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**AUSTRALIS**  
PHARMACEUTICALS

# Corporate Presentation

January, 2026

- Australis Pharmaceuticals (Australis) was founded to develop naturally derived compounds into **cancer therapeutics**
- The original compounds were discovered in honey bee propolis on Kangaroo Island in South Australia and characterized as **microtubule-destabilizing agents (MTAs)**
- **AUS\_001, oncology lead compound**, has demonstrated encouraging in-vitro and in-vivo data in efficacy and toxicity studies across multiple tumor lines
- Given the broad anti-cancer activity against solid tumors demonstrated by AUS\_001 and the ability to cross the **blood brain barrier**, the Phase 1 study will have a broad inclusion criteria for tumor types
- **Orphan Drug Designation** issued for AUS\_001 for **malignant gliomas** in February, 2025 and **Rare Pediatric Disease Designation** issued for AUS\_001 for **pediatric high grade gliomas** in March, 2025
- Pre-IND meeting completed with FDA: confirmed our pre-clinical approach
- Team in place has strong research and clinical development expertise

	Pancreatic Cancer <sup>1</sup>	Malignant Glioma <sup>2</sup>	Pediatric-type Glioma <sup>3</sup>
US Incidence	66,440	22,654	~8,800
Estimated Survival	12.8% (5 year survival)	~10% (5 year survival)	Most patients do not survive more than 1 – 2 years

**No therapies currently in clinical trials identified as having viable potential to address these great unmet needs for patients**

\*: these represent just a few of the potential tumor types that AUS\_001 can target  
<sup>1</sup> National Cancer Institute SEER database; <sup>2</sup> Mesfin, et al. 2024; Ostrom et al., 2022 <sup>3</sup> Gaijjar et al., 2022

## Efficacy

- **Pre-clinical efficacy:** Demonstrated across a range of tumor types, including **glioma and other tumor types representing unmet needs for patients**
- **Crosses the blood brain barrier**

## Safety

- **Safety margin:** ~20x more of AUS\_001 needed to inhibit growth of healthy non-neoplastic counterpart cells
- **Peripheral neuropathy:** Reversible neurotoxic effect
- **Drug-Drug interactions:** Poor inhibitor of CYP enzymes, common pathway for drug metabolism

## Patient Focus

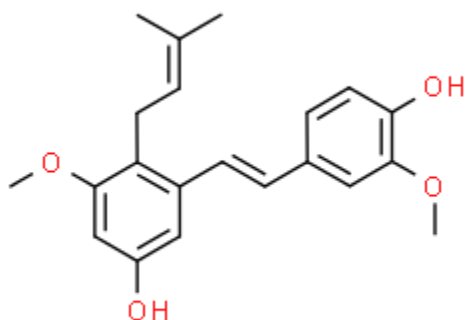
- **Developed an oral formulation\***
- **Potential balance of efficacy and safety,** based on pre-clinical therapeutic index

\*: Final decision still pending on encapsulation vs maintaining a liquid form for the clinical trial

*AUS\_001 extracted from the sedge plant  
(source of Kangaroo Island propolis)*

### **AUS\_001 Structure** (E-stilbene)

MW=340.42 g/mol



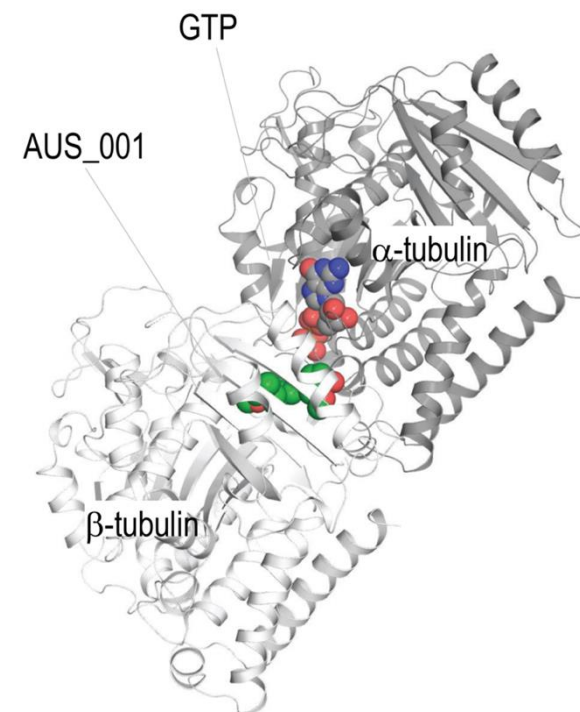
### **Common name:**

(3-[(E)-2-(4-Hydroxy-3-methoxyphenyl)vinyl]-5-methoxy-4-(3-methyl-2-buten-1-yl)phenol

### MECHANISM OF ACTION (MOA)\*:

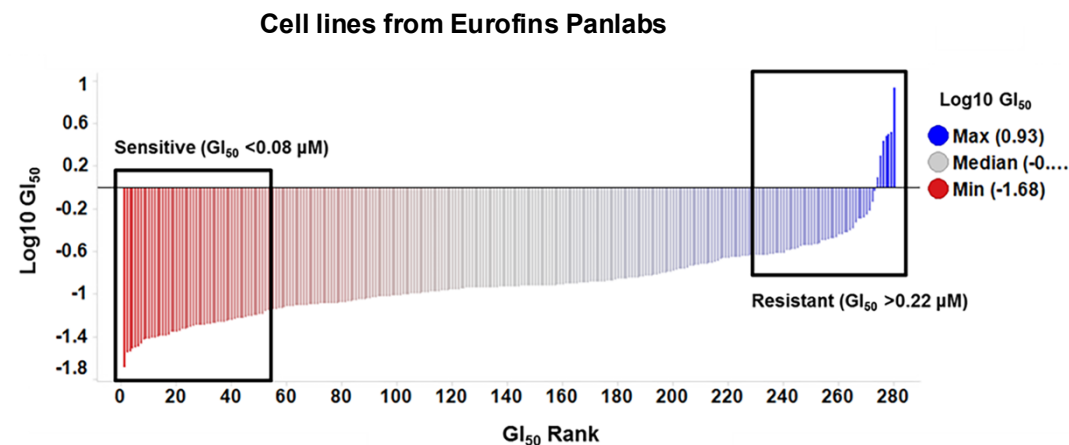
AUS\_001 is characterized as a microtubule-destabilizing agent and has demonstrated cell cycle inhibition and induction of programmed cell death. Reversible mode of target engagement and increased uptake by cancer vs. normal cells

