NOVOGEN

ASX: **NRT**
NASDAQ: **NVGN**

NOVOGEN ROADSHOW PRESENTATION

September 2014

Accompanying Notes

Slide 3 THE COMPANY

Novogen is an Australian/US biopharmaceutical company. Listed on both the Australian Securities Exchange (*NRT*) and NASDAQ (*NVGN*).

Headquarters - Sydney, Australia
US office - New Haven, CT

History

1994	Founded (by Graham Kelly); IPO/listed ASX Principal activity development of pharmaceutics (oncology, cardiovascular, inflammatory) based on benzopyran chemistry.
1998-2008	4x oncology drugs developed; 1x (phenoxodiol; IDRINOXIL) taken to pivotal Phase 3 multi-national trial in late-stage ovarian cancer.
2001	Form wholly-owned US subsidiary, Marshall Edwards Inc, (NASDAQ listing: *MEI*) to focus on oncology drug development; Novogen remains focused on cardiovascular/inflammatory diseases.
2006	G Kelly leaves Novogen.
2008	Phenoxodiol Phase 3 trial fails.
2011	Decision to focus on oncology; MEI renamed MEIPharma (*MEIP*); all IP transferred to MEIPharma; Novogen prepared for back-door listing.
2011	Kelly, Brown and Heaton establish Triaxial Pharmaceuticals with objective of developing super-benzopyrans (SBPs).
2012	Triaxial achieves breakthrough in SBP design and manufacture; Yale confirms high potency of SBPs against ovarian cancer stem cells.
2012	Novogen acquires Triaxial Pharmaceuticals in reverse take-over and demerges with MEIPharma.

Management:

Dr Graham Kelly	Executive Chairman and CEO, Novogen Group
Dr David Brown	Chief Scientific Officer, Novogen Group
Dr Andrew Heaton	Vice-President, Drug Discovery and Manufacture
Dr Justine Stehn	Director, Anti-Tropomyosin Program
Dr Stephen Palmer	Director, Degenerative Diseases Program
Ms Christine Bruce	Chief Financial Controller
Mr Lionel Mateo	Company Secretary

Key Metrics

168M ordinary shares and 4M options outstanding.

4000 (approx.) ASX shareholders
2300 (approx.) NASDAQ shareholders

1 ADR = 25 Ordinary ASX shares

Slide 4 Corporate Goal

Novogen is in the unique position of having two first-in-class anti-cancer drug technology platforms, each of which is capable of 'blockbuster' status, but which together, we believe, have the potential to change in a very substantial way the whole approach to cancer therapy.

Our goal is to see both drug platforms create a suite of drugs that will become standard 'go to' first-line therapies for most forms of cancer (in the way that the taxanes and platinums have) and be relied upon to prevent the development of recurrent disease with its high level of chemo-resistance and poor survival prospects.

This confidence and belief is based on three key factors:

1. The ability of the first technology (the super-benzopyrans) to kill the full hierarchy of cells within tumors, including both the tumor-initiating cells (cancer stem cells) and their daughter cells. **The first family of drugs capable of doing so.**

2. The ability of the second technology to destroy one of the two key components of the cytoskeleton of the cancer cell and to act synergistically with standard drugs (taxanes, vinca alkaloids) that target the other key cytoskeleton component in comprehensively destroying the cancer cell's skeletal structure and ability to survive.

3. The ability of both drug technologies to achieve these outcomes through high on-target activities and hence with minimal toxicities and no increased susceptibilities to infectious diseases, an emerging problem with chemotherapy.

Slide 5 Vision

Novogen has a 10-year growth plan to evolve into a major global bio-pharmaceutical player based on the extraordinary potential of its two first-in-class drug technologies that are targeting entirely new targets in cells associated with degenerative diseases including cancer and neuro-degenerative and musculo-degenerative diseases, and in the field of regenerative medicine where it has a unique opportunity to promote the activity of inherent normal neural stem cells in the repair of brain, spinal cord and peripheral nerves.

Slide 6 The Technologies

The Company has two core drug technology platforms, each first-in-class.

