Washington. D.C. 20549

Form 6-K

REPORT OF FOREIGN PRIVATE ISSUER PURSUANT TO RULE 13a-16 OR I5d-16
UNDER THE SECURITIES EXCHANGE ACT OF 1934

PE
9-1-02

For the month of **September, 2002**

Novogen Limited

(Translation of registrant's name into English)

140 Wicks Road, North Ryde, NSW, 2113, Australia
(Address of principal executive office)

[Indicate by check mark whether the registrant files or will file annual reports under cover of Form 20-F or Form 40-F.
Form 20-F √ Form 40-F ☐

[Indicate by check mark whether the registrant by furnishing the information contained in this Form is also thereby furnishing the information to the Commission pursuant to Rule 12g3-2(b) under the Securities Exchange Act of 1934. Yes ☐ No ☐

[If "Yes" is marked, indicate below the file number assigned to the registrant in connection with Rule 12g3-2 (b):
82-_____.1

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned, thereunto duly authorized.

Novogen Limited
(Registrant)

Date **5 September, 2002**

By

Ronald Lea Erratt
Company secretary

*Print the name and title under the signature of the signing officer.

GENERAL INSTRUCTIONS

A. Rule as to Use of Form 6-K.

This form shall be used by foreign private issuers which are required to furnish reports pursuant to Rule 13a-16 or 15d-16 under the Securities Exchange Act of 1934.

B. Information and Document Required to be Furnished.

Subject to General Instruction D herein, an issuer furnishing a report on this form shall furnish whatever information, not required to be furnished on Form 40-F or previously furnished. such issuer (i) makes or is required to make public pursuant to the law of the jurisdiction of its domicile or in which it is incorporated or organized, or (ii) files or is required to file with a stock exchange on which its securities are traded and which was made public by that exchange, or (iii) distributes or is required to distribute to its security holders.

SEC 1815 (7-91)

assets; bankruptcy or receivership; changes in registrant's certifying accountants; the financial condition and results of operations; material legal proceedings; changes in securities or in the security for registered securities; defaults upon senior securities; material increases or decreases in the amount outstanding of securities or indebtedness; the results of the submission of matters to a vote of security holders; transactions with directors, officers or principal security holders; the granting of options or payment of other compensation to directors or officers; and any other information which the registrant deems of material importance to security holders.

This report is required to be furnished promptly after the material contained in the report is made public as described above. The information and documents furnished in this report shall not be deemed to be "filed" for the purposes of Section 18 of the Act or otherwise subject to the liabilities of that section.

If a report furnished on this form incorporates by reference any information not previously filed with the Commission, such information must be attached as an exhibit and furnished with the form.

C. Preparation and Filing of Report.

This report shall consist of a cover page, the document or report furnished by the issuer, and a signature page. Eight complete copies of each report on this form shall be deposited with the Commission. At least one complete copy shall be filed with each United States stock exchange on which any security of the registrant is listed and registered under Section 12(b) of the Act. At least one of the copies deposited with the Commission and one filed with each such exchange shall be manually signed. Unsigned copies shall be conformed.

D. Translations of Papers and Documents into English.

Reference is made to Rule 12b-12(d) [17 CFR 240.12b-12(d)]. Information required to be furnished pursuant to General Instruction B in the form of press releases and all communications or materials distributed directly to security holders of each class of securities to which any reporting obligation under Section 13(a) or 15(d) of the Act relates shall be in the English language. English versions or adequate summaries in the English language of such materials may be furnished in lieu of original English translations.

Notwithstanding General Instruction B, no other documents or reports, including prospectuses or offering circulars relating to entirely foreign offerings, need be furnished unless the issuer otherwise has prepared or caused to be prepared English translations, English versions or summaries in English thereof. If no such English translations. versions or summary have been prepared, it will be sufficient to provide a brief description in English of any such documents or reports. In no event are copies of original language documents or reports required to be furnished.

AND MEDIA RELEASE **5 SEPTEMBER 2002**

RELEASE TO SHAREHOLDERS OF MARSHALL EDWARDS INC

Novogen Limited's subsidiary, Marshall Edwards Inc., (LSE-AIM: MSH), today the following letter to shareholders.

This letter is a report on the status of the development of the Marshall Edwards, Inc. (the Company) anti-cancer drug phenoxodiol, and the development strategy for the drug.

SUMMARY

The Phase I program for the intravenous dosage form of phenoxodiol is drawing to a close and phenoxodiol now has entered the formal approval stage for its Phase IIb clinical trials that will see the drug evaluated for efficacy. The existing program has met its objectives - confirming the drug's high level of safety, providing valuable information on how the drug behaves in the body, and providing early evidence of its anti-cancer action.

