Date: 7 August 2019 **Sydney, Australia**

ASX Limited
20 Bridge Street
SYDNEY NSW 2000

NOXOPHARM AUGUST 2019 CORPORATE PRESENTATION

- **Current clinical programs and therapeutic indications strategy remain ongoing**
- **Primary focus on use of Veyonda® and radiotherapy as first-in-class treatments for late-stage prostate cancer**
- **Commercial strategy to bring both DARRT and LuPIN programs as alternative treatments for late-stage prostate cancer**
- **Secondary focus on CEP program in sarcoma as strategic option offering expedited way to market approval with possible marketing/patent advantages**
- **Major discovery of idronoxil as a first-in-class STING modulator. STING proposed as essential companion treatment to immuno-oncology drugs**
- **Company see Veyonda® as leading, stand-out STING opportunity offering more effective and a well tolerated way of delivering STING compared to cyclic nucleotides**

Sydney, 7 August 2019: Noxopharm Limited (ASX: NOX) ('**Noxopharm'** or the '**Company'**) today releases its August 2019 Corporate Presentation ahead of Australian and U.S. roadshows.

The Presentation contains an update on recent studies showing Veyonda® to be a STING activator, and the impact this is expected to have on the Company's clinical development program and commercial opportunity.

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About Noxopharm
Noxopharm is a clinical-stage Australian drug development company with offices in Sydney and New York. The Company has a primary focus on the development of Veyonda® and is the major shareholder in Nyrada Inc, a spin-off company developing a pipeline of non-oncology drugs.

About Veyonda®
Veyonda® (previously known as NOX66) is a suppository dosage formulation of the experimental anti-cancer drug, idronoxil, that leads in the body to the formation of a proprietary pro-drug form. Idronoxil specifically inhibits the ability of cancer cells to respond to stress, such as that induced by radiation, leading to loss of pro-survival signaling via sphingosine-1-phosphate. Idronoxil is also a STING modulator, activating both the body's innate and adaptive immune systems.

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Roadshow Presentation

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ASX: NOX

DISCOVER DEVELOP DELIVER

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OUR VISION for

Veyonda®

A first-in-class, **dual-acting cytotoxic and immuno-oncology** molecule in a novel proprietary formulation

Putting **cGAS-STING** into the Tale

we believe that Veyonda ® has the potential to transform the survival and quality of life for a high proportion of cancer patients

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Relevance of STING …….. Why the pharmaceutical industry has STING in their sights

There are many different approaches to killing cancer cells, but one of the most recent ones is also one of the most elegant and rational ones:

killing cancer cells with our own immune cells!

An immune technology known as CAR-T has proven effective in some of the leukemias, but is very expensive, involves significant logistics, and is yet to show any benefit in solid cancers.

In the case of solid cancers, the challenge in using the body's immune system to fight the cancer is that the cancer cells either have eliminated all immune cells from the tumour or switched off their cancer-fighting ability.

These kind of tumours are called **COLD** tumours.

Tumours with some functioning immune activity are known as **HOT** tumours, and these are the tumours where the current immuno-oncology (i-o) drugs are able to work

However, the problem is that the majority of tumours are COLD!

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Relevance of STING …….. Why the pharmaceutical industry has STING in their sights

The proposed solution……

Converting **COLD tumours** into **HOT tumours**

In the case of the current i-o (checkpoint-inhibiting drugs), this conversion has been predicted to substantially boost their current **US$15 billion** p.a. sales to over **US$100 billion**

COLD tumour

HOT tumour

One promising method of achieving this conversion is known as STING agonism

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Relevance of STING …….. Why the pharmaceutical industry has STING in their sights

STING (Stimulation of Interferon Genes) is a fundamental first-line defense mechanism designed to eliminate cancer cells

STING involves the detection by the compound cGAS of damaged DNA in the cell's cytoplasm, a hallmark of cancer, setting in train a series of events that leads to the production of interferon by neighboring healthy cells. This interferon flags the cancer cell as needing to be removed, at the same time activating the local immune cells which then attack and kill the cancer cell

Switching STING back on so that COLD tumours become HOT tumours is considered vital for a successful immune-based cancer therapy

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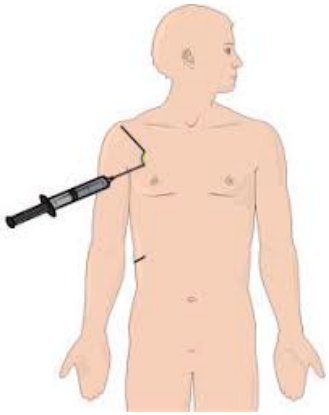
Relevance of STING …….. Why the pharmaceutical industry has STING in their sights

Multiple companies are working in the field of STING triggering (STING agonists) Their efforts are based on activating STING through the introduction of foreign DNA into the cancer cells. This is done with bacterial or viral cyclic nucleotides that are attempting to trigger a strong STING response in both the primary tumour and all secondary tumours

The limitation of these nucleotides is that they don't' distinguish between cancer and healthy cells. Exposing the whole body to these drugs runs the risk of inducing a so-called 'cytokine storm' of the sort that accompanies serious, overwhelming infections and which in itself can be fatal