Super-benzopyrans (SBPs)

The heritage of SBPs is a family of plant hormones with multiple functions in plants due both to regulation of specific biochemical effects and to gene regulation. 25% of human DNA is shared with plants and almost all biochemical processes within human cells have an evolutionary link to plant biochemical processes. It is unsurprising then that those same plant hormones have the ability to modify a wide range of biological processes and gene activities in humans.

SBPs are new chemical entities where the modest biological activity of the naturally occurring plant benzopyran hormone has been amplified some 100 to 1000-fold by classic medicinal chemistry processes.

The primary effect of SBPs is cytotoxicity of cancer cells due to inhibition of an oncogene enzyme critical to cancer cell survival. The presence of this oncogene in cancer cells of all phenotypes and critically important, in the full range of cancer cells within a tumor, means that the SBP family of drugs is the **first family of cytotoxics that kills both the (parent) cancer stem cells as well as the (daughter) somatic cancer cells. Thus offering for the first time the opportunity to prevent tumor relapse and the development of chemo-refractory cancer.**

Secondary biological effects are related to interaction with gene transcription factors leading to regulation of the activities of various genes. This novel function is being applied in two ways:

1. DEGENERATIVE DISEASES
   to promote the function of tissue stem cells associated with a range of degenerative diseases with a genetic basis () in bringing hope to sufferers of a wide range of conditions (muscular dystrophies, motor neurone disease, Alzheimer's etc) where no such hope currently exists, and
2. REGENERATIVE MEDICINE
   to promote the number and function of normal neural stem cells in order to repair injuries in the brain, spinal cord and peripheral nerves, and in so doing avoid the challenges associated with the use of transplanted stem cells.

Anti-tropomyosins (ATMs)

The ATMs target the microfilament component of the cytoskeleton of the cell. In this way they complement the action of the taxane and vinca alkaloid families of cytotoxic drugs that target the other main component of the cytoskeleton, the microtubules.

ONCOLOGY
The primary clinical indication is in cancer. Combining ATM treatment with that of vinca alkaloids leads to complete destruction of the cancer cell's cytoskeleton to an extraordinarily high level of synergy by eliminating the cancer cell's ability to switch signal transduction activity from the damaged microtubular component to the intact microfilament component.

Anti-microtubular drugs such as paclitaxel and docetaxel are still among the most prescribed drugs in oncology after 40 years. The company believes that combining them with an ATM, without exacerbating the toxicity of the anti-microtubular drugs, will lead to the combination becoming the 'go to' first-line therapeutic approach to most forms of cancer.

AUTOIMMUNE DISEASES
The broad function of the cytoskeleton across most cellular functions means that it likely to play a role in a broad range of degenerative diseases, but particularly autoimmune diseases where the role of the microfilaments in cell migration, adherence, mitosis and both internal and external communications is considered to be key in the autoimmune process.

The non-selective action of the anti-microtubular drugs makes them poorly suitable as potential therapeutics with their high level of toxic side-effects. The high (> 40) number of tropomyosin isoforms in the microfilaments and the ability of the ATM technology to target specific tropomyosin isoforms, makes these isoforms a novel drug target. That, and the ability of Novogen to produce ATM drugs with specific tropomyosin on-target activity, opens the possibility for an entirely novel approach to the treatment of autoimmune diseases.

Slide 7 SUPER-BENZOPYRANS Molecular target

The super-benzopyrans (SBPs) are small molecules.

They have a core benzopyran (diphenolic) scaffold to which is added various chemical moieties and where the overall steric shape of the molecule has been twisted to optimize its position in its protein target.

The SBP molecule has 5 key sites involved in target-binding. The considerable increase in the potency of the SBP molecule over earlier simple benzopyran shape is the result of the additional chemical moieties conferring vastly greater electron-transfer potential (both donating and receiving) to the molecule. The result is substantially greater binding affinity to the target.

The primary SBP target is the enzyme, tumor-associated NADH oxidase (tNOX).

