March 2019

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ASX: NOX

DISCOVER DEVELOP DELIVER

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We have a single objective:

To make ***Veyonda®*** (a radio-enhancing/immune-enhancing drug)

a standard companion drug

→ for all forms of radiotherapy

→ across most forms of solid cancer

in order to deliver

→ more potent, life-prolonging responses to radiotherapy

→ at lower, better tolerated dosages of radiotherapy

and in so doing

→ provide a transformative leap forward in the treatment of many cancers

Veyonda® - A New Improved Formulation of Idronoxil

Veyonda® delivers a proprietary pro-drug form of idronoxil* that delivers continuous anti-cancer activity for 12 hours

Veyonda® provides clinical benefit where earlier formulations did not

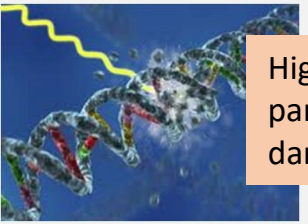
Veyonda® is a convenient-to-use, self-administered dosage form given twice daily to provide continuous 24-hour cover

* ***Patents pending***

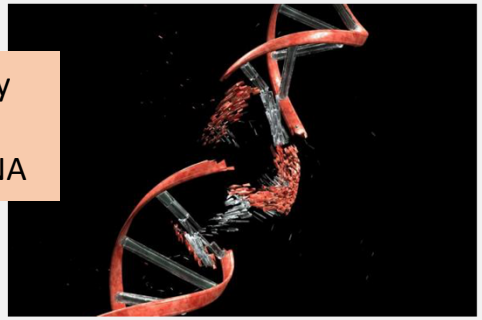
Examples of how a radio-enhancer might be used in prostate cancer

Stage of cancer	**Veyonda®** + Radiotherapy (RT)	Potential benefits
Early-stage, operable	→	Avoidance of surgery and attendant side-effects
Early-stage, inoperable or recurrence post-prostatectomy	→	Reduction in RT dosage to minimise pelvic tissue damage
Intermediate-stage, hormone sensitive or non-metastatic	→	Delay in start of castration therapy
Late-stage, palliative	→	• Better pain and symptom relief • Delay in tumour progression

How radiotherapy works

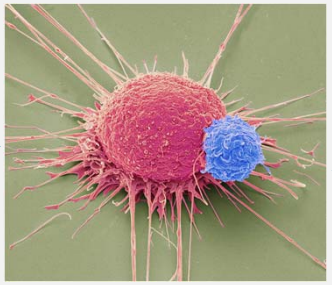


High energy particles damage DNA



Damaged DNA triggers release of **interferon** from neighboring healthy cells

Interferon activates innate immune cells (NK cells) within tumor. Activated NK cells attack cancer cells



