EDISON

Novogen

Asset acquisition

Acquires mid-stage cancer drug from Genentech

Pharma & biotech

Novogen has in-licensed global rights to the Phase II-ready drug GDC-0084 from Genentech. GDC-0084 is designed to treat brain cancers, including the rapidly fatal glioblastoma (GBM). Novogen plans to initiate a Phase II trial in GBM next year and positive trial results could allow filing for accelerated approval in 2020. The transaction will complement Novogen's preclinical assets and Cantrixil, which will enter Phase I in ovarian cancer in Q416. Our valuation of Novogen's existing programme is unchanged at A$112m (A$0.26/share). We value GDC-0084 under two different scenarios at A$11-54m, after accounting for A$35m of anticipated clinical trial costs.

Year end	Revenue (A$m)	PBT* (A$m)	EPS* (c)	DPS* (c)	P/E (x)	Yield (%)
06/15	1.6	(8.4)	(3.0)	0.0	N/A	N/A
06/16	3.7	(11.6)	(2.8)	0.0	N/A	N/A
06/17e	4.9	(19.9)	(4.6)	0.0	N/A	N/A
06/18e	4.7	(31.6)	(7.4)	0.0	N/A	N/A

Note: *PBT and EPS are normalised, excluding exceptionals and share-based payments.

GDC-0084: PI3K inhibitor targeting brain cancer

GDC-0084 is a small molecule phosphoinositide 3-kinase (PI3K) inhibitor, which readily crosses the blood-brain barrier and exhibited dose-dependent reductions in tumour growth in Phase I. The Phase II trial is likely to test GDC-0084 as first-line maintenance chemotherapy in newly diagnosed GBM following surgical resection and six weeks of chemoradiotherapy. The trial will recruit a biomarker-defined subgroup of patients who receive little benefit from standard chemotherapy.

Combination therapy may boost efficacy

Preclinical studies show that combining GDC-0084 with radiotherapy leads to improved efficacy. This raises the possibility that GDC-0084 may need to be administered from the start of radiotherapy treatment. If a combination trial needs to be carried out this could delay a potential approval filing by 2-3 years and would need to be funded by a partner or would entail higher costs in the absence of one.

Deal terms

Novogen will pay Genentech US$5m upfront, modest regulatory and commercial milestones, plus royalties in line with industry benchmarks (we assume 10%). Novogen will also purchase privately held Glioblast for A$2.1m (A$1.5m in shares) plus A$1.25m of shares payable at the start and successful completion of a Phase II trial. Two other milestones may be payable to Glioblast (no details disclosed – we model A$10.5m (5x the upfront payment), split between filing and approval).

Valuation: We value GDC-0084 at A$11-54m

We have valued GDC-0084 under two different development scenarios. Firstly, where only a single Phase II trial is required before filing for accelerated approval, our valuation is A$54m. Under a two-trial scenario and 2022 filing, our valuation is A$11m. We estimate that the cost of acquiring GDC-0084 and completing a Phase II trial would be A$46m, including A$42m in cash. With cash of A$33.5m on 30 June we estimate that Novogen will require A$20m of additional funding in FY18.

31 October 2016

Price	**A$0.09**
Market cap	**A$40m**
	A$/US$0.76
Net cash (A$m) at 30 June 2016	33.5
Shares in issue	429.7m
Free float	90%
Code	NRT
Primary exchange	ASX
Secondary exchange	NASDAQ

Share price performance

%	1m	3m	12m
Abs	(9.2)	(6.3)	(36.4)
Rel (local)	(7.2)	(2.2)	(36.4)
52-week high/low	A$0.17		A$0.09

Business description

Novogen is an ASX- and NASDAQ-listed biotechnology company. It is developing GDC-0084 for brain cancer and two in-house drug technology platforms: super-benzopyrans (SBPs) and anti-tropomyosins (ATMs). SBPs show activity against cancer stem cells and are active across many different cancers. ATMs show synergy with anti-mitotics and activity across many cancer types.

Next events

Initiate Cantrixil Phase I in ovarian cancer	Q416
Initiate GDC-0084 Phase II in GBM	2017
Submit Anisina IND to FDA	2017

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GDC-0084: Specifically developed to target GBM

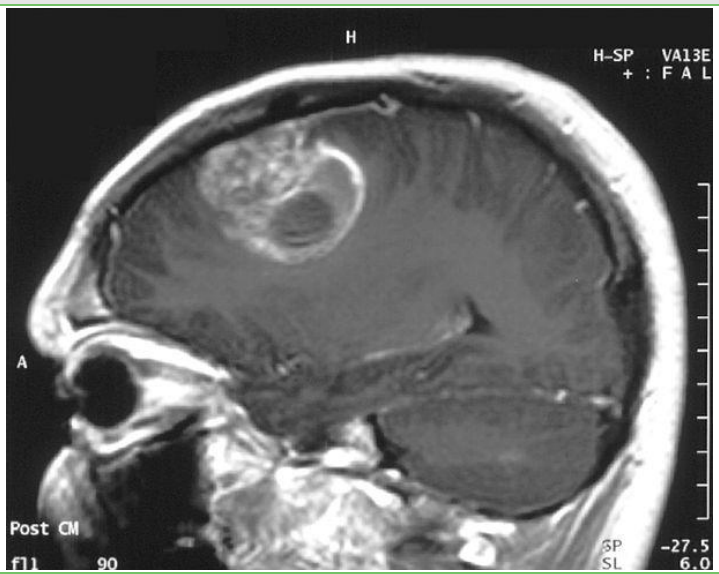
Novogen has in-licenced the Phase II-ready drug GDC-0084 from Genentech. GDC-0084 is an orally administered small molecule phosphoinositide 3-kinase (PI3K) inhibitor that targets an important growth signalling pathway in cancer cells. The drug was specifically developed to cross the blood-brain barrier and target the aggressive brain cancer glioblastoma, a disease with poor patient survival for which there are few effective therapies. The drug has completed a Phase I trial in patients with advanced disease, which confirmed that it readily crosses the blood-brain barrier and led to dose-dependent inhibition of tumour growth. Seven of the eight patients treated at the maximum tolerated dose of 45mg/day demonstrated levels of drug in the bloodstream that were associated with significant inhibition of tumour growth in preclinical models.

Glioblastoma: An aggressive brain cancer with few effective treatments

GBM is the most common and most aggressive primary malignant tumour of the brain and spinal cord (Exhibit 1). Approximately 11,500 patients are diagnosed with GBM each year in the US. GBM tumours are characterised by invasive and diffuse growth, which makes complete surgical removal difficult. Standard treatment for GBM entails maximal surgical resection of the tumour followed by radiotherapy with concurrent chemotherapy with temozolomide (TMZ) followed by adjuvant chemotherapy with the same drug to treat the residual infiltrative component of the tumour. Despite this aggressive treatment, the disease invariably returns resulting in a five-year survival rate of only 5%.[1]

Novogen intends to develop GDC-0084 as a treatment for newly diagnosed GBM patients (first line therapy). Development will target the 61%[2] of patients where the promoter of the O6-methylguanine methyltransferase (MGMT) gene in the tumour cells is unmethylated. These patients receive only minimal benefit from treatment with TMZ and are in urgent need of more effective therapies.