### X-ray Crystal Structure of the tubulin-AUS\_001 complex



## In Vitro Data

- High potency against **24 types of cancer\***, including **glioblastoma** and other tumor types representing unmet needs for patients
- Encouraging safety margin: **~20x** more of AUS\_001 needed to inhibit growth of healthy non-neoplastic counterpart cells
- Reduced concern for **peripheral neuropathy**: Drug treated midbrain and cortical neurons showed reversible neurotoxic effect for AUS\_001 but Paclitaxel-treated neurons suffered sustained neurotoxicity even after discontinuation of treatment. This is a key finding especially for the pediatric population
- Less susceptibility to **Drug Resistance-related mechanisms**

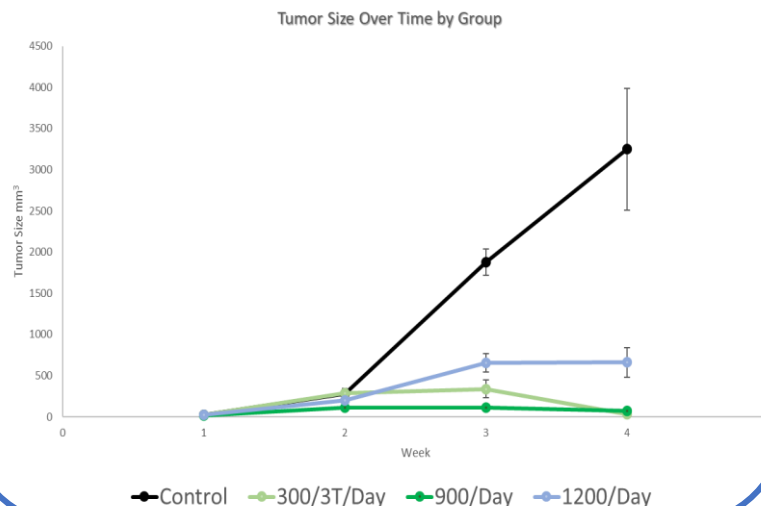


*The proliferation response of 280 cancer cell lines to AUS\_001 treatments as assessed by high-content fluorescence imaging (Eurofins Panlabs): All cell lines with cell count GI<sub>50</sub> < 0.08 μM were classified as sensitive to AUS\_001, while those with GI<sub>50</sub> > 0.22 μM were classified as resistant.*

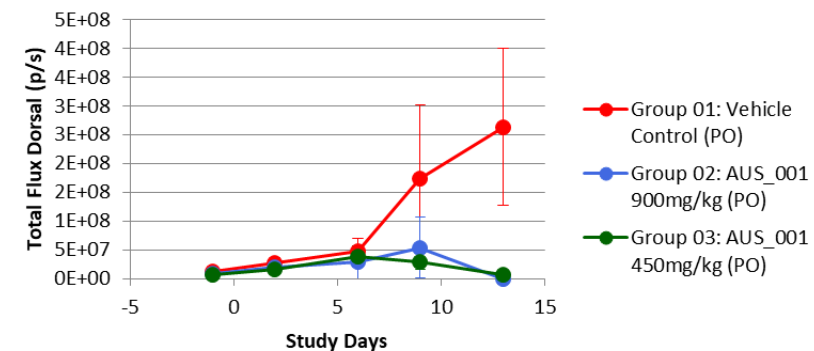
## In Vivo Data

- Efficacy established in **7 different in vivo cancer models\***
- Crossing of **blood-brain barrier\*\***
- Pharmacokinetics/Pharmacodynamics: **Accumulation** in tumors, organs and brain tissues
- **Lack of myelosuppression** or other overt toxicities in immunocompetent mouse study (21 days, P.O.)
- **Non-emetic** response in ferrets

B16F10 melanoma xenograft immunocompetent mice



GL261 Red-Fluc (i.c.) GBM model



\*: Figures to the right depict a few in vivo models; Additional data available upon request

\*\* : Assessed using the 3D Human Blood Brain Barrier Model

## Safety AUS\_001 Demonstrated an Encouraging Safety Profile in Predictive Toxicology Screen

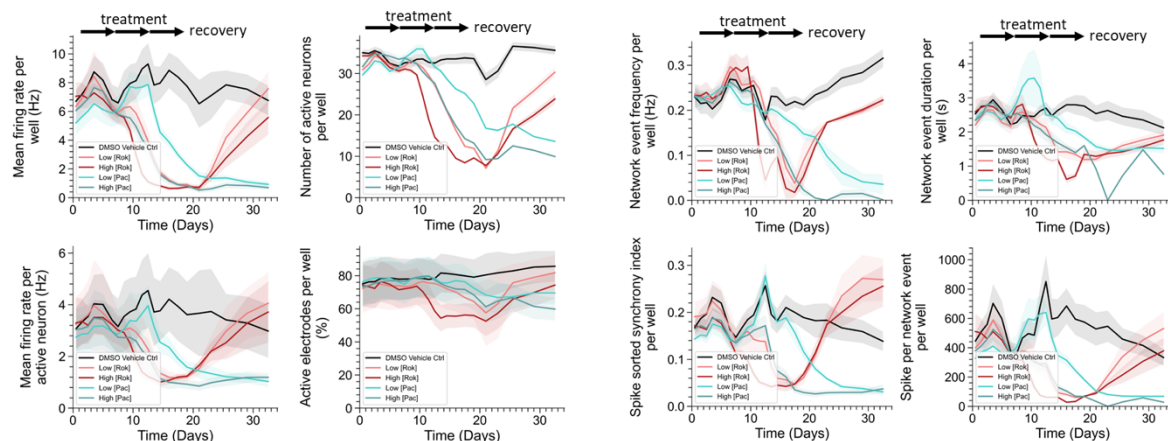
Study	Results
AMES Test	No mutagenic potential up to 100 µM doses of AUS_001
Cardiotoxicity	Low hERG-blocking liability with a half-maximal inhibitory concentration of 65 µM
<b>Hepatotoxicity</b>	<b>Poor inhibitor of CYP enzymes</b> , except for CYP1A
Vascular Toxicity	AUS_001 affects activated Human Umbilical Vein Endothelial Cells (HUVECs) at lower doses relative to those required for cytotoxicity induction of quiescent endothelial cells
P-glycoprotein (P-gp) model	P-gp overexpressing cellular models do not confer resistance to AUS_001
βIII-tubulin model	AUS_001 significantly retains its ability to sensitize βIII-tubulin overexpressing cells
<b>Neurotoxicity</b>	Drug treated midbrain and cortical neurons showed <b>reversible neurotoxic effect for AUS_001</b> , while Paclitaxel-treated neurons suffered sustained neurotoxicity after discontinuation of treatment



# AUS\_001 exerts reduced concern for neurotoxicity

## Midbrain Neurons Exposed to Paclitaxel and AUS\_001

Experiment started at Day 192 of Neuronal Maturation in plate NE245  
7-8 wells per condition



Drug concentrations increased roughly every 5 days

Low AUS\_001: 0.1uM → 2uM → 8uM    Low Pac: 0.1uM → 2uM → 8uM  
High AUS\_001: 1uM → 4uM → 16uM    High Pac: 1uM → 4uM → 16uM