The constitutive (normal) form of NOX (nicotinamide adenine dinucleotide oxidase; (or eNOX) is ubiquitous throughout living organisms. It is a hormone-responsive enzyme associated with cell membranes (principally plasma and mitochondrial membranes), where its primary function is the movement of hydrogen ions (protons) across membranes as part of redox mechanisms and the maintenance of the trans-membrane electrochemical potential. Removal of waste hydrogen from the cell and the transport of hydrogen within the mitochondria as part of ATP (energy) production are key functions of eNOX.

In tumor cells, eNOX is replaced with tNOX, a splice variant of eNOX. This is referred to as an 'enabling mutation', providing the tumor cell with a proton pump of greater capacity to meet the tumor cell's greater metabolic rate and ability to survive in a hostile (low pH, low oxygen) environment.

The SBPs bind to the quinone-binding motif of tNOX, effectively inhibiting all proton-pumping activity within the cancer cell, quickly leading to up-regulation of expression of the complement of pro-apoptotic factors (bid, bax, XIAP), DNA fragmentation and caspase-dependent apoptosis.

Simple benzopyran drugs, such as phenoxodiol (an early Novogen drug candidate), also target tNOX. What makes the SBPs 'first-in-class' is that the simple benzopyrans only kill the rapidly dividing cancer; the slowly-dividing progenitor cells (the cancer stem cells) are unaffected.

The SBPs kill both fast-dividing and slow-dividing sub-populations, making them the first molecules with the capacity **to kill the full hierarchy of cell within a tumor.**

Slide 8 SUPER-BENZOPYRANS Mechanism of Action

This slide summarises the key mechanism of action of the SBPs.

Inhibition of tNOX at the plasma membrane leads to a build-up of reduced quinone within the plasma membrane. This inhibits the sphingomyelin pathway within the plasma membrane, inhibiting the activity of (pro-survival factor) sphingosine-1-phosphate and promoting the production of (pro-apoptotic factor) ceramide.

Aptoptosis occurs principally via the release of caspase 2 and 3.

Slide 9 SUPER-BENZOPYRAN Oncology Pipeline

As a family, the SBP molecules are pan anti-cancer. This is the result of the molecular target, tNOX, being ubiquitous across all cancer phenotypes.

The central pharmacophore of the SBP molecule that is essential to its ability to bind to tNOX has been identified and is preserved in all members of the SBP family. However, chemical changes to the periphery of the molecule can result in significant changes in activity against specific cancer cell phenotypes. It is known that there are multiple isoforms of tNOX, and while this is yet to be confirmed, it is assumed that this preference for certain cancer phenotypes reflects preferential binding to certain tNOX isoforms.

Three SBP molecules have been selected for clinical development based on this phenotype preference.

TRX-E-005-1 shows an exceptionally high level of activity against ovarian cancer stem (CD44+) cells and ovarian somatic cancer (CD44-) cells. It is the active component of the product **Cantrixil**.

TRX-E-009-1 shows an exceptionally high level of activity against glioblastoma cancer stem cells. It is the active component of the product **Trilexium**.

TRX-E-022-1 shows an exceptionally high level of activity against prostate cancer cells. It is the active component of the product **Trx-7.**

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Slide 10 **CANTRIXIL** COMPOUND OVERVIEW

Cantrixil is owned by CanTx Inc, a New Haven, CT, based company that is a joint venture between Novogen (85%) and Yale University (15%). Novogen has licensed the active drug candidate to CanTx.

Cantrixil is a construct of **TRX-E-005-1** in **Captisol**, a cyclodextrin structure widely used for the delivery of water-insoluble drugs.

Cantrixil has been specifically developed for intra-peritoneal delivery where its primary purpose is to treat disseminated tumors wholly or largely confined to the abdominal cavity.

The two primary clinical indications are ovarian cancer and malignant ascites mainly associated with gastro-intestinal tumors following extensive invasion of the peritoneum and intestinal tract.

Animal studies to date have been limited to ovarian cancer, where it has been designed to seek out ovarian cancer stem cells contained in tumor masses varying in size from microscopic tumor spheroids, through to small tumors, and up to large tumor masses.

In animals whose abdominal cavity has been seeded with human ovarian cancer spheroids, the administration of Cantrixil into the peritoneal cavity effectively blocks the development of carcinomatosis, or the development of multiple tumors throughout the peritoneal cavity.