Exhibit 1: Contrast-enhanced MRI scan of a glioblastoma



Source: A Christaras, Wikimedia commons

1 CBTRUS Statistical Report: Primary Brain and Central Nervous System Tumors Diagnosed in the United States in 2008-2012. Ostrom et al *Neuro-Oncology* 17:iv1–iv62, 2015

2 Average of Chinot et al *N Engl J Med* 2014; 17:708-717 (67%) and Hegi et al *N Engl JMed* 2005; 352:997-1003 (55%)

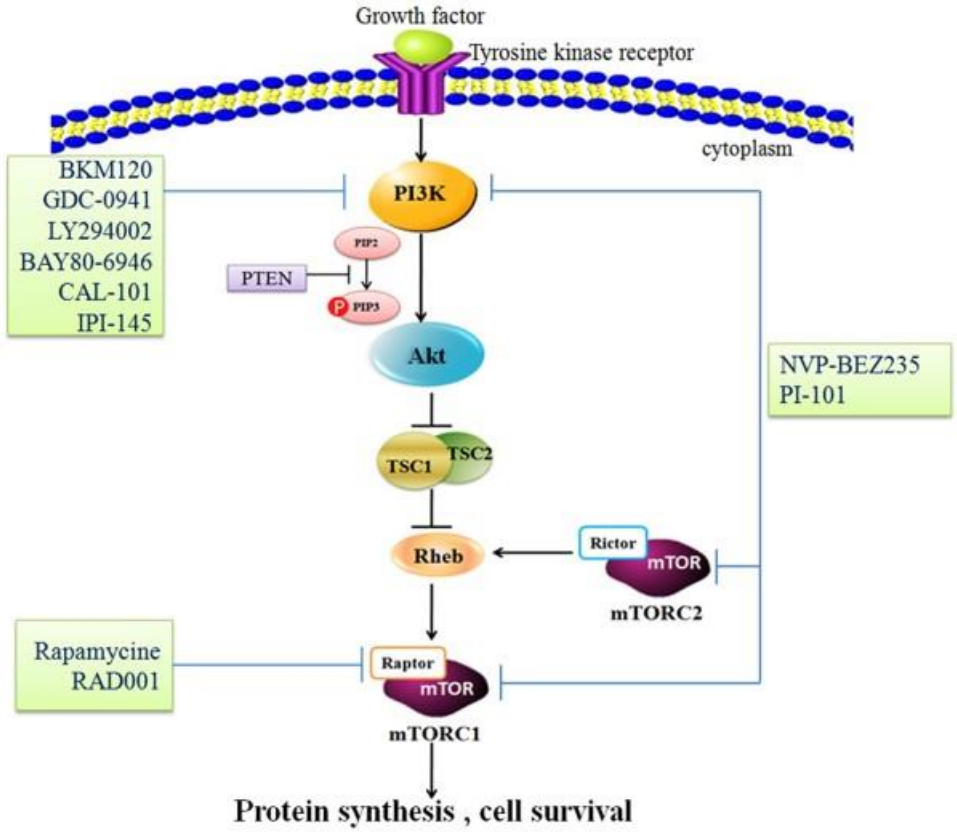
PI3K is a validated pathway targeted by a range of big pharma

The PI3K signalling pathway plays a crucial role in cellular proliferation, metabolism, survival and apoptosis (programmed cell death), as illustrated in Exhibit 2. PI3K signalling is initiated by receptor tyrosine kinases (RTK) or G-protein coupled receptors located at the cell surface, and by some oncogenic proteins such as Ras.

The PI3K pathway is frequently over-activated in cancer. The over-activation can occur through a variety of mechanisms including mutation and amplification of genes in the pathway, or by loss of function of the tumour suppressor PTEN, which is a negative regulator of PI3K signalling.

The first approved cancer drugs that target the PI3K pathway were the rapamycin analogues everolimus and temsirolimus, which inhibit mTORC1. The PI3K inhibitor idelalisib (Zydelig, Gilead Sciences) was first approved by the FDA in 2014 and is approved to treat several types of leukaemia and lymphoma, providing validation for PI3K as a target for anticancer drug development. Idelalisib is a selective inhibitor of the delta isoform of PI3K (PI3Kδ).

Exhibit 2: Overview of the PI3K/Akt/mTOR pathway



Source: Fang et al. Biomarker Research 2013, 1:30 http://www.biomarkerres.org/content/1/1/30. Note: The exhibit lists several PI3K and/or mTOR inhibitors that have been trialled in lymphoma.

PI3K is a promising target for GBM drug development

Inhibition of the PI3K pathway is a promising target for GBM drug development because abnormal PI3K signalling is associated with over 80% of cases of the disease.[3] These changes include: amplification of epidermal growth factor receptor (EGFR), which leads to activation of the PI3K pathway in ~45% of GBM cases; amplification or activating mutations of the genes encoding the

[3] The Cancer Genome Atlas Research Network. Comprehensive genomic characterization defines human glioblastoma genes and core pathways. Nature 2008, 455, 1061−1068.

PI3K subunits in ~15% of patients with GBM; and loss of PTEN function in ~40% of GBM cases. Loss of PTEN function has been shown to lead to poor survival.

Thorough preclinical development programme

Novogen will benefit from the thorough big pharma preclinical development programme that Genentech has conducted for GDC-0084.

GDC-0084 is a potent brain-penetrant small molecule inhibitor of PI3K (EC50 2nM) that was specifically designed for treatment of brain cancer. The drug is also deliberately designed to be a moderately potent inhibitor of mammalian target of rapamycin (mTOR) kinase (EC50 70nm) to avoid the toxicity seen with drugs that are potent inhibitors of both targets.

GDC-0084 was shown to freely cross the blood-brain barrier in the mouse, rat and dog, with brain to plasma ratios greater than 1 (Exhibit 3 upper section). Studies showed that the drug inhibited PI3K activity in mouse brain, reducing the phosphorylation of Akt (Exhibit 3, lower section)

Exhibit 4 shows that the drug strongly inhibited tumour growth in the GBM6 patient-derived tumour model.

Exhibit 3: GDC-0084 readily crosses blood-brain barrier and inhibits target in mouse brain

Table 1. Brain and CSF distribution of GDC-0084 in preclinical models

Species	Brain-to-Plasma Ratio	Free Brain-to-Free Plasma Ratio	CSF-to-Free Plasma Ratio
Mouse	1.3	0.40	N/A
Rat	2.4	0.68	1.5
Dog	2.2	0.45	1.03

Figure 1. Inhibition of p-AKT by GDC-0084 in mouse brain

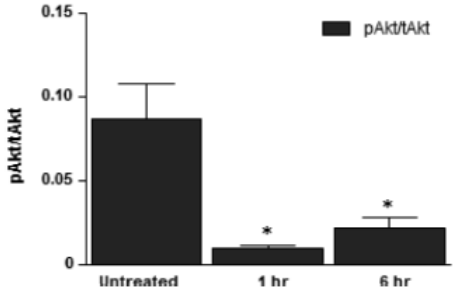
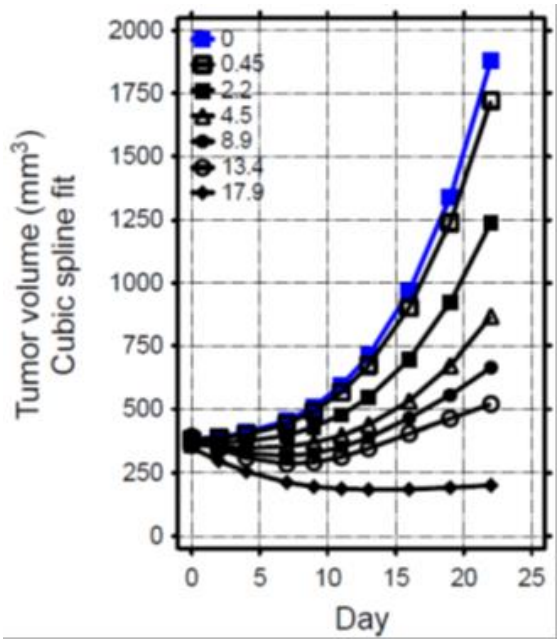


Exhibit 4: GDC-0084 inhibits tumour growth in mouse flank model of human GBM



Source: Morrissey, Vora et al ASCPT 2016 poster. Note: Figure 1 shows that GDC-0084 inhibits PI3K activity, reducing levels of phosphorylated Akt in the brain.

