

Bionomics Limited BNO Updated – Wednesday 27 June 2007

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Raises \$10m to take BNC105 into Phase I/IIa Clinical Trials

The Funds to Advance the Programmes

This week, the company raise \$10m to take the anti-cancer investigational drug BNC105 into Phase I/IIa clinical trials and undertake further development of the anti-anxiety investigational drug BNC210 through further preclinical development.

These funds will take the company to the next stock price re-valuation inflection point, being Phase I/IIa clinical trials.

The company now has estimated \$13m cash

Anticipated Events and Project Pipeline

The most advanced is the anticancer investigational drug, BNC105. The company has previously demonstrated in preclinical studies that BNC105 is a member of a new class of anticancer compounds called a Vascular Disrupting Agent (VDA). Bionomics will now commence manufacturing scale-up and formal toxicology studies with the objective of filing an IND application with the FDA. Bionomics intends to begin clinical trials of BNC-105 in late 2007.

BNC210 is the second most developed drug candidate in Bionomics pipeline.

Programme	Anticipated Event	Date
Cancer Programme (BNC105):	Preclinical Completed and IND submitted to FDA	Q3 07
	Phase I/IIa Initiated	Q4 07
MS Programme:	Drug Candidature Selection	Q4 07
Anxiety Programme (BNC210):	Large Scale Manufacture	Q1 08
	Toxicology Completed and IND Submission	Q3 08
	Phase I Initiated	Q4 08
Epilepsy Programme:	Drug Candidature Selection	Q2 08

Synopsis

Bionomics has focused on developing unique compounds with modes of action and clinical performance superior to the compounds in the market or undergoing clinical trials. The lead compound, BNC-105, is demonstrating qualities that could make it a highly successful compound, if future human clinical trials are successful.

- This investigational drug has blockbuster potential.
- The BNC-105 Phase I/IIa clinical trials are expected to be a revaluation trigger for the company.

The results for second most advanced compound, BNC210, for the treatment of anxiety and depression are very encouraging. Although the results are early stage, the data is statistically significant compared to the control. Separately, the speed of preclinical development of BNC210 highlights the value of Bionomics' investigational drug discovery and development platform.

Snapshot

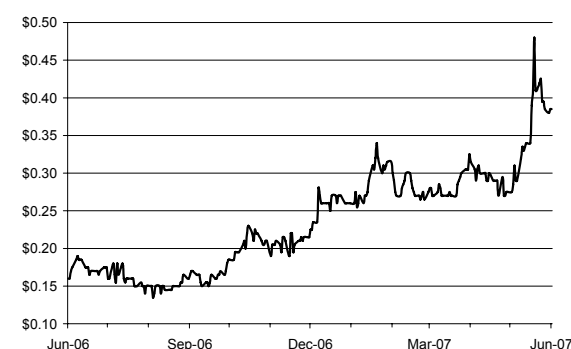
Last Price	\$0.39
Market Cap (m)	\$85.9m
52 Week High	\$0.48
52 Week Low	\$0.14
Sector	Pharmaceuticals & Biotechnology

Investment Fundamentals

Year-end June	FY05A	FY06A	FY07E	FY08E
NPAT (\$m)	(4.6)	(5.6)	(5.8)	(6.8)
EPS (c)	(4.1)	(3.5)	(2.6)	(3.1)
% Change	NA	NA	NA	NA
DPS (c)	0.0	0.0	0.0	0.0
Franking (%)	0.0	0.0	0.0	0.0
Yield (%)	0.0	0.0	0.0	0.0
PER (x)	NA	NA	NA	NA

Source: Intersuisse Estimates (accounting for recent capital raising)

Price Chart



Business Description

Bionomics Limited is a South Australian-based company with a promising preclinical pipeline of novel drug candidates. The most advanced compound, BNC-105; is an investigational anti-cancer compound targeting the blood vessels that feed tumours. Pre-clinical studies demonstrated that BNC-105 is more effective than more advanced similar drugs, currently in human clinical trials.

Analyst: Darren J. Grubb PhD MBA GDipAppFin

Strategic Direction

The 2006 acquisition of Iliad Chemicals proved to be a company-making deal. The promising lead compound BNC-105 came from Iliad's compound library. BNC-105 targets cancer, which is aligned with Bionomics board strategy. In addition, development of BNC-105 is not placing strain of the internal resources of the company.

The result has been the rapid preclinical development of BNC-105, which is anticipated (assuming it is successful in preclinical studies) to enter Phase I Clinical Trials around Q307.

