Sydney, 6 November 2017: Noxopharm is pleased to release the 3 presentations being made today in conjunction with the Company’s Extraordinary General Meeting.

Two transactions are being considered by shareholders which, if approved, will see a subsidiary company, Nyrada Inc, created as a drug development business with a commencing pipeline of 3 drug assets – NYX-104, NYX-205 and NYX-330.

The EGM is being held today (Monday 6 November) at 10 am at the Noxopharm offices: Level 4, Suite 3, 828 Pacific Highway, Gordon.

About NYX-104
NYX-104 is being developed to assist patient recovery after head and spinal trauma. It is a small molecule kinase-inhibitor that blocks TRPC class ion channel-regulated influx of calcium ions and mobilisation of calcium stores in axons exposed to glutamate overload.

About NYX-205
NYX-205 is being developed as an anti-inflammatory drug to treat inflammatory conditions of the central nervous system (eg. Alzheimer’s Disease, multiple sclerosis) and peripheral nerves (peripheral neuropathy), bowel (ulcerative colitis, sclerosing cholangitis) and liver (NASH). It is a small molecule inhibitor of thromboxane synthase.

About NYX-330
NYX-330 is being developed to treat hypercholesterolaemia. It is a small molecule inhibitor of PCSK9.

About Noxopharm
Noxopharm is an Australian drug development company with offices in Sydney and Hong Kong. The Company has a primary focus on the development of drugs to address the problem of chemotherapy- and radiation-resistance in cancer cells, the major hurdle facing improved survival prospects for cancer patients. NOX66 is the first pipeline product, with later generation drug candidates under development. The Company also has initiated a pipeline of non-oncology drugs.

About Nyrada Inc.
Nyrada Inc is a recently formed, New York-based biotechnology company established to house Noxopharm non-oncology drug assets.
Forward Looking Statements
This announcement may contain forward-looking statements. You can identify these statements by the fact they use words such as “aim”, “anticipate”, “assume”, “believe”, “continue”, “could”, “estimate”, “expect”, “intend”, “may”, “plan”, “predict”, “project”, “plan”, “should”, “target”, “will” or “would” or the negative of such terms or other similar expressions. Forward-looking statements are based on estimates, projections and assumptions made by Noxopharm about circumstances and events that have not yet taken place. Although Noxopharm believes the forward-looking statements to be reasonable, they are not certain. Forward-looking statements involve known and unknown risks, uncertainties and other factors that are in some cases beyond the Company’s control that could cause the actual results, performance or achievements to differ materially from those expressed or implied by the forward-looking statement. No representation, warranty or assurance (express or implied) is given or made by Noxopharm that the forward-looking statements contained in this announcement are accurate and undue reliance should not be placed upon such statements.
Targeting glutamate-mediated excitotoxicity in the brain using NYX-104

Gary Housley, Ph.D.
Translational Neuroscience Facility & Dept. Physiology, School of Medical Sciences, UNSW Sydney

Industry Partner: Noxopharm Pty Ltd
Contributors

Translational Neuroscience Facility
UNSW Sydney
Scientia Prof. Gary Housley
Dr. Nagarajesh Gorlamandala
Ms. Jasneet Parmar
Dr. Chamini Perera
Dr. Georg von Jonqueries

Noxopharm
Dr. Benny Evison
Dr. Graham Kelly

Declaration: Gary Housley is a co-inventor on a patent filed by New South Innovations Pty Ltd (UNSW Sydney) on TRPC ion channels as a therapeutic target for brain injury, and a patent filed by Noxopharm for drug action of NYX-104 in neuroprotection from stroke and related excitotoxic brain injury; Gary Housley holds shares in Noxopharm P/L. Research was funded by a project grant from Noxopharm to UNSW Sydney with Gary Housley as principal investigator.

- Procedures for animal models were approved by the UNSW Sydney Animal Care and Ethics Committee and follow the NHMRC guidelines for use of animals for biomedical research.
Noxopharm and UNSW combine on stroke project

- Noxopharm drug identified as potential brain/spinal cord injury preventative
- First-in-class drug to hasten rehabilitation from brain and spinal cord injury
- Made possible because of NOX66 drug delivery platform
- UNSW assisting in pre-clinical studies.

Sydney, 16 March 2017: Noxopharm Ltd (ASX: NOX) today unveils the first of its non- oncology drug pipeline, having entered into a research agreement with the University of New South Wales (UNSW) to develop a unique and highly sought preventative treatment for damage to the central nervous system. This includes stroke and spinal injury, and potentially a wide range of neurodegenerative diseases.