GDC-0084 combines with radiotherapy for increased efficacy

GDC-0084 showed increased efficacy when it was combined with radiotherapy under different treatment schedules in preclinical studies. This is important because radiotherapy is part of the standard treatment regimen for newly diagnosed GBM.

Two separate studies were performed in mice with GBM tumours implanted in the brain, which tests whether the drug crosses the blood-brain barrier in to the brain. The studies were conducted with two different GBM tumour lines in which the MGMT promoter is unmethylated. Both studies showed that combining GDC-0084 with radiotherapy increased efficacy, but differed in when in the course of treatment was the optimum time to administer the drug.

In one animal model the greatest efficacy was seen when GDC-0084 was administered either starting at the completion of radiotherapy or both during and after radiotherapy.

In the other animal model using different patient-derived GBM tumour cells, GDC-0084 as a single agent was more effective than radiotherapy alone, and the greatest efficacy was seen when GDC-0084 was administered at the same time as radiation therapy.

Given the mixed results from the two GBM animal models, it is not clear whether GDC-0084 will be effective when administered as a monotherapy at the completion of a course of radiotherapy or whether it will need to be administered at the same time as radiotherapy to give maximum benefit.

Phase I clinical trial shows a trend to efficacy at higher doses

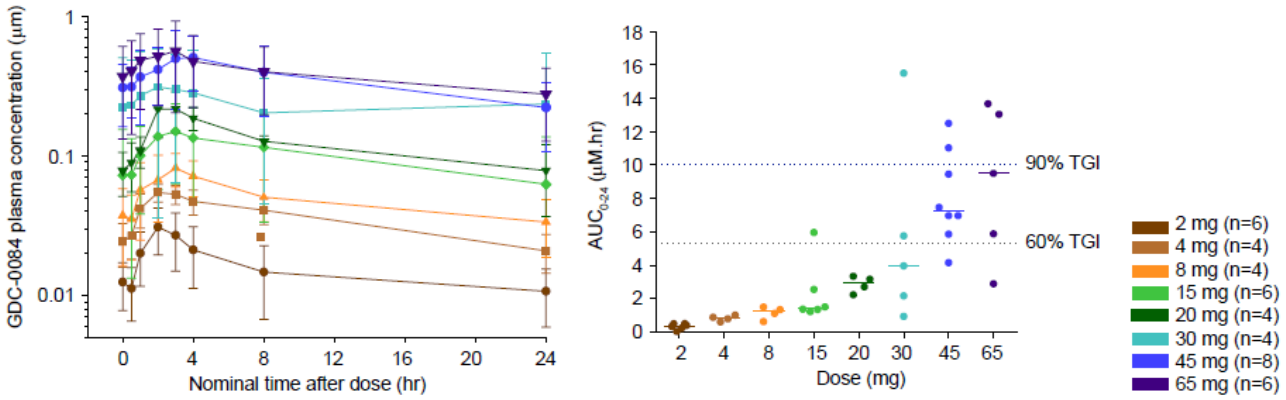
Genentech conducted a Phase I study of GDC-0084 in patients with progressive or recurrent high-grade gliomas (WHO Grade III–IV), including glioblastoma and malignant astrocytoma. The study enrolled 47 patients in eight successive dose escalation cohorts (2-65mg). The maximum tolerated doses (MTD) was identified as 45mg.

Overall, the adverse event profile was consistent with PI3K/mTOR class effects; adverse events at the MTD were amenable to monitoring, manageable and reversible upon dose hold or discontinuation. The most common Grade 3 adverse events related to GDC-0084 were hyperglycaemia (four patients) and mucositis (three patients).

Oral dosing of GDC-0084 demonstrated favourable pharmacokinetics in the Phase I trial, with sustained plasma levels following daily oral dosing (Exhibit 5, left-hand pane).

Seven of the eight patients treated at the MTD of 45mg/day demonstrated levels of drug in the bloodstream over the course of a day (area under the curve, or AUC) that were associated with at least 60% inhibition of tumour growth in preclinical models (Exhibit 5, right-hand pane).

Exhibit 5: GDC-0084 exhibited favourable pharmacokinetics and sustained drug exposure in Phase I



Source: Wen et al 2016 ASCO poster. Note: At the MTD of 45mg (blue bars), exposure to the drug in seven out of eight patients exceeds the levels that correlate with efficacy (60% tumour growth inhibition) in U87 GBM mouse model.

Analysis of tissue samples from a surgical brain specimen from one patient confirmed that the drug crosses the blood-brain barrier in humans, with concentrations seen in healthy brain and in tumour tissue higher than the levels in plasma.

Exhibit 6: GDC-0084 concentration in a surgical brain specimen*

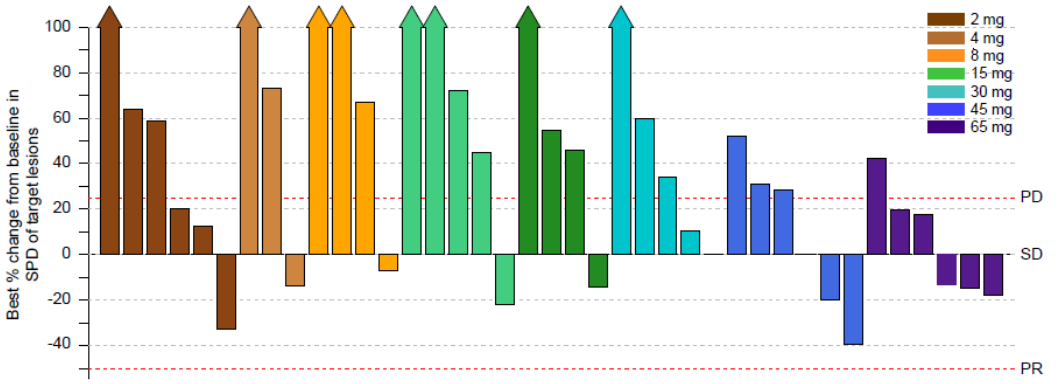
Sample	Total GDC-0084	Free GDC-0084
Plasma	0.56 uM	0.11 uM
Brain Tissue	0.86 uM	0.058 uM
Brain Tumour	0.80 uM	0.054 uM**

Source: Morrissey, Vora et al ASCPT 2016 poster. Notes: *Resection of brain tissue and tumour from a patient dosed at 45 mg QD; samples obtained 5.5 hours (plasma) and 7 hours (brain) post-dose. **Assumes same binding as brain.

Exhibit 7 summarises the tumour responses for the patients in the Phase I study, grouped by dose cohort. Although none of the tumours reached the 50% reduction in tumour size that would qualify as a partial response, a dose response in tumour growth was apparent, with much less tumour growth in patients treated at 45mg (the MTD) or higher doses.

It is important to note that the Phase I study was performed in patients with late stage disease and rapidly growing tumours. In the planned Phase II trial Novogen will be testing whether this reduction in tumour growth is sufficient to improve PFS and/or overall survival in patients with early stage disease who have undergone surgical resection and radiotherapy.

Exhibit 7: GBM patients in Phase I trial showed a trend to better disease control at higher doses of GDC-0084



Source: Wen et al 2016 ASCO poster. Note: Maximum tolerated dose was identified as 45mg (blue bars).

Targeting the majority of GBM patients who get minimal benefit from current drug therapy

Novogen anticipates conducting a randomised Phase II trial of GDC-0084 as adjuvant therapy in newly diagnosed GBM patients with unmethylated MGMT promoter who have undergone surgical resection of the tumour and a six-week course of radiotherapy (XRT) in combination with temozolomide.