While **Cantrixil** also is cytotoxic to the bulk of ovarian cancer cells (rapidly-dividing non-cancer stem cells), it is anticipated that **Cantrixil** will be used in combination with a standard of care drug in order to deliver a more comprehensive anti-cancer effect designed to achieve maximum cancer cell kill and to prevent the development of recurrent disease.

The rationale is that the prevention of recurrent disease following response to first-line therapy will require eradication of the full hierarchy of cells within the tumor, but particularly the ovarian cancer stem population, the long-lived progenitor cells responsible for propagation of the cancer.

Slide 11 **CANTRIXIL** In vitro data

Tumors are heterogenous tissues with multiple sub-populations of cells. Most tumors are now recognized as having two dominant sub-populations:

- the first is a relatively small population of poorly differentiated cells that proliferate slowly and which display features of tissue stem cells, such as the ability to undergo asynchronous mitosis, an ability for infinite cell division, and being long-lived. These have come to be regarded as ‘tumor-initiating

cells' or 'cancer stem cells' as the cells that propagate and spread the tumor. These cells generally are distinguished by their expression of the surface marker CD44.

- The second population is the product of the cancer stem cells and is the dominant population in a tumor. These cells divide rapidly, have a finite number of divisions, generally are relatively short-lived and generally are better differentiated. These cells are distinguished by lacking CD44 expression.

The laboratory of Professor Gil Mor of Yale Medical School is distinguished by being the first group to have successfully identified, isolated and propagated in the laboratory these two cell populations from ovarian cancer biopsies.

In the study shown here, CD44+ and CD44- cells have been isolated from an ovarian cancer biopsy and placed together in culture. The CD44+ cells are stained **GREEN** and the CD44- cell stained **RED**.

The starting co-culture contains approximately equal proportions of both types of cells.

After 72 hours in culture, the rapidly-dividing **RED** CD44- cells dominate the much slower-dividing **GREEN** CD44+ cells. This reflects the situation in an ovarian tumor in the patient.

Paclitaxel is the first-line drug of choice in the treatment of ovarian cancer. If it is introduced into the culture at the start, the **RED** CD44- cells are sensitive to this drug and are killed; the **GREEN** CD44+ cells are resistant and remain unaffected, even after 72 hours in culture. Again, this reflects the situation in the patient where chemotherapy with standard of care drugs such as paclitaxel might bring about apparent remission with shrinkage of the tumor following killing of the predominant daughter cancer cells. However, the parent cancer stem cells survive this chemical onslaught to go on some months later to produce a replacement generation of daughter cells that now show much reduced sensitivity to paclitaxel and all other drugs. This is recurrent disease and recurrent disease is invariably untreatable and fatal.

Slide 12 **CANTRIXIL** In vitro data

The top 3 graphs in this slide compare the response of human ovarian cancer stem cells derived from 6 patients to paclitaxel, carboplatin and **TRX-E-005-1**.

Even at doses of paclitaxel and carboplatin that would be lethal to a patient (20 ug/mL), their ability to kill the cancer stem cells is very low (20%). Essentially, these cells are refractory (completely insensitive) to paclitaxel and carboplatin.

In contrast, the same cells are sensitive to the cytotoxic effect of **TRX-E-005-1** at doses down to 0.1 ug/mL.

The bottom graph plots the effect of **TRX-E-005-1** on the growth of an ovarian cancer stem cell line over 48 hours. At doses as low as 0.0125 ug/mL, this drug is inhibiting the growth of the cells compared to the rapid rise in cell numbers (blue line) of untreated, control cells. Withdrawal of the drug after this time is associated with no recovery of the cells, indicating that they have been killed.

Slide 13 CANTRIXIL In vitro data

A key step in the development of Cantrixil was its ability to kill ovarian cancer cell spheroids.

These are 3-dimensional clusters of cancer cells that are found in the peritoneal cavity of women with ovarian cancer and are considered a prime means of spread of the cancer throughout the abdominal cavity.

Cantrixil proved highly cytotoxic against these structures, confirming the ability of the drug to efflux from the Captisol construct and to penetrate multi-cellular structures.