As a result, these STING agonists are being injected directly into single tumours, thereby potentially limiting their effectiveness



First clinical report (June 2019) of this approach in a study conducted by Aduro Biosciences Inc and Novartis produced disappointing data

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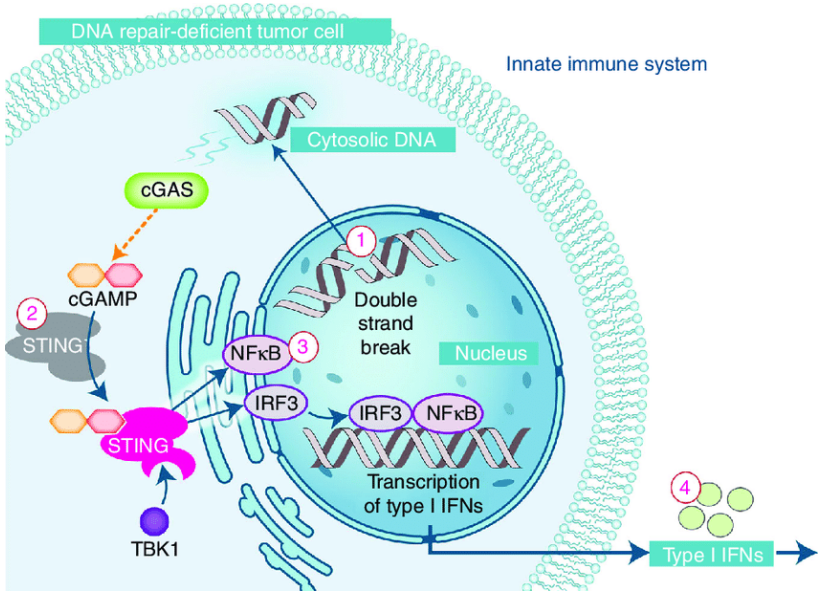
Veyonda® *(previously known as NOX66; suppository dosage form of idronoxil (IDX))*

A first-in-class cGAS-STING modulating agent

IDX switches STING on!

It amplifies STING where it already exists



Veyonda®

Active ingredient idronoxil (IDX)

Competitive advantages

- IDX does not trigger STING. It amplifies STING where it already exists
- IDX relies on the presence of '*self DNA*' to amplify STING, not on foreign DNA, thereby limiting its effect to cancer cells
- IDX is <u>not</u> a bacterial or viral cyclic nucleotide and therefore will <u>not induce a potentially fatal '*cytokine storm*' syndrome.</u>
  Veyonda® has proven to be well-tolerated in clinical trials to date
- IDX up-regulates both the innate and the adaptive immune system: Natural Killer (NK) cells, monocytes, CD4+ (Helper T cells), CD8+ (cytotoxic T cells)
- IDX is not restricted to being injected into a single tumour. It can be given systemically via Veyonda® giving it the chance to reach all tumours

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Contents

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1

Veyonda® Clinical Development Program Summary

Impact of IDX-STING discovery on Veyonda® development strategy

DARRT, LuPIN and CEP programs to remain on-track

STING amplification now considered a key mechanism of action in the DARRT program. Radiotherapy also known to activate STING, with Veyonda® now believed to be boosting this effect to yield the abscopal responses being seen with this combination

The IONIC Program *(Immuno-Oncology with NOX66 in Combination)* added as a fourth clinical program

STING, and converting COLD tumours to HOT tumours, currently proposed as a means of boosting the benefit of current i-o drugs. The Company believes that the key competitive advantages of Veyonda® in the STING response offer an important opportunity in a looming major new field of cancer therapy

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1 Veyonda® - Clinical Program

Unique 4-way development program

4-way use of a STING agonist to attack metastatic disease

CEP Chemo-Enhancement Program	**Chemo-enhancement:** **Soft tissue sarcoma: Veyonda + doxorubicin**
DARRT Direct and Abscopal Response to Radiotherapy	**Radio-enhancement:** **Prostate Cancer: Veyonda + radiotherapy**
LuPIN Lutetium-PSMA in Combination with NOX66	**Radio-enhancement:** **Prostate Cancer: Veyonda + ^{177}lutetium-PSMA-617**
IONIC Immuno-Oncology with NOX66 in Combination	**Immuno-oncology:** **Veyonda + Immuno-oncology agent**

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1 Veyonda® - Clinical Program

STRATEGY

CEP
Chemo-Enhancement Program

- Demonstrate the value of Veyonda by improving outcomes in under-served cancers e.g. Sarcoma
- Likely quickest way to achieve NDA (2025/26)

DARRT
Direct and Abscopal Response to Radiotherapy

- Prostate cancer is large market opportunity
- Potential future use in early-stage prostate cancer
- Potential future use in diverse late-stage cancers

LuPIN
Lutetium-PSMA in Combination with NOX66

- ^{177}lutetium-PSMA-617 registration forecast for 2021
- Establishes Veyonda's potential as adjunct to a whole range of radiopharmaceuticals