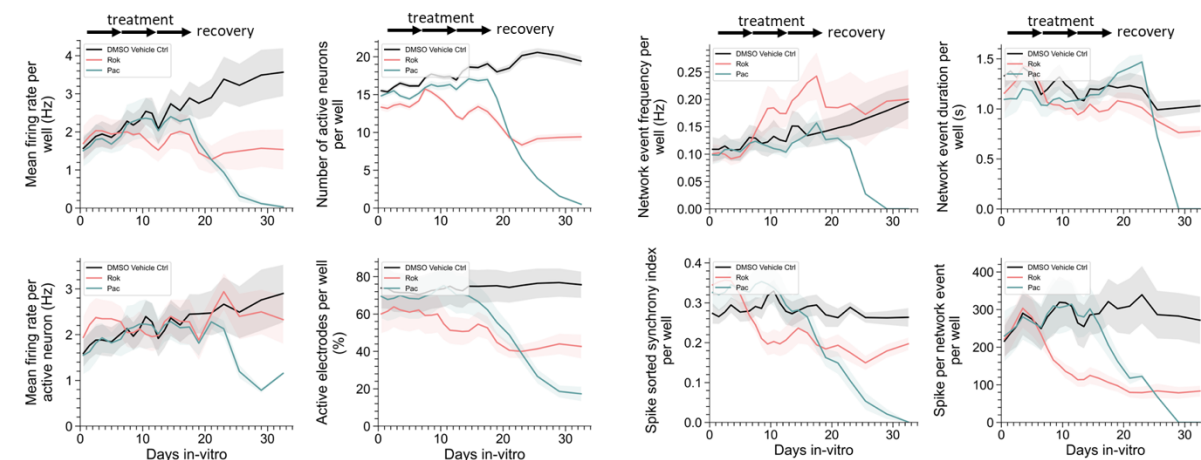


- ✓ Drug treated midbrain and cortical neurons showed reversible neurotoxic effect for AUS\_001 but paclitaxel-treated neurons suffered sustained neurotoxicity even after discontinuation of treatment

**Methods:** The cells were maintained in 16 electrode per-well 48-well MEA plates and were fed by half-media change three times per week. Culture media and method for midbrain and cortical neurons is as defined previously in Milky et al 2022. Paclitaxel and AUS\_001 treatment media was prepared fresh for every feed using individual frozen drug aliquots to avoid repeated freeze-thaw cycles. The DMSO vehicle control media was matched to the highest concentration of DMSO in the drug media (the High Paclitaxel condition). When drug treatment was initiated, the midbrain neurons had been maintained in culture for 192 days and the cortical for 137 days. Drug treatments increased in concentration roughly every 5-6 days depending on the feeding schedule. A full media change was performed to wash out the drugs to begin the recovery period. Recordings were taken daily during drug exposure, and at least twice a week during recovery, by a Maestro pro MEA system (Axion Biosystems). The MEA maintained a 37°C and 5% CO2 environment for the recordings. MEA Recordings were single-cell spike sorted using Plexon Offline Sorter version 4.5 (Plexon Inc) to isolate individual neurons from the electrodes and analyzed with Neural Metric Tool (Axion Biosystems).

## Cortical Neurons Exposed to Paclitaxel and AUS\_001

Experiment started at Day 137 of Neuronal Maturation in plate NE258  
5-6 Wells per Condition

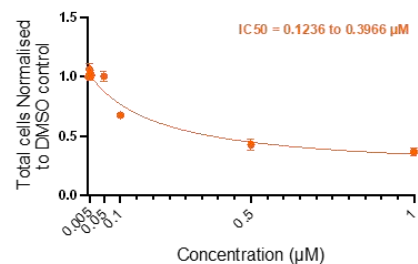
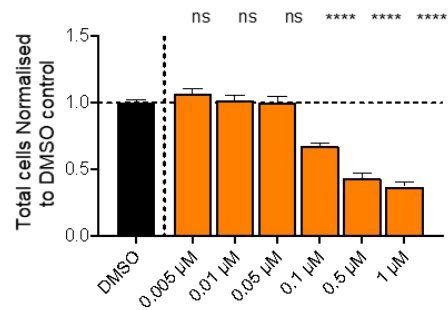


Drug concentrations increased roughly every 5 days

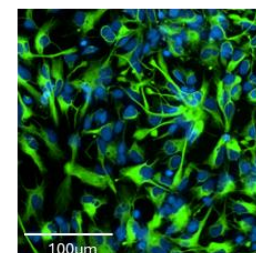
AUS\_001: 0.1uM → 1uM → 2uM    Pac: 1uM → 2uM → 4uM

# Human Neural Midbrain Progenitors Tolerate Higher Concentrations of AUS\_001 Relative to Other Microtubule Targeting Agents

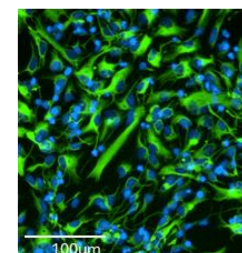
Aus001



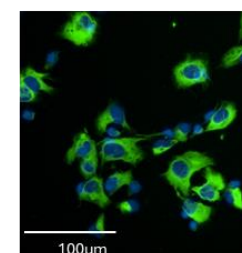
DMSO control



0.1 μM

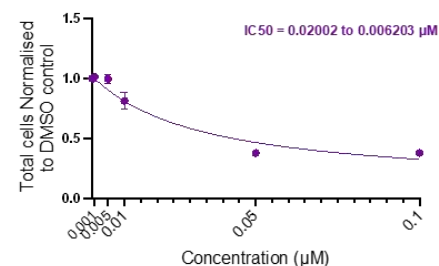
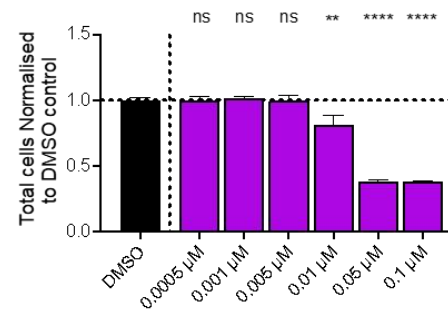


