ABN 16 165 160 841

ASX RELEASE **24th Aug 2023**

CEO & Managing Director's Presentation to Annual General Meeting

Melbourne, Australia: Amplia Therapeutics Limited (ASX: ATX) ("Amplia" or the "Company") is pleased to release the CEO & Managing Director's presentation to the Company's Annual General Meeting (YE 31 March 2023) to be held today.

This ASX announcement is authorised for release by the Company Secretary.

Investor Contact:
Dr Chris Burns
Chief Executive Officer
chris@ampliatx.com

About Amplia Therapeutics Limited

Amplia Therapeutics Limited is an Australian pharmaceutical company advancing a pipeline of Focal Adhesion Kinase (FAK) inhibitors for cancer and fibrosis. FAK is an increasingly important target in the field of cancer immunology and Amplia has a particular development focus in fibrotic cancers such as pancreatic cancer. FAK also plays a significant role in a number of chronic diseases, such as idiopathic pulmonary fibrosis (IPF). For more information visit www.ampliatx.com and follow Amplia on Twitter (@ampliatx) and LinkedIn.

Ampliа

Annual General Meeting

CEO Presentation

24th August 2023

ampliatx.com | @ampliatx

DISCLAIMER

The information contained in the presentation is not intended to be an offer for subscription, invitation or recommendation with respect to shares of Amplia Therapeutics Limited ("Amplia") in any jurisdiction. This presentation is not a prospectus, product disclosure statement or other offering document under Australian law (or any other law). No representation or warranty, express or implied, is made in relation to the accuracy or completeness of the information contained in this document or opinions expressed in the course of this presentation. The information contained in this presentation is subject to change without notification.

This presentation may contain forward-looking statements which can be identified by the use of words such as "may", "should", "will", "expect", "anticipate", "believe", "estimate", "intend", "scheduled" or "continue" or similar expressions. Any forward-looking statements contained in this presentation are based on Amplia's good-faith assumptions as to the financial, market, regulatory and other risks and considerations, and are subject to significant risks, uncertainties, assumptions, contingencies and other factors (many of which are outside the control of, and unknown to Amplia, and its officers, employees, agents or associates), which may cause the actual results or performance to be materially different from any future result so performed, expressed or implied by such forward-looking statements. There can be no assurance or guarantee that any of the assumptions will prove to be correct or that actual outcomes will not differ materially from these statements, and the differences may be adverse. Accordingly, you should not place undue reliance on these forward-looking statements.

To the maximum extent permitted by law, Amplia disclaims any obligation or undertaking to publicly update or revise any forward-looking statements contained in this presentation, whether as a result of new information, future developments or a change in expectations or assumptions, and disclaims all responsibility and liability or any loss arising from this presentation or reliance on anything contained in or omitted from it.