IONIC
Immuno-Oncology with NOX66 in Combination

- Further enhance the revolutionary effects of technological mega-trends in cancer treatment e.g. immuno-oncology drugs

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DARRT-1
DARRT-2

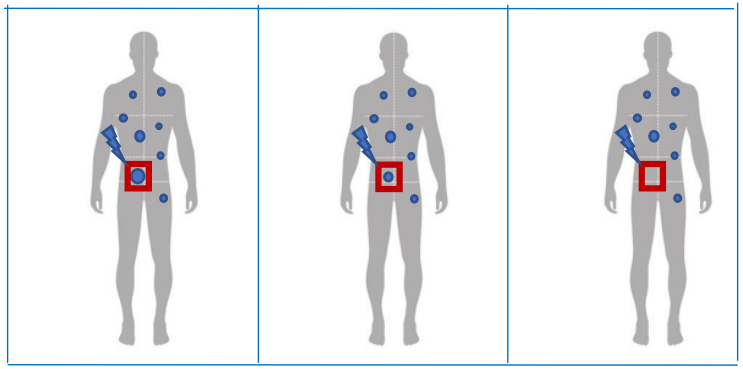
2

DARRT
Direct and Abscopal Response to Radiotherapy

DARRT-1 *(Current)*

- Late-stage mCRPC ; no remaining approved Rx options
- Progressive disease; post-taxane; post-androgen-ablation; eligible for palliative RT for symptomatic relief
- **DARRT Regimen:** Single course of palliative (20-25 Gy) EBRT to a single lesion + 10 days Veyonda®

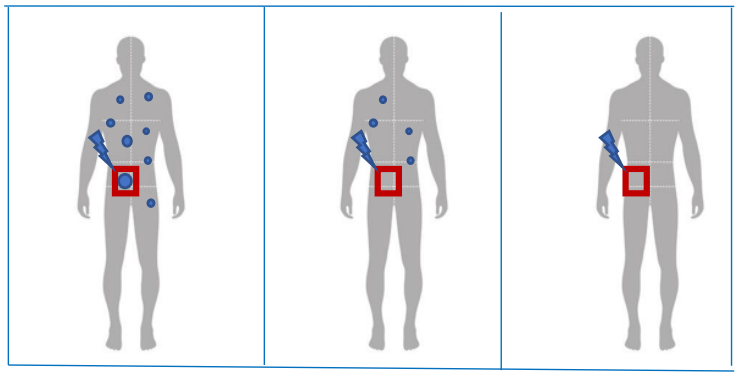
Standard response



Shrinkage of Irradiated tumor

Complete resolution of Irradiated tumor

Abscopal response



Partial abscopal response

Complete abscopal response

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DARRT
Direct and Abscopal Response to Radiotherapy

DARRT-1 *(Cont)*

- Open label study; 24 patients with mCRPC post-taxane and abiraterone/enzalutamide
- Treatment regimen = 10 days Veyonda®: palliative EBRT (20-25 Gy) in 5 fractionated doses to a single lesion
  - Dose escalation arm: 12 patients: 400 mg/800 mg/1200 mg Veyonda®
  - Dose expansion arm: 12 patients: 1200 mg Veyonda®

At **24 weeks** of follow up of 12 patients in the Dose Escalation Arm:

- **Safety and tolerability:** All three doses continued to be well tolerated and no serious side-effects were reported as being related to Veyonda®
- **Tumour size:** Disease control (stabilization of tumour volume) was highly durable with 57% of patients remaining progression-free at 6 months
- **Pain:** 5 of 7 patients maintained pain responses (≥ 30% falls) at 6 months as compared to 3 months, with two of these patients being completely pain-free at 6 months
- **PSA:** 5 of 14 patients (36%) experienced a clinically meaningful PSA response (≥ 50% fall) during the follow up period

Note: PSA responses in trials of palliative external beam radiotherapy alone range from 5-9%[1,2]

1. Din OS, et al. Radiother Oncol. 2009;93:192-6. 2. Kwon ED, et al. Lancet Oncol. 2015;15:700-12.

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DARRT
Direct and Abscopal Response to Radiotherapy

DARRT-2 *(planned)*

- **Prostate Cancer**
- **Double-blind, randomized, controlled trial**
- **Phase 2 with possible Phase 3 adaptation**
- **Veyonda plus radiotherapy**
- **Repeat courses of 1200 mg Veyonda® daily/7 days each month for 6 months**
- Expected commencement H2 2020
- Multisite
- Powered to achieve statical significance on several efficacy parameters
- Designing study in collaboration with world-renowned prostate cancer experts in Australia and USA

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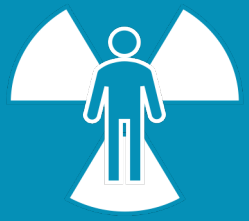
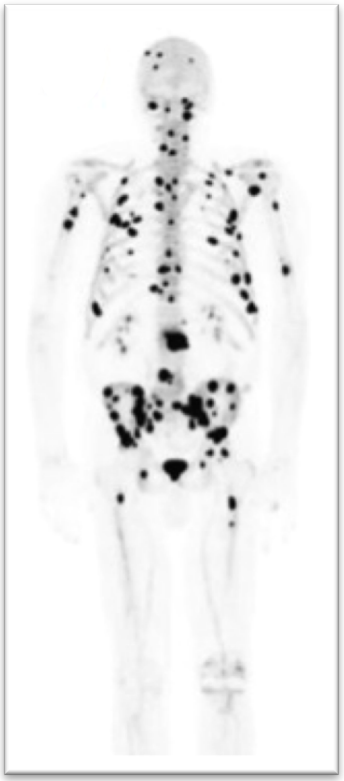