1 μM

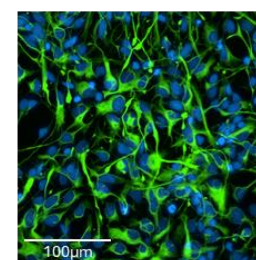


Nestin/DAPI

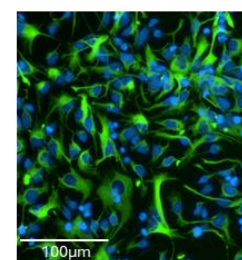
Avanbulin



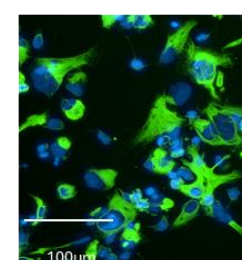
DMSO control



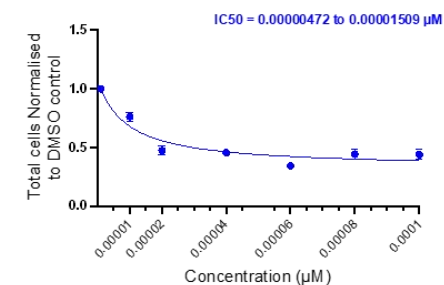
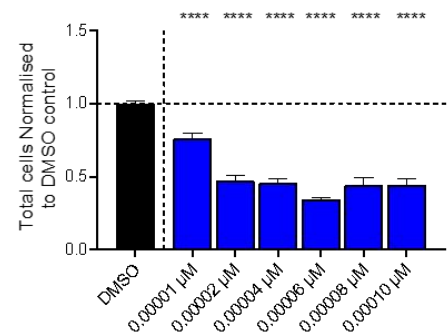
0.01 μM



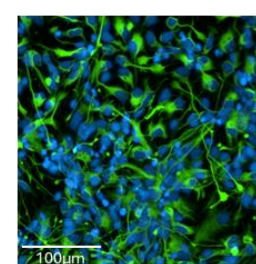
0.1 μM



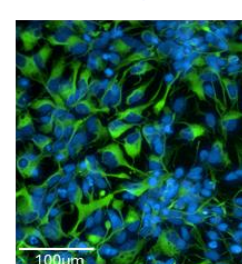
Vincristine



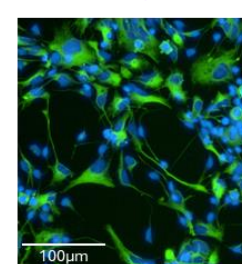
DMSO control



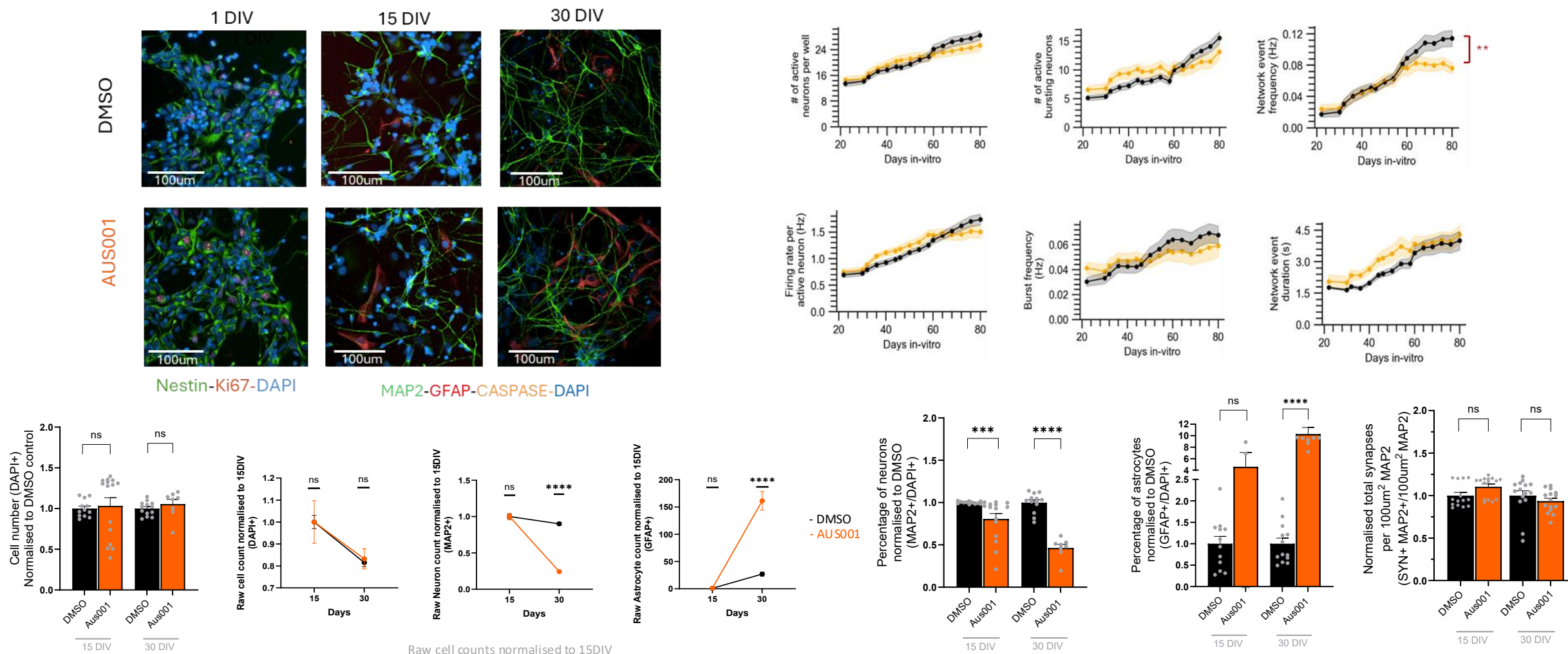
0.00001 μM



0.00002 μM



# Recovery of AUS\_001-treated Midbrain Neural Progenitor Cells Points to Maintenance of Functional Integrity - Potential Clinical Relevance for Patients with Pediatric Gliomas

