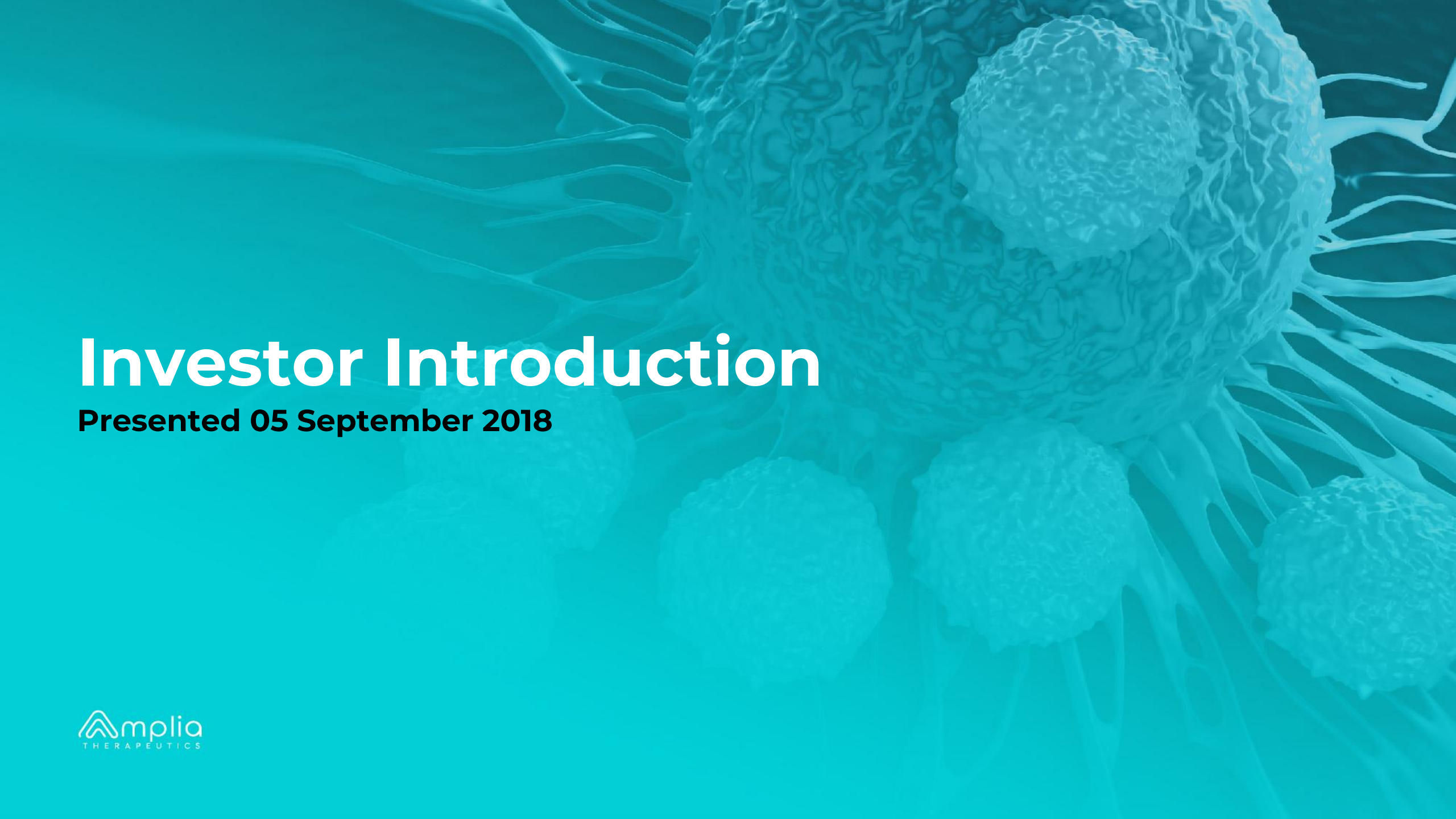


Investor Introduction

Presented 05 September 2018

Amplia THERAPEUTICS

Notice

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. 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.

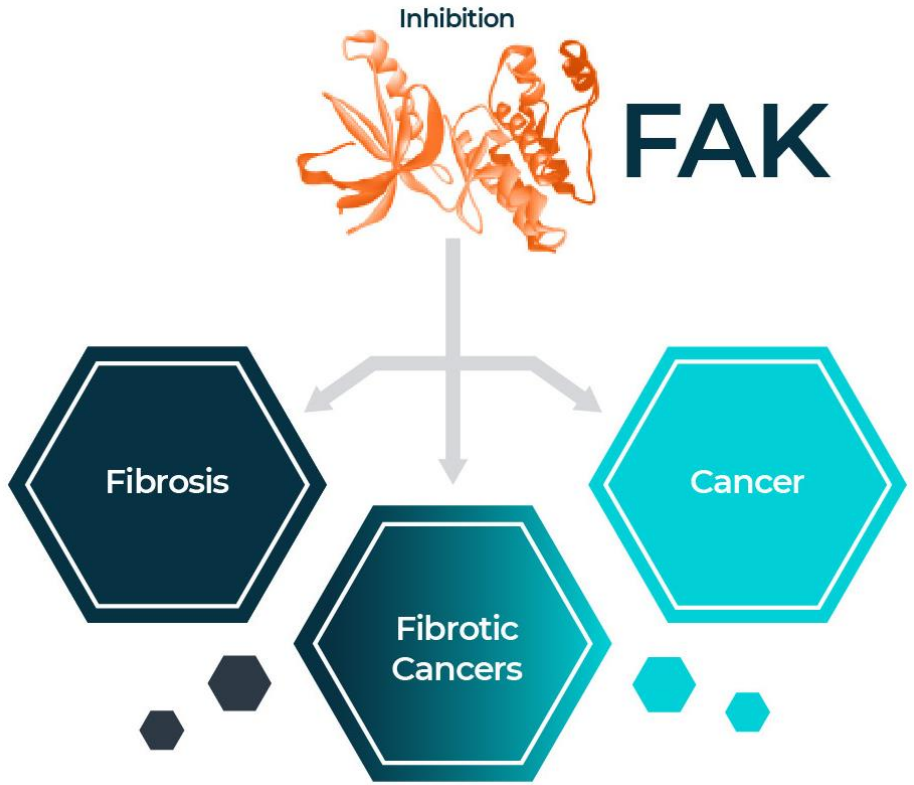
This presentation contains forward-looking statements which can be identified by the use of words such as "may", "should", "will", "expect", "anticipate", "believe", "estimate", "intend", "scheduled" or "continue" or similar expressions. Any forward-looking statements contained in this presentation are 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 actual outcomes will not differ materially from these statements. The data and results pertaining to clinical subjects used in this presentation are illustrative of medical conditions and outcomes associated with potential applications of Amplia's acquired product pipeline. Actual results from clinical trials may vary from those shown.