COMPANY SNAPSHOT

Amplia THERAPEUTICS

CORPORATE

Share Price: 8c

Valuation: $16m

Smooth CEO transition

ACCENT TRIAL

Seven sites open - VIC, NSW, QLD

13 patients dosed - Phase 1b

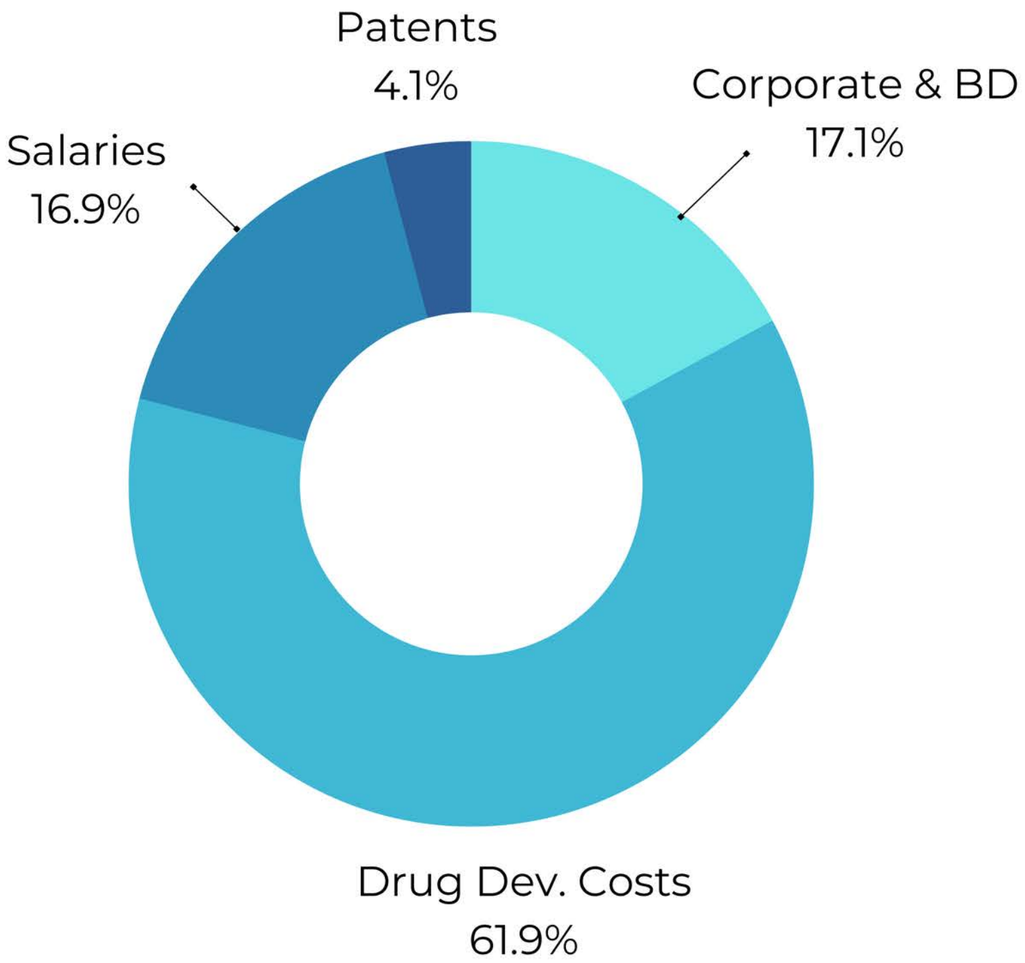
PRECLINICAL

CSIRO collaboration - wound healing

AMP945 with FOLFIRINOX

CORPORATE

USE OF FUNDS



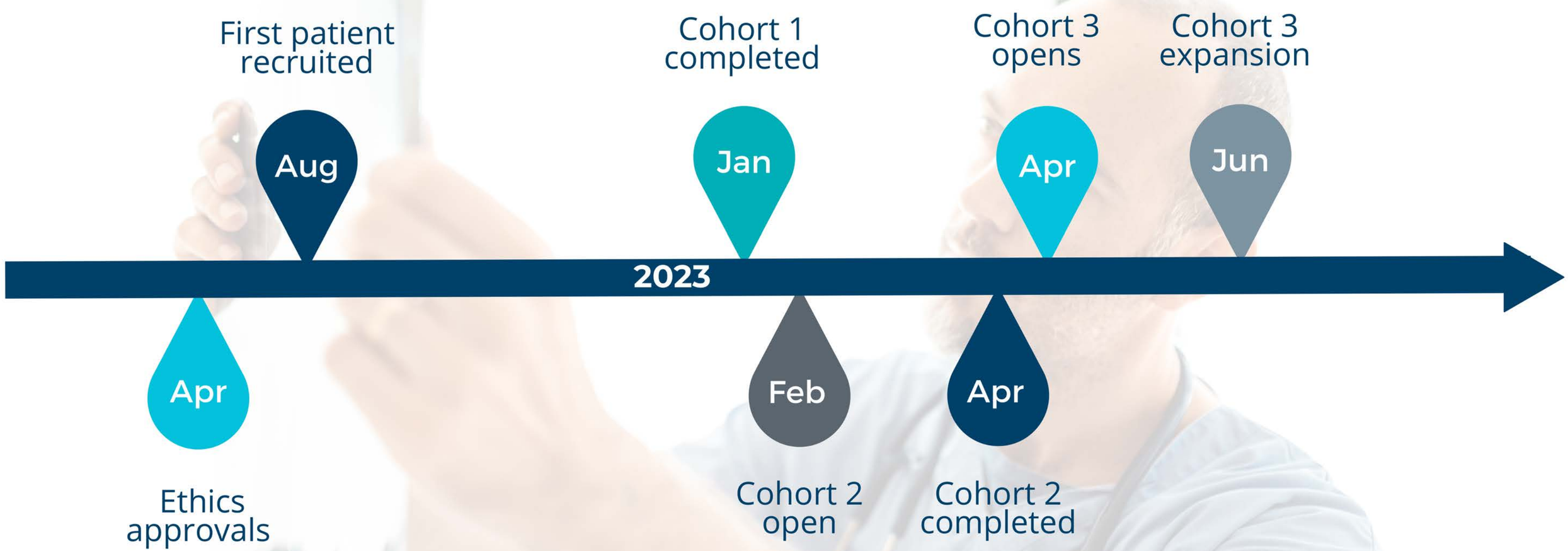
INCREASED PROFILE

- Good publicity on ACCENT trial
- Presentations at international scientific and industry conferences
- Active in local ecosystem