SURVIVING CELLS killed by immune cells

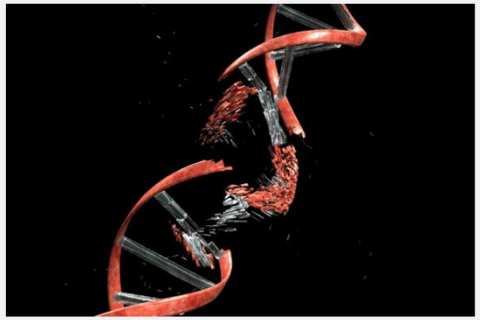
Cell attempts to repair DNA damage

Too much damage » Repair unsuccessful » CELL DIES

Repair successful » CELL SURVIVES

How *Veyonda*® radio-enhances

BLOCKS DNA REPAIR MECHANISMS



Damaged DNA triggers release of **interferon** from neighboring healthy cells

Cell attempts to repair DNA damage

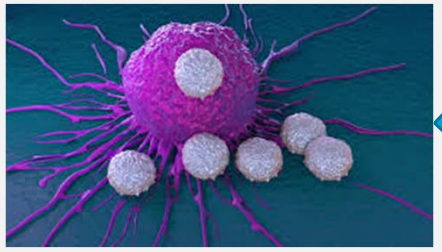
Repair unsuccessful
CELL DIES

FEWER CELLS SURVIVE

SURVIVING CELLS more effectively killed by amplified immune response

Veyonda® BOOSTS INTERFERON RESPONSE

INCREASED ACTIVITY OF IMMUNE CELLS



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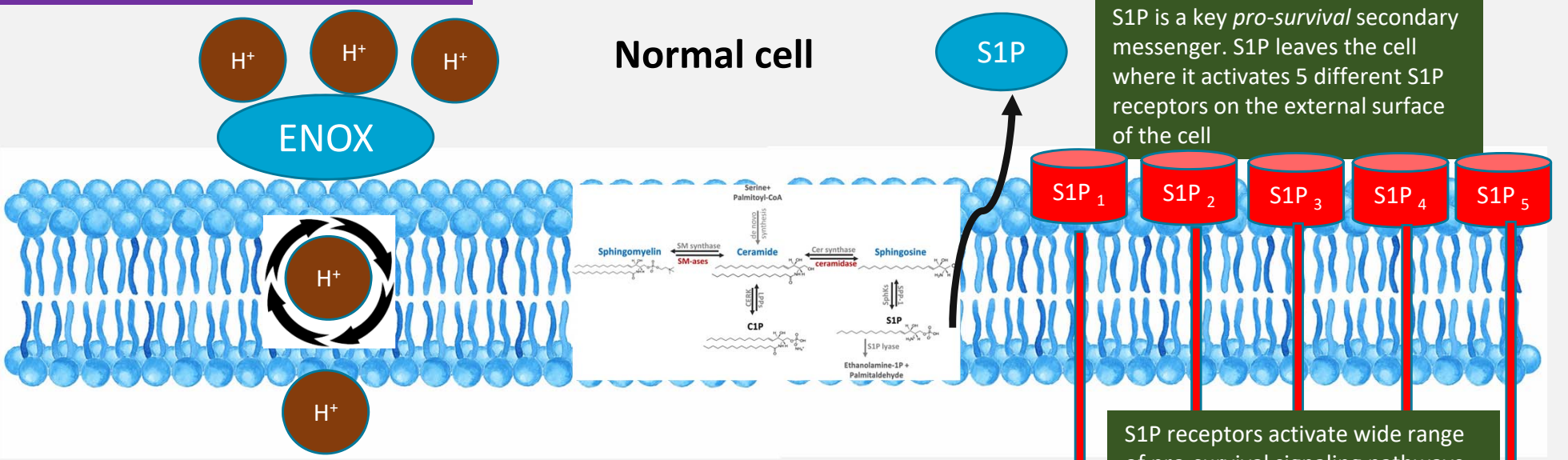
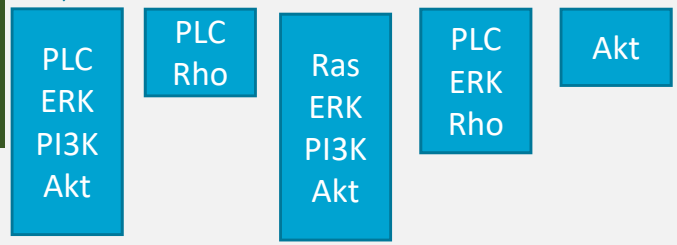
Veyonda® MoA



S1P is a key *pro-survival* secondary messenger. S1P leaves the cell where it activates 5 different S1P receptors on the external surface of the cell

S1P receptors activate wide range of pro-survival signaling pathways, one of which is DNA repair capacity



External NADH oxidase (ENOX) enzyme regulates pump that transfers protons (H^+) to the outside of the cell. This creates transmembrane electron potential that powers membrane functions such as the 'sphingomyelin cycle'.