The focus of the drug development program remains the intravenous dosage form of phenoxodiol. An oral dosage form also is being evaluated clinically, but it is the intravenous form that is the most advanced and that will be taken immediately into full Phase II testing.

Concurrent with the clinical program, ongoing laboratory studies are being conducted to define the mechanism of action of phenoxodiol. Those studies now have confirmed phenoxodiol causes the cancer cells to undergo apoptosis (cell death) by activating the cancer cell's self-destruct mechanism. The way that phenoxodiol does is this unique and has been recognized for some time now as an important goal for the next generation of anti-cancer drugs. Phenoxodiol causes death of cancer cells even when the cells are highly resistant to the lethal effects of standard anti-cancer drugs.

The ability of cancer cells to survive indefinitely through inactivation of proteins known as death receptor proteins is common to many human cancers. Approximately half of the cancers that arise in such tissues as the ovary, kidney, pancreas and gall bladder, plus the common forms of leukemia, are able to survive because they have inactivated their death receptors. Reactivating those death receptors results in the cancer cell immediately self-destructing. Recent laboratory studies conducted at the Yale University Medical School have discovered that the anti-cancer action of phenoxodiol lies in its ability to reactivate the death receptors on cancer cells in a very potent manner. Just as importantly, this effect is limited to cancer cells with the death receptor mechanism in non-cancer cells being unaffected by phenoxodiol.

This means that phenoxodiol has the potential to be an effective anti-cancer agent in its own right for a wide range of human cancers.

However, recent research has shown a way by which phenoxodiol works effectively even in those cancers where inactivation of the death receptors is not a primary cause of the cancer. The Phase II stage of the program will incorporate this recent important discovery and will compare the efficacy of phenoxodiol alone and in combinational therapy.

This is the first known trial in humans of anti-cancer therapy that is targeting reactivation of the death receptors in a highly selective manner.

The cancer types that the Phase II program for the intravenous dosage form will focus on are ovarian carcinoma, renal carcinoma and leukemia.

MECHANISMS OF ACTION

Identifying the best uses for phenoxodiol hinges on an increased understanding of it's mechanisms of action.

For that reason, the Company is putting considerable effort into this area. We are pleased to report that those studies have delivered some important achievements that confirm our early view that phenoxodiol heralds a new class of anti-cancer drug and represents a major new therapeutic opportunity, particularly with cancers that have proven highly resistant to standard therapies.

We now know that two key targets of phenoxodiol in cancer cells are the death receptors and an enzyme known as sphingosine kinase.

Target # 1. *Death receptors*

The death receptors are a family of proteins on the surface of all cells that, when activated, lead immediately to the death of the cell. These receptors (Fas, TNFR1, DR3-DR6) trigger the cell to self-destruct within one to two days through a process of auto-digestion (known as apoptosis). The death receptors and apoptosis are important to our health, allowing the body's immune system to kill a cell whenever it is damaged or when it is required to die as part of normal tissue remodeling.

Normal, healthy cells prevent accidental triggering of this mechanism by producing blocking proteins (known as *anti-apoptosis proteins*) (e.g. c-FLIP) that block low level activation of the death receptors. A damaged cell normally shuts off production of these anti-apoptosis proteins, thereby allowing the immune system to trigger apoptosis by contacting the death receptors.

Cancer cells resist this process by producing large amounts of blocking proteins such as c-FLIP. In this way, cancer cells are protected from the body's immune system. Switching off the production of these blocking proteins in cancer cells leads immediately to their death. For this reason, these blocking proteins have become recognized as an important new target for a new generation of anti-cancer drugs. However, the challenge has been how to knock out c-FLIP production in cancer cells without having a similar effect in non-cancer cells.

Phenoxodiol now is confirmed as the first drug to achieve this outcome. Phenoxodiol potently switches off the production of anti-apoptotic proteins in human cancer cells in a highly selective manner. A research team at **Yale University Medical School** is responsible for discovering this mechanism and Marshall Edwards, Inc. is continuing to work closely with them on this aspect of phenoxodiol's function.

Target # 2. *Sphingosine kinase.*

Phenoxodiol inhibits the activity of the enzyme, sphingosine kinase. Sphingosine kinase is a key enzyme in enabling a cell to respond to growth signals. It is essential to the survival of all cells.

As with the anti-apoptosis proteins, sphingosine kinase activity is highly elevated in many forms of human cancer. In particular, it is an enzyme on which many forms of human cancer (particularly

ovarian cancer and leukemia) are dependent for their ability to grow and to metastasize. However, sphingosine kinase also is an oncogene, which means that abnormally high activity of this enzyme will cause cancer.