INCREASED BD ACTIVITY

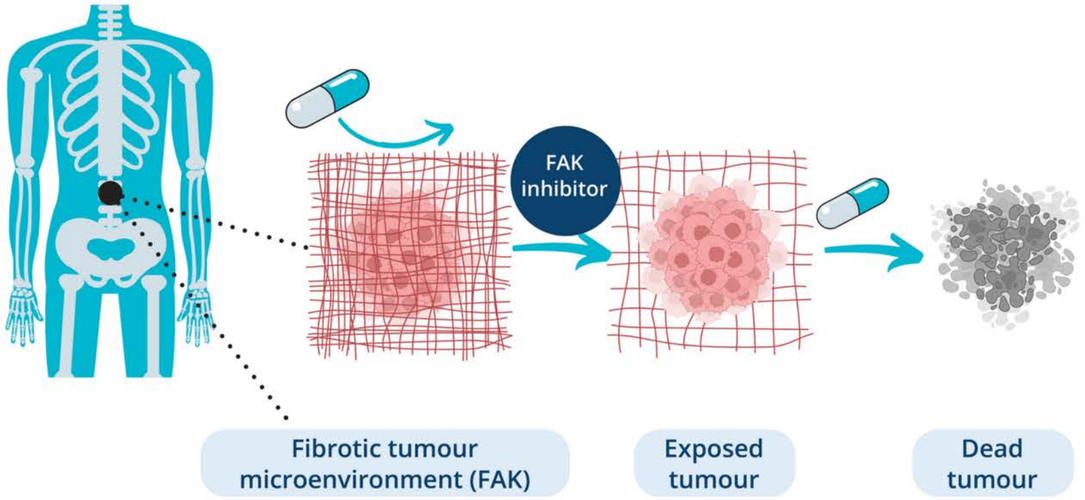
- Participation at AusBIO, BIOKorea, BIO
- Preliminary discussions with Pharma/biotech partners