'Sphingomyelin cycle': Sphingomyelins constituting about 25% of membrane phospholipids are in constant state of flux between sphingosine ⟺ ceramide ⟺ sphingosine-1-phosphate (S1P).

Activate DNA repair enzymes:
- Topoisomerase 2
- PARP 1 and 2

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Veyonda® MoA

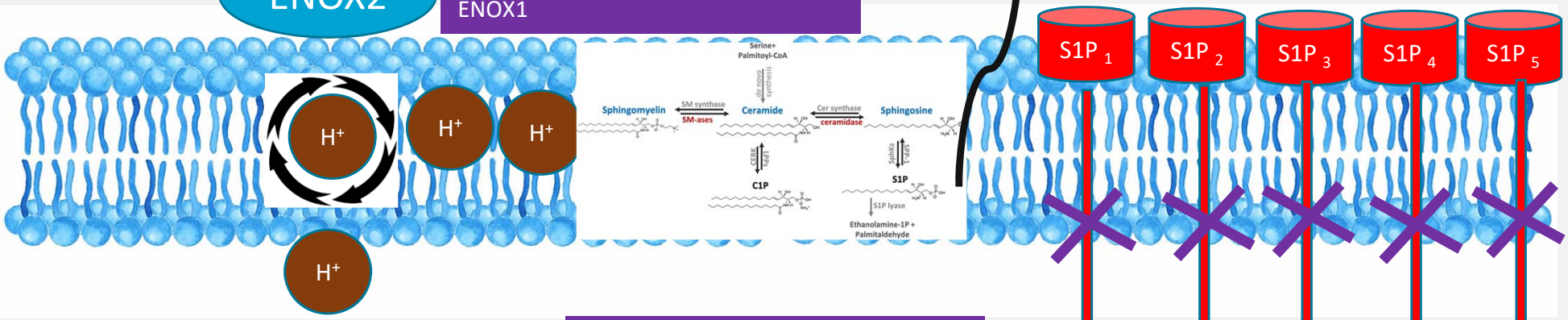
Cancer cell

Human cells express RNA for two forms of ENOX - ENOX1 and ENOX2. ENOX2 protein only expressed on cancer cells where it dominates over ENOX1

ENOX2

S1P

Declining S1P levels deprive S1P receptors of activation



Idronoxil (Veyonda active ingredient) only binds to ENOX2. ENOX2 inhibition leads to accumulation of protons in plasma membrane.

Sphingosine kinase function inhibited by elevated proton levels. Ceramide (*pro-apoptotic*) levels rise and S1P (*pro-survival*) levels fall.

Key pro-survival signaling pathways down-regulated

PI3K Akt

PI3K Akt

DNA repair enzyme activity inhibited

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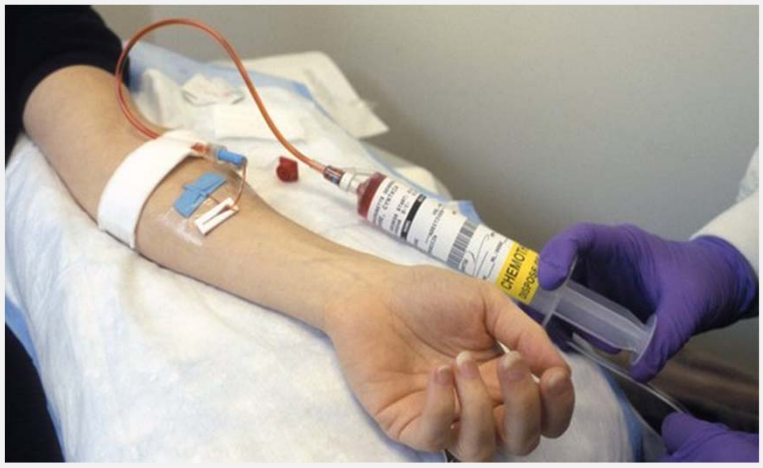
Veyonda® + Radiotherapy