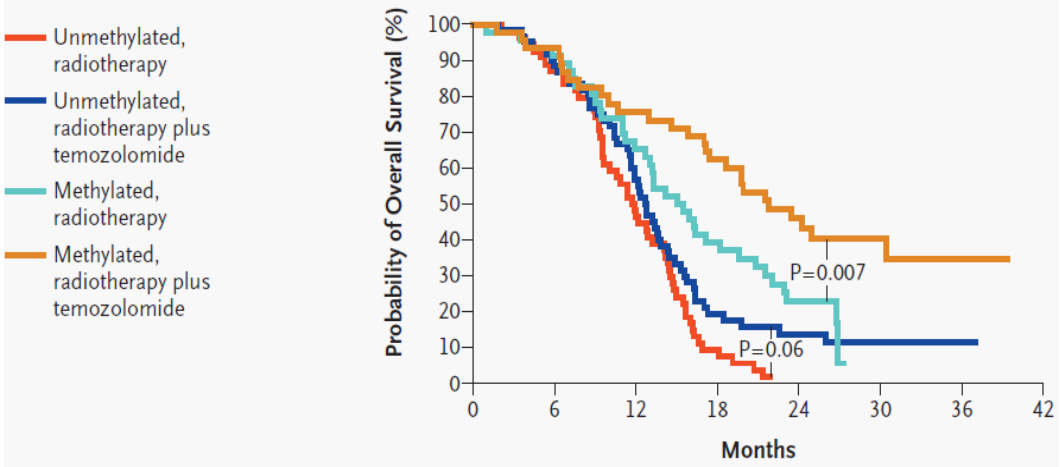
In a seminal study reported in 2005, Hegi et al[4] found that GBM patients with an unmethylated MGMT promoter received only minimal additional benefit when standard of care drug temozolomide was added to radiotherapy in first line treatment of newly diagnosed disease (Exhibit 8).

Exhibit 8 shows that patients whose tumour contained a methylated MGMT promoter received a survival benefit when temozolomide was added to standard radiotherapy; their median survival was 21.7 months, as compared with 15.3 months among those who received only radiotherapy (HR 0.51, P=0.007). In patients with an unmethylated MGMT promoter, there was a smaller and statistically insignificant difference in survival between patients who received temozolomide and radiotherapy (orange line) compared to radiotherapy alone (blue line, 12.7 vs 11.8 months, HR 0.69, P=0.06).

TMZ is an alkylating agent that adds a methyl group to purine bases of DNA, blocking DNA replication and leading to cell cycle arrest and apoptosis. MGMT can repair the DNA by removing the added methyl group, counteracting the cytotoxic effect of TMZ. Methylation of the MGMT promoter silences the gene, reducing MGMT activity in the cell and allowing TMZ to have its desired cytotoxic effect. Low levels of MGMT in tumour tissue are associated with longer survival among patients regardless of whether or not they receive TMZ chemotherapy.

4 Hegi et al *N Engl J Med* 2005;352(10):997-1003

Exhibit 8: Adding temozolomide to radiotherapy in first line GBM provides little benefit to patients with unmethylated MGMT promoter



Source: Hegi et al *N Engl J Med* 2005;352(10):997-1003

As Exhibit 9 shows, the median PFS among five studies of GBM patients with unmethylated MGMT promoter the average median PFS was only 5.2 months and the median OS was 13.8 months. This means that a two-month improvement in either PFS or OS would be a clinically meaningful benefit. It also means that a study in this patient group will be relatively quick to conduct, with PFS data expected to mature less than six months after the completion of recruitment.

Exhibit 9: Overall survival and PFS in GBM with unmethylated MGMT promoter treated with radiotherapy plus TMZ

	Median overall survival (months)	Median PFS (months)	PFS at 6 months	2-year survival rate
Hegi et al NEJM 2005	12.7	5.3	40%	14%
Nabors et al, Neuro-Oncology 2015	13.4	4.1	N/A	N/A
Gilbert et al, JCO, 2013	14.0	5.7	N/A	N/A
AVAGLIO ASCO, 2013	14.6	5.8	N/A	N/A
RTOG-0825, ASCO, 2013	14.3	N/A	N/A	N/A
Average	**13.8**	**5.2**		

Source: Edison Investment Research; Hegi et al *N Engl J Med* 2005;352(10):997-1003; Nabors et al. *Neuro-Oncology* 2015 17(5):708-717; Gilbert et al. *J Clin Oncol* 2013 31(32):4085-4091. Note: RTOG = Radiation Therapy Oncology Group

Brain metastases offer potential upside

Although initial development of GDC-0084 will target GBM, it also has the potential to treat brain metastases for a range for different cancers. Brain metastases are quite common, but there are few drugs available to treat them. Lung, breast and melanoma represent the majority of brain metastases. Genentech has done preclinical studies showing that GDC-0084 improves survival in mouse models of brain metastases in HER2+ breast cancer.

Additionally, even though GDC-0084 has been specifically designed to cross the blood-brain barrier, it is also likely to be effective against tumours elsewhere in the body. However, at this stage we have not included potential applications of GDC-0084 for any indications other than glioblastoma in our valuation model.

IP protection to 2032 in the US

Patent protection for GDC-0084 extends to 2032 in the US and 2031 in other major markets. Given the potential for patent term adjustment to compensate for the time required for clinical studies and regulatory review, we model cash flows for GDC-0084 out to 2035.

Other PI3k inhibitors on the market or under development

Exhibit 10 summarises some of the other PI3K drugs that are on the market or in development.

Idelalisib (Zydelig, Gilead) was approved in 2014 as a second line treatment for chronic lymphocytic leukaemia (CLL) and lymphoma. In March 2016 Gilead halted 6 combination trials of Idelalisib in newly diagnosed patients due to serious side effect including deaths. Idelalisib inhibits the delta isoform of PI3K and inhibiting strongly PI3Kdelta affects the immune system. In contrast, GDC-0084 strongly inhibits PI3Kalpha and only weakly inhibits PI3Kdelta, and has not caused similar side effects in clinical trials. Bayer is undertaking a Phase III study of Copanlisib, which also strongly inhibits PI3Kdelta, in non-Hodgkin's lymphoma.

A number of PI3K inhibitors have been trialled in GBM, but development of most has been discontinued, including voxtalisib (Sanofi/Exelixis) and PX-866 (Cascadian Therapeutics). Other tha2 GDC-0084 the only other PI3K inhibitor that continues to be developed for GBM is BKM120 (buparlisib; Novartis).

BKM120 is being tested in three Phase I/II trials in recurrent GBM in combination with different drugs. A Phase III trial of BKM120 in advanced breast cancer met its primary endpoint of improving PFS (6.9 vs 5.0 months, HR 0.76, p=0.014). However, the high rate of serious adverse events (77% vs 32%) raised questions about the overall benefit of the treatment; mood disorders were common eg depression 26% vs 9%. Mood disorder is an adverse event that is specific to BKM120 and not observed with other PI3K inhibitors. Efficacy was greater in patients with mutations in the PIK3CA gene (PFS 7.0 vs 3.2 months), so there may be further development of BKM120 in this subgroup of patients.

Toxicity was also an issue when BKM120 was trialled as a first line therapy in newly diagnosed GBM patients. Safety data from the Phase I study that reported in 2015 showed that a tolerable RP2D was not determined for concurrent administration of BKM120 in combination with TMZ during radiotherapy due to challenging toxicity. However, a RP2D was identified for adjuvant use in combination with TMZ after radiotherapy was completed, although 6 of the 16 patients developed dose limiting toxicities. No efficacy data was reported.

Novartis' latest pipeline update indicates that it anticipates filing BKM120 for approval in unspecified solid tumours some time from 2020 onwards. Although the target indication has not been specified, we believe that it is most likely to be in breast cancer.

Genentech is continuing to develop the PI3K inhibitor Taselisib (GDC-0032), which inhibits the alpha, delta and gamma isoform of the disease. Taselisib is in a Phase III trial in breast cancer and a Phase II/III trial in squamous cell lung cancer. Both of these diseases have **much** larger patient populations than GBM. We do not know why Genentech chose to develop Taselisib in preference to GDC-0084, but the larger addressable market for Taselisib may have played a role in the decision.

Novartis anticipates filing the PI3Kalpha inhibitor BYL719 for the treatment of advanced breast cancer in combination with fulvestrant in 2019, and for other solid tumours from 2020 onwards. However, this drug is not being developed in GBM.