05 September 2018

Focal Adhesion Kinase - 101

- Fibrosis is the formation of excess fibrous connective tissue which can adversely affect normal organ or tissue architecture and function
- In cancer, fibrosis triggered by tumours can interfere with existing chemotherapies or rendered them completely ineffective
- Fibrosis can hide certain tumours from the immune system rendering breakthrough immuno-oncology (I-O) treatments ineffective
- Cell adhesion molecules regulate fibrotic processes
- Focal adhesion kinase (FAK) is an important protein involved in cell adhesion, migration and invasion as well as immune responses to tumours and is thus a significant target in both cancer and fibrotic diseases
- Blocking / inhibiting FAK safely could make difficult to treat tumours responsive to existing chemotherapeutics or provide a much needed breakthrough in the treatment of tumours that are resistant to I-O therapies



Amplia THERAPEUTICS

Value Proposition

- Amplia has an exclusive worldwide licence to develop and commercialise two best-in-class FAK inhibitors (FAKi) developed by the Cancer CRC : AMP945 and AMP886
- AMP945 is a “pure play” FAKi with superior specificity. AMP886 is a multi-action molecule that hits two other important cancer pathways – VEGFR3 and FLT3
- Two molecules, multiple indications = multiple “shots on goal”
- The immediate objective is to have AMP945 (US) ‘IND Phase II ready’ in the second half of 2019 = significant value inflection point
- The clinical pathway to achieve this should be low risk given that two 3rd party FAKi drug candidates have successfully completed Phase I safety trials
- Experienced drug development team and strong academic partners, backed by the Cancer CRC (Australia) and Cancer Research UK

Exclusive Worldwide Licence

- Amplia has in-licensed AMP945 and AMP886 from Cancer Research Technology (CRT), the development and commercialisation arm of Cancer Research UK – the world's leading cancer research charity
- AMP945 and AMP886 were originally developed by Melbourne based Cancer Therapeutics CRC (CTx), a collaborative partnership of leading Research Institutes, Universities, and Biotechs, supported by the Australian Cooperative Research Centre (CRC) Program
- CTx "is in the business of finding cures for cancer" and until mid 2014 promising candidates were licensed to CRT for further development and commercial licensing
- In 2016 CRT licensed a CTx developed protein inhibitor (PRMT5) to MSD (Merck) with an upfront of US$15 million, plus potential milestones of US$500 million, plus royalties on sales
- Dr Warwick Tong (then CTx CEO) said of the PRMT5-MSD transaction, "This is a great result for Australian science … and further demonstrates what can be achieved when science and commercialisation capabilities unite"

Experienced Team

Warwick Tong

MB ChB MPP GAICD
Chairman

Ex-GSK, experienced drug developer. Former CEO and Director, Cancer CRC.

Simon Wilkinson

Managing Director & CEO

20 yrs biotech executive management & capital markets experience.

Robert Peach

PhD
Independent Director

25+ yrs experience including senior executive and scientific positions at Apoptos, Biogen Idec, IDEC and Bristol-Myers Squibb.

Co-founder and CSO of Receptos (2009) which was acquired by Celegen for US$7.8B in 2015.

Christian Behrenbruch

D.Phil (Oxon) MBA JD
Director

Seasoned biotech entrepreneur.

Director, Factor Therapeutics (ASX : FTT)

CEO of Telix Pharmaceuticals (ASX : TLX)

Experienced Team

Chris Burns

PhD, FRSC FRACI Director

Over 20yrs experience in Pharma, biotech and academia.

Discovered clinically trialled drugs momelotinib and lexibulin. 50+ scientific publications, 30+ patents.

Mark Delvin

PhD, Chief Scientific Advisor

Experienced drug discovery biologist.

COO for Cancer Research CRC.

Previously Director of Translational Cancer Biology for the CTx where he has worked extensively on FAK inhibitors.

Mark Sullivan

Regulatory Affairs Adviser

Experienced drug development professional (ex GSK, Gilead).

Founder and Managing Director, Medicines Development for Global Health.

Damian Slizys

BSc(Hons), LLB(Hons), PhD
Intellectual Property Adviser

Principal Falkenheim Advisory.

Previously FPA Patent Attorneys.

Amplia THERAPEUTICS

Scientific Advisors

Prof. Margaret Frame

OBE, PhD

Science Director and Chair of Cancer Biology, University of Edinburgh.

Global thought leader in FAK.