LuPIN

2

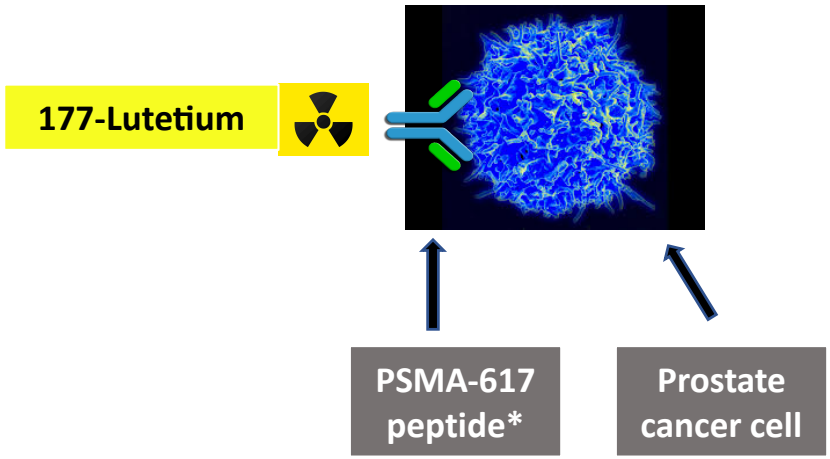
LuPIN

^{177}Lutetium-PSMA-617 is a radiopharmaceutical comprising the radionuclide^{177}lutetium (a beta emitter) attached to the monoclonal antibody peptide (617) against the prostate surface membrane antigen (PSMA). Enabling the access of a low dose of radiation to all prostate cancer cells within the body



Given as 4-6 single intravenous injections 6-weeks apart

^{177}Lu-PSMA-617 has shown encouraging benefit in the treatment of patients with mCRPC[1]



*PSMA-617 is owned by NOVARTIS

1. Bräuer A, et al. Eur J Nucl Med Mol Imaging. 2017;44(10):1663-1670..

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2

LuPIN

AIM

To show that Veyonda® boosts the efficacy of radiopharmaceutical treatment which is limited by the degree of expression of the target receptor on cancer cells

RATIONALE

To do so through

- DNA repair inhibition to boost DNA-damaging effect of isotope on cancer cell
- STING agonist effect to stimulate innate immune cell function in all lesions

STUDY Objectives in LuPIN-1: To achieve

- **higher response rates**, with more patients able to complete the 6-course Lu-PSMA treatment without relapsing
- **greater depth of response** as measured by PSA levels
- **more durable responses** as measured by improved time to progression and overall survival.

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LuPIN

LuPIN-1 *(Current)*

Investigator-initiated, open-label, Phase 1 study to determine if Veyonda® increases the response to ^{177}Lu-PSMA-617 in men with mCRPC[1]

PATIENTS* (N=56)

- mCRPC previously treated with abiraterone and/or enzalutamide and taxane-based chemotherapy
- Evidence of disease progression
- PMSA-positive disease
- Adequate hematologic, hepatic and renal function

- Veyonda® daily for 9 days
- ^{177}Lu-PSMA-617 iv on Day 0
- Up to 6 x 42-day cycles

Cohort 1 (n=8)
Veyonda® **400mg**

Cohort 2 (n=24)
Veyonda® **800mg**

Cohort 3 (n=24)
Veyonda® **1200mg**

Endpoints

- Toxicity (primary)
- Efficacy via composite of QoL, pain, PSA, medical imaging (primary)
- Tissue bio-distribution
- PFS & OS (12 months)
- Change in serum PSMA

Status

- Cohorts 1 and 2 - fully enrolled
- Cohort 3 – open for enrolment

1. ANCTR. Trial registration number ACTRN12618001073291. Available at: www.anzctr.org.au/Trial/Registration/TrialReview.aspx?id=374100. Accessed March 2019.