This complements the company's endeavours into other fields of cancer research, which in February 2006 lead to the licensing of cancer targets to Genmab for antibody therapeutic development.

The company also has a strong focus on epilepsy and anxiety, with promising compounds in preclinical and lead optimisation development stage, as well as generating revenue from licensing of epilepsy diagnostic tests to LABCORP® and Athena Diagnostics Inc.

Summary of Drug Development

	Screening	Chemistry	Preclinical	Clinical
BNC105 (Cancer)	██████████	██████████	██████████	██████████
BNC210 (Anxiety Disorders)	██████████	██████████	██████████	
Kv1.3 Blockers (MS)	██████████	██████████	██████████	
GABA-A Agonists (Epilepsy)	██████████	██████████	██████████	

Cancer Investigational Drugs

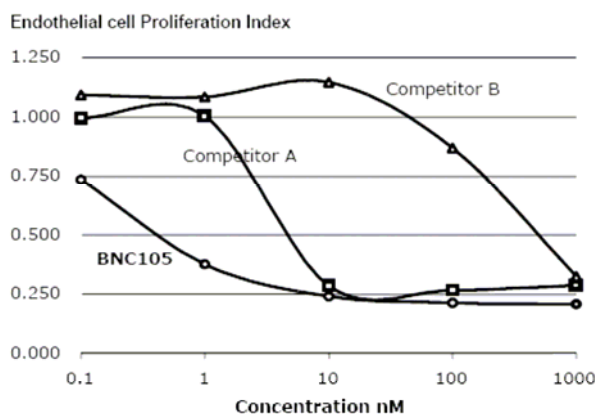
The company's lead compound is BNC-105, as been accelerated internally because of very promising preclinical and *in vitro* results. BNC-105 is a type of compound referred to as a Vascular Disrupting Agent (VDA)

BNC-105 for Cancer

Selected as the lead compound in April 2006, BNC-105 is currently in animal and functionality *in vitro* studies. BNC 105 is a powerful VDA, which prevents cancer growth by disrupting blood vessels around solid tumours. The compound does not disrupt 'normal' blood vessels.

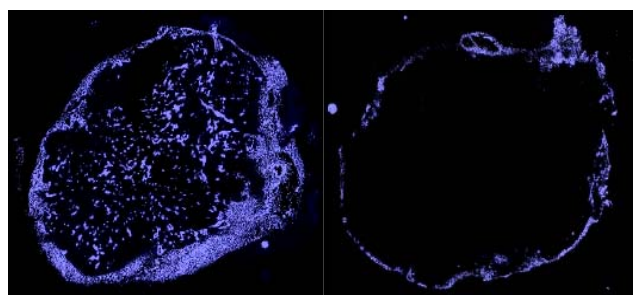
Therapeutic Synergies

Importantly, BNC-105 is 10 fold more active and less toxic than a competitive product currently in later-stage clinical trials. Preclinical studies comparing BNC-105 against CA4P and ABT-751 suggested that BNC-105 has greater efficacy to its competitors.



The graph demonstrates the increased potency of BNC-105 in an *in vitro* assay, tested alongside two competitive vascular inhibitors. Competitor A is CA4 and competitor B is ABT-751. The data suggests that BNC-105 may have great efficacy and a wider therapeutic window than its competitors.

VDAs are more effective when used in combination with other anti-cancer agents that target the peripheral rim of malignant tumour. Animal studies with BNC-105 in combination with Avastin, 5-FU or doxorubicin not only slowed tumour growth, but prevented tumour growth. Animal studies demonstrated that BNC-105 was capable of necrosing the central core of a tumour, whilst Avastin, 5-FU and doxorubicin targeted the perimenteral cancer cells.



The photo demonstrates the efficacy of the compound. The photo on the left is solid tumour. The photo on the right is tumour after treatment of a mouse with BNC-105. The central core of the tumour is necrosed and the peripheral remains of the tumours may be further reduced by treatment with conventional chemotherapeutics.

Mode of Action

BNC-105 binds tubulin at the colchicine site. At low concentrations it interferes with microtubule dynamics inhibiting their ability to go through cycles of tubulin polymerisation. Tubulin polymerisation and degradation is an essential process in cell stability and mostly active in dividing cells. The process of preventing division of tumour cells is a strategy used by the marketed taxane and vinca-binding drugs, although taxanes and vinca alkaloids bind to different sites of the tubulin heterodimer, preventing formation of polymerised tubulin.

