Nyrada (ASX:NYR) – The Best Risk-Reward Biotech on the ASX

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Investing in biotech companies is inherently risky because the outcome is usually binary - either a drug will be efficacious [work] and the investment will be outstanding, or it will not, and a large loss will be incurred. When biotech investments go well, the outcome can be life-changing, not only for the patients who receive new, more effective treatments for diseases and conditions, but also for the investors who back these companies, with the potential for returns of many, many multiples. Neuren (ASX: NEU) $0.30 to $25.00, and Telix (ASX: TLX) $0.60 to $29.00, are two recent good examples of what can happen when things go well. Opthea (ASX: OPT) $3.60 to $0.60 is an example of what happens when things go wrong, with the company now in long-term suspension.

In this update, Canary Capital explains why we believe Nyrada Inc (ASX: NYR) offers the most compelling risk/reward profile for a biotech investment on the ASX. Nyrada is developing Xolatryp, a novel small-molecule drug designed to inhibit TRPC ion channels - specifically TRPC 3, 6, and 7. By blocking these channels, Xolatryp prevents excessive calcium influx into heart, brain, and kidney cells following the initial injury. Unchecked calcium overload leads to toxicity, cell death, and long-term organ damage, with serious health consequences for patients.

Xolatryp – A Potential New Frontier in Cardioprotection

In an announcement released on 9th June 2023, Nyrada stated that it had selected a new drug candidate, NYR-BI03 (now called Xolatryp) due to superior potency and safety profile, overcoming limitations identified with the previous candidate, NYR-BI02[1].

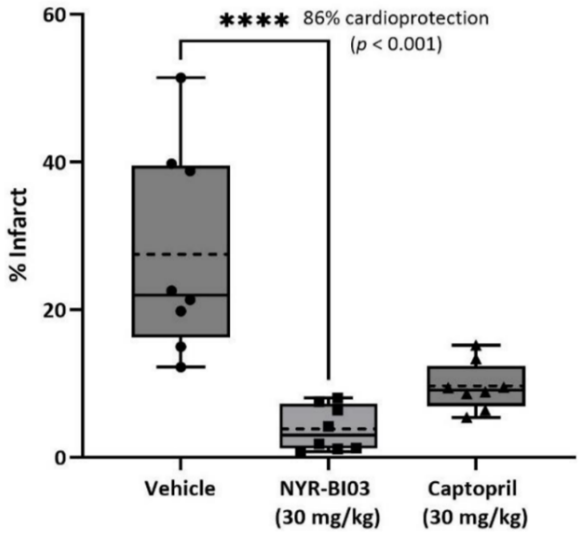
"Following a review of over 200 available compounds generated by the Company's medicinal chemistry program, NYR-BI03, a closely related analogue of NYR-BI02, was identified as having a superior safety profile along with better potency on the TRPC ion channel protein target, securing its selection as Nyrada's new lead brain injury drug candidate."

Heart attacks affect 15-20 million people globally each year, with up to 15% mortality within 30 days due to complications like heart failure and significant risks of mortality during the following 12 months from cardiac arrest caused by arrhythmias (irregular beating of the heart). By some assessments, 50-70% of heart tissue damage is caused by reperfusion injury, where restoring blood flow after a heart attack causes further significant damage to the heart.

Cardiovascular diseases (CVDs) are the leading cause of death globally, taking an estimated 17.9 million lives each year[2]. The global market for myocardial infarction (heart attack) therapies is expected to reach US$3.7 billion by 2032[3].

On 1st October 2024, Nyrada announced that Xolatryp demonstrated significant cardioprotection[4]. In a preclinical rat model, the drug limited cardiovascular damage associated with myocardial ischemia-reperfusion injury following clearance of the blockage in the artery. Xolatryp (30 mg/kg) demonstrated strong and statistically significant cardioprotection, reducing cardiac tissue damage by 86 per cent (see diagram 1 NYR-BI03 versus Vehicle) (*p* value* < 0.001, n=8 per group)[5] following a 24-hour infusion of the drug. Captopril, a drug that reduces blood pressure, was used as a positive control and given to animals prior to myocardial infarction, but it is not a drug that is used in the emergency setting.