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LuPIN-1 *(Current)*

Interim Safety data on first 16 patients[1]

- All 16 patients received at least 2 doses; 4 (25%) patients received 6 cycles
- Treatment was well tolerated; 1 patient (6.3%) reported an SAE due to pneumonitis and continued on trial without Veyonda®

AEs ≥ Grade 2, n (%)	Cohort 1 400mg **Veyonda®** + RT (n=8)	Cohort 2 400mg **Veyonda®** + RT (n=8)	Overall (n=16)
Total	4 (50.0)	1 (12.5)	5 (31.3)
Haematologic	3 (37.5)	-	3 (18.8)
Fatigue	3 (37.5)	1 (12.5)	4 (25.0)
Other (pneumonitis)	1 (12.5)	-	1 (6.3)

1. Emmett LE, et al. Abstract submitted to SNMMI 2019.

2

LuPIN

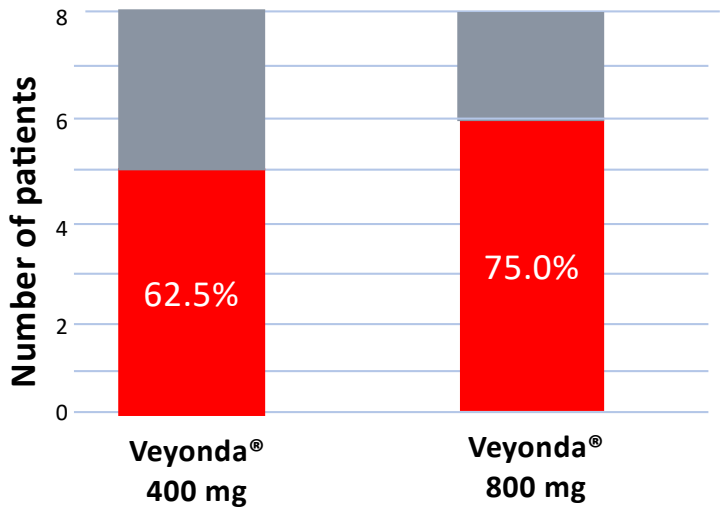
LuPIN-1 *(Current)*

Interim Efficacy data on first 16 patients

First **16 subjects:**

- ✓ All of whom have received ≥ 2 doses of ^{177}Lu-PSMA-617
- ✓ 4/16 have already completed the planned 6 cycles
- ✓ Patients 1-8 received 400 mg of Veyonda®
- ✓ Patients 9-16 received 800 mg of Veyonda®
- ✓ 3 (19%) patients exited the trial early due to progressive disease

PSA% response by Veyonda® dose



Adapted from Emmett L, et al. 2019[1]

69% overall PSA response rate (≥ 50% PSA fall) and 75% response rate with 800 mg Veyonda® compares favourably with PSA response rates of ^{177}Lu-PSMA-617 alone, ranging between 31 - 61%[2-5]

1. Emmett LE, et al. Abstract submitted to SNMMI 2019. 2. Hofman MS, et al. Lancet Oncol. 2018;19(6):825-33. 3. Kratochwil C, et al. Journal Nuc Med. 2016;57(8):1170-6. 4. Emmett L, et al. J Med Rad Sci. 2017;64(1):52-60. 5. Emmett L, et al. Clin Genitourin Cancer. 2019;17:15-22.

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CEP-1
CEP-2

Why Chemo-Enhancement?

Because:

- Chemotherapy remains the mainstay treatment of most types of cancer in all stages of the disease[1]
- However, **toxicity and the development of drug resistance** make **chemotherapy** less practical and less attractive in late-stage disease[1,2]
- **IDX enhances the anti-cancer activity of many cytotoxics by up to 2,000x** and reverses resistance to drugs including alkylating agents and anthracyclines[3]
- We believe that CEP addresses a significant unmet patient need and represents the likely **quickest way to achieve an NDA**

The objective of the CEP program is to demonstrate that Veyonda® plus chemotherapy can improve efficacy and/or achieve the same efficacy with a reduced, safer chemotherapy dose and can re-sensitise patients who have become resistant to chemotherapy.

1. Rumpold H & Wimder T. Magazine of Eur Med Oncol. 2017;10:119-120. 2. Gupta S, et al. Ann N Y Acad Sci. 2011;1215:150-60. 3. Brown D, et al. Drugs Fut. 2008;33(10):844-860.

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CEP
Chemo-Enhancement Program

CEP-1 *(Completed)*

Phase 1, open label, first in human study of Veyonda® in combination with carboplatin in heavily pre-treated patients with end-stage metastatic disease

N=19: multiple tumour types: **breast, prostate, lung, ovarian**

- Locally or metastatic advanced solid tumours; no CNS involvement
- ≥1 measurable lesion via CT/MRI
- ECOG PS 0-1
- Adequate haematologic, hepatic and renal function
- Life expectancy ≥12 weeks

Cohort 1
Veyonda® **400mg (1x daily)**
(n=8)

- Monotherapy Arm: 3 weeks Veyonda®
- Combination Arm: Veyonda®
- \+ monthly carboplatin
  - 3 cycles 50% standard dose
  - 3 cycles 75% standard dose

Cohort 2
Veyonda® **800mg (400 mg 2x daily)**
(n=11)

Endpoints
- Overall response
- Percent change in target lesions
- PFS & OS
- PK
- Safety

CEP-1 concluded in April 2019

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CEP
Chemo-Enhancement Program

CEP-1 *(Completed)*

Dose cohort	Assessment time point*	Count (n)	Partial response	Stable disease	Progressive disease
Cohort 1 Veyonda® 400mg, n (%)	Cycle 3	5	0 (0.0)	4 (80.0)	1 (20.0)
	Cycle 6	2	0 (0.0)	1 (50.0)	1 (50.0)
Cohort 2 Veyonda® 800mg, n (%)	Cycle 3	9	0 (0.0)	7 (77.8)	2 (22.2)
	Cycle 6	6	1 (16.7)	4 (66.7)	1 (16.7)