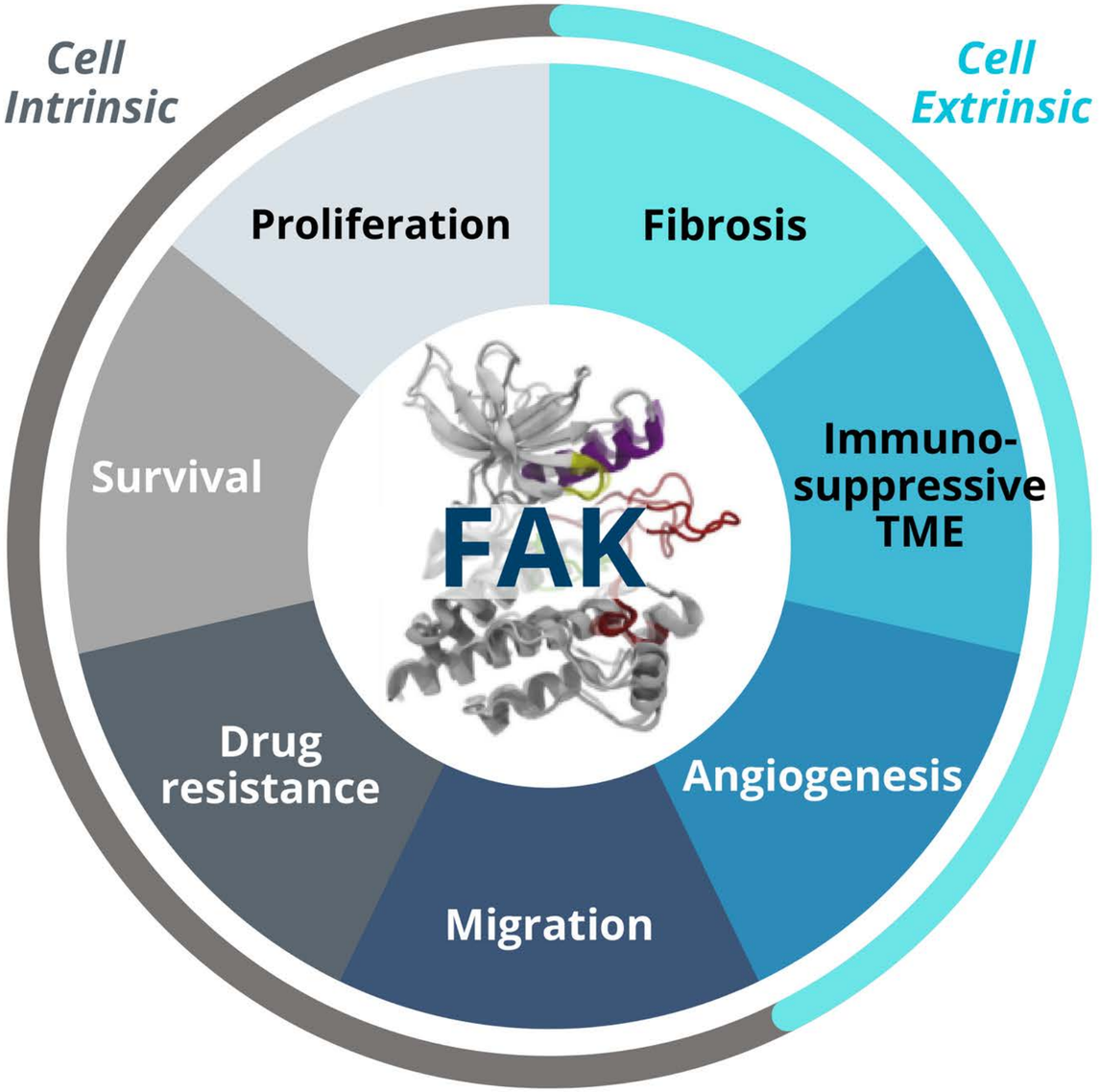
ACCENT TRIAL



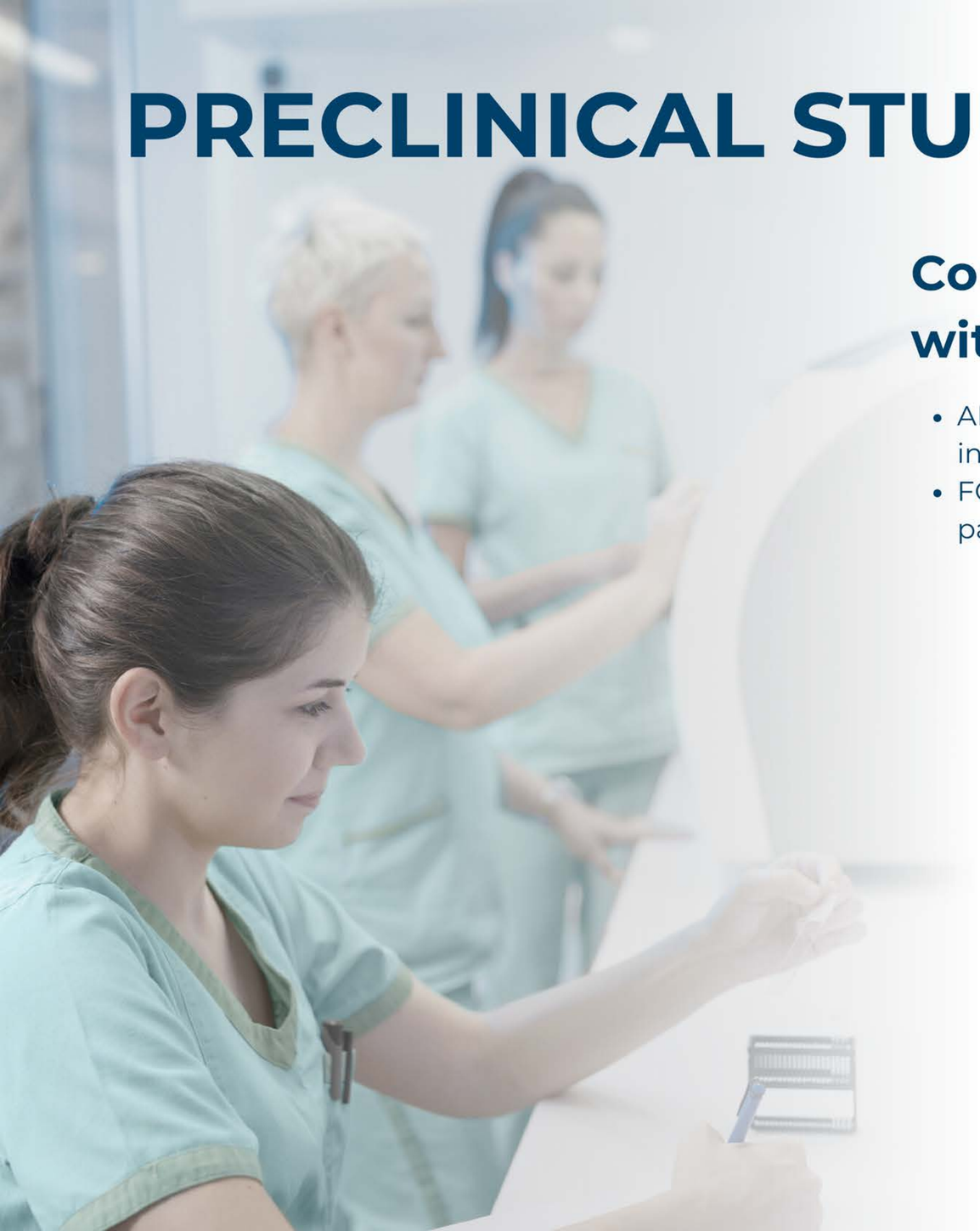
FAK INHIBITION IN CANCER

- Preclinical and clinical evidence of role of FAK in tumour growth, resistance to chemotherapy and radiotherapy, in immuno-modulation and in formation of fibrotic tissue
- FAK activity promotes cancer both within cancer cell and in tumour microenvironment