Neil Carragher

PhD

Professor of Drug Discovery and Director of Edinburgh Cancer Discovery Unit.

Experienced FAK researcher.

Alan Serrels

PhD

Research Fellow, MRC Centre for Inflammation Research.

Identifying FAK's role in suppressing ant-tumour responses and strategies for combination treatment strategies to overcome this cancer defense mechanism.

What does FAK do?

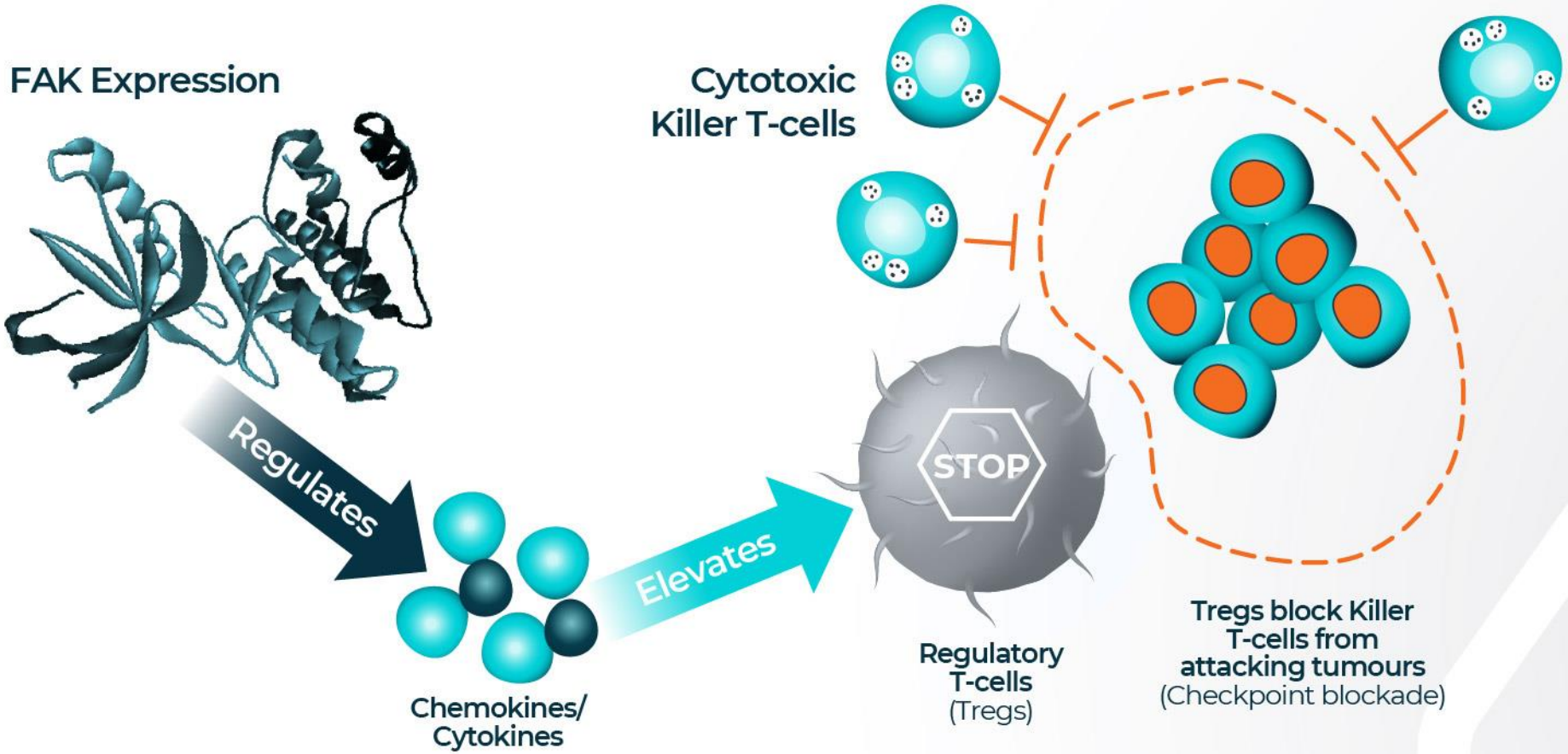
- Focal Adhesion Kinase is frequently up-regulated in many cancer types
- Signals functions that are important for cancer development and metastatic disease
- Co-opted by cancer to assist spread and to develop resistance to therapy
- FAK-dependent signaling drives elevated levels of regulatory T-Cells which in turn suppress the all important anti-tumour T-cell response
- Role in fibrosis has impact in both “fibrotic cancers” (e.g. pancreatic/ovarian), as well as non-cancer fibrotic diseases (e.g. lung/liver fibrosis)

Key Messages

1. FAK has a fundamental role in cancer. Blocking FAK opens the door for new combination therapies with approved drugs.
2. FAK plays a major role suppressing the immune attack on tumours. Blocking FAK allows immuno-oncology drugs (e.g. checkpoint inhibitors) to work in tough cancers like pancreatic cancer.
3. The planned clinical development pathway creates the opportunity for multiple indications in both cancer and non-cancer applications.

Amplia THERAPEUTICS

FAK expression contributes to tumour immunity in fibrotic cancers



Drugs that target FAK are novel because they target the tumour micro-environment, the supportive infrastructure that enables cancer cells to "hide" from the immune system

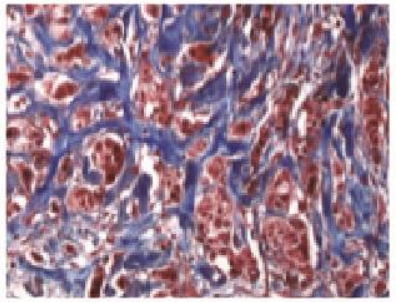
Inhibiting FAK appears to unlock the potency of checkpoint inhibitor drugs (i.e.PD-1/PD-L1, CTLA-4), particularly in fibrotic cancers like pancreatic cancer.