The collaboration brings together key Australian intellectual properties owned by Noxopharm and UNSW that together form the basis for a potential treatment to prevent the debilitating after-effects of brain and spinal cord injury. The ultimate objective is a drug that would be delivered following acute brain or spinal cord injury to prevent further spread of that injury.

The aim of the proposed drug is not to treat the original injury (eg. trauma, stroke etc), which often is limited enough to self-repair, but to stop the cascade of death of nerve cells (known as excitotoxicity) that occurs in the brain and spinal cord after the initial injury and which typically leads to an area of cell death that is too large to be repaired. This ‘follow-on damage’ typically accounts for most of the loss of function following such injuries. Limiting this ‘follow-on damage’ is expected to make a significant difference to the recovery prospects of people following brain and spinal cord injury. There currently is no effective treatment of excitotoxicity.
Translational Neuroscience – identification of NYX-104 as a neuroprotective drug to reduce excitotoxic brain injury

- Identification of a novel Ca\(^{2+}\) permeable ion channel (TRPC3) activated by mGluR as a new drug target for reducing the expansion of the penumbra following stroke.

- *In vitro* assays: HEK293 cells (genetically-encoded Ca\(^{2+}\) imaging)

- *In vivo*: photothrombotic infarcts; intravital multiphoton imaging
Epidemiology of Stroke

- Annually, 15 million people worldwide suffer a stroke.
- Stroke is Australia’s second single greatest killer after coronary heart disease and a leading cause of disability.
- 65% of stroke survivors suffer a disability which impedes their ability to carry out daily living activities unassisted.
- Stroke kills more women than breast cancer and more men than prostate cancer.
- In 2017 Australia, there will be almost 56,000 new and recurrent strokes – that is one stroke every nine minutes.
- In 2017 Australia there will be more than 475,000 people living with the effects of stroke. This is predicted to increase to one million by 2050.
- Around 30% of stroke survivors are of working age [under the age of 65].
- The financial cost of stroke in Australia is estimated to be $5 billion each year.

Brain ischemia from stroke and other brain injuries and pathologies causes excess release of the excitatory neurotransmitter – Glutamate – which chronically activates neurons and drives spread of brain injury.
Current treatments for Stroke are directed to re-perfusion and are time-critical

- tPA - thrombolytic agents are the principal recourse for restoring perfusion (< 15% of thrombotic strokes; within 3 hours)
- Interventional radiology
TARGET BACKGROUND

Glutamate released in excess with brain ischaemia (Stroke)
- Activates mGluR (G protein-coupled glutamate receptor)
- Leads to activation of PLCB
- Leads to production of DAG and IP3
- DAG activates TRPC3 ion channels
  - sole effector of mGluR-mediated sustained depolarizing post-synaptic current in cerebellar Purkinje neurons (pn)
- Block of sustained neuron depolarization and Ca2+ entry via the TRPC ion channels is a therapeutic target.

We discovered that alternative splicing of the TRPC3 ion channels in the brain produces a higher Ca2+ entry into neurons than was previously known (Kim et al. 2012 J Neuroscience).

This led us to realise that targeting the neuronal TRPC3 channels was likely to be neuroprotective in stroke and other brain injury etiologies where glutamate excitotoxicity contributes to pathology.
Transient Receptor Potential Canonical Channel (TRPC) - activation by mGluR

Adapted from Fowler MA et.al, 2007

TPC3c isoform lacks a functional CIRB domain – resulting in sustained Ca^{2+} entry with elevated cytosolic Ca^{2+}
Localization of TRPC expression in cerebral cortex & cerebellum

*In situ* hybridisation

TRPC3 variant expression confirmed in the human brain

Dong, 2008
Activation of TRPC3 channels by the mGluR1 agonist DHPG causes sustained Ca\(^{2+}\) entry. The figure shows Fluo 4 microfluorometry Ca\(^{2+}\) imaging data using HEK293 cells co-expressing mGluR1 and TRPC3c channels. DHPG is initially in Ca\(^{2+}\)-free media to deplete IP\(_3\) -mediated Ca\(^{2+}\) store release. When Ca\(^{2+}\) is returned to the chamber, Ca\(^{2+}\) entry through the activated TRPC channels is prominent (red highlight). The TRPC3-mediated Ca\(^{2+}\) entry is blocked by genistein (200 µM, lower trace). - after Kim et al. 2012.
Drug Discovery program based on rHEK293-GCaMP5G Ca$^{2+}$ reporter cell line

