroke





FAVOURABLE RESULTS TO DATE



ARG-007 showed a **67%**reduction in brain tissue death
(infarct volume) for at least 28
days after stroke¹.



Pre-clinical studies showed ARG-007 does not exacerbate bleeding in hemorrhagic stroke model, meaning it could be **safe to administer in the field** by first responders².



No adverse effects were observed in preliminary PK and toxicology animal studies, indicating that ARG-007 is potentially safe and well-tolerated at the relevant doses³.

OFFERS NEUROPROTECTION EVEN WHEN CO-ADMINISTERED WITH CLOT DISSOLVING DRUGS 4

These findings are preliminary in nature. A larger dataset will be required for clinical validation.

^[1] Preclinical animal stroke model

^[2] Preclinical study

 $[\]hbox{\it [3] ASX Announcement 'Argenica completes pilot pre-clinical pharmacokinetics study' 01 July 2021}$



NEAR-TERM CLINICAL TRIAL CATALYSIS



GLP* SAFETY & TOXICOLOGY STUDIES

ANIMAL STUDY

Characterise the safety profile of **ARG-007** by identifying its impact on organ structure and / or functionality at a range of doses, including maximum tolerated dose.

COMPLETED



FINAL PHARMACOKINETIC STUDIES

ANIMAL STUDY

Determines how ARG-007 is absorbed, distributed, metabolised, and excreted by the body, and is essential for establishing dosing regimes for the Phase 1 clinical trial.

COMPLETED

PRE-STUDY ACTIVITIES

Completion of preliminary study activities, including clinical site management setup, preparation of ethics submission and healthy volunteer patient recruitment.

Q4 CY21 – Q2 CY22



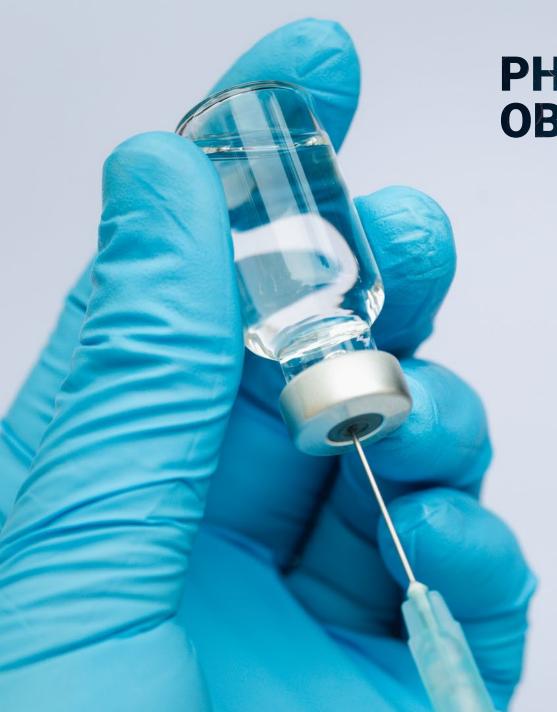
PHASE 1 CLINICAL TRIAL

IN-HUMAN STUDY

Preparation of ethics submission, ethics approval by ethics committee, initiate volunteer recruitment, dosing of all cohorts

Q2 - Q4 CY22





PHASE 1
OBJECTIVES

IMPROVE THE

UNDERSTANDING

OF HOW ARG-007

EFFECTS THE BODY

SAFETY OF ARG-007
WHEN ADMINISTERED

DETERMINE THE IDEAL SAFE DOSAGE

IDENTIFY ANY POSSIBLE

ADVERSE REACTIONS



PROPOSED TRIAL DESIGN*



4 cohorts, with each cohort receiving a different dose of **ARG-007** (8 participants in each cohort, 2 placebo and 6 receiving **ARG-007**).

LOCATION

Single site study conducted at the Linear Clinical Research facility in Western Australia.

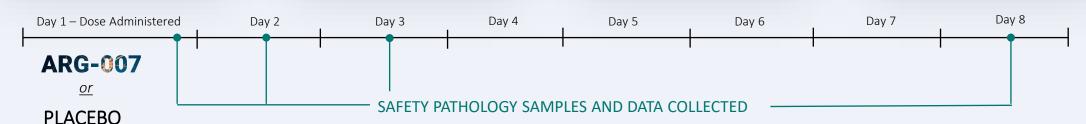


Estimated 6 months, with preliminary findings expected following completion of volunteer dosing.



Double-blind, randomised study where participants either receive a dose of **ARG-007** or a placebo.

PROCEDURE OUTLINE FOR EACH PARTICIPANT



*the trial design is subject to approval by a Human Research Ethics Committee (HREC). Trial design documentation and preclinical safety and toxicology data will be submitted to a HREC shortly.





PHASE 2 STUDIES



Data collected from the Phase 1 clinical trial will be critical to progress into Phase 2 trials, where **ARG-007** will be administered to **stroke patients**.