(Diagram adapted from Symeonides et. al. J Immunotherapy Cancer. 2017; 5: 17.)

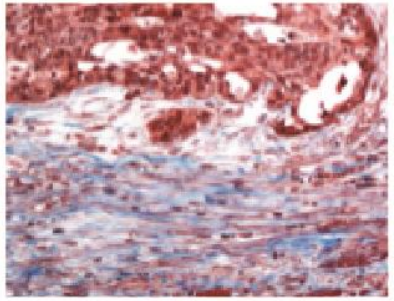
Amplia THERAPEUTICS

Fibrotic Cancers

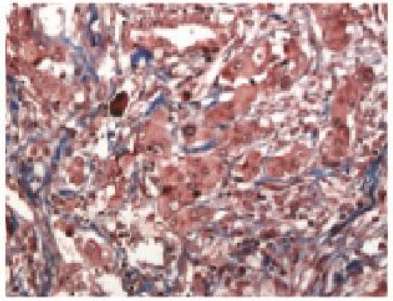
Breast Cancer



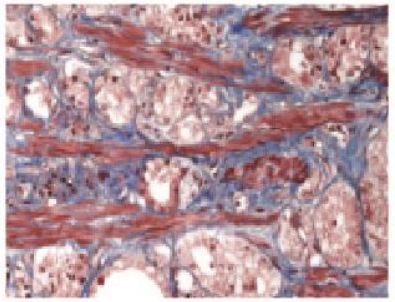
Esophageal Cancer



Lung Cancer

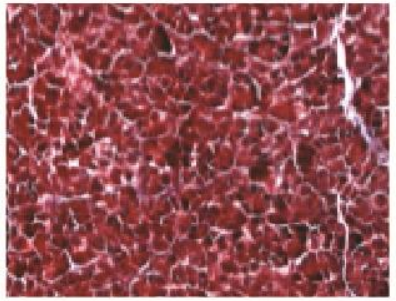


Prostate Cancer

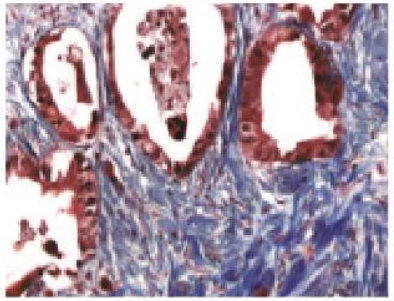


Fibrotic cancers have a very different immunologic profile. Fibrotic cancers use the tumour micro-environment as part of their defence against the immune system

Normal (Pancreas)



Pancreatic Cancer



The biggest unmet need in cancer therapy is pancreatic cancer. Pancreatic cancer is the epitome of a fibrotic tumour that can potentially be attacked with a FAK inhibitor in combination with other immuno-oncology therapies

Pancreatic Cancer:

- worst survival outcome of the 21 most common cancers
- death rates are increasing while most other cancers declining
- predicted to overtake breast cancer as the 4th most common cancer killer by 2030

Amplia THERAPEUTICS

FAK promotes tumour evasion by inducing an immunosuppressive microenvironment

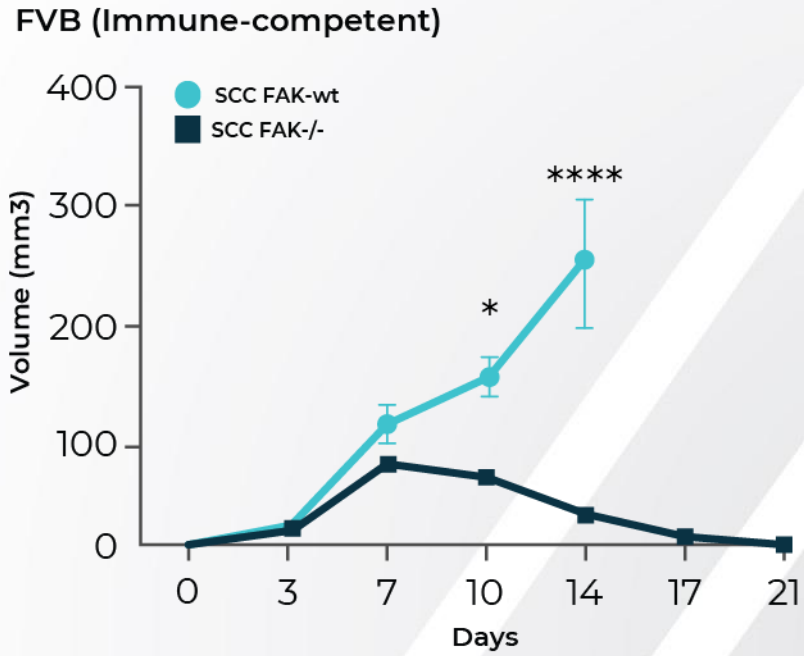
Cell

Article

Nuclear FAK Controls Chemokine Transcription, Tregs, and Evasion of Anti-tumor Immunity

Alan Serrels,[1,7,*] Tom Lund,[1,7] Bryan Serrels,[1] Adam Byron,[1] Rhoanne C. McPherson,[2] Alexander von Kriegsheim,[1] Laura Gómez-Cuadrado,[1] Marta Canel,[1] Morwenna Muir,[1] Jennifer E. Ring,[3] Eleni Maniati,[4] Andrew H. Sims,[1] Jonathan A. Pachter,[3] Valerie G. Brunton,[1] Nick Gilbert,[5] Stephen M. Anderton,[2] Robert J.B. Nibbs,[6] and Margaret C. Frame[1,*]

Inhibiting FAK decreases immunosuppressive cell populations in tumours and may enable immunotherapy to work in cancer populations that have a generally poor response to checkpoint inhibitor drugs.