Potential use across broad range of radiotherapy practice

Externally-delivered radiotherapy



Internally-delivered radiopharmaceuticals

Clinical Programs

DARRT — Direct and Abscopal Response to Radio-Therapy

LuPIN — ^{177}Lutetium-PSMA In Combination With VeyoNda

Initial focus on **late-stage prostate cancer**

- Metastatic, castrate-resistant disease
- Post-docetaxel and abiraterone/enzalutamide
- No remaining standard treatment options
- Progressive disease
- Anticipated survival of > 3 months
- Patient eligible for palliative treatment

DARRT

Direct and Abscopal Response to Radio-Therapy

Rationale

1. Use of palliative (low) dose of radiation minimizes damage to innate immune cells within the irradiated lesion.

2. Veyonda® amplifies radiation-induced DNA damage in cancer cell by:
- *blocking cell division, thereby exposing the DNA to greater damage, and*
- *blocking the ability of the cancer cell to repair that damage*

3. Amplified radiation-induced DNA damage then:
- *Increases likelihood of irradiated cancer cell dying*
- *Enhances response of local innate immune cells*

Objectives

<u>Local effect.</u> Greater shrinkage of the irradiated target lesion (DIRECT RESPONSE)

<u>Systemic effect</u>. An anti-cancer response in non-target, non-irradiated lesions stemming from enhanced innate immune and epigenetic responses in the irradiated lesion (ABSCOPAL RESPONSE)

DARRT

Direct and Abscopal Response to Radio-Therapy

External Beam RT
or
Stereotactic Body RT

- Patients with multiple lesions requiring palliative radiotherapy
- Irradiate one lesion(*20-25 Gy in 5 fractionated doses*)
- **Veyonda®** 13-16 days
- Assessments at 6 , 12 and 24 weeks
  - PSA
  - Pain Score
  - QoL Score
  - Time to progression
  - RECIST* (where possible)

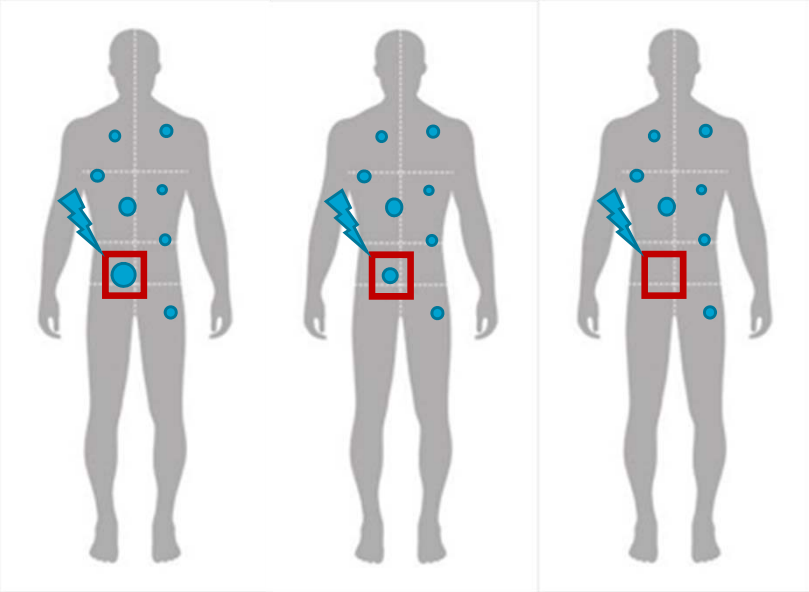
*Patients in dose-finding phase needed to have at least 2 measurable lesions as per RECIST v1.1

DARRT

Direct and Abscopal Response to Radio-Therapy

DIRECT RESPONSE

At a minimum, Veyonda® is expected to lead to better **DIRECT response** to radiotherapy by functioning as a **radio-enhancer**