While stroke is the current corporate and commercial focus, safety data from the Phase 1 clinical trial can potentially be used to move directly into other Phase 2 trials in other types of brain injury, including;

- Hypoxic ischemic encephalopathy (HIE) A type
 of brain dysfunction that occurs when the brain doesn't
 receive enough oxygen or blood flow for a period of time.
- Traumatic brain injury (TBI) & concussion An injury resulting from a violent blow or jolt to the head or body.
- **Surgically induced stroke** stokes sustained during surgery including endovascular aneurysm repair, aortic repair, and transcatheter aortic valve implantation.



PIPELINE OF POTENTIAL APPLICATIONS

1		PRECLINICAL EFFICACY	PRECLINICAL SAFETY	PHASE 1	PHASE 2	PHASE 3
STROKE	Damage to the brain from interruption of its blood supply.			•		
SIS	Surgically Induced Stroke (SIS) occurs during or after surgery.					
HIE	Hypoxic ischemic encephalopathy (HIE) occurs when the brain doesn't receive enough oxygen or blood flow.					
SEVERE TBI	Trauma to the head or body causes a loss of consciousness.					
HEART ATTACK	Heart suddenly stops pumping blood around your body, the brain is starved of oxygen.				 	
MILD TBI	Brain injuries that result in concussions or feelings of confusion.					



ARG-007 POTENTIAL APPLICATIONS

SURGICALLY INDUCED STROKE



Stroke during surgery (perioperative stroke) can occur in up to 10% of patients undergoing high risk cardiac or brain surgery¹.



Close to 95% of these strokes are ischemic in nature, and outcomes tend to be worse than in non-surgical stroke patients¹.



Given patients are under anaesthetic, it is difficult to identify whether a patients has sustained a perioperative stroke as there are no clear clinical signs.



ARG-007 could provide protection to brain cells following perioperative stroke, similar to non-surgical stroke.
Following completion of the Phase 1 clinical trial, Argenica could move into a Phase 2 trial in perioperative stroke.





ARG-007 significantly reduced the volume of brain tissue death (infarct volume) for HIE Not significant (p=0.22)Very Significant (p=0.0009)Infarct Volume Significant (p=0.03)Est. 40% reduction vs. Hypothermia INJURY + ARG-007 **INJURY + SALINE INJURY + HYPOTHERMIA** (Saline used as Placebo) (Current standard of care) (300nmol/kg)

ARG-007 POTENTIAL APPLICATIONS

PERINATAL BRAIN INJURY



Hypoxic ischemic encephalopathy (HIE) is a type of brain dysfunction that occurs when the brain doesn't receive enough oxygen or blood flow for a period of time.



HIE may develop during pregnancy, labour and delivery, or in the postnatal period.



HIE is the leading cause of mortality and morbidity in newborn children, with survivors suffering significant neurological outcomes including cerebral palsy, epilepsy, intellectual disability and autism spectrum disorders.



Preclinical studies have shown Argenica's ARG-007 provides neuroprotection in an animal model of Perinatal hypoxicischemic encephalopathy (HIE).

7 Preclinical study www.argenica.com.au



ARG-007 POTENTIAL APPLICATIONS

TRAUMATIC BRAIN INJURY



Traumatic brain injury (TBI) usually results from a violent blow or jolt to the head or body.



Preclinical studies show that ARG-007 reduces neuronal injury after TBI.

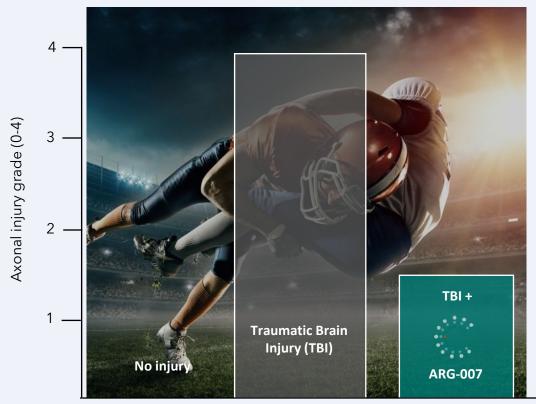


Neuroprotective pharmacological agents aimed at minimising harm to the brain and improving patient outcomes after a TBI are currently lacking.



TBI places a massive burden on society and the economy, a situation further compounded by its rising incidence.

Significant protective effect in other brain injury models

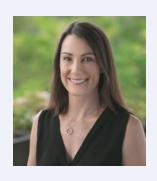


Protective effect of ARG-007 using the Traumatic Brain Injury preclinical model. ARG-007 significantly reduced axonal injury 5 days after a single i.v. injection

Preclinical study www.argenica.com.au



CREDENTIALLED & EXPERIENCED TEAM



Dr Liz Dallimore CEO & Managing Director

- PhD in Neuroscience (UWA) and an MBA (AGSM)
- Over 20 years' experience in R&D, technology commercialisation and management consulting, including at KPMG, EY and PWC
- Extensive background in stroke and spinal cord regeneration research at the Australian Neuromuscular Research Institute, UWA and Oxford University



Dr Samantha South Executive Director

- Extensive background in CNS medical research at Weill Medical College at Cornell University (NY), The University of Queensland and The Garvan Institute
- 10 years of Director experience at multiple companies
- Over 13 years' experience in technology transfer in medtech / biotech sector, at UQ, QUT and UWA.