**Gq activation using carbachol**

**Ca$^{2+}$ free perfusion**

**Block of Ca$^{2+}$ re-entry using PyR3 (10 µM) – a TRPC3 channel blocker**

FlexStation 3 multi-modal microplate reader - Molecular Devices

Disposable pipette tips minimize reagent cross-contamination between wells and experiments.
NYX-104 blocked Ca$^{2+}$ dynamics of Gq signalling in HEK293 cells

NYX-104 (4 µM)

Noxopharm unpubl. data
NYX-104 provided optimal block of Ca\(^{2+}\) dynamics of Gq signalling in HEK293 cells

Noxopharm unpubl. data
Photo-thrombotic stroke model

5 min illumination @ 561 nm

Yao et.al. 2003 Stroke

Rose bengal
4DPI WT mouse
Photothrombotic lesion

Gorlamandala - Housley et al. unpubl
Surface infarct comparison

\[ p = 0.002 \]

\[ p = 0.016 \]

Infarct surface area (mm\(^2\))

- 2 HPI carrier
- 5 DPI carrier
- 5 DPI NYX-104

Noxopharm unpubl. data
Lesion volume comparison

$p = 0.010$

$p < 0.001$

$p = 0.025$

Infarct lesion volume (mm$^3$)

2 HPI carrier

5 DPI carrier

5 DPI NYX-104

NYX-104

Noxopharm unpubl. data
CONCLUSIONS

• NYX-104 is very effective at blocking neuronal Ca\(^{2+}\) dynamics mediated by a dominant cell signalling pathway in neurons that is over-driven during brain injury associated with excessive release glutamate neurotransmitter. This arises in conditions such as stroke, epilepsy and brain trauma.

• NYX-104 can be delivered as a therapeutic treatment after brain injury via a Noxopharm – proprietary platform for systemic delivery, where it is effective in reducing the on-going glutamate excitotoxicity; the outcome in an experimental model of stroke is significantly reduced infarct size (neuroprotection).
References


NYX-205 program and peripheral neuropathy

Dr Benny J. Evison
Noxopharm Non-Oncology Program Director
Central nervous system (CNS) = brain and spinal cord

Peripheral nervous system = nerves outside CNS

From: www.study.com
Peripheral neuropathy

- Damage to these nerves = peripheral neuropathy
- Sensory symptoms:
  - tingling,
  - numbness,
  - burning sensations,
  - considerable pain

From: www.study.com
Peripheral neuropathy

• Associated with:
  • diabetes,
  • inflammatory bowel disease (IBD),
  • traumatic injury,
  • treatment with certain cancer medications
Chemotherapy induced peripheral neuropathy (CIPN)

- Acute or chronic
- Affects 30 – 70% of people receiving chemotherapy
- Can result in chemotherapy cessation
- No effective prophylactic or therapeutic treatment
- Nyrada Inc’s initial goal is to deliver an effective treatment

From: www.veteranslawblog.org
Chemotherapy induced peripheral neuropathy (CIPN)

Nerve cell = “neuron”

From: www.pixabay.com
Chemotherapy induced peripheral neuropathy (CIPN)

• Neurons are susceptible to cancer chemotherapies, e.g. paclitaxel
Chemotherapy induced peripheral neuropathy (CIPN)

- Neurons are susceptible to cancer chemotherapies, e.g. paclitaxel

From: www.pixabay.com
Chemotherapy induced peripheral neuropathy (CIPN)

- Neurons are susceptible to cancer chemotherapies, e.g. paclitaxel
Chemotherapy induced peripheral neuropathy (CIPN)

• Neuroinflammation
NYX-205 as a treatment for CIPN

- NYX-205 has shown promise as a therapy for inflammatory diseases
- But not neuroinflammatory diseases

From: www.pixabay.com
NYX-205 as a treatment for CIPN

- Why?
- Blood-nerve barrier
NYX-205 as a treatment for CIPN

- LIPROSE technology as a solution
- Lipid protective shield
  - Used by Noxopharm to deliver idronoxil into the brain
NYX-205 as a treatment for CIPN

- LIPROSE technology as a solution
NYX-205 as a treatment for CIPN

- LIPROSE technology as a solution
- Lipid = NYX-205
NYX-205 as a treatment for CIPN