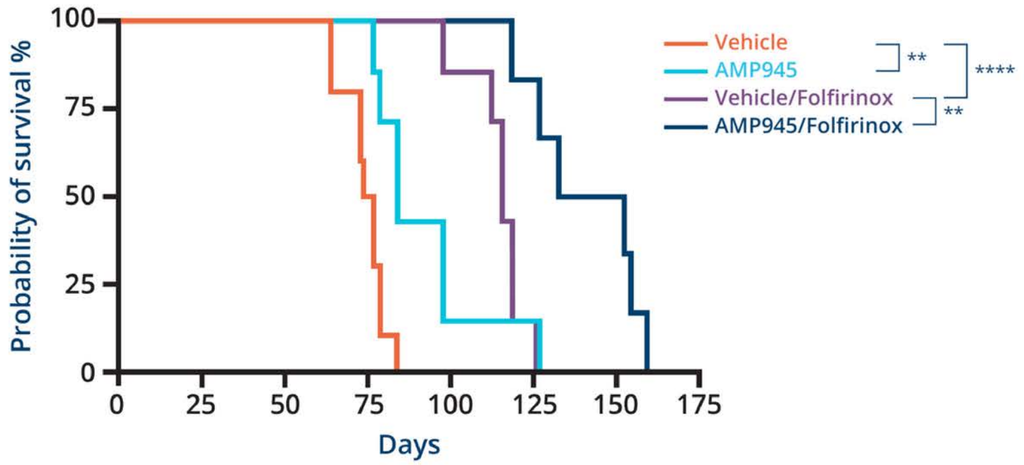


PRECLINICAL STUDIES



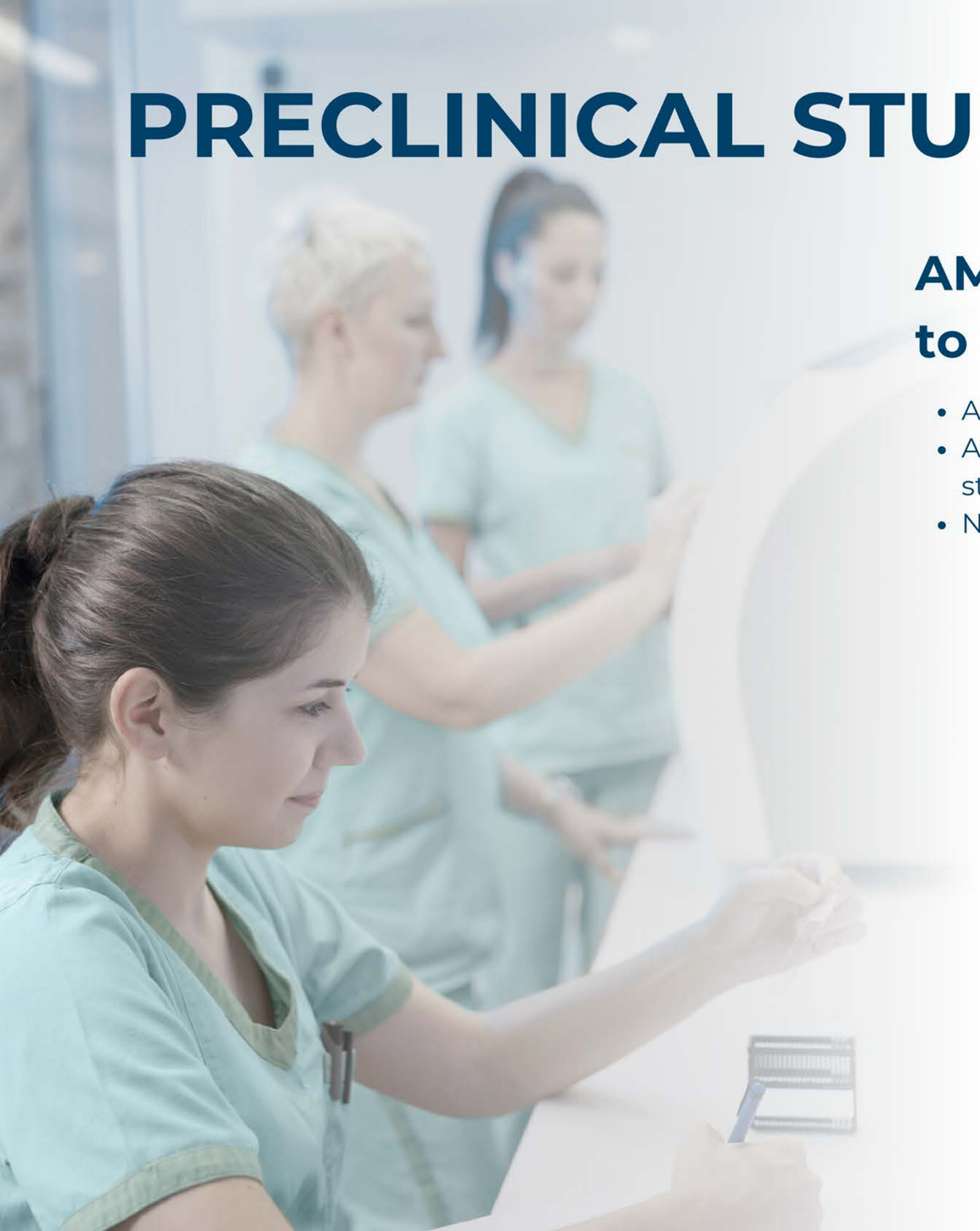
Combination benefit demonstrated with FOLFIRINOX