Slide 14 CANTRIXIL In vitro data

The next key step in the development of Cantrixil was its ability to block the development of carcinomatosis, the primary rationale for using Cantrixil in the clinic.

Yale has developed a mouse model considered highly representative of ovarian cancer. Human ovarian cancer CD44+ (cancer stem) cells grown as spheroids are injected into the abdomen of athymic mice where they spread into a disseminated tumor masses over a period of 3 weeks comprising a mixture of CD44-positive and CD44-negative tumor cells. The organ distribution of the tumors and their histological appearance mirrors that seen in humans.

Cantrixil injected into the peritoneal cavity of these mice once the tumors have established results in a highly significant reduction in tumor load. In most animals tumors completely regress, with the least response substantial inhibition of growth. A scan of a mouse with the least response is shown.

Cantrixil is the first product to have achieved any meaningful anti-cancer effect in this highly stringent model.

Slide 15 CANTRIXIL Clinical development plan

Cantrixil currently is undergoing its IND process ahead of a proposed Phase 1a/Phase 1b study anticipated to commence mid-2015.

The primary trial site will be the Yale-New Haven Cancer Center in conjunction with the Smilow Cancer Hospital at Yale-New Haven. Additional sites to be added.

The Principal Investigators are Paul Eder MD and Elena Ratner MD of Yale University Medical School.

Patients will have late-stage, platinum- and taxane-refractory epithelial ovarian cancer.

Cantrixil will be administered intra-peritoneally via a portacath over a 4-week treatment course comprising 5 consecutive days treatment per week for 3 weeks.

Slide 16 TRILEXIUM Compound overview

Trilexium is the SBP drug, **TRX-E-009-1**, prepared in a proprietary formulation designed to optimize its bio-availability.

Like **TRX-E-005-1 (Cantrixil), TRX-E-009-1** is pan anti-cancer and is being developed as a general anti-cancer agent.

However, it has been selected as a lead candidate from the Company's library of SBP analogs because of its stand-out potency against glioblastoma stem cells and because it has the chemical properties required to cross the blood-brain barrier.

For this reason, the primary clinical strategy is to seek its development for the treatment of cancers of neural origin in both adults and children. Glioblastoma multiforme, astrocytoma, pineoblastoma, diffuse intrinsic pontine glioma, medulloblastoma and neuroblastoma are to be targeted primarily in first-in-man studies.

As with **Cantrixil**, the rationale behind **Trilexium** is its ability to kill cancer stem cells as well as their daughter cells, thereby delaying or reducing the risk of tumor recurrence.

Slide 17 TRILEXIUM In vitro data

This slide compares the cytotoxicity of **TRX-E-009-1** against glioblastoma multiforme (GBM) cells with that of **temozolomide**, the only cytotoxic drug approved for the treatment of GBM because of its ability to cross the blood-brain barrier.

Two cell lines have been used in this study: one from a patient who responded initially to **temozolomide** therapy (*TMZ-sensitive*) and one from a patient who failed to respond to **temozolomide** (*TMZ-resistant*).

Both cell lines show poor response to **temozolomide** with very high IC50 values of 609 and 1828 uM respectively. (IC50 refers to the amount of drug required to kill 50% of cancer cells in culture, a standard reference point for comparing the relative strength of anti-cancer drugs). Drug levels of 609 and 1828 uM are far too high to ever be achieved clinically.

In contrast, both cell lines show strong response to **TRX-E-009-1** (0.043 and 0.060 uM respectively), drug levels readily achieved in vivo.

Three conclusions to be drawn from studies such as this are:

- GBM somatic cancer cells are far more sensitive to **TRX-E-009-1** than to **temozolomide**
- **TRX-E-009-1** is unaffected by **temozolomide** resistance
- **TRX-E-009-1** would be appropriate to use in patients following **temozolomide** Rx regardless of tumor-response to **temozolomide**

Slide 18 TRILEXIUM In vitro data

Further studies confirming the ability of **TRX-E-009-1** to kill GBM cells from patients whose tumor failed to respond to **temozolomide**. In this case the mean IC50 values are 0.017 um (**TRX-E-009-1**) versus 918 uM (**temozolomide**).