- ✓ **Efficacy:** Of the 9 patients allocated to the higher dosage (800 mg) of Veyonda®, **5/9 (56%) showed stable disease (no tumour growth and no new tumours) or a partial response after 6 cycles**

- ✓ **Safety:** Veyonda® was generally well tolerated with only one SAE (anaemia) considered possibly related to Veyonda®

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CEP
Chemo-Enhancement Program

CEP-2
(Proposed)

- **Soft tissue sarcomas:** cancer type with poor therapeutic options
- Opportunity for **Accelerated Approval**, **Orphan Drug designation**, **Pediatric Review Voucher**
- **Timetable:**
  - Submit IND to FDA Q3/4 2019
  - NDA grant 2025/26 assuming accelerated approval

Pre-clinical studies: Potent monotherapy effect of IDX and additive effect of IDX + doxorubicin against both soft tissue and hard tissue sarcomas. (*Data on file*)

- Metastatic soft tissue sarcoma (mSTS) is invariably fatal
- Technically rare, yet 21,000 patients are diagnosed with mSTS in the G7 every year[1,2]
- Doxorubicin (Standard of care for mSTS) was discovered in 1969[3]

1) Amankwah et al (2013). Epidemiology and therapies for metastatic sarcoma. Clinical Epidemiology, *5, 147–162.*
2) Soft Tissue Sarcoma: Epidemiology. DRG. (2018). Incidence in mature markets
3) Cassinelli, G. (2016). The Roots of Modern Oncology: From Discovery of New Antitumor Anthracyclines to Their Modern use. *Tumori Journal, 102, 226-235*
4) Miller, K. (2017). Do investors value the FDA orphan drug. *Orphanet Journal of Rare Diseases*, 12:114

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CEP-2
(Proposed)

Phase 1b, open label; Veyonda® + doxorubicin; metastatic soft tissue sarcomas

- ✓ **Design:** Dose escalation and dose expansion arms
  - — Dose escalation: 400, 600, 800, 1200, 1800 mg Veyonda® (6 patients per dose)
  - — Dose expansion: MTD Veyonda® dose (16 patients)
  - — Standard 3 cycles of doxorubicin
- ✓ **Title:** *A dose escalation and dose expansion study of Veyonda® (idronoxil suppository) plus doxorubicin in anthracycline-naïve, adult patients with soft tissue sarcoma*
- ✓ **Sites:** U.S.; multiple
- ✓ **Eligibility:** Anthracycline naïve adult patients with metastatic soft tissue sarcoma for whom treatment with doxorubicin is considered to be appropriate
- ✓ **Objectives:** MTD, safety, PK, QoL, efficacy (RECIST, PFS, OS)
- ✓ **Pivotal trial**: End-points to be determined on basis of Phase 1b outcome

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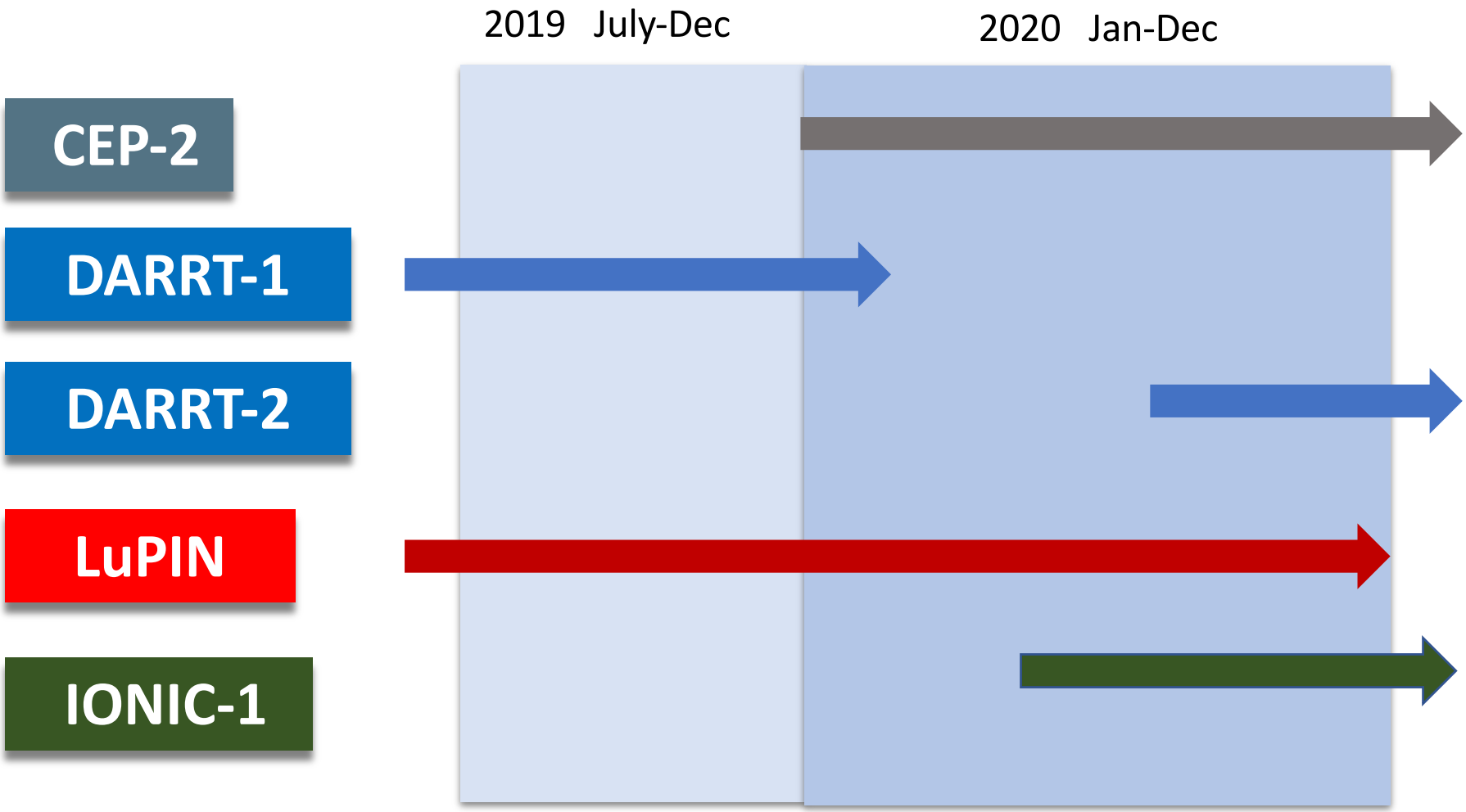
- Timetable
- News Flow