- AMP945 combines with FOLFIRINOX chemotherapy to improve survival in a mouse model of aggressive patient-derived pancreatic cancer
- FOLFIRINOX chemotherapy is preferred treatment for advanced pancreatic cancer in many countries, including US and Europe



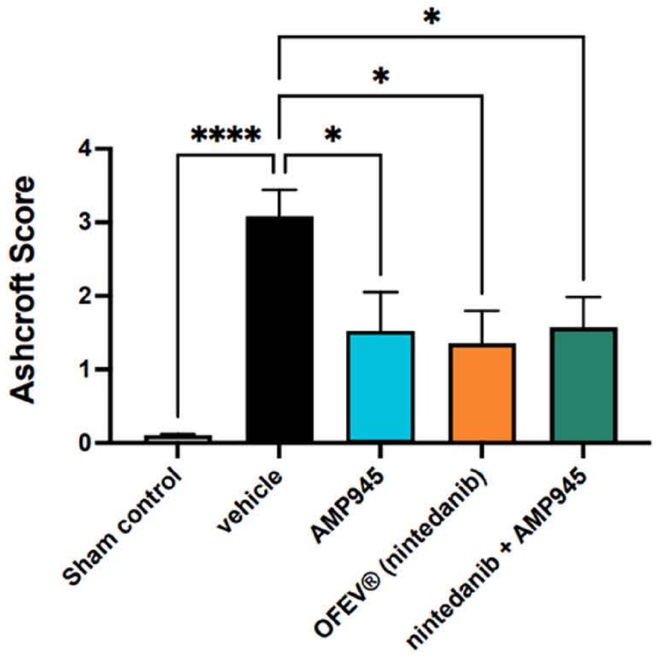
AMP945 treatment for 3 days preceding FOLFIRINOX treatment, on an 14 day cycle