Diagram 1: Injury Protection Following Ischemic-Reperfusion



Source: Nyrada Inc

On 23rd October 2024, Nyrada announced supplementary data from the rat study providing further confirmation of Xolatryp's efficacy in treating ischemic-reperfusion injury associated with heart attack[6]. Echocardiography, which is used to measure the performance of the heart, showed significant improvements in heart function.

Animals treated with Xolatryp had a substantial 43% increase in left ventricular ejection fraction (p <0.0001)[5], a key indicator of the ability of the heart to pump, significantly improving overall cardiac function.

The same animals also showed a 50% increase in fractional shortening (p = 0.0002)[5], indicating that Xolatryp preserved the heart's ability to contract, indicating prevention of damage to the left ventricle.

*A **p-value** (short for *probability value*) is a number used in statistics to help decide whether the results of a study or experiment are likely due to chance.

Left ventricular dimensions were reduced by 13% during diastole (p = 0.0072)[5] and 22% during systole (p = 0.0006)[5], highlighting Xolatryp's role in preventing harmful stretching of the heart muscle. Animals treated with Xolatryp also had an increase in the left ventricular posterior wall thickness by 25% (p = 0.0346)[5], reinforcing the structural integrity of the heart and potentially improving resilience against further injury. The levels of three key blood biomarkers that elevate in response to ischemic reperfusion injury to the heart were assessed. Xolatryp reduced the levels of Aspartate Aminotransferase (AST - which is an enzyme found in the blood following injury) by 42% ($p < 0.05$)[7], Lactate Dehydrogenase (LDH – an enzyme which is a general marker of cell or tissue damage) by 45% (p = 0.0285)[8] and Troponin I (a cardiac-specific protein injury marker that is released into the blood following injury following a heart attack) by 32% (no p value due to low sample size).

The images below show sections of rat heart tissue from sham (normal), injury plus vehicle (damaged), Xolatryp treated (protected), and injury plus Captopril (existing standard of care therapy) for visual comparison. Red-stained areas of the heart tissue indicate healthy, uninjured tissue, whilst grey-stained areas represent dead heart tissue. The images visually highlight that outcomes for Xolatryp (NYR-BI03)-treated animals were far superior to control and Captopril-treated animals. (Note- In Sham treatments, the doctor goes through the motions without actually performing the treatment)

Rat Heart Tissue Samples Images

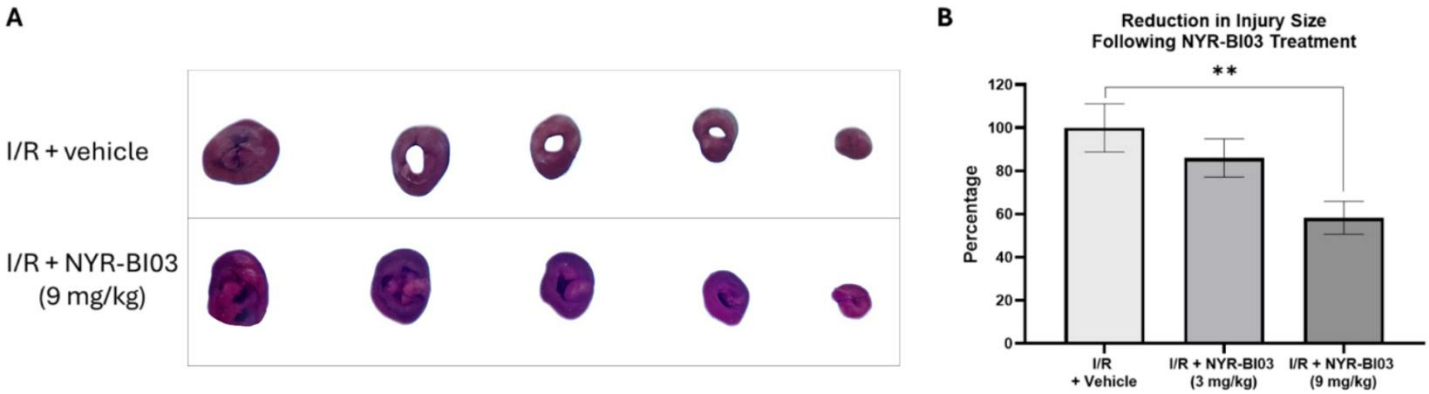
Source: Nyrada Inc

On the 8th of May 2025, Nyrada announced that cardioprotection had been extended to arrhythmia control (irregular heartbeats) in another preclinical study[9]. This additional preclinical study demonstrated Xolatryp provided a strong level of cardioprotection when administered as a short-duration (3-hour) intravenous infusion following myocardial infarction.