- Lipid-NYX-205 traverses blood nerve barrier
NYX-205 as a treatment for CIPN

- Neuroinflammation subsides
Summary

• CIPN associated with neuroinflammation,
• No therapy exists,
• NYX-205 has established anti-inflammatory properties,
• LIPROSE will be applied to help NYX-205 traverse blood nerve barrier:
  • To enable a therapeutic effect
An eye on the future

• Other diseases are neuroinflammatory,
  • Alzheimer’s disease,
  • motor neuron disease,
  • multiple sclerosis, etc.
• Nyrada Inc is evaluating these indications as targets of NYX-205/LIPROSE
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Although the Company believes that the expectations reflected in the forward looking statements included in this presentation are reasonable, none of the Company, its Directors or officers can give, or gives, any assurance that the results, performance or achievements expressed or implied by the forward-looking statements contained in this document will actually occur or that the assumptions on which those statements are based are exhaustive or will prove to be correct beyond the date of its making. Readers are cautioned not to place undue reliance on these forward-looking statements. Except to the extent required by law, the Company has no intention to update or revise forward-looking statements, or to publish prospective financial information in the future, regardless of whether new information, future events or any other factors affect the information contained in this presentation.

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Cardio Therapeutics Pty Ltd
and the PCSK9 inhibitor drug program

Cardio Therapeutics is part of the Altnia Group and subject to acquisition by Nyrada Inc.
Forward Looking Statements

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The PCSK9 story outline

An important & valuable new treatment for atherosclerosis and cardiovascular disease

- Who
- What
- Why
- How
Our goal is to:

Have a tablet (oral) drug to target and inhibit PCSK9-LDL Receptor interaction to treat hypercholesterolemia (high LDL-cholesterol levels) and cardiovascular diseases by daily use.

- Statins to treat high LDL-cholesterol levels
  - In market in 1987

- Injectable PCSK9 Monoclonals to treat high LDL-cholesterol levels
  - In market 2015

- PCSK9 inhibitor oral tablet to treat high LDL-cholesterol
  - In development
Why is this important

The medical need is large

Hypercholesterolemia - also called high cholesterol:-

- cholesterol (a sterol) is essential for our body
- cholesterol is carried in our blood in lipoproteins (fat protein)
- lipoproteins are named after their density - very low density lipoprotein (VLDL), low density lipoprotein (LDL), intermediate density lipoprotein (IDL) and high density lipoprotein (HDL)
- HDL is “good” lipoprotein – the rest are “bad”
- high levels of LDL-cholesterol are linked to an increased risk of atherosclerosis and coronary heart disease
- the National Institutes of Health says that 34 million Americans have elevated “bad” blood cholesterol levels *

* https://ghr.nlm.nih.gov/condition/hypercholesterolemia#statistics
Why is this important

The medical need is large

Atherosclerosis (arteriosclerotic vascular disease) *

- Atherosclerosis is a disease in which plaque builds up inside your arteries
- Arteries are blood vessels that carry oxygen-rich blood to your heart and other parts of your body
- Cells in your body (brain, muscles etc.) rely upon oxygen to function
- Atherosclerosis limits the flow of oxygen-rich blood to cells that make your body work well
- Atherosclerosis starts off as debilitating and restricting activity
- More severe blockage can lead to serious problems, including heart attack (ischemic heart disease (IHD)), ischemic stroke, and death
- Ischemic heart disease remains the leading cause of premature adult mortality worldwide **

* https://www.nhlbi.nih.gov/health/health-topics/topics/atherosclerosis
Global, regional, and national age-sex specific all-cause and cause-specific mortality for 240 causes of death, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013
Why is this important

- The medical need is large

Atherosclerosis (arteriosclerotic vascular disease)

https://library.med.utah.edu/WebPath/ATHHTML/ATH006.html

www.nhlbi.nih.gov/health/health-topics/topics/atherosclerosis
Why is this important

- The medical need is large

Coronary heart disease – the most common cause of heart attacks

- Stable plaques cause angina and pain, anxiety and discomfort – and in more severe cases a loss of mobility and independence
- Unstable plaques can lead to a blood clot and heart attack or stroke
- Causes about one-third of all deaths in people older than 35 years *
- The 2016 Heart Disease and Stroke Statistics from the American Heart Association (AHA) has recently reported that 15.5 million persons ≥20 years of age in the USA have CHD **
- CHD affects around 1.2 million Australians, is the single leading cause of death in Australia, claimed the lives of 19,777 Australians (12% of all deaths) in 2015 and kills one Australian every 27 minutes ***