4 Clinical timetable 2019 - 2020



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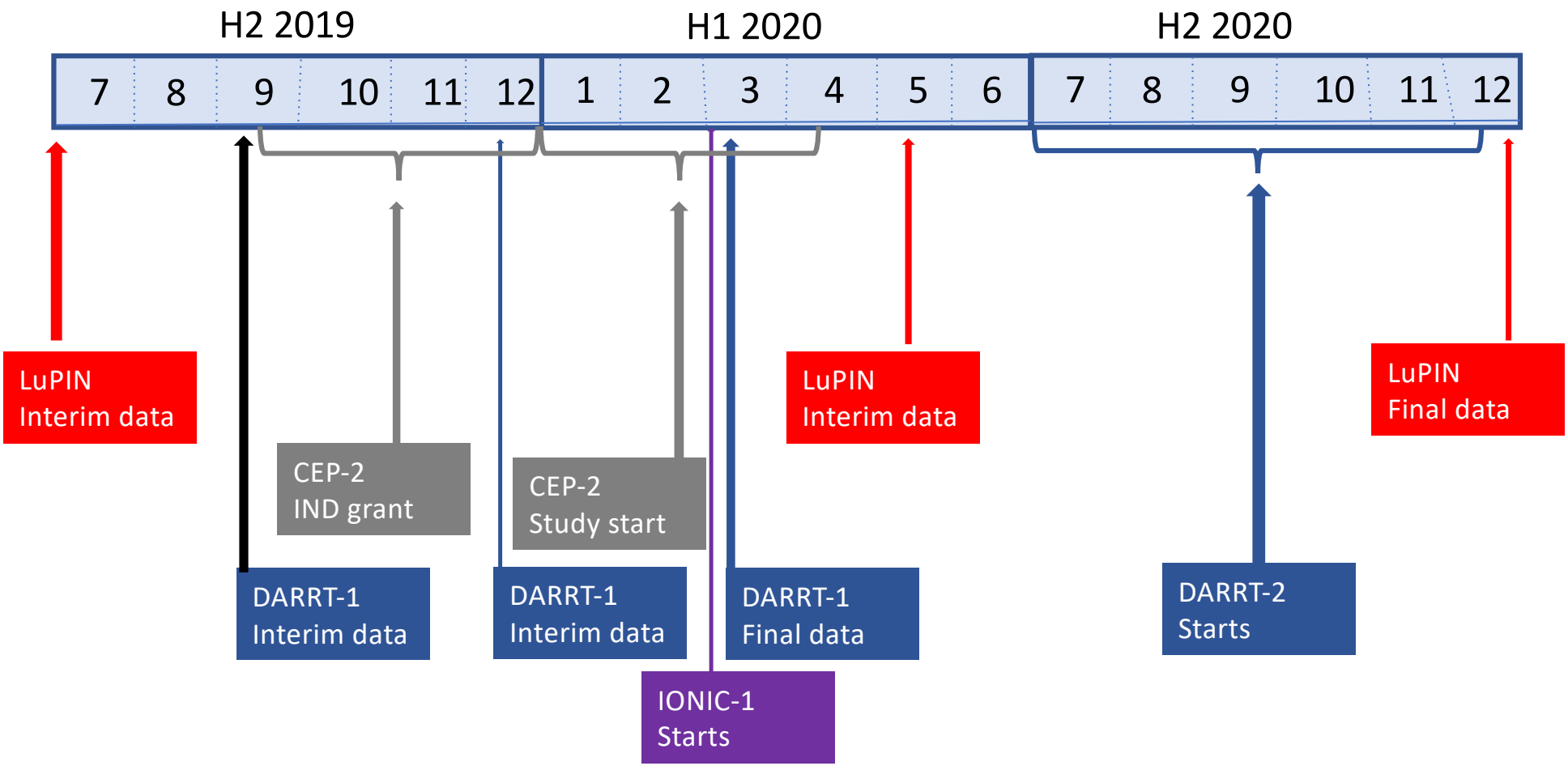
Anticipated clinical news flow