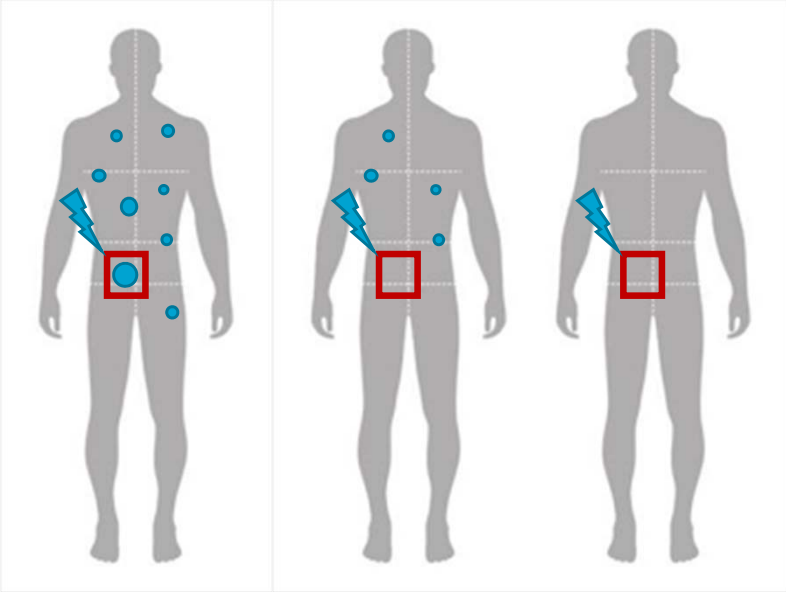


Shrinkage of Irradiated tumor

Complete resolution of Irradiated tumor

ABSCOPAL RESPONSE

The best expected outcome would be an improved DIRECT response, plus shrinkage of non-targeted lesions



Partial abscopal response

Complete abscopal response

DARRT-1 Study

Details:

- Phase 1b multi-national study (Australia, NZ, Georgia)
- Open label, single-arm study
- 24 patients; metastatic, castrate-resistant prostate cancer
- **Veyonda®** + external beam RT to 1 lesion*
- Part 1: Dose-finding (constant RT dose)
  - 400 mg Veyonda (4 patients)
  - 800 mg Veyonda (4 patients)**
  - 1200 mg Veyonda (4 patients)***
- Part 2: 1200 mg Veyonda
  - 12 patients

*Patients in dose-finding needed to have one measurable lesion as per RECIST v1.1
**2 non-evaluable patients were replaced
*** 1 patient was not evaluable at 12 weeks

DARRT-1 Study

12-week data for Part 1 patients:

	400 mg n=4	800 mg n=4	1200mg n=4 (3 evaluable)
PSA response*	0	2	2
Pain response**	2	3	2
RECIST response***	4 SD	1 PR 2 SD 1 PD	3 SD

* > 50% decline
** > 30% decline
*** aggregate of all measurable lesions

24-week data Part 1 patients – late May 2019
12-week data Part 2 patients – July 2019
24-week data Part 2 patients – Q1 2020

PD = Progressive disease
SD = Stable disease
PR = Partial response
CR = Complete response

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DARRT-1 Study

Interim conclusions:

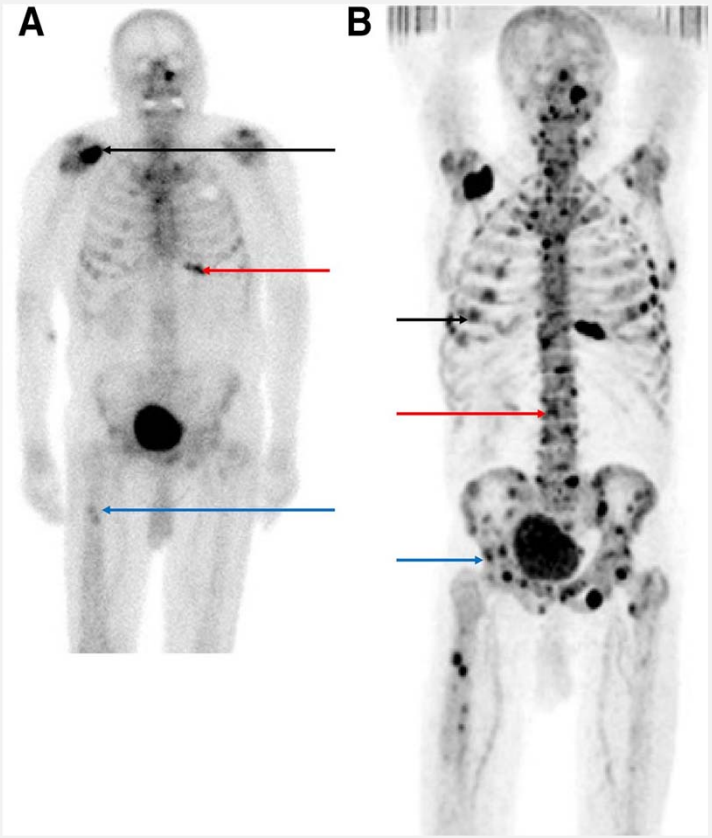
- **Veyonda®** + palliative dosages of radiotherapy well tolerated
- 400 mg dose of **Veyonda®** sub-therapeutic
- No notable difference between 800 and 1200 mg doses
- In the 7 evaluable patients in the 800 and 1200 mg cohorts*
  - 4/7 achieved PSA falls >50%
  - 5/7 achieved decrease in pain levels >30%
  - 1/7 showed partial response (RECIST) and 5/7 showed stable disease

The significant reductions in PSA, pain levels and the tumour control rate^ is suggestive of systemic (off-target) responses at 3 months men with advanced mCRPC.

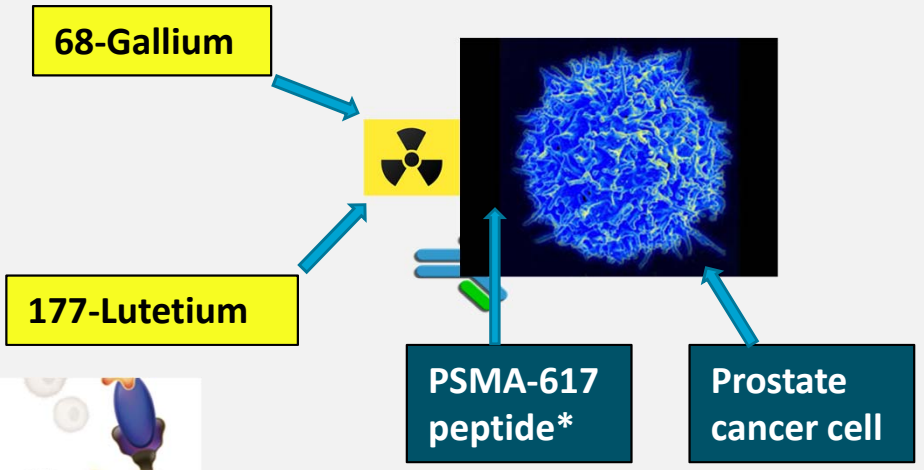
* Of the 10 patients enrolled, 7 were evaluable at 12 weeks
^ Tumour/disease control rate = proportion with SD or PR or CR

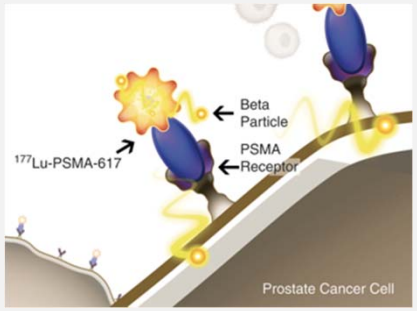
LuPIN

^{177}Lutetium-PSMA-617 In Combination With VeyoNda



Aim of ^{177}lutetium-PSMA-617 therapy is to deliver a low dose of radiation to all cancer cells within the body





* PSMA-617 is owned by NOVARTIS

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LuPIN

^{177}Lutetium-PSMA-617 In Combination With VeyoNda

Rationale

1. Use of radiopharmaceutical maximises synergy between **Veyonda®** and radiation in the broad spread of cancer cells throughout the body.