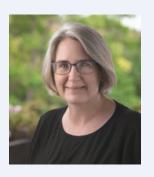
Emma Waldon Company Secretary

- Experienced Company Secretary with ASX listed and private companies
- Over 18 years' corporate advisory, capital markets and corporate governance experience
- Current co-sec of medical device developer EMVision (ASX: EMV) and previous co-sec of Hazer Group (ASX: HZR)



Geoff Pocock Non-Executive Chairman

- 20 years' experience in commercialisation of emerging technologies and capital markets
- Non-Executive Director of EMVision Medical Devices Ltd (ASX:EMV)
- Co-Founder / Former Managing Director of Hazer Group (ASX: HZR)



Liddy McCall Non-Executive Director

- Over 25 years' experience of senior Board and Management roles and has a strong history of success with early-stage Biotechnology companies
- Co-founded 3 biotechnology companies which have successfully achieved 3 FDA drug registrations and 1 FDA/CE Mark medical device approval
- Co-founder of iCeutica Inc group (acquired in 2011 achieving a ten-fold uplift on the valuation) and Dimerix Limited (ASX:DXB)



Terry Budge Non-Executive Director

- 25 years with National Australia Bank in senior executive roles before serving as managing Director of Bankwest from 1997 to 2004
- Previously a member of the Fundraising Committee of the Perron Institute, and formerly an independent director for Westoz Investment Company (ASX:WIC)



LEADING RESEARCH & CLINICAL TEAM



Prof. Bruno Meloni CSO & Research Lead

Head of Stroke Laboratory Research at UWA and the Perron Institute. Professor Meloni has over 25 years experience as a research scientist, the last 20 in the field of stroke/cerebral ischaemia. Research in the stroke/cerebral ischaemia field has focused on understanding the mechanisms associated with ischaemic brain injury, the identification of potential neuroprotective targets and the development of new therapies. A/Prof Meloni has experience with designing preclinical stroke trials, and the use of peptides as neuroprotective agents.



Geoffrey Donnan Member - CAC

Professor of Neurology at The University of Melbourne and former Director of The Florey Institute of Neuroscience and Mental Health. His research interest is clinical stroke management. He was co-founder, with Professor Stephen Davis, of the Australian Stroke Trials Network (ASTN) within which there have been conducted numerous investigator driven and other stroke trials. He was Editor-in-Chief of the International Journal of Stroke and is Past President of the World Stroke Organization.



Dr David Blacker Chairman - CAC

Acute stroke clinician/neurologist who has previous experience initiating neuroprotection clinical stroke trials in Western Australia and being the local Principal Investigator of a number of national and international acute and secondary prevention stroke studies. Prof Blacker is the Perron Institute Medical Director and consultant neurologist and stroke physician.



Paul Bailey Member - CAC

Medical Director for St John Ambulance Western Australia. Paul's research focus has been in the areas of out of hospital cardiac arrest, anaphylaxis, emergency department systems and trauma - with 29 papers published in the scientific literature since 2015. Paul and his team are active participants in the WA Stroke Advisory Group - which has transformed the clinical approach to stroke patients in the prehospital environment in WA.



Prof. Neville Knuckey Clinical Lead

Head of Stroke Research at the Perron Institute. Professor Knuckey is a neurosurgeon, whose interest for medical research began in the United States over 20 years ago. Prof Knuckey's main area of expertise is the development and use of stroke, global cerebral ischaemia and more recently perinatal hypoxia and traumatic brain injury models to explore neuronal ischaemic damaging events, and for the assessment of potential neuroprotective therapies.



Tim Phillips Member - CAC

Dr Tim Phillips is an Interventional Neuroradiologist with 15 years' experience, currently working at the Neurological Intervention and Imaging Service of Western Australia (NIIS WA) and the Perth Children's Hospital. Prior to returning to Perth he undertook post-specialist fellowship training at the Royal Melbourne Hospital, The Royal London Hospital, Queens Hospital Romford, The National Hospital for Neurology and Neurosurgery, and Great Ormond Street Hospital in London.



INVESTMENT HIGHLIGHTS

3#

PRE-CLINICAL DATA

Positive pre-clinical results provide encouraging dataset leading into in-human trials.

CLEAR PATHWAY TO TRIALS

Clear clinical pathway and robust capital position to execute Phase 1 clinical trials.

2#

UNMET CLINICAL NEED

There is an urgent unmet need and priority to search for widely applicable and effective neuroprotective solution.

EXCEPTIONAL TEAM

4#

An exceptional team with expertise in drug development commercialisation and capital markets.

NEAR TERM CATALYSIS

Value enhancing milestones expected in the near term including, Phase 1 clinical trial initiation and first data read outs from the trial.