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- Cash position
- Interim funding
- IPO

5 Current cash position

Capital raisings since ASX listing August 2016

IPO	**AU$6.0 M**
Aug 2017	**AU$5.5 M**
April 2018	**AU$11.5 M**
TOTAL	**US$23.0 M**

Cash position at 31 July 2019 = **US$6.5 M**

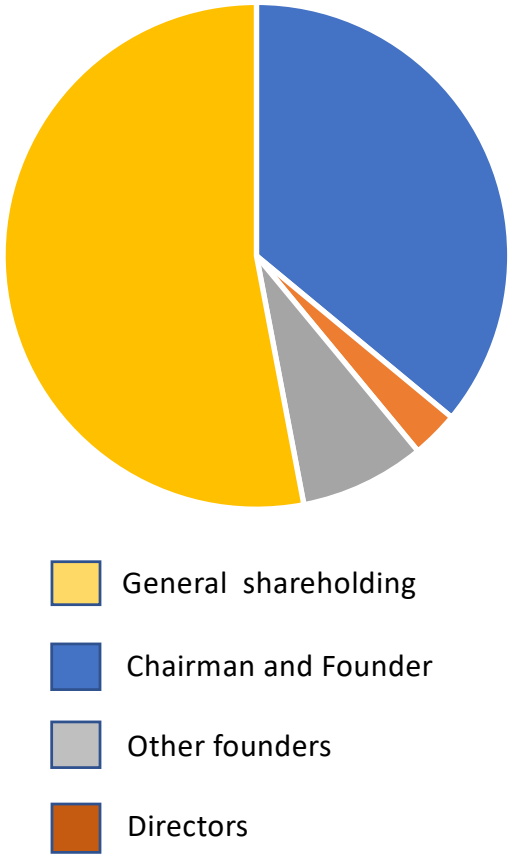
Current monthly burn rate = **US$800K**

Anticipated Australian Govt 43% R&D cash rebate.
August 2019 = **US$3.6 M**

NOXOPHARM

5 Key Metrics

Number of Shares	125.6 million shares outstanding 30.5 million options (expiring 2020-22)
Market Cap (3 August 2019)	AU$56.6M
IPO price **12 month high/low**	20 cents $1.64/0.36
Cash position	• AU$ 6.2M (31 July 2019) • AU$3.6M *anticipated* (Aust Govt R&D Rebate)



NOXOPHARM

Our People

Graham Kelly Executive Chairman

BSc, BVSc, PhD

Graham has 25 years' experience in founding and leading biotech companies. He founded Novogen Ltd (ASX:NRT. NASDAQ: NVGN)(now Kazia Therapeutics, KZA) in 1994; Marshall Edwards Inc (NASDAQ:MEI) (now MEI Pharma, MEIP) in 2001; Noxopharm (ASX:NOX) in 2016. He oversaw NVGN from A$12M market cap in 1994 to A$900M in 2003.

Greg van Wyk CEO

MBBCh, BBA, MEc

Greg is a medical doctor who prior to joining NOX had a 11-year career as a Medical Director in Eli Lilly, leading medical teams in Australasia and North-Western Europe across a range of therapeutic areas including oncology. Greg also has post-graduate degrees in management and economics.

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Our People

John Wilkinson Chief Scientific Officer

BSc Hons, PhD

John brings over 30 years of experience from both pharmaceutical and research settings in Australia and the United Kingdom. His research interests include Drug Development, Virology, Oncology and Immunology, translating novel laboratory findings into human clinical trials that have previously resulted in numerous peer-reviewed publications.

Gisela Mautner Global Medical Director

MD-PhD (*TU-LMU Munich*) MPH (*Harvard*) MBA (*Kellogg*) FACPE (Australia)

Gisela is a medical doctor with over 20 years' experience in the pharma/biotech industry in Europe, USA and Australasia. In her early career Gisela was a research fellow at NIH, Bethesda, MD. Since then she has focused on bringing new drugs and therapies to market in senior roles in Medical Affairs at Merck/MSD, Bayer and Amgen.

Jeanette Bell Chief Operating Officer

BMedSc, MScM, PhD candidate

Jeanette has more than 30 years' experience in healthcare including 15 years at Eli Lilly in senior leadership roles in Europe, Asia Pacific and Japan. The scope of work in Asia Pacific involved co-developing the clinical strategy and operations plans for registration studies in China and Japan. In the role as COO, Jeanette will drive the delivery of the Noxopharm Clinical Development Plan.

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