When FAK is genetically ablated ($FAK^{-/-}$) from malignant squamous cell carcinoma (SCC) cells transplanted into immune-competent FVB mice, the immune response leads to significant tumour regression compared to SCC cells with wild type FAK cells present (FAK-wt).

Amplia THERAPEUTICS

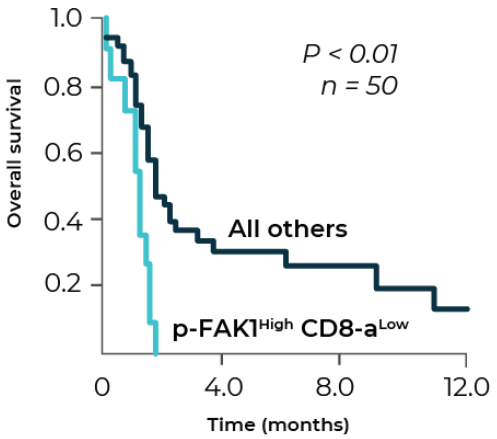
Pancreatic Cancer responsive to FAKi + combination therapies

nature medicine

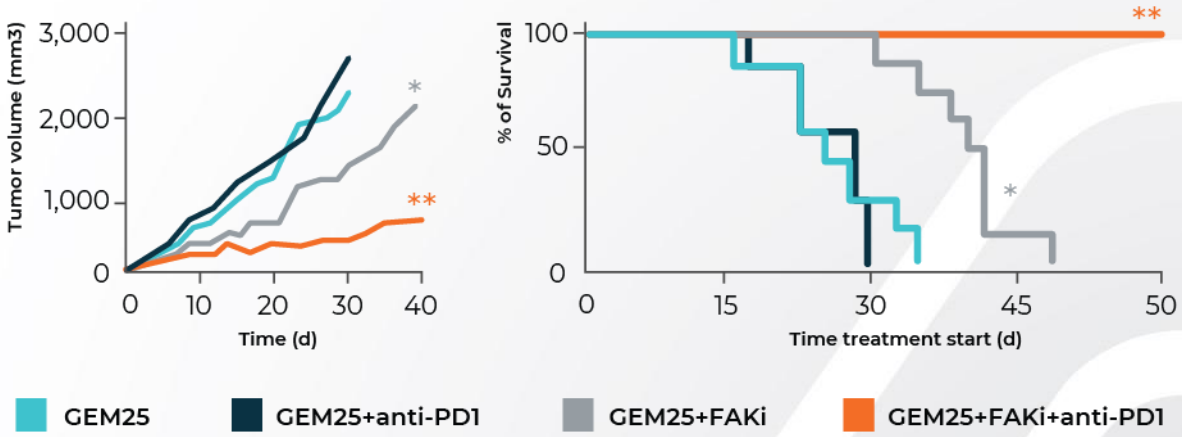
Targeting focal adhesion kinase renders pancreatic cancers responsive to checkpoint immunotherapy

Hong Jiang[1,2], Samarth Hegde[1,2], Brett L Knolhoff[1,2], Yu Zhu[1,2], John M Herndon[1,2], Melissa A Meyer[1,2], Timothy M Nywening[3], William G Hawkins[3,4], Irina M Shapiro[5], David T Weaver[5], Jonathan A Pachter[5], Andrea Wang-Gillam[1,4] & David G DeNardo[1,2,4,6]

VOLUME 22 | NUMBER 8 | AUGUST 2016



In 50 patients with Pancreatic ductal adenocarcinoma (PDAC), high FAK activity and related poor CD8+ cytotoxic T cell infiltration, was associated with poor overall survival



In KPPC transgenic mice, FAK inhibition rendered PDAC tumours responsive to both Gemcitabine chemotherapy and checkpoint immunotherapy with 3-way combination treatment conferring 100% survival