* https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4958723
Why is this important

- The medical need is large

Cardiovascular disease *

- kills one Australian every 12 minutes.
- affects one in six Australians or 4.2 million people
- was the main cause for 490,000 hospitalisations in 2014/15.
- claimed the lives of 45,392 Australians (nearly 30% of all deaths) in 2015 - deaths that are largely preventable

Why is this important

- The medical need is large

**Ischemic stroke** occurs when the blood supply to part of the brain is suddenly blocked

“In the U.S. it is estimated that 6.4 million adults have suffered a stroke and another 13 million may have experienced a “silent stroke,” loss of brain cells without visible symptoms.

In the U.S., the financial burdens associated with stroke treatment and recovery is estimated at $73 billion dollars in 2010”
Why is this important

- The present treatments to reduce LDL-cholesterol
  
  - Diet, smoking & lifestyle (exercise)
    - Some people have a genetic predisposition that diet etc. cannot resolve, others are unable to achieve control
  
  - Statins – Atorvastatin (Lipitor™), Fluvastatin, Lovastatin, Pravastatin, Rosuvastatin, Simvastatin
  
  - Biologic PCSK9 (Proprotein convertase subtilisin/kexin type 9) inhibitor drugs approved by FDA (USA) and EMA (Europe)*
    - evolocumab (Repatha™) (Amgen),
    - alirocumab (Praluent™) (Sanofi/Regeneron)

* Approved for use in patients with heterozygous and homozygous familial hypercholesterolemia and for patients intolerant of statins or those with a major risk of cardiovascular disease (CVD) but unable to lower their LDL cholesterol (LDL-C) to optimal levels with statins and ezetimibe www.ncbi.nlm.nih.gov/pubmed/27186592
Why is this important

It is a huge commercial opportunity

- **Statins**
  - were originally discovered from fungi
  - are now off patent (generics) and cheap
  - reached a peak of US$19 billion sales p.a. (2005)
  - have safety and toxicity problems *“a sizable proportion of statin-treated patients does not achieve recommended target LDL cholesterol levels, and some discontinue treatment owing to drug-related side effects”*
  - work as HMG-CoA reductase inhibitors **

- **Biologic PCSK9 drugs are**:
  - new (approved in 2015 & 2016) but expensive (~ US$14,000 p.a.) ***
  - require injection every 2 weeks
  - have demonstrated excellent safety and efficacy data since approvals in USA and EU
  - work by inhibiting PCSK9 binding to LDL Receptor

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What we are doing

How new drugs are brought forward

❖ The old approach
  ▪ many old drugs came from medicines derived from natural sources (hit-n-miss medicine over many years) e.g. penicillin from mould (antibiotic) and quinine from bark of the cinchona tree (anti malaria)
  ▪ then there is High Throughput Screening (HTS) - laboratory-based screening libraries (thousands to millions) of compounds tested against disease-specific tests (“assays”) against a “target” and looking for “hits” that could become a drug to treat a disease (also hit-n-miss)

❖ The newer approach
  ▪ based upon the dramatic reduction in cost & time in genomic sequencing (US$2.7 billion and 15 years (starting 1990) vs. ~ US$1,000 and weeks (now)
  ▪ looking at the differences between patient-groups and their genomics
  ▪ the idea is to identify genes (and gene products) – “targets” - that were critical to disease
  ▪ and then develop drugs that would act on those targets using structure based design (SBD)
  ▪ not so hit-n-miss – so risks are reduced

Our PCSK9 program comes from this more modern genomic and SBD approach

https://www.cnbc.com/2015/12/10/unlocking-my-genome-was-it-worth-it.html
Genomics and hypercholesterolemia

2003 – enter PCSK9 (Proprotein convertase subtilisin/kexin type 9)

- the PCSK9 gene is associated with increased risk of coronary artery disease *
- the PCSK9 gene produces a protein (PCSK9) that is produced by liver cells and enters the blood to help regulate LDL cholesterol levels
- a gain-of-function mutation in PCSK9 causes genetic (familial) hypercholesterolemia *
- humans with an absence of PCSK9 (or loss of function mutations) are healthy AND have significantly reduced LDL cholesterol levels AND a “strikingly reduced frequency of reduced coronary heart disease” *

Our PCSK9i program comes from understanding the importance of PCSK9 in this disease and the expected safety profile for a PCSK9i drug

* https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2386899/
1. With PCSK9 bound to the LDL Receptor, there are less LDL Receptors to traffic LDL cholesterol out of the blood and into the cell – because more of the LDL Receptors are trafficked to lysosomes for degradation 2.