Utilising the same rodent model from Nyrada's October 2024 study but conducted by a different contract research organisation (CRO), rodents were administered Xolatryp at doses of 3.0 and 9.0 mg/kg over 3 hours following acute myocardial ischemia (AMI).

This trial confirmed Xolatryp provided cardioprotection with a short-duration treatment with a significant reduction in both heart muscle injury size and Troponin I, which is a clinically relevant biomarker that is released into the blood following heart damage. High Troponin I levels are strongly associated with larger injury size, impaired cardiac function and a higher risk of mortality. The size of injury was determined via a technique called TTC staining, where viable tissue is stained 'red' and metabolically 'dead' tissue remains unstained (A). A dose-dependent reduction (the higher the dose, the greater the reduction in injury size) was observed following 3 hours of Xolatryp continuous intravenous infusion, which was statistically significant at the 9 mg/kg dose compared with vehicle (42% reduction; p = 0.008, one-way ANOVA, Bonferroni t-test post-hoc, n = 10; B) (see diagram below).

Rat Heart Tissue Samples Images and Results Over 3 Hour Dosing Period*



* Note: Vehicle = Placebo

Source: Nyrada Inc

Significant Reduction in Injury Biomarkers

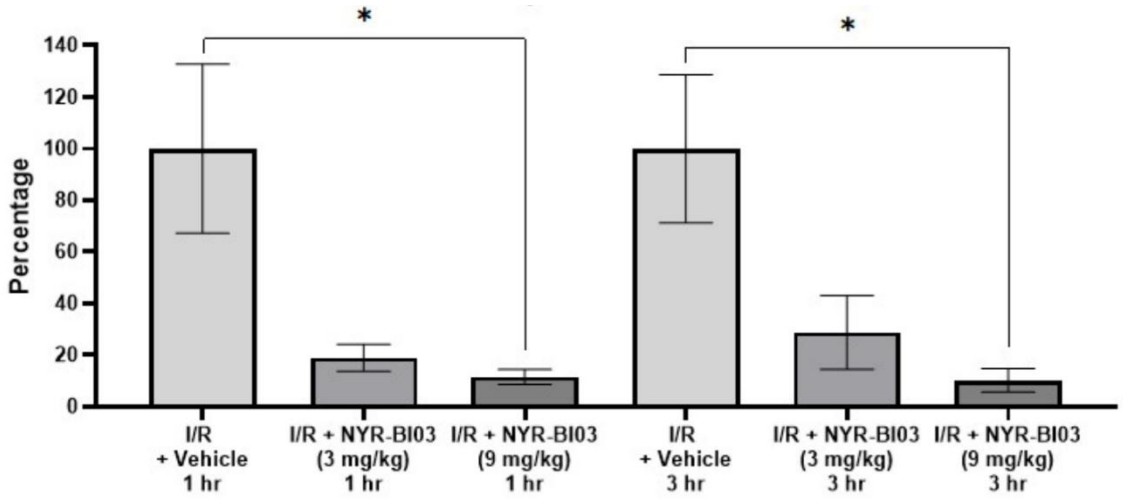
A dose-dependent reduction was observed in plasma Troponin I levels, which were statistically significant at 9 mg/kg dose (32% reduction; p = 0.014[5 7], n = 10).

Following a myocardial infarction (heart attack), there is a significant reduction in cardiac output and the ensuing blood flow to the organs. The biomarker alanine aminotransferase (ALT) elevates due to liver damage resulting from low blood supply following heart attack. Xolatryp reduced ALT levels in a dose-dependent manner, with a significant reduction observed at the 9 mg/kg dose (21% reduction; p = 0.0202, Student's t-test, n = 10).