Manufacture

On 25 October 2006, Bionomics and SAFC Pharma (Sigma-Aldrich Corporation) signed an agreement where SAFC will produce 1½ kilograms of BNC-105. This is enough material to complete preclinical and Phase I/IIa Clinical Trials. The compound is relatively easy to synthesize. In April 2007, the company reported it completed GMP manufacture of BNC105. Bionomics remains on track to file an Investigational New Drug (IND) application with the US FDA and to commence human trials of BNC105 later in 2007.

BNC105 synthesis is the relatively simple 6-reaction synthetic pathway, of which only reactions 4 to 6 must be performed under GMP conditions. A small amount of the 1.5kg yield is currently being used in toxicology testing and the remainder will be used in the first clinical trial.

Competition

Vascular targeting agents (VTAs) and vascular disrupting agents (VDAs), have two distinctly different modes of action. VTAs are anti-angiogenesis compounds designed to keep new blood vessels from forming and do not directly destroy the blood vessels that nurture tumours. On the other hand, VDAs operate by destroying the endothelium of solid tumours resulting in starvation of tumour cells and resulting in widespread necrosis of established tumours¹.

VTAs are the predominant field of investigation, spurred on by the success of agents such as Avastin®. There is significant research being undertaken in this field.

Angiogenesis inhibitors interfere with new vessel formation and have a preventative action. These require chronic administration, and are likely to be of particular benefit in early stage or asymptomatic metastatic disease. On the other hand VDAs target the established tumour blood vessels, resulting in tumour ischemia and necrosis. These agents are therefore given acutely, show immediate effects and may have particular efficacy against advanced disease².

There are two classes of VDA's, the Flaviniods and the Tubulin-Binding Agents

DMXAA (AS1404)

DMXAA is the only Flaviniod demonstrated to be a VDA and induces the synthesis of TNF- α in tumours, causing vascular shutdown.

A successful Phase I clinical trial was completed in 63 patients. DMXAA is currently in Phase II clinical trials in the United States for the treatment of prostate cancer (sponsored by Antisoma Research).

In this trial, a DMXAA/docetaxel regimen is being compared with docetaxel alone. Furthermore, DMXAA is undergoing three Phase II clinical trials in combination with carboplatin and paclitaxel for the treatment of lung, ovarian and prostate cancer.

On 19 April 2007, Novartis and Antisoma signed a deal worth up to US\$890m for DMXAA

Tubulin-Binding Agents

Chemotherapeutics targeting the tubulin are currently used in standard anti-cancer therapy. Many of the anticancer agents act on microtubules and arrest mitosis. Microtubule effectors interfere with microtubule assembly. There are three major classes of microtubule effectors that bind tubulin in different sites. Taxanes stabilise microtubules by blocking disassembly.

The other two classes are vinca alkaloids and colchicine analogues. These compounds destabilise microtubules by inhibiting assembly of tubulin molecules³. The VDAs bind to the Colchicine site of tubulin, resulting in destabilisation of the polymerised protein resulting in cell death. This is similar to BNC-105's mode-of-action¹.

CA4P

CA4P is a phosphate pro-drug of the tubulin-binding agent combretastatin A-4 (CA4), an inhibitor of tubulin polymerisation. Systemic administration of CA4P causes rapid and selective vascular shutdown in a range of human xenografted and rodent tumours. Dephosphorylation of CA4P by endogenous phosphatases yields CA4, which has a high-affinity for tubulin at (or near) the colchicine-binding site, causing depolymerisation of tubulin dimers in the cellular cytoskeleton.

The sensitivity of the immature tumour vasculature to CA4P probably relates to not only differences between newly formed endothelial cells, but also to characteristics of the tumour microcirculation. CA4P significantly reduces vascular function, even at low doses.

It is of interest that, despite extensive cancer cell death observed after the vascular shutdown, the tumour returned. This has been attributed to rapid re-growth from the rim of viable tumour cells surviving at the tumour periphery. These remaining cells are the most sensitive to conventional cytotoxic, which is why CA4P is an effective adjunct therapy with other chemotherapeutics. In order to combat the

¹ Bioorganic & Medicinal Chemistry 15 (2007) 605–615

² Bioorganic & Medicinal Chemistry 15 (2007) 605–615

³ British Journal of Pharmacology (2005) 145, 1076–1083.

doi:10.1038/sj.bjp.0706276; published online 13 June 2005

tumour cells which remain, CA4P has also been studied in combination therapies with conventional chemotherapeutic agents such as cisplatin, taxol, doxorubicin and 5-fluorouracil, as well as radiation. However, this could also be due to the tumours becoming dormant during therapy.