A small number of drugs are known to be under development to target this important enzyme, but phenoxodiol is unique in inhibiting sphingosine kinase only in cancer cells. As with c-FLIP levels, sphingosine kinase activity in non-cancer cells is unaffected by phenoxodiol. **Phenoxodiol is the first inhibitor of sphingosine kinase activity to be tested in humans.**

Studies are continuing in collaboration with a number of centers including the **Medical University of South Carolina** to fully describe this effect and to explore the likely link between sphingosine kinase activity and the production of anti-apoptosis proteins.

The studies being conducted at **Yale University Medical School** and the **Medical University of South Carolina** are part of an integrated research program involving a number of research institutions designed to characterize the way in which phenoxodiol exerts its unique effects. The **National Institutes of Health (NIH)** is part of that international co-coordinated program, bringing to the program particular expertise in how anti-cancer drugs affect the ability of cancer cells to proliferate, and the development of drugs for treatment of head and neck cancers, a particularly difficult cancer type to treat.

PHASE I CLINICAL PROGRAM

The current Phase I program is evaluating two dosage forms of phenoxodiol – an intravenous form and an oral form. The main objective of this program is to provide information on such matters as the safety of the drug and its behavior in the body.

The issue of whether or not the drug is producing an anti-tumor response is normally not a key objective in Phase I, as a significant anti-tumor effect is not normally anticipated at this level of study. In fact even anti-cancer drugs that ultimately go on to receive marketing approval because of their potent anti-cancer effect, on average show very poor tumor response in patients in Phase I programs. This low response rate in other drugs is due to various reasons: the advanced nature of the disease in the patients being used, the fact that the cancers have become unresponsive to standard chemotoxic drugs, and the fact that a broad variety of dosages and treatment schemes (including ineffective ones) are being evaluated.

Despite this, the Company was sufficiently confident in phenoxodiol that it sought to look for preliminary evidence of efficacy at this level of trialing. For this reason, the program was designated Phase Ib/IIa.

This program has involved five separate studies. Three of these are using the intravenous formulation of phenoxodiol and two are using the oral dosage formulation. This unusually large number of clinical studies has been possible because of the anticipated low toxicity of the drug. The strategy behind this multi-trial program was to expedite the drug's development by seeking answers to a number of key therapeutic questions at the one time, such as weekly versus continuous intravenous injection, oral versus intravenous dosage, and discovering which tumor types are the most sensitive.

All patients enrolled in these studies must have had advanced, metastatic cancers and have failed standard anti-cancer therapies. Phenoxodiol is the only anti-cancer drug given during the trial.

Weekly intravenous treatment. This treatment schedule was examined in a study conducted at an Australian hospital (St. George Hospital, Sydney). It was completed recently and the large amount of data is in the process of being evaluated. That process is expected to provide a final report within the next few months.

In this study, phenoxodiol was administered as a single weekly intravenous injection to 21 patients (with a variety of solid tumors) for treatment cycles of six weeks. Doses of between one and 30 mg/kg were used. Treatment was continued indefinitely provided that there was no toxicity and no disease progression.

Interim results of this study have been reported to a November 2001 meeting and the April 2002 annual meeting of the American Association for Cancer Research.

No significant toxicity was observed. None of the usual toxicities associated with many anti-cancer drugs involving the bone marrow (anemia), skin (hair loss), gut (nausea, diarrhea) or nervous system (loss of function) were encountered.

Also confirmed was the fact that phenoxodiol was released into the body in a form that was available to act against cancer cells, that phenoxodiol was not broken down (metabolised) in the body to any significant extent, and that levels of phenoxodiol reached in the body were up to 15 times those required to kill cancer cells in the laboratory.

A number of patients were able to continue on treatment beyond 12 weeks, with one patient (with metastatic renal carcinoma) remaining on the drug for 20 months.

Continuous intravenous treatment. This method of administration was studied at two sites - Australia's largest tertiary-care hospital (Royal Prince Alfred Hospital, Sydney), and the Taussig Cancer Care Center, Cleveland Clinic Foundation, Cleveland, USA.

In this approach, the drug is infused on a continuous basis via a pump into the bloodstream over seven days. Whereas the weekly intravenous injection approach delivers high levels of drug in the body over no more than several hours, the continuous intravenous infusion approach delivers a constant, lower drug level over days or weeks. The two different methods of administration were being evaluated for possible use in the Phase II clinical trials.

Patients with various forms of advanced, solid tumors were treated for six cycles, comprising seven days on and seven days off per cycle. Patients received dosages ranging from 0.3 – 47 mg/kg/day. Treatment was continued until patients showed toxicity or disease progression.