Exhibit 10: Selected PI3K inhibitors approved or in development

Product	Class	Stage	Indication	Comment
Idelalisib (CAL-101; GS-1101; Gilead/Calistoga)	PI3Kdelta	Marketed	chronic lymphocytic leukaemia (CLL), lymphomas	FDA approved in 2014 for relapsed CLL and lymphoma. In March 2016 Gilead halted 6 combination trials in first line therapy due to serious side effect including deaths.
BKM120 (buparlisib; Novartis)	Pan-class I PI3K	Phase III	breast cancer, lung cancer, head and neck cancer, advanced solid tumours,	BKM120 plus fulvestrant met its primary endpoint of improving PFS in the BELLE-2 Phase III trial in ER+/HER2- advanced breast cancer (6.9 vs 5.0 months, HR 0.76, p=0.014). Efficacy was greatest in patients with PIK3CA mutations (PFS 7.0 vs 3.2 months), but there was a high rate of serious adverse events (77 vs 32%). Three Phase I/II trials of BKM120 in recurrent GBM in combination with different drugs are underway. Based on challenging safety profile in Phase I/II trial development in lung cancer was discontinued (ASCO 2016)
Taselisib (GDC-0032, Genentech)	PI3Kalpha, delta, gamma	Phase III	Metastatic breast cancer, squamous cell lung cancer	Genentech continuing development. Phase III underway in mBC. Phase II/III underway in squamous cell lung cancer
BYL719 (Novartis)	PI3Kalpha	Phase II	Advanced solid tumours (including those with PIK3CA alteration)	Anticipates filing in 2019 for mBC (+ fulvestrant) and for other solid tumours in 2020 onwards. Phase II trials underway include breast, lung, colorectal, head and neck, myeloma
Copanlisib (BAY80-6946; Bayer)	Pan-class I PI3K	Phase III	Advanced solid tumours, non-Hodgkin lymphoma	Phase III in non-Hodgkin's lymphoma
Apitolisib (GDC-0980, RG7422, Genentech)	PI3K/mTOR	Phase II (discontinued)	Lymphoma, breast cancer, prostate cancer, mesothelioma, mRCC.	Not included in Genentech's latest pipeline. In Phase I in solid tumours (n=120) 10 RECIST partial responses, 5 confirmed including 4 in mesothelioma. In Phase II in mRCC n=85 randomised vs everolimus, results favoured everolimus; (PFS 3.7 vs 6.1 months, HR=2.12 P<0.01). In an open label Phase II in endometrial carcinoma (n=56) ORR 6%, median PFS 3.5 months.
Gedatolisib (PF-05212384, Pfizer)	PI3K/mTOR	Phase II	AML, breast cancer, advanced solid tumours	Phase II in AML, Phase 1b underway in breast cancer. Phase I trials underway in combination with docetaxel, cisplatin, dacomitinib, paclitaxel, carboplatin
Voxtalisib (SAR245409, XL765; Sanofi/Exelixis)	PI3K/mTOR	Phase II (discontinued)	Advanced solid tumours, CLL, lymphoma, ovarian cancer	Sanofi has discontinued development. A combination phase 1b/2 trials with temozolomide in glioblastoma showed 4% ORR. Has been tested in a single agent phase 2 trial in Non-Hodgkin's lymphoma, combination studies in mBC, lymphoma, leukaemia.
GDC-0084 (Novogen/Genentech)	PI3Kalpha/ mTOR	Phase I	Glioblastoma	
Pictilisib (GDC-0941; Genentech)	Pan-class I PI3K	Phase II (discontinued)	Breast cancer, NSCLC	No benefit of adding Pictilisib to fulvestrant in ER positive, aromatase resistant BC (HR 0.74 and 1.07 in parts 1 and 2 of the trial. Roche has discontinued this indication. High proportion discontinued due to toxicity
MLN1117 (INK1117; Intellikine)	PI3Kalpha	Phase II	Advanced solid tumours with PIK3CA mutation	Phase II underway in mRCC and endometrial carcinoma; Phase Ib in NSCLC.
GSK2636771 (GSK)	PI3Kbeta	Phase I/II	Gastric adenocarcinoma	Phase I/II underway in advanced gastric adenocarcinoma sponsored by Yonsei University
AMG319 (Amgen)	PI3Kdelta	Phase II	SCCHN	Phase II in head and neck cancer initiated 2015, sponsored by Cancer Research UK
PX-866 (Cascadian Therapeutics (formerly Oncothyreon))	Pan-class I PI3K	Phase II (discontinued)	Advanced BRAF-mutant cancers, NSCLC, prostate cancer	Development discontinued. Phase II in glioblastoma missed primary endpoint; ORR 3%, 6-month PFS 17%. Phase 2 trial (for NSCLC) concluded : "The addition of PX-866 to docetaxel did not improve PFS, response rate, or OS in patients

Source: Edison Investment Research, clinicaltrials.gov, company announcements

Financial impact of GDC-0084 licensing deal

Payment to Genentech

Consideration for the worldwide licence to develop and commercialise GDC-0084 includes a US$5m upfront payment and modest regulatory and commercial milestones (value of milestones not disclosed - we model US$5m payable on market approval). Novogen will also pay royalties in line with industry benchmarks – we assume an average royalty rate of 10% of net sales is payable to Genentech.

Acquisition of Glioblast

As part of the transaction Novogen will also purchase the privately held company Glioblast. The principals of Glioblast, Paul Hopper, an experienced life sciences executive, and Leslie Chong, who

was formerly clinical programme lead for GDC-0084 at Genentech, have strong relationships with the investigators at Genentech and were instrumental in negotiating the transaction; they will become consultants to Novogen on an ongoing basis. Novogen will acquire Glioblast for A$2.1m, comprising A$0.6m in cash and A$1.5m payable as equity.

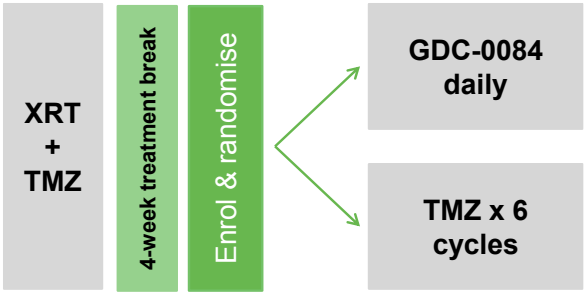
The Glioblast shareholders will also be entitled to four further payments in cash or equity on the achievement of performance-related milestones. The first two milestones of A$1.25m each, payable as equity, will become payable on the commencement and successful completion of a Phase II clinical trial of GDC-0084. The details of the performance hurdles and the payment amount for the final two milestones have not been disclosed, although the company has disclosed that the payments may be made in cash or equity at Novogen's sole discretion. In our forecasts we assume that these payments total A$10.5m (five times the upfront payment), split between approval filing and first marketing approval.

Development plan and trial costs

Novogen anticipates conducting a randomised Phase II trial of GDC-0084 as adjuvant therapy in patients with unmethylated MGMT promoter who have undergone surgical resection of the tumour. We assume that the trial design will be similar to Exhibit 11, where patients undergo a standard six-week course of radiotherapy (XRT) in combination with temozolomide followed by a four-week treatment break, before being randomised to receive maintenance therapy of either daily oral GDC-0084 or six cycles (24 weeks) of temozolomide. One advantage of this design is that GDC-0084 would be used as a monotherapy just as it was in the Phase I trial, so there would be no need for additional safety studies before the Phase II trial could commence.