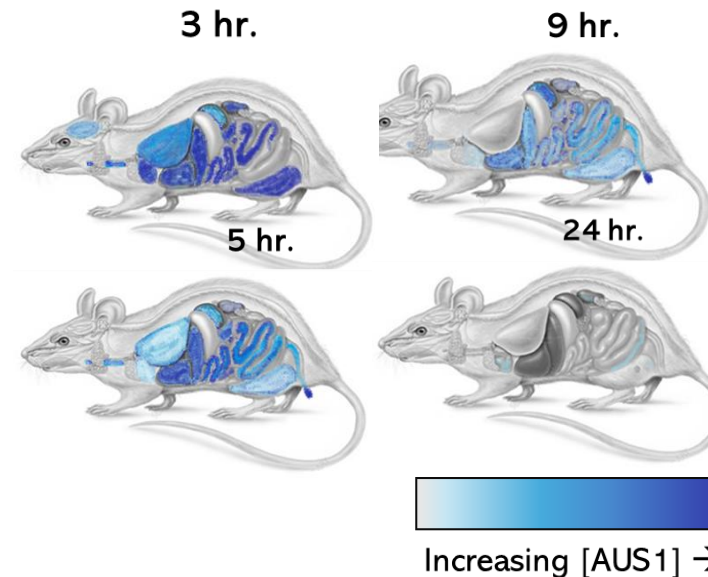
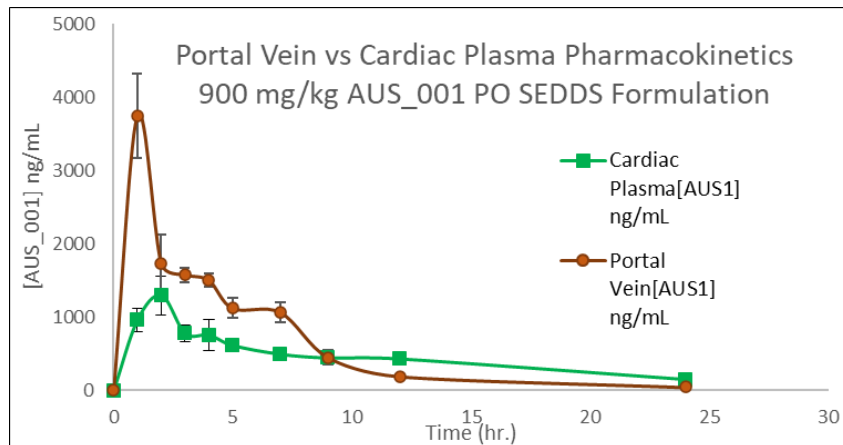




# Characterized the PK/PD Profile for the Oral Administration of SEDDS-formulated AUS\_001

## PK/PD Data for the AUS\_001 Self-Emulsifying Drug Delivery System (SEDDS) Formulation Development and Efficacy

- Solubility up to 400 mg/mL
- T<sub>max</sub>: 3 hrs
- T<sub>1/2</sub> elimination: **8.29 hr**
- 72% escapes first pass-metabolism
- Bioavailability: 15-20%
- Excipients inhibit UGT enzymes

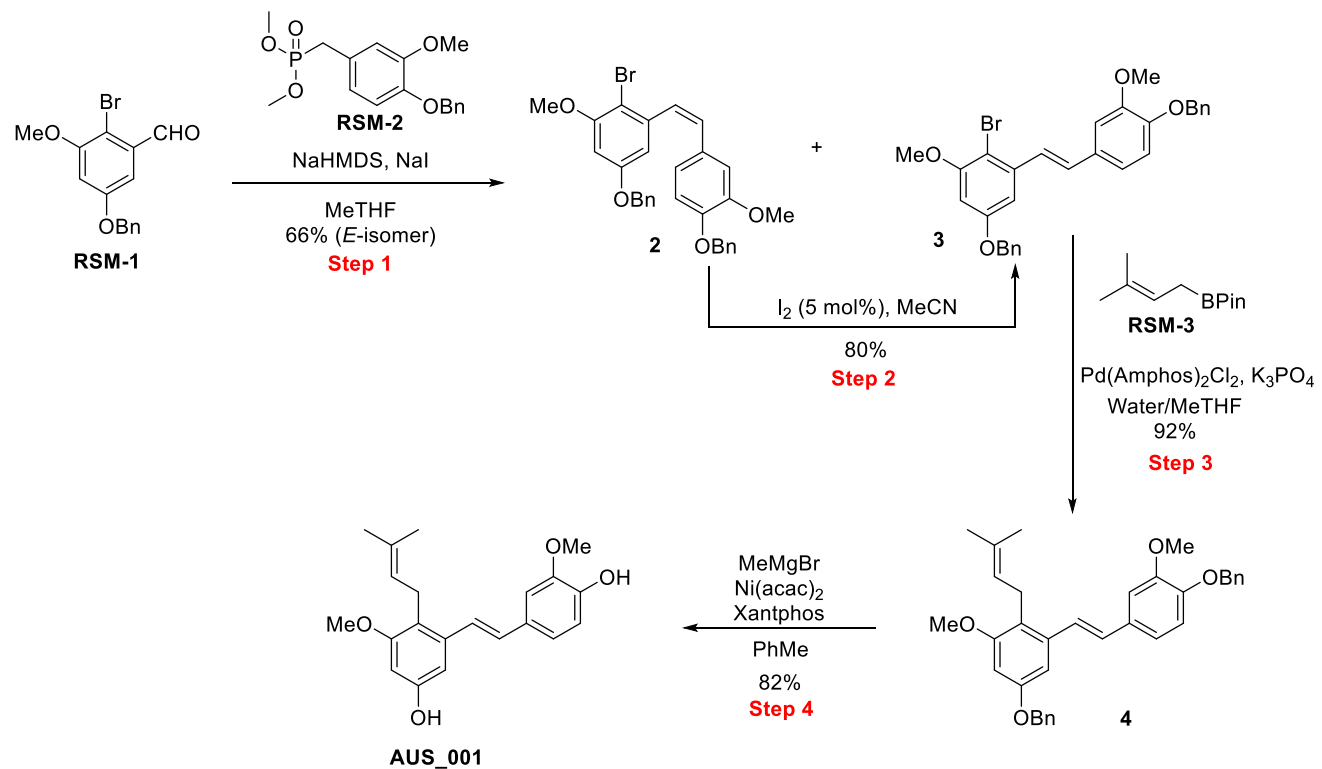


## AUS\_001 Demonstrated an Encouraging Safety Profile in *In Vivo* Studies

- Upon single oral administration, there were **no deaths, and no overt toxicity** observed for the SEDDS formulated AUS\_001 **at the 2,000 mg/kg dosage** in Balb/c mice
- Of note, the **vehicle does not exhibit any toxicity** on its own at **>10,000 mg/kg** (highest dose tested)
- A **repeat dose range-finding (DRF) study in mice** was conducted with a 14 daily repeat dosing of the SEDDS formulated AUS\_001 at 100, 500, or 1,500 mg/kg. Generally, **oral doses up to 1,000 mg/kg were well-tolerated in mice**, with no myelotoxicity toxicity and no toxicity detected in all tissues analyzed except the cecum. Soft stools were noted 1-5 hours post dose.
- The **non-GLP DRF 7-day toxicology study in rats and the 28-day GLP toxicology study** indicates that **oral doses up to 1,000 mg/kg/day were well tolerated**. At the highest dose tested (1,000mg/kg), gas in caecum and intestine, as well as soft stools were noted.

## Synthesis and Manufacturing

- 4 step scalable GMP manufacturing synthesis of drug substance established.