Providing further evidence of the high sensitivity of GBM cells to **TRX-E-009-1.**

Slide 19 TRILEXIUM In vitro data

This study shows that the cytotoxic effect of **TRX-E-009-1** on GBM cells extends to the tumor-initiating cells within this form of brain cancer – the stem-like GBM cancer cells.

In this library of cells established in the laboratories of Dr Moonsoo Jin (Weill Cornell Medical School), GBM cells taken from tumor biopsies are grown in culture conditions that promote the de-differentiation of the GBM cells into cells with stem-like characteristics. These cells, in common with cancer stem cells associated with other forms of cancer, are resistant to standard chemotherapies.

TRX-E-009-1 achieved universal killing of this library of cells, the first time any drug has delivered any meaningful cytotoxic effect in the hands of these researchers.

Slide 20 TRILEXIUM Clinical development plan

Trilexium currently is completing its pre-clinical program.

It is proposed to conduct a Phase 1b study in Australian hospitals, commencing 2Q2015.

This will be a standard pharmacokinetic/pharmacodynamic/safety/maximum tolerable dose-determining study.

Patients will be enrolled with a variety of solid tumors, but with the intention of enrolling as many cases of GBM post-temozolomide/Avastin therapy as possible.

A treatment course will be 4 weeks, comprising twice-daily dosing, daily for 21 days followed by 7 days rest.

Treatment will continue until disease progression.

Slide 21 Anti-Tropomyosin (ATM) Drug Technology

The **ATM** drug platform targets the cytoskeleton of the cancer cell and introduces an entirely new and exciting anti-cancer target.

The cytoskeleton provides an essential framework for any cell, underpinning its ability to move, to adhere, to communicate with neighboring cells, to conduct internal signal transduction pathways, and to divide.

The cytoskeleton has two major components: the microtubules and the microfilaments.

While each of these two structural components have discrete functions, they also overlap to a large degree, meaning that there is a high degree of co-dependency and cooperation between them.

To date, drugs targeting the cancer cell's cytoskeleton have targeted the microtubules specifically, and in doing so, have spared the microfilaments.

The Novogen **ATM** drug technology now provides the ability to destroy both components of the cytoskeleton, robbing the cancer cell of its ability to detour function from one component to the other.

Slide 22 ATM Platform Proven efficacy of targeting the cytoskeleton

The microtubules have served for 40 years (and continue to serve) as a key cancer drug target. The taxanes (paclitaxel, docetaxel) and vinca alkaloids (vincristine, vinblastine) continue to be among the most widely prescribed drugs in oncology, in many cases as first-line therapies of choice.

Destruction of the microtubules serves to block mitosis (cell division) and leads to cell death through blockage of key internal signal transduction pathways, preventing the cell from managing its internal and external communication requirements.

The success of the anti-microtubule drugs has been offset by a number of negatives:

- not all cancer phenotypes are sensitive to anti-microtubule drugs and not all patients with generally sensitive cancer phenotypes (eg ovarian, lung, prostate cancer) are individually sensitive
- tumor cells rapidly develop resistance to these drugs
- cancer stem cells are completely insensitive to these drugs
- their anti-mitotic effect is not limited to cancer cells, resulting in significant dose-limiting toxicity.

Slide 23 **ATM Platform** The Tm5NM1 isoform

The microfilaments are linear, rope-like structures comprising two proteins: actin and tropomyosin.

Actin is an unsuitable drug target because of the ubiquity of actin in the body, particularly in muscle (smooth, cardiac, striated). Drugs targeting actin are highly toxic, blocking muscle contraction of the heart. With only two isoforms of actin having been identified, there was no opportunity to avoid this lethal side-effect.

The breakthrough in using the microfilaments as an anti-cancer drug target came from two discoveries.

The first discovery was that there are > 40 different isoforms of tropomyosins and that they can be differentiated into muscle- and non-muscle isoforms.

That initial discovery led to the second key discovery that one of those non-muscle tropomyosin isoforms, Tm5NM1, has been commandeered by cancer cells to the extent that tumor cells are highly dependent on that isoform for their function and survival.