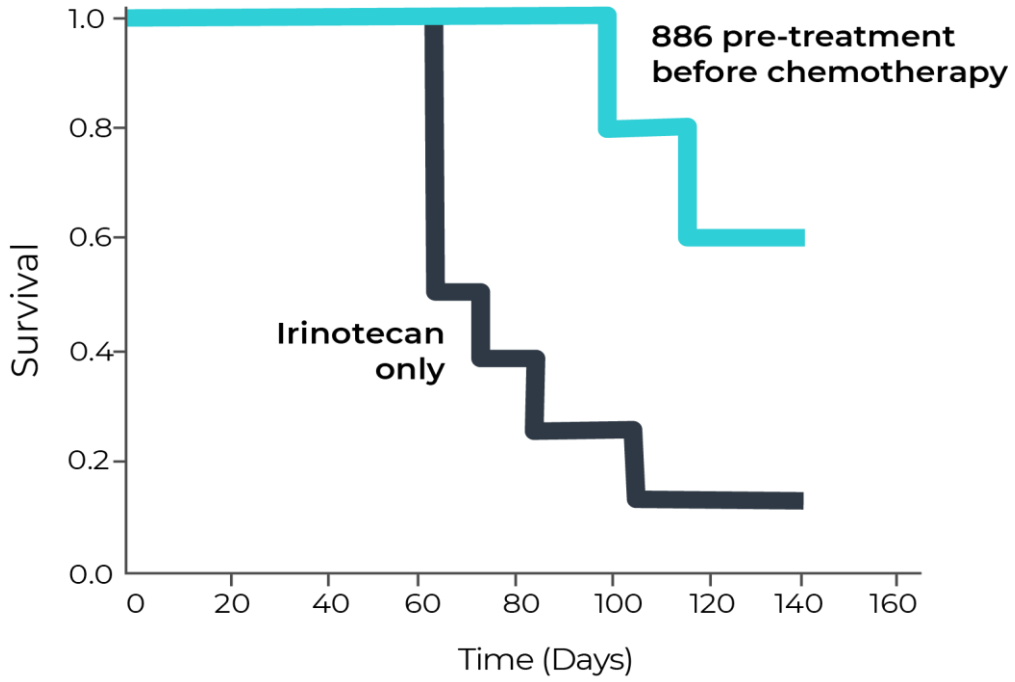
Amplia THERAPEUTICS

The Amplia FAK Assets

- Novel orally available small molecule FAK inhibitors
  - **AMP945** – Highly selective
  - **AMP886** – Highly differentiated, multi-action (FAK/FLT3/VEGFR3)
- Excellent potency and selectivity, very promising pharmacokinetics and physical-chemistry properties
- Expected superior safety profile in I-O combo therapy compared to I-O plus immuno-stimulators
- Scale-up (kg scale) chemistry already developed in partnership with an international commercial contract manufacturer
  - Including methods that are suitable for GMP production of clinical trial material
  - Highly cost effective common synthetic routes for both molecules
- Strong intellectual property position
  - National phase filing in commercially important jurisdictions
  - Clear strategy to expand scope of existing IP

AMP886 “Triple kinase FAKi” sensitizes tumours to chemotherapy

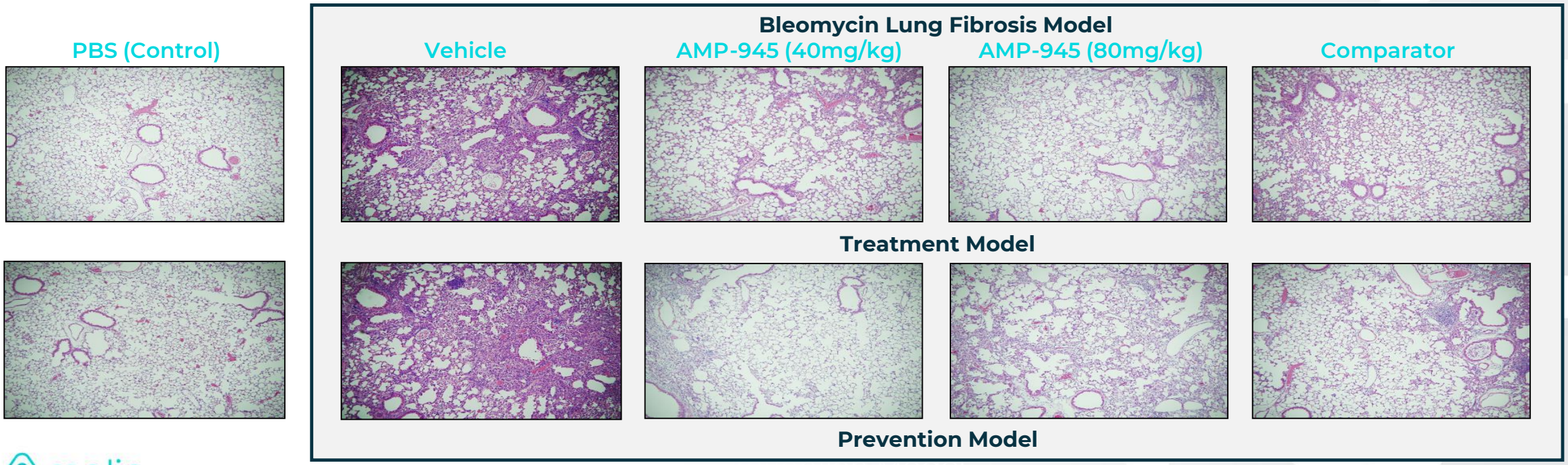
Inhibiting FAK with the multi-action AMP886 agent combined with Irinotecan, has a significant impact on tumour growth and survival in mouse models of pancreatic cancer (human PANC-1)



Amplia THERAPEUTICS

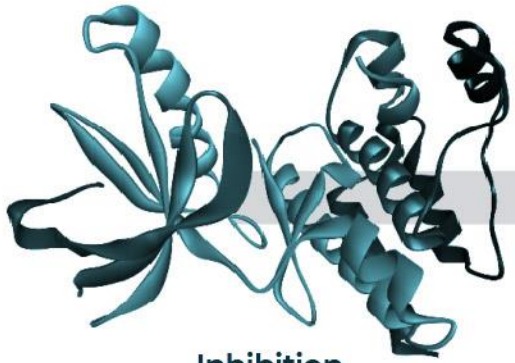
Non-cancer indications: FAK and fibrosis*

- AMP945 has been extensively evaluated in both prevention and treatment animal models of fibrosis*, including on a comparative basis with other FAK inhibitors under development
- In these models, AMP945 has demonstrated excellent efficacy
- Current poor treatment options in serious chronic fibrotic diseases provides a significant parallel opportunity



Leads to a clinical development roadmap with multiple “shots on goal”