Xolatryp Provides Arrhythmia Protection

As part of this study, cardiac arrhythmias (irregular heartbeats) were monitored via electrocardiogram (ECG). Arrhythmias are a common cause of further cardiac arrest events and mortality in the 12 months following a heart attack. Animals treated with Xolatryp showed statistically significant superior outcomes. In this model, significant arrhythmia events were observed at 1 hour and 3 hours post-injury. A dose-dependent reduction was observed in ventricular premature beats (VPB), which, following ischemia, trigger ventricular tachycardia (VT) and ventricular fibrillation (VF), the leading causes of sudden cardiac death in the 12 months following a heart attack. A statistically significant reduction in VPB events was noted at the 9 mg/kg dose (88% reduction; $p = 0.04$[5 7] at 1 hour and 90% reduction; $p = 0.010$[5 7] at 3 hours versus control, n = 9 – 10).

Reduction in Ventricular Premature Beats Following NYR-BI03 Treatment



* Note: Vehicle = Placebo

Source: Nyrada Inc

This study built upon Nyrada's earlier successful cardioprotection studies reported in October 2024[4 6]. Significantly, these two trials in October 2024 and May 2025 were conducted by two different Contract Research Organisations (CRO's), both looking at infarct size and injury blood biomarkers like Troponin I. These studies have confirmed the replicability in animal models of AMI, with both confirming Xolatryp protects heart cells from damage resulting from pathological calcium ion influx following ischemia reperfusion (unblocking of the artery) in the heart.

It is important to note that TRPC channels expressed in mice and rats are also expressed in humans. This will be covered in the Scientific Risks section of this report.

Phase I Safety Trial Now Complete

Nyrada announced the completion of its Phase I safety study in humans on 6th August 2025[10]. The trial's Safety Review Committee reviewed safety and pharmacokinetic data (how a drug is absorbed, moves through, is broken down, and exits the body) from all six cohorts, and no dose-limiting adverse effects (side effects of a drug or other treatment that are serious enough to prevent an increase in dose or level of that treatment) were observed in trial participants with Xolatryp deemed safe and well tolerated. A final report on the Phase I trial is due to be released in September 2025.

Phase IIa Trial Into Cardioprotection to Commence in Q1, 2026

An Announcement to the ASX on 23rd of July 2025, titled "Nyrada Advances Xolatryp Towards Phase IIa Trial in Cardioprotection", confirmed that the company is now preparing to commence a Phase IIa trial in patients who have had a myocardial infarction[11]. The indicative study design incorporates 150 patients in a randomised placebo-controlled, double-blind clinical trial in Australia, testing two different doses of Xolatryp administered as a 6-hour infusion. Key endpoints will be confirming safety as well as cardioprotection following ischemic reperfusion, which is where oxygen-rich blood begins to flow again after the blockage in the artery is removed.

In response to an injury (ischaemic phase = oxygen and energy deprivation), stress hormones such as adrenaline and angiotensin II are generated, and these activate (open) TRPC channels, allowing calcium influx into cells. During the reperfusion phase, many different Reactive Oxygen Species (ROS) are produced and diffuse into heart cells, causing the TRPC channels to be upregulated (with more on the surface of each cell) and activated in the open position, allowing high levels of calcium ions to flood into cells, ultimately leading to toxicity and cell death. The calcium ions flooding into the cells also cause more ROS to be created, leading to uncontrolled toxicity. By temporarily inhibiting the TRPC channels using Xolatryp, this toxic process is avoided, and critical organ tissue is preserved, reducing the infarct or injury size.

The trial is also likely to examine the potential of the drug to significantly reduce arrhythmias. Another endpoint is likely to be cardio function, measuring the volume of blood being pumped by the heart, including left ventricular ejection fraction and fractional shortening (measures of how well the heart is pumping). Key enzyme levels in the bloodstream, including Troponin, may also be measured.