Synthesis Process

# AUS\_001 Data Accepted for Presentation at Multiple Peer Reviewed Meetings

AACR 2024

AACR 2024

ASCO 2024

SITC 2024

ASCO GI 2025

## The novel microtubule-destabilizing compound AUS\_001 maintains unique binding to the colchicine site of tubulin and elicits reversible cellular effects relative to other anti-tubulin agents

Abstract No: 7141 Herman Lelie<sup>1</sup>, Yao-Chieh Chou<sup>2</sup>, Alastair J. King<sup>2</sup>, Zlata Bojarska<sup>3</sup>, Andrea E. Prota<sup>4</sup>, Michel O. Steinmetz<sup>2</sup>, Marina Koutsoumpa<sup>1</sup>

<sup>1</sup>Australis Pharmaceuticals, Inc., Simi Valley, CA, USA, <sup>2</sup>Pharmacology Discovery Services Taiwan, Ltd

Our previous work showed that the proliferative responses, as assessed by microtubule destabilization activity

### 1. AUS\_001

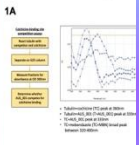


Figure 1. (A) Direct absorbance reaction curves showing the effect of AUS\_001 on tubulin polymerization. The graph shows a decrease in absorbance over time for AUS\_001 compared to control, indicating microtubule destabilization.

### 3. AUS\_001 is a

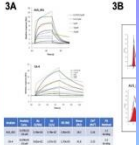


Figure 3. (A) Surface Plasmon Resonance (SPR) sensorgrams showing the binding of AUS\_001 to tubulin. The graph shows a concentration-dependent increase in SPR response, indicating binding to the colchicine site.

### 2. Orally administered AUS\_001 is well-tolerated in

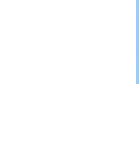


Figure 2. (A) BALB/c mice treated with AUS\_001. The graph shows tumor growth delay in mice treated with AUS\_001 compared to control, indicating antitumor activity.

## A novel microtubule disruptor exerts broad anticancer efficacy with a tolerable safety profile

Herman Lelie<sup>1</sup>, Inger Brandsma<sup>2</sup>, Giel Hendriks<sup>2</sup>, Lee R. Cavedine<sup>3</sup>, Brogan A. Ekins<sup>3</sup>, Steven M. Garner<sup>3</sup>, Andrew J. Cook<sup>3</sup>, Muthukrishnan Renganathan<sup>3</sup>

<sup>1</sup>Australis Pharmaceuticals, Inc., Simi Valley, CA, USA, <sup>2</sup>Department of Pharmacology, University of Texas Health Science Center at San Antonio, San Antonio, TX, USA, <sup>3</sup>College of Medicine and Public Health, Flinders University, South Australian Health and Medical Research Institute, Adelaide, SA, Australia, <sup>4</sup>Adelaide Medical School

Abstract No: 4701

<sup>1</sup>Australis Pharmaceuticals, Inc., Simi Valley, CA, USA, <sup>2</sup>Department of Pharmacology, University of Texas Health Science Center at San Antonio, San Antonio, TX, USA, <sup>3</sup>College of Medicine and Public Health, Flinders University, South Australian Health and Medical Research Institute, Adelaide, SA, Australia, <sup>4</sup>Adelaide Medical School

Initial evaluation of the prenylated hydroxy-stilbene isolated from bee propolis, in a cell-based profiling screen identified that AUS\_001 exerts a high degree of potency across 30 cancer types with site of tubulin and elicited the reversible nature of target engagement.

Microtubules are a well-established target for cancer treatment and the ability to also retain efficacy in a subset of 15 established glioma cell lines with

### 1. AUS\_001 exerts broad *in vitro* anti-cancer activity

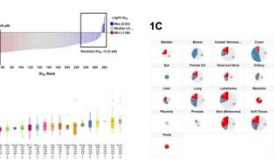


Figure 1. The proliferation response of cancer cell lines to AUS\_001 was assessed by high-content fluorescence imaging (Eurofins Panlabs). (A) Lines with cell count GI50 <0.08 uM were classified as sensitive to AUS\_001, while those with GI50 >0.22 uM were classified as resistant. (B) Dose-response curves for 50% of maximal inhibition of cell growth (GI50) from cell lines of the 19 most sensitive cancer types (site of primary tumor breakdown of cell lines classified as sensitive, resistant, or intermediate each cancer type represented in the OncoPanel™ Profiling Analysis. PI are relative to the total number of cell lines for each tissue/tumor type.

### 2. Orally administered AUS\_001 is well-tolerated in

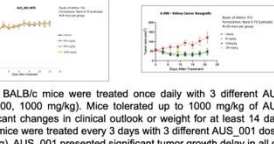


Figure 2. (A) BALB/c mice treated with AUS\_001. The graph shows tumor growth delay in mice treated with AUS\_001 compared to control, indicating antitumor activity.

## The microtubule-destabilizing agent AUS\_001 is an attractive candidate for glioblastoma therapy

Abstract No: 3114 Marina Koutsoumpa<sup>1</sup>, Aaron L. Carlson<sup>2</sup>, Teresa M. DesRochers<sup>2</sup>, Peter Y.W. Chan<sup>3</sup>, April L. Risinger<sup>3</sup>, Robert Adams<sup>4</sup>, Cedric Bardy<sup>4</sup>, Daniel Thomas<sup>5</sup>, Herman Lelie<sup>1</sup>

<sup>1</sup>Australis Pharmaceuticals, Inc., Simi Valley, CA, USA, <sup>2</sup>Kiyatec, Inc., Greenville, SC, USA, <sup>3</sup>Department of Pharmacology, University of Texas Health Science Center at San Antonio, San Antonio, TX, USA, <sup>4</sup>College of Medicine and Public Health, Flinders University, South Australian Health and Medical Research Institute, Adelaide, SA, Australia, <sup>5</sup>Adelaide Medical School

Microtubules are a well-established target for cancer treatment and the ability to also retain efficacy in a subset of 15 established glioma cell lines with

### 1. AUS\_001 crosses the Blood Brain Barrier

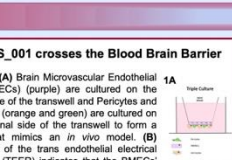


Figure 1. (A) Brain Microvascular Endothelial Cells (BMECs) (purple) are cultured on the luminal side of the transwell and Pericytes and Astrocytes (orange and green) are cultured on the abdominal side of the transwell to form a barrier that mimics an in vivo model. (B) Evaluation of the trans endothelial electrical resistance (TEER) indicates that the BMECs co-culture possesses reasonable barrier tightness (TEER >15040 Ohm/cm²) on Day 5 upon system activation. BMECs treated with 1uM AUS\_001 for 6h exhibited strikingly decreased TEER across 2 independent biological replicates. Drug passage through the membrane was confirmed using LC-MS and the apparent permeability coefficient (Papp) was calculated based on the permeation rate and compound concentration.