While Tm5NM1 is found in non-cancer cells, healthy cells are not dependent on that isoform for survival, so allowing the cancer cell to be selectively targeted.

Slide 24 **ATM Platform** Inhibiting Tm5NM1

ATM compounds have been designed specifically to bind to the C-terminus of Tm5NM1, thereby:

- blocking the ability of the Tm5NM1 to dimerise

- thereby preventing the assembly of the microfilament

- leading to disassembly of the microfilaments

- leading to cell death.

The first ATM drug (TR100) has provided proof-of-concept that it is possible to deliver a potent anti-tumor effect (melanoma) in mice by targeting Tm5NM1 without adverse side-effects.

Second generation ATM drugs with greater on-target specificity and considerably greater anti-cancer potency have been developed and are undergoing pre-clinical development.

Slide 25 **ATM Platform** A new paradigm in chemotherapy

ATM drugs deliver a potent anti-cancer effect on their own.

However, their proposed method of use is in combination with standard of care anti-microtubule drugs.

The rationale being that a combination of the two drugs, each targeting their respective component of the cytoskeleton, will deliver comprehensive destruction of the cancer cell's cytoskeleton, leaving no capacity for the cancer cell to switch between the two cytoskeleton components.

This graph shows the 'predicted' (mathematical modeling) and the 'actual' effect on neuroblastoma cells of combining an ATM drug with an anti-microtubular drug (vincristine), with profound synergy resulting.

Notable features of an ATM / anti-microtubular drug combination are:

- there is no increased toxicity in animals despite the high level of synergy when used in combination with anti-microtubular drugs, highlighting the on-target specificity of the anti-Tm5NM1 action

- ATM drugs are unaffected by efflux pump mechanisms associated with resistance to anti-microtubular drugs.

Slide 26 **ATM Platform** Clinical strategy

ATM drugs deliver a potent anti-cancer effect on their own. However, their use in combination therapy with vincristine remains the preferred clinical strategy for those cancers (eg. prostate, ovarian, lung and breast cancer) where there is responsiveness to anti-microtubule drugs.

In other cancers (eg. melanoma, neuroblastoma) which typically show little or no response to anti-microtubule drugs, ATM drugs will be used on their own.

To date the pre-clinical focus has been on prostate cancer, melanoma and neuroblastoma, with each of these phenotypes showing potent response to ATM therapy.

Novogen currently is identifying the lead ATM drug candidate from a library of highly active compounds. This process is expected to conclude by end of September 2014.

ATM drugs are orally dosed with the formulation currently the subject of optimsation.

A first-in-man clinical study is anticipated being conducted in 2H15.

Slide 27 VALUE DRIVERS

Priority #1
The primary focus of the Company is to progress both **Cantrixil** and **Trilexium** into first-in-man studies.

Both products currently are in their pre-clinical, IND processes with the aim of being in the clinic in each case by about mid-2015.

Priority #2
Trx-7 and the lead **ATM** drug will continue with their pre-clinical development, but the need to allocate resources will see the Company focus on bringing **Cantrixil** and **Trilexium** into the clinic first, with **Trx-7** and the **ATM** candidate entering the clinic in about 4Q 2015.

Priority #3
The Company's neurodegenerative disease, musculodegenerative disease, and regenerative medicine programs are all early-stage, drug discovery programs requiring modest R&D expenditure.

Slide 28 VALUE PROPOSITION

The Company has two novel, 'first-in-class' drug technologies, a significant risk-reduction situation.

One of these technologies, the SBP technology platform, is the first drug technology with proven capacity to kill the full range of cancer cell sub-populations within ovarian and glioblastoma tumors. The long-sought goal of preventing tumor relapse is raised for the first time with this breakthrough technology.

The second technology offers the ability to elevate standard of care treatment with anti-microtubular drugs to a new level through the comprehensive destruction of the cancer cell's cytoskeleton.

The timetable is to have 2 drug candidates in the clinic by mid-2015 and 4 by end of 2015.

The Company owns both technologies and has an aggressive patenting program in place.

Novogen runs on a project management basis, thereby minimizing overheads and allowing tight financial control and budgeting.

The CEO and senior management are highly experienced in drug development and the running of public companies.