Key Risks and Why Canary Capital Believes They Have Been Mitigated

Xolatryp also has a low nanomolar potency, meaning that it only takes a low dose to inhibit TRPC channels. The drug has a fast onset to therapeutic levels and a short half-life, meaning once dosing is finished, the drug [and its effects] leave the body relatively quickly (within around 6 hours). Because the drug is infused, it is ideal for use in emergency departments or critical care ambulances.

Scientific Risks

The development of a TRPC knockout mouse occurred in 2013[12]. These mice were genetically bred without the expression of these channels, hence the name "knockout mice". The mice were viable, had normal lifespans, and were fertile. These mice enabled researchers to study the role of TRPC channels in disease and injury and gave confidence to pursue research into potential treatments in humans.

Since the creation of these TRPC knockout mice, multiple animal models in rodents have confirmed cardioprotection and neuroprotection *in vivo* (in a living animal). In a study titled "Major contribution of the 3/6/7 class of TRPC channels to myocardial ischemia/reperfusion and cellular hypoxia/reoxygenation injuries" researchers wanted to find out how these calcium ions get into myocytes, also known as heart cells[13]. TRPC channels 3, 6 and 7 are a group of protein "gates" located on the surface of cells that regulate the flow of positively charged ions such as calcium and sodium into cells.

In laboratory experiments, they stressed heart-like cells by providing low oxygen levels and then restored oxygen. In mice, they blocked blood flow to the heart for 30 minutes, then restored it, mimicking a heart attack. Some mice lacked TRPC 3, 6 and 7 channels ("knockout mice"), so researchers could see what happened without the TRPC channels.

In normal mice with TRPC 3, 6 and 7 channels, harmful amounts of calcium entered the heart cells during stress. This calcium overload damaged the mitochondria (the cell's "power plants"), triggering inflammation, which ultimately led to cell death.

Mice without the TRPC 3, 6 and 7 had significantly smaller heart injuries, healthier heart tissue, and better heart pumping capacity after the simulated heart attack. Blocking these gates, either with drugs or genetically, helped to protect the heart.

The conclusion was that drugs that block TRPC channels represent promising targets for reducing heart damage following a heart attack or ischemic reperfusion surgery to remove the blockage.

Boehringer Ingelheim Trial in Human Cells Grown in Laboratory

Boehringer Ingelheim, which is a large private pharmaceutical company based in Germany with 54,000 employees in 78 countries, completed a Phase II trial with its drug BI 764198 into Focal Segmental Glomerulosclerosis (FSGS, a kidney disease) on 1st March 2025. They are due to report results at any time. If the trial is successful, this will be the first Phase II trial to demonstrate efficacy in disease using a TRPC6 channel inhibitor in humans. Canary Capital recommends monitoring the company's website for the release of the results from Boehringer's phase II trial[14].

As stated earlier, it is important to note that TRPC channels expressed in mice and rats are also expressed in humans. As is described below, research conducted by Boehringer Ingelheim has confirmed the translation of TRPC6 inhibition from knockout rodents to human cells.

In a Boehringer Ingelheim abstract presented at Kidney Week 2024, results from a study on primary human podocyte (kidney) cells and glomerular endothelial cells (GECs) cultured in high glucose and exposed to angiotensin II, with or without Boehringer Ingelheim's TRPC6 inhibitor BI 764198, were presented[15].

Kidneys filter waste from the blood using a very fine barrier made up of special cells called podocytes. If podocytes are damaged, the filter leaks, and proteins spill into the urine – a key sign of kidney disease.

A problem protein ion channel, called TRPC6, lets too much calcium ion into podocytes, which makes them lose their structure and function. This damage weakens the kidney filter. Researchers tested a new drug called BI 764198, which blocks TRPC6, to see if it could protect podocytes.

The researchers grew human kidney cells in the lab and stressed them with high sugar and a hormone (angiotensin II) that normally harms podocytes. They measured how much calcium entered the cells, and how well the kidney filter worked in a laboratory model of a "kidney-on-a-chip." They used advanced genetic and protein tests to see how cells responded.

The researchers found that the podocytes (but not other kidney cells) had more TRPC6 activity under stress. The new drug stopped the harmful calcium overload in these cells. It also kept the filter from leaking proteins in the "kidney-on-a-chip."