2. **Veyonda®** amplifies radiation-induced DNA damage in cancer cell by:
- *blocking cell division, thereby exposing the DNA to greater damage, and*
- *blocking the ability of the cancer cell to repair that damage*

3. Amplified radiation-induced DNA damage then:
- *Increases likelihood of irradiated cancer cell dying*
- *Enhances response of local innate immune cells*

Objectives

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

LuPIN-1 Study

- Phase 1/2 study; investigator-initiated; Australia
- Open label, single arm
- PSMA-positive, late-stage mCRPC patients
- 6 courses of ^{177} lutetium-PSMA-617 administered intravenously every 6 weeks
- **Veyonda®** administered for 10 days, starting day 1 of every course
- 8 patients 400 mg **Veyonda®**; 24 patients 800 mg **Veyonda®**
- 30/32 patients enrolled 1 March 2019*

*Clinical data from first 16 patients to be presented at SNMMI Conference, Anaheim, June 2019.

Planned Expanded Clinical Study Program 2019-2020

DARRT - mCRPC
- Late-stage mCRPC patients eligible for palliative radiotherapy therapy
- Phase 2 (Proof-of-concept), Global including US
- Double-blind, 2-arm study
- End-points: PSA response, pain response, QoL, rPFS, OS

CEP – sarcoma
- Patients with Sarcoma eligible for treatment with doxorubicin
- Phase 1, US
- Open label, multiple dose combinations
- End-points: Pharmacokinetics, Tolerability, Safety, DCR, ORR, PFS, OS

DARRT – rare cancers
- Rare cancers eligible for palliative radiotherapy therapy
- Phase 1, Australia
- Open label
- End-points: Correlative data analysis, DCR, ORR, PFS, OS

Veyonda® + immuno-oncology drug
- NSNSC lung cancer patients
- Previously treated with platinum containing chemotherapy regimen
- Phase 1b, open label, Australia
- End-points: Pharmacokinetics, Tolerability, Safety, irDCR, irORR, irPFS, OS

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External radiotherapy in cancer (all forms) treatment*

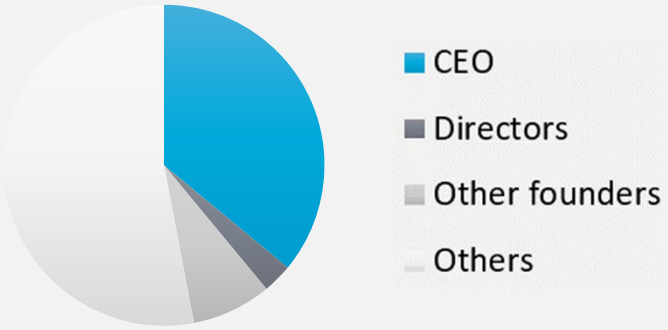
	Africa	Asia Pacific	Europe	Latin America	North America
Population	1070	4108	893	601	350
No. radiation centres	140	2590	1430	620	2790
No. radiotherapy courses	0.4M	3.3M	1.9M	0.6M	0.9M
Cost per course (US$)	1,300	2,120	3,490	2,080	7,050

RADIO-ENHANCER OPPORTUNITY Total 7.1 M courses of radiotherapy = **US$70 billion** (US$10K per course)

* Zubizaretta E et al. Clinical Oncology (2017) 29, 84-92

Key metrics

Number of Shares	**121.9M** : Free float 66.8%
Market Cap (1 March 2019)	AU$53M
IPO price **12 month high/low**	20 cents $1.64/0.36
Cash position	AU$ 9.6M (31 Dec 2018)



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