### 4. $\beta$ III-tubulin overexpression confers limited resistance to AUS\_001

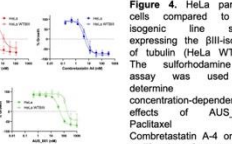


Figure 4. HeLa pan cells compared to isogenic line of expressing the  $\beta$ III-iso of tubulin (HeLa WT). The graph shows that the  $\beta$ III-iso of tubulin confers limited resistance to AUS\_001, as indicated by the similar growth curves.

## The microtubule-destabilizing agent AUS\_001 acts as an immunogenic cell death inhibitor

Abstract No: 976 Marina Koutsoumpa<sup>1</sup>, Herman Lelie<sup>1</sup>

<sup>1</sup>Australis Pharmaceuticals, Inc., Simi Valley, CA, USA, <sup>2</sup>Pharmacology Discovery Services Taiwan, Ltd

Microtubule-targeting agents (MTAs) are a class of cancer drugs that can affect the body's immune responses. Previous studies support that I) upon integrity, generating microtubular DNA which can activate the cGAS-Stimulator of Interferon Genes (STING) pathway known to stimulate macrophage leading to cGAS-STING-dependent interferon-stimulated gene induction (Fermat et al., 2021) ii) guanine nucleotide exchange factor-H1 (GEF-H1) is cells (DCs) activation and enhancement of cross-presentation of tumor antigens to CD8-T cells (Kashyap et al., 2019) iii) several MTAs have the ability to Emerging evidence shows that initiation of specific cell death modalities such as ICD, increase the immunogenicity of tumors. An assorted set of therapeutic molecular patterns that produce neantigens and stimulate adaptive immunity. We recently discovered a novel microtubule disruptor, stilbene isolated from bee propolis, in a cell-based profiling screen identified that AUS\_001 exerts a high degree of potency across 30 cancer types with site of tubulin and elicited the reversible nature of target engagement.

The goal of the current study was to explore the potential of AUS\_001, an effective microtubule-destabilizing antineoplastic agent, to also maintain control

Background

Microtubule-targeting agents (MTAs) are a class of cancer drugs that can affect the body's immune responses. Previous studies support that I) upon integrity, generating microtubular DNA which can activate the cGAS-Stimulator of Interferon Genes (STING) pathway known to stimulate macrophage leading to cGAS-STING-dependent interferon-stimulated gene induction (Fermat et al., 2021) ii) guanine nucleotide exchange factor-H1 (GEF-H1) is cells (DCs) activation and enhancement of cross-presentation of tumor antigens to CD8-T cells (Kashyap et al., 2019) iii) several MTAs have the ability to Emerging evidence shows that initiation of specific cell death modalities such as ICD, increase the immunogenicity of tumors. An assorted set of therapeutic molecular patterns that produce neantigens and stimulate adaptive immunity. We recently discovered a novel microtubule disruptor, stilbene isolated from bee propolis, in a cell-based profiling screen identified that AUS\_001 exerts a high degree of potency across 30 cancer types with site of tubulin and elicited the reversible nature of target engagement.

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Microtubule-targeting agents (MTAs) are a class of cancer drugs that can affect the body's immune responses. Previous studies support that I) upon integrity, generating microtubular DNA which can activate the cGAS-Stimulator of Interferon Genes (STING) pathway known to stimulate macrophage leading to cGAS-STING-dependent interferon-stimulated gene induction (Fermat et al., 2021) ii) guanine nucleotide exchange factor-H1 (GEF-H1) is cells (DCs) activation and enhancement of cross-presentation of tumor antigens to CD8-T cells (Kashyap et al., 2019) iii) several MTAs have the ability to Emerging evidence shows that initiation of specific cell death modalities such as ICD, increase the immunogenicity of tumors. An assorted set of therapeutic molecular patterns that produce neantigens and stimulate adaptive immunity. We recently discovered a novel microtubule disruptor, stilbene isolated from bee propolis, in a cell-based profiling screen identified that AUS\_001 exerts a high degree of potency across 30 cancer types with site of tubulin and elicited the reversible nature of target engagement.

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## Implication of the novel microtubule targeting agent, AUS\_001, in pancreatic cancer cellular signaling

Abstract No: 748 Marina Koutsoumpa<sup>1</sup>, Herman Lelie<sup>1</sup>, Kun-Yuan Lin<sup>2</sup>, Pony Yu-Ling Lee<sup>2</sup>

<sup>1</sup>Australis Pharmaceuticals, Inc., Simi Valley, CA, USA, <sup>2</sup>Pharmacology Discovery Services Taiwan, Ltd

Pancreatic cancer (PanCa) is considered a major therapeutic challenge due to its poor prognosis, high mortality rate and resistance to most therapies, underlining the need for new treatment approaches. Our previous investigations revealed that the prenylated hydroxy-stilbene, AUS\_001, inhibits  $\beta$ -tubulin polymerization via its unique binding in the trans configuration to the colchicine site of tubulin. A cell-based profiling platform (OncoPanel™ Profiling Analysis, by Eurofins Panlabs) identified sensitivity to AUS\_001 in 12 out of 13 PanCa lines included with a median concentration causing 50% growth inhibition of 0.227uM.

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Aim

The goal of the current study was to elucidate the signaling activity and cellular responses following treatment with AUS\_001 and explore the prospect of the latter in PanCa treatment.