This study showed the drug helped prevent podocyte injury in human cells in vitro (in the laboratory). This study was important because it provided strong evidence that BI 764198 could be a promising treatment to protect human kidney cells, which filter blood and slow down kidney disease by reducing podocyte cell death before it causes serious leakage and loss of kidney function.

Why is the Boehringer Laboratory trial Relevant to Nyrada?

Following a myocardial infarction, particularly during the ischaemic phase - when tissue oxygenation and energy supply are compromised - stress hormones such as epinephrine (adrenaline) and angiotensin II are released. These hormones activate TRPC channels, facilitating the influx of calcium into cells.

Upon reperfusion, as blood flow and oxygenation are restored, reactive oxygen species (ROS) are generated within cardiac myocytes (heart cells). ROS subsequently induce the upregulation and activation of TRPC channels, further enhancing calcium entry into cells. Excessive intracellular calcium leads to toxicity and cell death, while simultaneously promoting additional ROS production - a cycle that amplifies myocardial (heart) injury. Inhibition of TRPC channels with Xolatryp can disrupt this pathological cascade, thereby preserving organ function and minimising tissue damage and reducing infarct size.

Given Nyrada's Xolatryp drug knocks out TRPC channels 3, 6 and 7, the effect of the drug is likely to be broader in terms of potential diseases and conditions it could treat.

Safety Risks

Nyrada has completed a full battery of preclinical safety, dose escalation and toxicology studies[16]. The company recently completed a Phase I safety study in humans, which confirmed the drug is safe and well-tolerated with no dose-limiting adverse effects[10].

Regulatory Risks

Xolatryp is a first-in-class small molecule drug developed to treat life-threatening conditions such as heart attacks, stroke and traumatic brain injury. The US Food and Drug Administration (FDA) tends to look favourably upon new classes of drugs for unmet needs (a condition where there is no treatment or medicine currently available), particularly for life-threatening or terminal conditions. Where no existing treatments are available, such as is the case for cardioprotection, there is no need to show that it performs better than existing alternatives.

The bar for FDA approval also tends to be set lower for new drugs addressing critical unmet needs. For example, a 5% reduction in infarct (injury) size is expected to deliver a 20% reduction in the risk of sudden cardiac death or hospitalisation due to heart failure in the 12 months following a myocardial infarction[17]. Furthermore, if an infarct is in the upper quartile in terms of size, this translates to a 7 times greater risk of sudden cardiac death or hospitalisation due to heart failure in the 12 months following a myocardial infarction. Because a relatively small reduction in infarct size can translate into significantly improved health outcomes for patients, the scope to secure regulatory approval is high. There are also mechanisms to secure accelerated approval from the FDA, to shorten the pathway to commercialisation.

Trial Execution Risks

Australia is a favoured jurisdiction for conducting early-stage clinical trials, with many CRO's capable of running the Phase IIa trial for Nyrada. The company is in the process of identifying and evaluating several CRO's before a choice will be made for the upcoming trial.

Nyrada CEO, James Bonnar, is a qualified chemist, and he has designed and executed phase I and II trials under the guidelines of the FDA. Prior to joining Nyrada, James worked for Neuren (ASX: NEU) and designed late-stage trials for their drug called Daybue (trofinetide). Daybue has now been commercialised as a treatment for Rett Syndrome. Dr Alexandra Suchowerska, who has a PhD in neuroscience, successfully ran the Phase I safety trial for Nyrada and will be responsible for overseeing the Phase IIa cardioprotection trial for the company.

Commercialisation Risks

While Nyrada may be small by global standards, its board and management team bring heavyweight expertise, with a proven track record in running clinical trials, successfully commercialising new drugs, and securing major licensing deals with leading multinational pharmaceutical companies.

Nyrada CEO, James Bonnar, played a key role in developing Neuren's Daybue drug for Rett Syndrome. Chairman John Moore co-founded Trialogics, a global clinical trial technology firm, bringing deep insight into best practices worldwide.