3. Without PCSK9, more of the LDL Receptors traffic back to the cell membrane 4. ready to shuttle more LDL cholesterol out of the blood and into the cell and then to lysosomes for degradation 5.

6. Even if PCSK9 is present, a drug can inhibit PCSK9 binding to LDL Receptor so that more LDL Receptors shuttle more LDL cholesterol out of the blood and into the cell 3.

Adapted from [https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2386899/figure/fig1](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2386899/figure/fig1)
What we are doing

Next-generation inhibition of PCSK9

- Genomics and the success of the existing biologic PCSK9 inhibitors tells us that PCSK9 is a good “target” i.e. something to inhibit to treat atherosclerosis and vascular disease.
- Oral drugs are much more acceptable to patients and cost-effective for patients and payers.
- Some experts think that small molecules (i.e. those that are orally available as a tablet taken daily) cannot effectively inhibit the PCSK9-LDL-Receptor interaction.

* Nature Reviews Drug Discovery Published online 28 Apr 2017; doi:10.1038/nrd.2017.83
What we are doing

- Next-generation inhibition of PCSK9

Cardio Therapeutics has shown that a small molecule from structure based design (e.g. ALT-30) can indeed effectively inhibit the PCSK9-LDL-Receptor interaction in *in vitro* assays and in-silico screening.

Model of PCSK9 (some residues omitted for clarity) with surface plot (charge)

Groove filled with a computational prototype compound (shown in yellow)

- It is possible to use a small molecule to inhibit PCSK9 – LDL-R binding

Surface for LDL-R interaction

Courtesy H. Treutlein, J. Zeng, Computist Bio-Nanotech
Who we are

Dr Ian Dixon
Director and Founder of Altnia Group

- Co-inventor of Cardio Therapeutics Pty Ltd PCSK9 technology
- PhD in Biomedical engineering from Monash University
- MBA and Engineering experience
- Director of Noxopharm (ASX-NOX) a listed clinical-stage anticancer company
- Founder of Cynata (ASX-CYP) – a clinical-stage stem cell company now worth ~$70m and partnered with Fuji Film regenerative medicine company
- Co-inventor of ATM-3507 anticancer drug
- Founder and CEO of Exopharm Pty Ltd (www.exopharm.com)
Other members of the team

<table>
<thead>
<tr>
<th>Person</th>
<th>Key roles</th>
<th>Relationship or affiliation</th>
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<tbody>
<tr>
<td>Dr Jim Palmer</td>
<td>Medicinal chemistry</td>
<td>Consultant</td>
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<td>Dr Herbert Treutlein *</td>
<td>Structure-based and fragment-based drug design tools based on their MFMD technology</td>
<td>Sanoosa <a href="http://www.sanoosa.com">www.sanoosa.com</a></td>
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<tr>
<td>Dr Jun Zeng *</td>
<td>Structure-based and fragment-based drug design tools based on their MFMD technology</td>
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<td>Dr Ian James</td>
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<td>Chempartner (Shanghai, China)</td>
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<td>Jubilant Biosys (Uttar Pradesh, India)</td>
<td>Drug synthesis</td>
<td>Contractor</td>
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* Also as co-founders of Okedro Pty Ltd and Cardio Therapeutics Pty Ltd and experts in structure-based drug discovery
How - The pathway to our PCSK9i as a drug

Further optimisation
Preclinical testing
Clinical trials
Registration
Sales

Further rounds of synthesis and testing
- Manufacture
- GMP-grade drug
- Phase I

Animal testing of compounds for efficacy and safety
- Extensive preclinical safety and efficacy testing in two species
- Phase IIa

Oral availability of lead compound
- Preparation for Phase I clinical trials
- Phase IIb

Preclinical candidate selection
- Approval to run clinical trial
- Phase III

Preclinical candidate selection
- Approval to run clinical trial
- Phase III
The PCSK9 story outline

[Diagram showing the outline with questions 'Who', 'What', 'Why', 'How']

An important & valuable new treatment for atherosclerosis and cardiovascular disease
Thank you for your attention