PRECLINICAL STUDIES

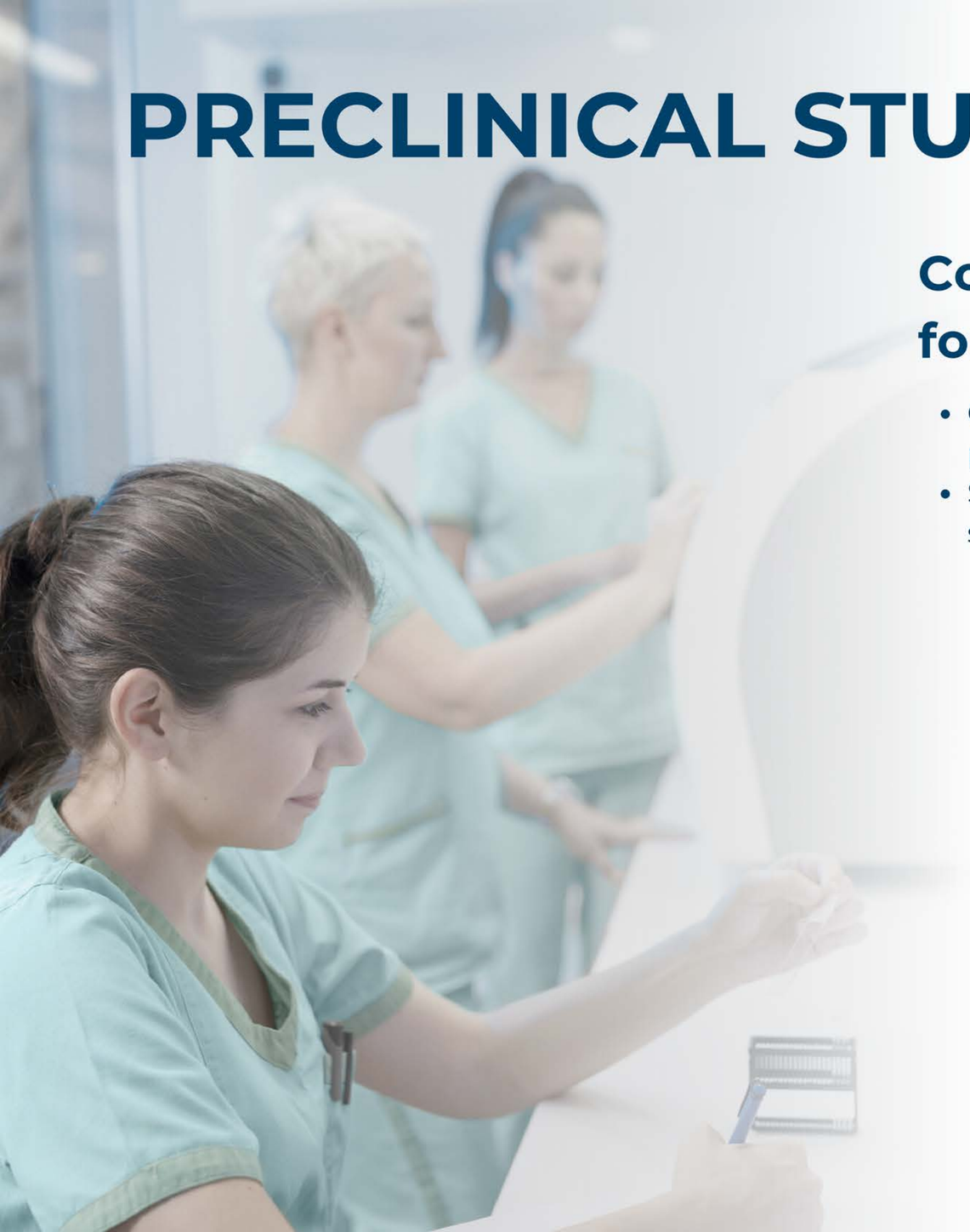


AMP945 shows activity equivalent to standard-of-care in IPF model

- AMP945 is effective in the industry standard model of IPF in the mouse
- AMP945 reduces lung fibrosis (Ashcroft score) to the same level as current standard-of-care drug nintedanib
- No combination effects observed indicating anti-fibrotic effect is maximised



PRECLINICAL STUDIES



Collaboration with CSIRO on formulation for topical delivery

- Collaboration with CSIRO to develop topical formulation of our drugs for potential treatment of wounds and burns
- Strong scientific rationale for improved wound healing and reduced scarring with FAK inhibition

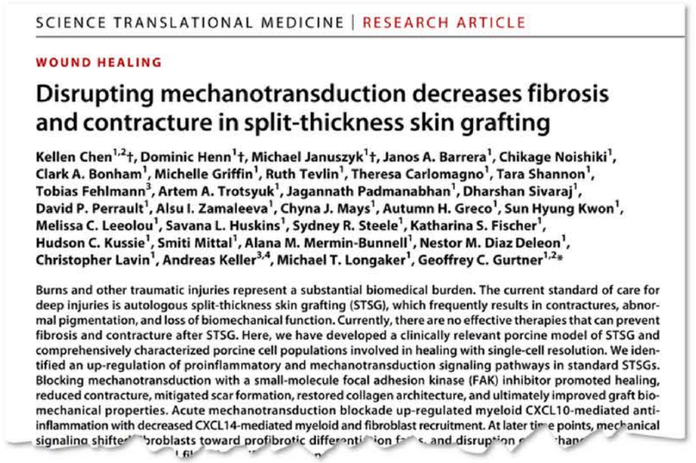
nature medicine

Focal adhesion kinase links mechanical force to skin fibrosis via inflammatory signaling

Victor W Wong[1], Kristine C Rustad[1], Satoshi Akaishi[1], Michael Sorkin[1], Jason P Glotzbach[1], Michael Januszyk[1], Emily R Nelson[1], Kemal Levi[1], Josemaria Paterno[1], Ivan N Vial[1], Anna A Kuang[2], Michael T Longaker[1] & Geoffrey C Gurtner[1]

Exuberant fibroproliferation is a common complication after injury for reasons that are not well understood[1]. One key component of wound repair that is often o mechanical force, which regulates cell-matrix through intracellular focal adhesion componer focal adhesion kinase (FAK)[1,2]. Here we repor is activated after cutaneous injury and that thi potentiated by mechanical loading. Fibroblast knockout mice have substantially less inflamn fibrosis than control mice in a model of hypert formation. We show that FAK acts through extr kinase (ERK) to mechanically trigger the secre chemoattractant protein-1 (MCP-1, also know

candidate pathways driving early scar formation, we performed a genome-wide microarray analysis on wild-type mouse scars that had



SCIENCE TRANSLATIONAL MEDICINE | RESEARCH ARTICLE

WOUND HEALING

Disrupting mechanotransduction decreases fibrosis and contracture in split-thickness skin grafting