The Australian arm of the study used dosages ranging from 2- 40 mg/kg/day. That study finished in July 2002, and the data currently is being evaluated. The results of this study have not been made public by the hospital concerned pending presentation of the data at a future scientific conference and publication in a scientific journal.

However, it is possible to confirm that no toxicities were reported in the 24 patients in the study and that the study met its objectives in confirming achievement of steady-state levels of drug over the period of treatment. A number of patients also were able to continue on therapy beyond six treatment cycles without disease progression.

The U.S. arm of the study is nearing completion, with 18 patients enrolled and six patients still to be enrolled. This study has been extended recently to provide greater focus on the lower (0.3- 3.3 mg/kg/day) and upper (up to 47 mg/kg/day) ends of the dosage range.

Interim results of the first 10 patients in this study were reported to the American Association for Cancer Research in April 2002, noting that there was no significant toxicity and that six of the 10 patients treated with dosages ranging between 0.6 – 3.3 mg/kg/day showed stabilized tumors over the period of treatment.

This study should be fully recruited by November 2002.

Oral dosage treatment. Two studies are being conducted at two Australian hospitals. One study involves patients with advanced prostatic cancer, and the second study involves patients with various forms of leukemia.

Both of these studies are in their early stages and interim results are expected in 2003.

PHASE II CLINICAL PROGRAM

This next phase of development of the intravenous dosage form will be conducted in patients with specific tumor types – ovarian carcinoma, renal carcinoma and leukemias. These tumor types are thought to be particularly appropriate because of their particular dependency for survival and growth on sphingosine kinase function and on blockage of death receptor activity.

It is proposed to conduct the Phase II program as multi-center trials using both U.S. and Australian hospitals. Hospitals in other territories will be added as needed.

The Phase II program will be conducted in two parts over the next 15 months and is expected to involve several hundred patients. In the first part, phenoxodiol will be used as a monotherapy. In the second part, phenoxodiol will be used in combination with another agent.

The first of these trials is planned to involve ovarian cancer patients in both Australian and U.S. hospitals and currently is being considered by the relevant Institutional Review Boards. This trial is expected to commence in November 2002.

ONCOLOGY DRUG PROGRAM

Marshall Edwards, Inc. has an active interest in the oncology drug program that Novogen Limited is pursuing and from which phenoxodiol derives. Under the terms of its license agreement with Novogen, the Company has an option over any new oncology drugs emanating from that program that advance to human clinical trials. It is worth highlighting a number of features of that program that are of ongoing interest to Marshall Edwards, Inc.

A large number of analogues of phenoxodiol have been synthesized. These are compounds based on the phenoxodiol structure, but modified to varying degrees. The effect of that structural modification is to produce new drugs with different anti-cancer effects compared to phenoxodiol. Of particular interest is the finding that it is possible to produce drugs with specific activity against specific types of cancer.

This is an entirely novel and exciting finding, pointing to the ability of Novogen scientists to customize drugs for specific cancer types.

Marshall Edwards, Inc., in conjunction with Novogen, has adopted a strategy of developing drug candidates for specific tumor types such as breast cancer, neuroblastoma (the major form of cancer in children) and mesothelioma. Collaborations are being formed with world-leading research institutions in each of these areas in order to take new drugs into the clinic for specific forms of cancer for which there are few current treatment options.

The goal of Marshall Edwards, Inc. and Novogen is to develop a family of anti-cancer drugs, all based on the phenoxodiol structure, with complementary activity that will provide treatment options across most forms of human cancer.

I hope that you agree with me that this is an exciting program and that the Company is well positioned with its intellectual property position to become a significant force within the oncology field.

ISSUED FOR	:	**NOVOGEN LIMITED**
LISTINGS	:	**ASX (CODE NRT), NASDAQ (CODE NVGN).**
FOR FURTHER INFORMATION	:	**DR GRAHAM KELLY, PHENOXODIOL PROGRAM DIRECTOR** **CHRISTOPHER NAUGHTON, MANAGING DIRECTOR** **NOVOGEN LIMITED** **TEL (02) 9878 0088** http://www.novogen.com
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Statements herein that are not descriptions of historical facts are forward-looking and subject to risk and uncertainties. Actual results could differ materially from those currently anticipated due to a number of factors, including those set forth in the Company's Securities and Exchange Commission filings under "Risk Factors", including risks relating to the early stage of products under development; uncertainties relating to clinical trials; dependence on third parties; future capital needs; and risks relating to the commercialisation, if any, of the Company's proposed products (such as marketing, safety, regulatory, patent, product liability, supply, competition and other risks).