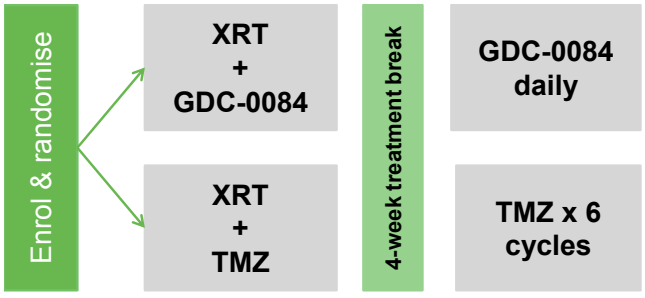
Exhibit 12 shows an alternative trial design where GDC-0084 replaces temozolomide entirely, both during and after the six-week course of radiotherapy. However, at this stage it is not clear whether an additional Phase I study would be needed to confirm that GDC-0084 can be safely administered concurrently with radiotherapy at the MTD of 45mg. In this regard it is encouraging that no additional toxicity was observed when GDC-0084 was combined with radiotherapy in preclinical studies, but this question will need to be discussed with the FDA and other regulators.

Exhibit 11: Presumed Phase II design: GDC-0084 replaces temozolomide during maintenance phase



Source: Edison Investment Research. Note: TMZ=temozolomide; XRT=radiotherapy

Exhibit 12: Alternative Phase II design: GDC-0084 replaces temozolomide entirely



Source: Edison Investment Research. Note: TMZ=temozolomide; XRT=radiotherapy

In our forecasts we take a conservative approach and assume that the Phase II trial follows the design shown in Exhibit 11. Based on discussions with the company and its advisors we assume that the trial will commence recruiting patients in Q2 of CY17, complete recruitment within 15-18 months, and report data for a progression free survival (PFS) primary endpoint in Q1 of CY19. Data for an overall survival secondary endpoint should mature in late CY19. The estimated cost of the 160-patient trial is US$25m.

In this scenario, we assume that the regulators advise that an additional safety study is required before GDC-0084 can be administered during the six-week course of radiotherapy. We assume that in order to satisfy this requirement, such a Phase Ib safety study is conducted while the Phase II

trial is underway at an estimated cost of US$1-1.5m. Successful completion of the Phase Ib study would clear the way to conduct a trial where GDC-0084 is used in conjunction with XRT during the first six weeks of treatment as well as during the subsequent maintenance phase, as shown in Exhibit 12. The longer period of treatment with GDC-0084 could potentially lead to greater efficacy.

Exhibit 13 shows that we estimate that the total cost of the acquiring GDC-0084 programme and completing the Phase II trial and additional Phase Ib trial would be US$35.1m (~A$46m). We note that A$4m of the Glioblast upfront and milestone payments would be payable in scrip and not cash.

Exhibit 13: Summary of costs to acquire GDC-0084 and complete Phase II trial in GBM

	US$m	A$m
Upfront payment to Genentech for GDC-0084 licence	5.0	6.6
Glioblast acquisition and first two milestone payments	3.6	4.6
GDC-0084 trial costs to FY19	26.5	34.9
Total expenditure	35.1	46.1

Source: Novogen company announcement, Edison Investment Research estimates. Note currency converted at A$/US$0.76.

Valuing GDC-0084

GBM revenue opportunity

There are approximately 11,500 new cases of GBM in the US each year[1]. Sixty-one per cent[2] of patients have unmethylated MGMT promoter and would be eligible for first line therapy with GDC-0084. As the only currently-approved chemotherapy for first line GBM has only minimal efficacy in this group of patients, we assume GDC-0084 would get 80% uptake among the eligible patients if approved. Assuming a price of US$50,000 per patient and assuming 4% annual market growth, we estimate peak sales potential in the US in 2030 at US$525m. We estimate that global peak sales would be double the US sales, giving global peak sales of US$1,050m in 2030.

Two development/valuation scenarios for GDC-0084

We have assessed the impact of the GDC-0084 licence deal under two different development scenarios; the first scenario assumes accelerated approval at the completion of the Phase II trial, and in the second scenario an additional clinical trial is required to be performed by a partner prior to approval filing.

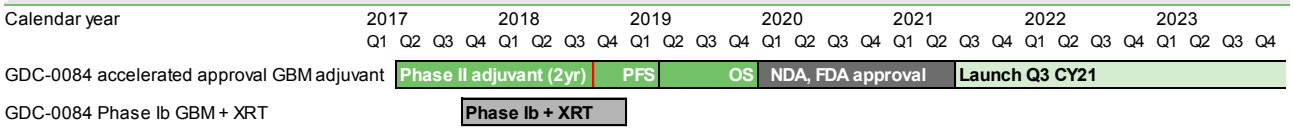
Approximately 60% of drugs that enter clinical development fail in Phase II, which makes this the riskiest stage of development. If the Phase II trial results are positive we assume that Novogen would out license the drug to a partner, which would complete clinical development (if further trials were required) and market the drug if approved.

If the trial results are positive we envisage two potential development scenarios.

In scenario 1 there is a statistically significant improvement in PFS, which is sufficiently meaningful to justify filing for accelerated approval. Although PFS data could be available in Q119, we take a conservative approach to timing and assume that Novogen (or a partner) waits for OS data to mature in Q419 before filing for approval. This scenario could potentially see the drug launched in Q321 as shown in Exhibit 14. We note that if the approval filing is based on PFS data alone the launch could potentially come nine months earlier, in Q420.

We assume that a Phase Ib trial to identify the MTD for GDC-0084 when used in combination of radiotherapy will be conducted while the Phase II trial is underway. This would ensure that a Phase II combination therapy trial could commence in 2019 if required. We expect 6-12 patients would be recruited for this trial.

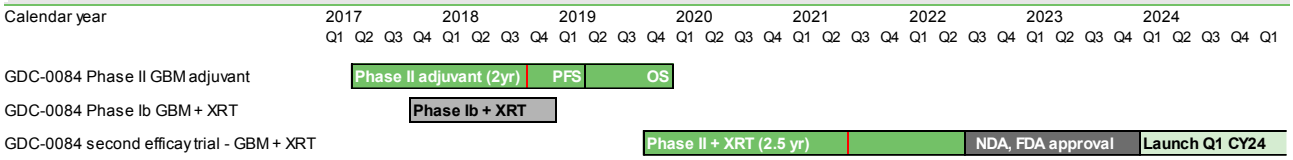
Exhibit 14: Assumed clinical trial and approval timeline for GDC-0084 under two scenarios



Source: Edison Investment Research

Scenario 2 assumes that the results of the first Phase II trial indicate that GDC-0084 is effective against GBM, but that an additional clinical trial is required before filing for approval. The additional study could be a Phase III trial to demonstrate an overall survival benefit, or it could be a pivotal Phase II study of GDC-0084 used during the six-week course of radiotherapy as well as during the maintenance phase after radiotherapy is completed. We anticipate that this second trial could be completed in 2.5 years if it uses the same trial sites as the initial trial.

Exhibit 15: Assumed clinical trial and approval timeline for GDC-0084 under two scenarios



Source: Edison Investment Research

Our valuation of GDC-0084 includes risk-adjusted upfront payments and clinical/regulatory milestones (but not sales milestones) from potential licensing deals. We assume that GDC-0084 is sub-licensed to a marketing partner in mid-2019 after reporting PFS data from the Phase II trial.

For scenario 1 we assume that the licence deal includes a US$40m upfront payment and US$120m in clinical and regulatory milestone payments (we risk-adjust milestone payments in our forecasts). These payment amounts are based on a review of Phase II oncology drug licencing deal metrics from BioCentury. Upfront payments from these deals averaged US$38m, and total clinical, regulatory and sales milestones averaged US$300m. We assume 40% of the milestone payments (US$120m) are for clinical and regulatory milestones.

We assume Novogen receives a 20% royalty on net sales under this scenario because the data is ready for filing, and that Novogen pays a royalty of 10% to Genentech.

Exhibit 16 shows that under scenario 1 we value GDC-0084 at A$54m, with royalties contributing 67% of the value.