FAK



Inhibition

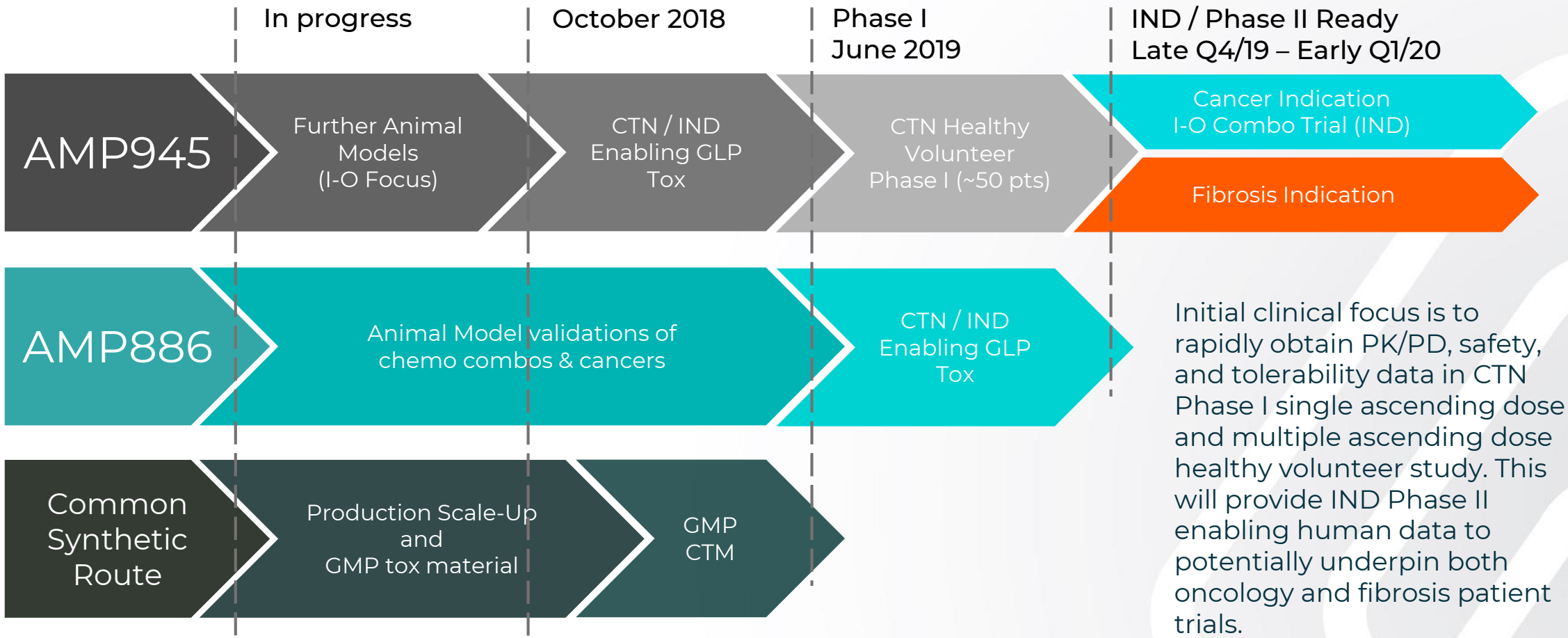
AMP886 + Chemo (mTOR/BRAF, etc) → Refractory/Progressive Cancer (Ovarian Cancer)

AMP945 + Checkpoint Inhibitors → Fibrotic Cancers (Pancreatic Cancer)

AMP945 → Fibrosis (Idiopathic Pulmonary Fibrosis - IPF)

Amplia THERAPEUTICS

High-level Development Plan (18 months)



Initial clinical focus is to rapidly obtain PK/PD, safety, and tolerability data in CTN Phase I single ascending dose and multiple ascending dose healthy volunteer study. This will provide IND Phase II enabling human data to potentially underpin both oncology and fibrosis patient trials.

Core IP Granted or 'National Phase'

Patent / Application number			Filing Date		Status	
Selective FAK Inhibitors [AMP945]	FAK Inhibitors [AMP886]	Territory	AMP945	AMP886	AMP945	AMP886
US 61/443,773	US 61/443,773	USA	17th Feb, 2011	17th Feb, 2011	Priority Filing	Priority Filing
US 61/523,489	US 61/523,503	USA	15th Aug, 2011	15th Aug, 2011	Supplemental	Supplemental
US 61/579,729	US 61/579,719	USA	23rd Dec, 2011	23rd Dec, 2011	Supplemental	Supplemental
PCT/GB2012/000176	PCT/GB2012/000175	UK	17th Feb, 2012	17th Feb, 2012	PCT filing	PCT filing
US 9120761	US 9012461	USA	6th July, 2012	6th July, 2012	Granted	Granted
EP 12705384.1	EP12705383.3	Europe	Sept, 2013	Sept, 2013	National phase filing	Granted
US 9174946	US 9421205	USA	15th Aug, 2013	11th Feb, 2015	Granted	Granted
AU 2012216894	AU 2012216893	Australia	12th Aug, 2013	13th Aug, 2013	Granted	Granted
CA 2827172	CA2827171	Canada	12th Aug, 2013	12th Aug, 2013	National phase filing	National phase filing
[JP] 5937112	[JP] 5937111	Japan	19th Aug, 2013	Aug, 2013	Granted	Granted
IN 1744/MUMNP/2013	IN 1743/MUMNP/2013	India	16th Sept, 2013	16th Sept, 2013	National phase filing	National phase filing
ZL201280018816.6	ZL201280018969	China	16th Oct, 2013	17th Oct, 2013	Granted	Granted