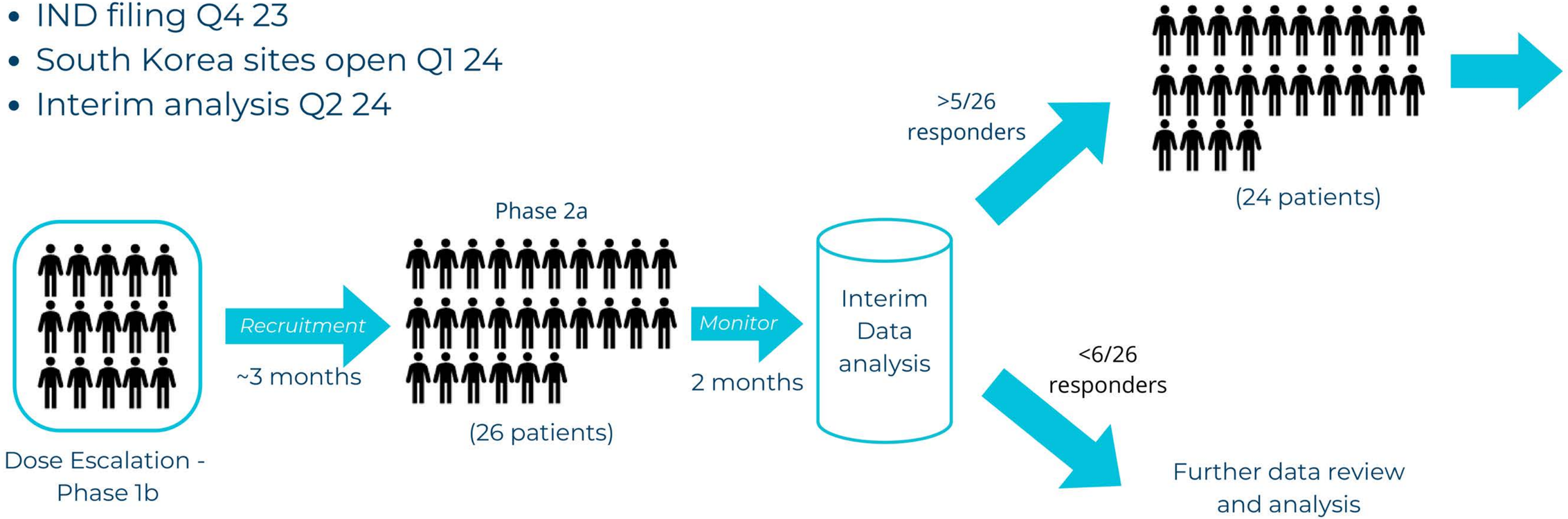
Kellen Chen[1,2]†, Dominic Henn[1]†, Michael Januszyk[1]†, Janos A. Barrera[1], Chikage Noishiki[1], Clark A. Bonham[1], Michelle Griffin[1], Ruth Tevlin[1], Theresa Carlomagno[1], Tara Shannon[1], Tobias Fehlmann[3], Artem A. Trotsyuk[1], Jagannath Padmanabhan[1], Dharshan Sivaraj[1], David P. Perrault[1], Alsu I. Zamaleeva[1], Chyna J. Mays[1], Autumn H. Greco[1], Sun Hyung Kwon[1], Melissa C. Leeolou[1], Savana L. Huskins[1], Sydney R. Steele[1], Katharina S. Fischer[1], Hudson C. Kussie[1], Smiti Mittal[1], Alana M. Mermin-Bunnell[1], Nestor M. Diaz Deleon[1], Christopher Lavin[1], Andreas Keller[3,4], Michael T. Longaker[1], Geoffrey C. Gurtner[1,2]*

Burns and other traumatic injuries represent a substantial biomedical burden. The current standard of care for deep injuries is autologous split-thickness skin grafting (STSG), which frequently results in contractures, abnormal pigmentation, and loss of biomechanical function. Currently, there are no effective therapies that can prevent fibrosis and contracture after STSG. Here, we have developed a clinically relevant porcine model of STSG and comprehensively characterized porcine cell populations involved in healing with single-cell resolution. We identified an up-regulation of proinflammatory and mechanotransduction signaling pathways in standard STSGs. Blocking mechanotransduction with a small-molecule focal adhesion kinase (FAK) inhibitor promoted healing, reduced contracture, mitigated scar formation, restored collagen architecture, and ultimately improved graft biomechanical properties. Acute mechanotransduction blockade up-regulated myeloid CXCL10-mediated anti-inflammation with decreased CXCL14-mediated myeloid and fibroblast recruitment. At later time points, mechanical signaling shifted fibroblasts toward profibrotic differentiation

FUTURE MILESTONES

ACCENT TRIAL

- Dose escalation complete Q4 23
- IND filing Q4 23
- South Korea sites open Q1 24
- Interim analysis Q2 24



2023 | 2024

FUTURE MILESTONES

PRECLINICAL STUDIES - EXPANDING CLINICAL POTENTIAL AND COMMERCIAL OPPORTUNITIES

Data from ovarian cancer studies **AMP945**

Data from final IPF studies **AMP945**

2023

2024

Data from cancer studies **AMP886**

Preliminary data from wound healing studies **AMP945**

Mission

Developing life-changing medicines for people with cancer and fibrotic diseases

Vision

> To be a world class clinical stage biotechnology company with a pipeline of innovative medicines that improve patients' lives

> To be the definitive partner for translating medical discoveries to clinical proof of concept

> To have a workplace that inspires

Values

Patients first | Integrity | Respect | Performance | Innovation | Accountability | Excellence

Thank you.

Amplia Therapeutics Limited
ABN 16 165160 841
ASX: ATX
info@ampliatx.com
ampliatx.com

Chris Burns PhD GAICD
CEO and MD

chris@ampliatx.com