Exhibit 16: Valuation GDC-0084 under scenario 1 – accelerated approval in FY21

	Risk-adjusted NPV		Unrisked NPV	
	US$m	A$m	US$m	A$m
Royalties (receives 20% royalty, pays 10% to Genentech)	40.8	53.7		
Upfront payments and milestones, net of final two Glioblastoma milestones	20.2	26.6		
Acquisition and trial costs	-20.4	-26.8		
Total GDC-0084 valuation under scenario 1	40.7	53.5	212	279

Source: Edison Investment Research. Note: NPVs are after 30% corporate tax, adjusted for accumulated losses; discount rate 12.5%. Global peak sales US$1,050m in 2030.

For scenario 2 we assume a smaller US$20m upfront payment due to the additional clinical trial required to be conducted by the licensee, but the same US$120m in milestones. We assume Novogen receives a typical Phase II royalty of 15% and pays a 10% royalty to Genentech.

Exhibit 17 shows that under scenario 2 we value GDC-0084 at A$11m, with royalties contributing 48% of the value.

Exhibit 17: Valuation GDC-0084 under scenario 2 – approval in FY24 after a second trial

	Risk-adjusted NPV		Unrisked NPV	
	US$m	A$m	US$m	A$m
Royalties (receives 15% royalty, pays 10% to Genentech)	14.0	18.4		
Upfront payments and milestones, net of final two Glioblastoma milestones	14.9	19.6		
Acquisition and trial costs	-20.4	-26.8		
Total GDC-0084 valuation under scenario 2	8.5	11.2	79	104

Source: Edison Investment Research. Note: NPVs are after 30% corporate tax, adjusted for accumulated losses; discount rate 12.5%. Global peak sales US$1,050m in 2030.

Our analysis suggests that the most attractive aspect of GDC-0084 is the potential for it to gain accelerated approval following a single Phase II trial, which offers the potential for substantial financial returns to shareholders. If a second efficacy trial is required, delaying approval to 2024, then the returns would be more modest.

The first drug from Novogen's in-house pipeline is ready to enter the clinic

Novogen is currently developing two classes of drugs that have performed strongly in preclinical efficacy studies (Exhibit 18). Its super-benzopyran drugs, Cantrixil and Trilexium, have performed strongly in an ovarian cancer stem cell model developed by Yale University, and have shown strong efficacy in cell lines from a range of cancers. Novogen's second drug class, anti-tropomyosins (ATM), including lead candidate Anisina, show strong synergy with anti-mitotic drugs, such as vinca-alkaloids, that are standard-of-care for a number of cancers. The chemo-sensitising effect of the ATMs in combination with vinca-alkaloids is expected to have a significant clinical benefit, as it may reduce the dose of anti-mitotic drugs that are administered, which would result in a corresponding decrease in associated toxicity. Combination therapy may also slow the development of drug resistance.

While Novogen's drug candidates have shown a considerable promise in preclinical studies to date, they are still at a relatively early stage of development. Its most advanced in-house drug, Cantrixil, is scheduled to commence a Phase I trial in ovarian cancer in Q416. The company announced in early September that the FDA has allowed the IND submission, clearing the way for the drug to be administered to patients in clinical trials.

Exhibit 18: Novogen's in-house product pipeline

Drug candidate	Indication	Stage	Next steps
Cantrixil	Ovarian cancer	Phase I ready	Complete preclinical toxicology studies. First-in-man study in Q416.
Anisina	Melanoma	Preclinical	Complete manufacturing scale-up and animal efficacy studies. Initiate preclinical toxicology studies. First-in-man study in 2017.
	Prostate cancer	Preclinical	
	Neuroblastoma	Preclinical	Obtain dedicated third-party funding for Phase I study.
Trilexium	Melanoma	Preclinical	Complete animal efficacy studies. Initiate preclinical toxicology studies.
	Brain cancer	Preclinical	Complete animal efficacy studies.

Source: Edison Investment Research

Valuation

Our valuation of Novogen's existing pipeline is virtually unchanged at A$112m (previously A$113m) or A$0.26/share (undiluted, unchanged) and A$0.23/share after diluting for options and convertible notes, based on a risk-adjusted discounted cash flow model. Novogen is also listed on NASDAQ under the code NVGN, with each NASDAQ-listed ADR representing 25 ordinary shares. Our undiluted valuation equals US$4.95 per ADR at current exchange rates (previously US$4.97 per ADR). Note that the per-share value does not account for the shares that will be issued as part of the acquisition of Glioblast (A$1.5m in shares) or the Glioblast milestone payments (potentially

A$1.25m of shares in FY17 on initiation of Phase II, and a further A$1.25m in FY19 on successful completion of the Phase II).

The benefit from the higher 10% success probability assigned to Cantrixil, now that the FDA has accepted the IND for a Phase I trial, expected to commence in Q416, has been offset by a reduction in our assumed market growth rate from 5% to 4%.

Our valuation reflects our understanding of the likely development path for Novogen's three lead drugs Cantrixil, Anisina and Trilexium. Although we believe that our valuation model represents a likely development scenario, it should be considered as indicative because the choice of cancer indications that are eventually developed will be influenced by the ongoing preclinical efficacy studies, as well as by which patients show initial signs of efficacy in the pending Phase I trials.

Our cash flow forecasts extend out to 2035, but do not include any terminal valuation and apply a 12.5% discount rate. In calculating the diluted NPV/share, we assume that the A$1.5m Triaxial convertible note is converted to 60m shares (the A$1.5m convertible note was issued as part of the purchase of Triaxial and its SBP technology). The conversion of the notes is subject to achieving specified clinical milestones: 20m can be converted on IND allowance; 16m on completion of Phase I trials; and 24m on completion of Phase II trials.

Exhibit 19 shows our market assumptions for Cantrixil, Trilexium and Anisina and the contribution of product royalties and milestone payments to the rNPV. We have offset the risk-adjusted trial cost against milestone revenue for each drug, rather than against royalty revenue. This understates the contribution of the milestone payments to the rNPV and overstates the contribution of royalties.

Exhibit 19: Novogen sum-of-the-parts DCF

	Base case likelihood (%)	rNPV (A$m)	rNPV/sh (A$)	Assumptions
Ovarian and other abdominal cancers: Cantrixil	10%	28.6	$0.07	Global peak sales* of US$680m from ovarian cancer (14,300 US deaths/yr, 30% penetration) and bowel cancer (50,300 US deaths, 25% develop malignant ascites, 20% penetration); pricing of US$50k. Global sales 2x US sales; launch 2024; assume receives 15% royalty on net sales, pays away 5% of revenue to Yale.
Prostate cancer: Anisina	7.5%	22.5	$0.05	Global peak sales of US$880m assuming 29,500 US deaths/yr; 30% penetration; pricing of US$50k. Global sales 2x US sales; launch 2025; assume receives 13% net royalty.
Melanoma: Anisina	7.5%	7.1	$0.02	Global peak sales of US$$300m assuming 9,700 US deaths/yr; 30% penetration; pricing of US$50k. Global sales 2x US sales; launch 2025; assume receives 13% net royalty.
Paediatric neuroblastoma: Anisina	7.5%	0.6	$0.00	Global peak sales of US$25m assuming annual US incidence of 700 cases, 45% moderate to high risk, 80% penetration; pricing of US$50k. Global sales 2x US sales; launch 2025; 13% net royalty on sales.
Melanoma: Trilexium	5.0%	4.6	$0.01	Global peak sales of US$$300m assuming 9,700 US deaths/yr; 30% penetration; pricing of US$50k. Global sales 2x US sales; launch 2026; assume receives 15% royalty on net sales.
Brain cancer: Trilexium	5.0%	4.3	$0.01	Global peak sales of US$300m assuming annual US incidence of GBM of 11,500 cases, 25% penetration; DIPG US incidence 275, 80% penetration; pricing of US$50k. Global sales 2x US sales; DIPG launch 2026; 15% royalty on net sales.
Cantrixil milestones		12.2	$0.03	Assumes potential licensing upfronts and milestones total US$140m (US$23m after risk adjustment); assume 5% of upfront and milestone payment paid away to Yale.
Anisina milestones		9.2	$0.02	Assumes potential licensing upfronts and milestones total US$140m (US$23m risk adjusted).
Trilexium milestones		4.0	$0.01	Assumes potential licensing upfronts and milestones total US$140m (US$14m risk adjusted).
SG&A to 2020		-14.5	-$0.03	
Portfolio total		**78.6**	**$0.18**	
Cash (30 June 2016)		33.5	$0.08	
Enterprise total		**112.1**	**$0.26**	

Source: Edison Investment Research. Note: *Peak sales in 2015 dollars based on current addressable market. Actual peak sales forecast is higher due to market growth. We assume that the addressable markets grow at 4% per year.