Where Nyrada truly stands out is in licensing - the critical step once a drug shows efficacy. Board member Ruediger Weseloh, PhD, has closed nearly 100 commercial deals at Merck KGaA. Chris Cox is an experienced Wall Street M&A lawyer who has successfully overseen US$200 billion of transactions. He was Executive Vice President and Chief Corporate Development Officer of The Medicine Company, and in his new role at Population Health Partners, he works with Sir Clive Meanwell. He led the licensing of Inclisiran to Novartis for US$9.7B. Marcus Frampton, CIO of the Alaska Permanent Fund, backed Juno Therapeutics' record biotech seed round before its US$9B sale to Celgene.

Nyrada's leadership combines proven trial execution with world-class commercial dealmaking - a rare mix that positions the company to generate substantial shareholder value.

Competition Risks

Nyrada has developed a strategy around novel TRPC channel therapies, which means there is potential for further growth in the future by targeting additional indications or health issues. The completed Phase I trial for Xolatryp supports 3 indications or development pathways so far: Cardioprotection, stroke, and traumatic brain injury. Subject to preclinical studies, additional indications could be pursued. Xolatryp inhibits TRPC channels 3, 6 and 7, whereas Boehringer Ingelheim's BI 764198 drug only inhibits the TRPC 6 channel. For this reason, Xolatryp is likely to convey broader action in treating disease or injury.

Faraday Pharmaceuticals is currently conducting a Phase III trial into cardioprotection using its drug candidate FDY-5301. The drug has been designed to reduce the injury from ischemic reperfusion by targeting and destroying the ROS, hydrogen peroxide. This is a downstream approach compared to how Xolatryp works. Many ROS types can cause damage following ischemic reperfusion, and Faraday's drug targets the elimination of only one. In contrast, Xolatryp's mechanism of action focuses on inhibiting the TRPC channels, stopping the overloading of all ROS and calcium into heart cells, thereby preventing toxicity and stopping cell death from reperfusion. Therefore, Xolatryp works upstream of Faraday Pharmaceutical's FDY-5301, so the drug is likely to provide superior heart protection with stronger efficacy compared to Faraday.

Nyrada has applied for an international composition of matter patent over its Xolatryp molecule. This type of patent offers the broadest level of protection for the company. An international patent search has found that the drug is novel and inventive and has utility (applicability). The conclusion from this work on protecting the IP is that the molecule is patentable. International patent applications have been lodged and reviewed, with the current status being awaiting grant. Upon granting, Nyrada will be able to defend against any infringement where an entity tries to develop a similar molecule.

Financial Risks

The company is exposed to some foreign exchange risk due to the drug for the Phase IIa trial being manufactured outside Australia. There is also some risk of costs in relation to the trial being higher than anticipated, although the company is in advanced discussions with several CRO's and the company is confident that the funds currently available will be sufficient to complete the planned Phase IIa trial into cardioprotection.

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[8] t-test between injury + vehicle and injury + NYR-BI03

[9] Nyrada Announcement 8th May 2025: NYRBI03 Extends Cardioprotection to Arrhythmia Control

[10] Nyrada Announcement 6th August 2025: Positive Final Review of Nyrada Phase I Clinical Trial

[11] Nyrada Announcement 23rd July 2025: Nyrada Advances Xolatryp™ Towards Phase IIa Trial in Cardioprotection

[12] From GTP and G proteins to TRPC channels: a personal account

[13] Major contribution of the 3/6/7 class of TRPC channels to myocardial ischemia/reperfusion and cellular hypoxia/reoxygenation injuries

[14] A Study to Test BI 764198 in People With a Type of Kidney Disease Called Focal Segmental Glomerulosclerosis

[15] Kidney Week 2024 – Boehringer Ingelheim Abstract, Targeting Podocyte Calcium Influx with TRPC6 Inhibitor BI 764198: Implications for Glomerular Filtration Barrier Protection

[16] Nyrada Announcement 16th October 2024: Nyrada Successfully Completes Safety Studies Ahead of Phase I Clinical Trial

[17] Relationship Between Infarct Size and Outcomes Following Primary PCI: Patient-Level Analysis From 10 Randomised Trials

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