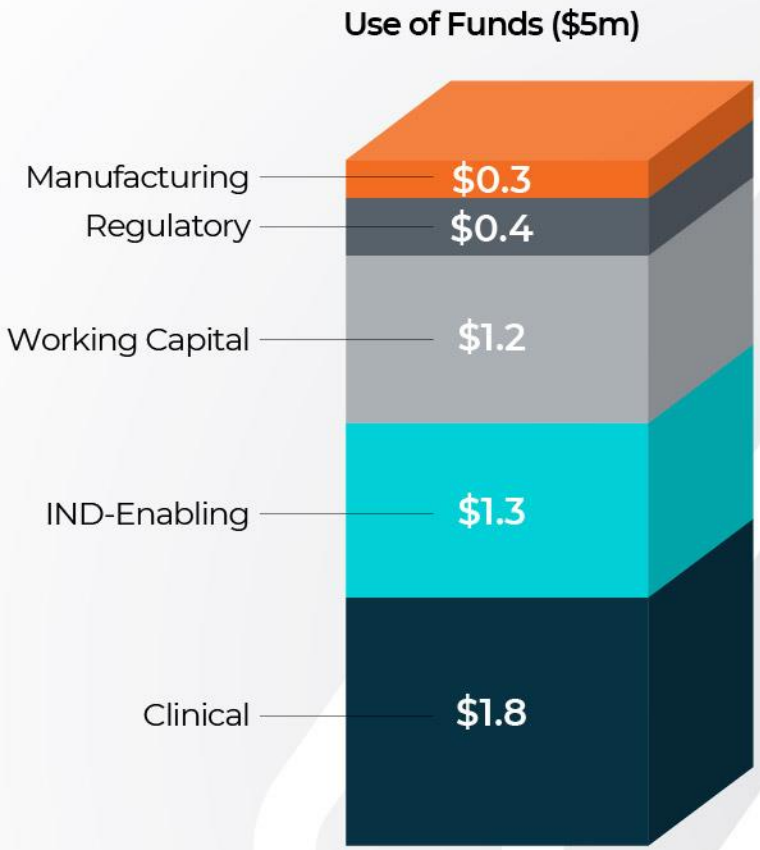
An Evolving Asset Class: Competitive Landscape

Agent	Company	Status	Notes
VS-4718 (PND-1186)	Verastem	PI	First generation candidate
VS-6063 (PF-04554878)	Verastem	PI/PII	Combo with Pfizer/Merck
GSK-2256098	GSK	PI	Paused
CT-707	Centaurus Pharma	PI-Not yet recruiting	Questions about selectivity
BI-853520	Boehringer-Ingelheim	PI	Two PI trials completed
ASN-006	Asana	Discovery	Early stage

- Established target but still relatively little commercial congestion around novel FAKi compounds
- Increased interest in the use of FAK inhibitors in the immuno-oncology setting as a combination therapy (*Verastem-Pfizer partnership*)
- Verastem (NASDAQ: VSTM, Market Cap USD $600m) is our nearest comparator
- Our differentiation is:
    - AMP945 is extremely selective / “clean” for FAK inhibition compared with competitive products – important in immuno-oncology combo applications to maximise safety
    - AMP866 has useful multi-target properties for chemotherapy combination use

Capital Requirements and Use of Funds

- Current balance sheet sufficient to complete remaining Phase I enabling preclinical activities
- Running 'virtually' to minimise cash burn during preclinical and Phase 1 development with team build-up occurring pre Phase II
- Projected ~AUD $5m requirement to complete initial human studies and be Phase II ready (Phase II planning assumes US IND)
- Completion of a capital raise targeted by end Q3/2018
- In parallel, several business development discussions with global pharmaceutical companies
    - Potential collaborations (pre-clinical and clinical)
    - Cancer and Fibrosis

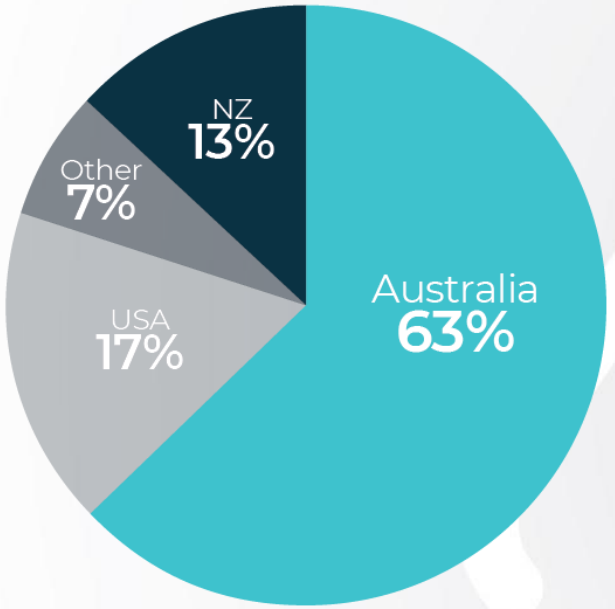


Corporate Snapshot (ASX:ATX)

Key Statistics as at 04 September 2018	
Share price	**A$0.34 [post share consolidation]**
Shares on issue	**41,023,303**
Market Cap	**A$14 million**
Options	**4,240,000 (A$0.60 to US$4.00)**
Cash	**A$2.0 million (Appendix 4C 30/06/18)**

Significant Holders	
Citicorp Nominees Pty Ltd	**15.4%**
CTxT Pty Ltd	**11%**
Elk River Holdings (Chris Behrenbruch)	**6.1%**
34th Avenue Pty Ltd (Mark Devlin)	**5.4%**
Christopher Burns	**5.4%**
Warwick Tong and Mark Sullivan (each)	**4%**

Register	
Top 20	**68%**
Total shareholders	**3,096**
Voluntary Restricted Shares (May 2020)	**18,460,308**



Amplia THERAPEUTICS

For further information please contact

Simon Wilkinson

CEO

simon@ampliatx.com

+64 21 661 850

www.ampliatx.com

Stock ASX:ATX

Amplia THERAPEUTICS