Under development scenario 1 our valuation for Novogen including the GDC-0084 transaction would increase to A$165.5m or A$0.39/share (undiluted) and A$0.34/share after diluting for options and convertible notes.

Under development scenario 2 our valuation including GDC-0084 would be to A$123.3m or A$0.29/share (undiluted) and A$0.25/share after diluting for options and convertible notes.

Sensitivities

The key question regarding GDC-0084 is whether it works sufficiently well as a single agent in adjuvant therapy to justify accelerated approval in line with scenario 1. If it needs to be used concurrently with radiotherapy to deliver sufficient efficacy in the target population then one or more additional efficacy trials may be required, as outlined in scenario two, delaying potential launch until 2024. There is a significant risk that GDC-0084 does not provide sufficient survival benefit to justify approval either as a single agent or combination therapy.

We have assumed that a Phase Ib study of GDC-0084 in combination with radiotherapy is conducted in parallel with the planned Phase II trial, but that will be dependent on adequate funding.

We do not know the details of the final two milestones payable to Glioblast and have had to rely on our own assumptions. From our discussions with management we believe that our analytical assumptions (A$10.5m combined value, payable on filing and approval) gives a reasonable guide to the potential economic impact of the Glioblast milestones.

While Novogen has funds to initiate the Phase II study of GDC-0084 in GBM, it would require additional funds of A$42m over 2018 and 2019 to complete all the planned trials from the existing portfolio (including GDC-0084), which could result in significant dilution of existing shareholders.

Our valuation includes revenues from the development of four drugs in six disease indications, as well as (risk-adjusted) upfront and milestone payments for three licensing deals. While each of these targeted indications is supported by the current preclinical efficacy studies, and evidence of a dose response in the GDC-0084 Phase I trial, the company may not ultimately pursue development of the drugs for all of these indications. On the other hand, ongoing preclinical efficacy studies could identify additional disease indications that should be investigated in clinical trials. While we believe that the drug development timelines used in our forecasts are achievable, at this early stage it is hard to accurately predict how long it will take to get the drugs to market.

Financials: Additional funds likely required in FY18

FY16 results (year ending 30 June) showed a net loss of A$12.1m, 69% larger than the previous corresponding period (pcp). Normalised after tax loss of A$11.6m was 38% larger than the previous year. R&D expense was A$9.9m compared to A$5.9m in FY15, reflecting the ramp-up of R&D activities, including preparations for the planned Cantrixil clinical trial. Administration expenses were A$5.8m vs A$3.8m pcp. Cash at 30 June was A$33.5m.

We have updated our financial forecasts to reflect the FY16 results and the payments associated with the in-licensing of GDC-0084, the acquisition of Glioblast and funding the proposed Phase II trial. This has increased forecast R&D expenditure by 50% to A$19.6m in FY17 and by 90% to A$30.5m in FY18. We have assumed that the development of Novogen's in-house drug candidates continues in line with our existing timelines. We estimate that Novogen has sufficient funds to support operations to the end of FY17, but will require addition funds of ~A$20m in FY18 and a further A$22m in 2019 if all three of Cantrixil, Trilexium and Anisina progress to clinical trials in addition to the GDC-0084 Phase II. Note that we include unrisked clinical trial costs in our financial forecasts to show the potential funding requirement if the clinical trial programme is conducted in line with our expectations (trial costs risk-adjusted for NPV calculation).

Exhibit 20: Financial summary

A$'000s	2014	2015	2016	2017e	2018e
Year end 30 June	AASB	AASB	AASB	AASB	AASB
PROFIT & LOSS					
Sales, royalties, milestones	0	0	0	0	0
Other (includes R&D tax rebate)	342	1,637	3,665	4,900	4,725
Revenue	342	1,637	3,665	4,900	4,725
R&D expenses	(2,476)	(5,935)	(9,894)	(19,579)	(30,474)
SG&A expenses	(3,695)	(3,269)	(5,120)	(5,744)	(6,010)
Other	0	0	0	0	0
EBITDA	(5,829)	(7,567)	(11,349)	(20,423)	(31,759)
Operating Profit (before GW and except.)	(5,291)	(7,572)	(11,421)	(20,541)	(31,853)
Intangible Amortisation	(570)	(570)	(570)	(66)	(61)
Exceptionals	0	1,116	(569)	0	0
Operating Profit	(5,861)	(7,026)	(12,560)	(20,607)	(31,914)
Net Interest	(628)	(280)	406	669	282
Profit Before Tax (norm)	(7,569)	(8,422)	(11,586)	(19,938)	(31,632)
Profit Before Tax (reported)	(7,569)	(7,306)	(12,154)	(19,938)	(31,632)
Tax benefit	0	0	0	0	0
Profit After Tax (norm)	(7,569)	(8,422)	(11,586)	(19,938)	(31,632)
Profit After Tax (reported)	(7,569)	(7,306)	(12,154)	(19,938)	(31,632)
Average Number of Shares Outstanding (m)	156.7	238.4	427.4	429.7	429.7
EPS - normalised (c)	(4.76)	(2.99)	(2.84)	(4.64)	(7.36)
EPS – diluted (c)	(4.76)	(2.99)	(2.84)	(4.64)	(7.36)
Dividend per share (A$)	0.0	0.0	0.0	0.0	0.0
BALANCE SHEET					
Fixed Assets	2,021	1,491	1,427	1,243	1,188
Intangible Assets	1,960	1,390	822	756	696
Tangible Assets	14	85	592	473	479
Investments	47	16	13	13	13
Current Assets	2,638	44,649	34,090	14,723	3,548
Stocks	0	0	0	0	0
Debtors	66	151	199	199	199
Cash	2,502	44,371	33,453	14,086	2,912
Other	70	127	438	438	438
Current Liabilities	(3,247)	(1,777)	(1,432)	(1,432)	(1,432)
Creditors	(259)	(1,619)	(1,300)	(1,300)	(1,300)
Short term borrowings	(2,707)	0	0	0	0
Other	(281)	(159)	(132)	(132)	(132)
Long Term Liabilities	0	0	(154)	(154)	(20,154)
Long term borrowings	0	0	0	0	(20,000)
Other long term liabilities	0	0	(154)	(154)	(154)
Net Assets	1,412	44,362	33,931	14,380	(16,850)
CASH FLOW					
Operating Cash Flow	(5,709)	(5,759)	(12,383)	(20,036)	(31,356)
Net Interest	0	0	405	669	282
Tax	0	0	0	0	0
Capex	(27)	(97)	(525)	0	(100)
Acquisitions/disposals	0	8	3	0	0
Equity Financing	2,793	47,415	782	0	0
Dividends	0	0	0	0	0
Other	0	0	0	0	0
Net Cash Flow	(2,943)	41,566	(11,719)	(19,367)	(31,175)
Opening net debt/(cash)	(1,323)	205	(44,371)	(33,453)	(14,086)
HP finance leases initiated	0	0	0	0	0
Other	1,416	3,011	800	0	0
Closing net debt/(cash)	205	(44,371)	(33,453)	(14,086)	17,088

Source: Novogen accounts, Edison Investment Research

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