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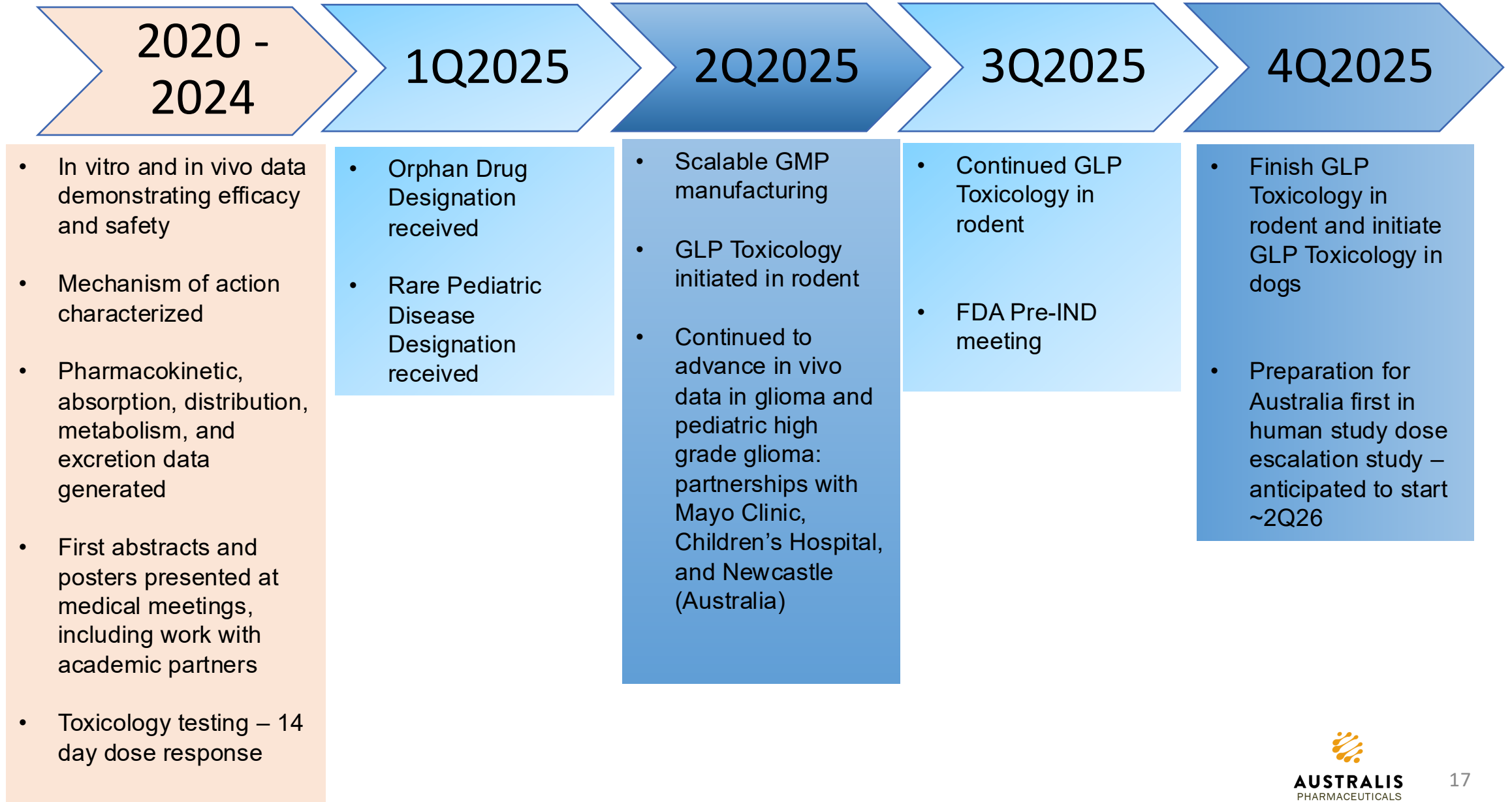
Background

**Conclusions**  
Our findings provide critical insights into the molecular events triggered by AUS\_001 in PanCa cells and serve as a valuable resource for future exploration of AUS\_001 as a novel PanCa therapeutic agent

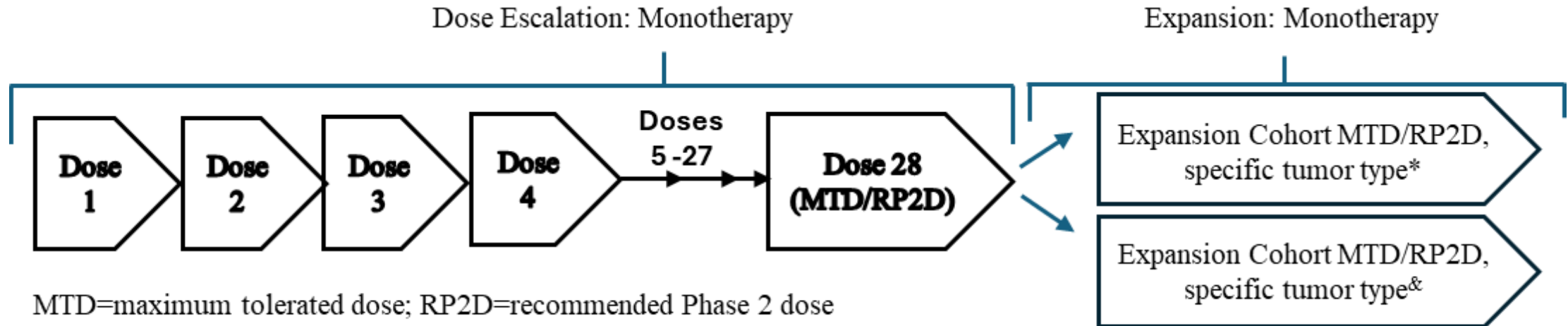
Global Patents/Exclusivity	Expiry
2012: Use and synthesis of AUS_001 and one pipeline asset	2032
2020: Covers 4 pipeline assets, new synthesis route and 5000+ potential new chemical entities	2040
Orphan Drug and Rare Pediatric Designations	~ 7 year extension following approval



## Australis Key Achievements To Date



## *A Multi-Institutional Study of AUS\_001, as Monotherapy in Patients With Solid Tumors*



\*Adults with metastatic and/or locally advanced solid tumors

&Pediatrics aged 5-17 years with pediatric-type diffuse high-grade glioma

- The Dose Escalation Phase will be a 3+3 design and is expected to enroll patients at approximately 2-4 sites in Australia
- The Expansion Phase would also be planned to include US sites
- Proposed clinical paths for glioma will focus on pediatric high grade glioma and adult glioma with an initial focus on relapsed patients, including Temozolomide resistant, then moving to first line

**Successfully raised ~\$30M over the last 10+ years**

**No debt on the balance sheet**

**Enough cash on hand to proceed through Phase 1**

**Currently seeking to enhance our capital with ~\$15 Million  
raise in 2026 for clinical trials beyond Phase 1**

## Australis Pharmaceuticals Executive Team

Team Member	Role	Expertise	Past Experiences
Michele Korfin, RPh, MBA	CEO	~30 years as a Biotech leader focused on clinical development, regulatory, and commercialization	Merck, Celgene, Kite, TYME, Gamida Cell
Caroline Carr, CPA	CFO	Finance leader in Biotechs and Pharma companies with expertise in investor relations	Mycovia, Dara, Pfizer, Deloitte
Marina Koutsioumpa, PhD	VP, Cancer Biology	PhD-trained cancer biologist specializing in molecular pharmacology and preclinical development ( <i>in vitro</i> and <i>in vivo</i> models)	UCLA Center for Systems Biomedicine
Herman Lelie, PhD	VP, Research and Development	PhD with expertise in analytical chemistry and pre-clinical development	MIT, UCLA, Bruin Biometrics, Constitution Labs
John Heyburn, MBA	Clinical Operations	Expertise in hematology/oncology clinical trials	Morphotek, Advaxis, Tmunity, Gennao Bio
Todd Robinson	Founder	Entrepreneur with business and financial expertise. Owner of the land where AUS_001 was discovered	Building and leading organizations across many industries

- AUS\_001 has demonstrated **encouraging pre-clinical efficacy with a large safety margin**
- Completing IND enabling studies this year and will plan to be **ready for first in human studies in ~2Q 2026**
- **Experienced scientific team** who have partnered very effectively with academic institutions
- Leadership team with **expertise** in advancing oncology therapies through FDA approval and launch