In a Phase I clinical trial in 34 patients was completed. CA4P was rapidly converted to CA4. CA4P was well tolerated with moderate side effects.

Clinical trials have been conducted on (or are in process of) assessing the treatment of advanced solid tumours using CA4P in combination with the chemotherapy drugs Avastin, carboplatin and paclitaxel (sponsored by Oxigene). Furthermore, CA4P is also undergoing two Phase II clinical trials (sponsored by the National Cancer Institute) to test CA4P's ability to stop the growth of anaplastic thyroid cancer alone and in combination therapy with doxorubicin, cisplatin and radiation therapy. To date there have been nine clinical trials involving CA4P worldwide¹.

BNC-105 as been tested in preclinical trials against this compound and BNC-105 demonstrates greater efficacy and a higher potential therapeutic window.

ABT-751

ABT-751 is an oral antimetabolic agent that binds to the colchicine site on tubulin. In preclinical studies, ABT-751 inhibited cellular proliferation of a broad range of human tumour cell lines and xenograft models, including those that are paclitaxel and doxorubicin resistant due to the multi-drug-resistant phenotype.⁴

Although Phase I Clinical Trials identified toxicology as a potential issue, Phase II Clinical Trials (sponsored by Abbott laboratories) is in trials targeting colorectal cancer, lung cancer and renal cell carcinoma. However the company has not reported on the progress of these trials nor the state of current development for the last 18 months.

BNC-105 as been tested in preclinical trials against this compound and BNC-105 demonstrates greater efficacy and a higher potential therapeutic window.

ZD6126

ZD6126 is a pro-drug of the tubulin-binding agent N-acetylcolchicinol, an inhibitor of tubulin polymerisation. ZD6126 causes selective contraction of the proliferating, immature endothelial cells lining tumour blood vessels causing disruption of blood flow.

Despite a large reduction in vascular volume and extensive tumour necrosis after a single dose, a thin viable rim of tumour cells remained adjacent to normal tissue. The surviving tumour cells caused regeneration of the tumour.

However, combining ZD6126 with cisplatin, taxol or radiation eliminated the viable tumour rim resulted in tumour regression

In a Phase I clinical trial, 27 patients were treated with ZD6126 and adverse effects did not appear to be dose related. Phase II clinical trials were conducted for the treatment of metastatic renal cell carcinoma (sponsored by AstraZeneca) and an additional combination trials with oxaliplatin, 5-fluorouracil and leucovorin in subjects with metastatic colorectal cancer.

The trial was suspended due to adverse toxicology¹.

TZT1027

TZT1027 has demonstrated potent and broad-spectrum anti-tumour activity against human tumour xenografts in mice, including refractory ovarian and renal cancers. The agent is a mitotic spindle poison, which inhibits microtubule assembly by interacting with tubulin in the vinca alkaloid-binding domain

In a Phase I clinical trial, 17 patients received TZT1027 and the side effects were high.

A Phase II clinical trial has also been completed within the United States (sponsored by Daiichi Pharmaceuticals and the National Cancer Institute) targeting advanced or metastatic soft tissue sarcoma.

Furthermore, TZT1027 was in two (now suspended) Phase I and Phase II Clinical Trials, sponsored by Daiichi Pharmaceuticals.

The Phase I trial was studying the side effects of TZT1027 with gemcitabine targeting advanced or metastatic solid tumours. The Phase II trial was studying the effectiveness of TZT1027 in treating patients who have progressive locally advanced or metastatic non-small cell lung cancer.

Teikoku Hormone Manufacturing Company, which holds the rights to TZT1027, ended into an agreement with Daiichi Pharmaceuticals in May of 2005 and is seeking a new partner for further clinical research. In addition, on 1 October 2005 Teikoku Hormone Manufacturing Company and Grelan Pharmaceutical Company merged to form ASKA Pharmaceutical Company.

The development status of the compound is unknown¹.

AVE8062

AVE8062 is a pro-drug of the CA4 and the parent compound is released upon exposure to amino peptidase. AVE8062 was found to have more powerful tumour blood flow stasis and anti-tumour effects compared with CA4P. AVE8062 causes rapid shutdown of tumour blood flow and extensive necrosis in experimental tumour models. However, despite the strong tumour-suppressing qualities of AVE8062 it does not produce an immediate reduction in tumour size upon administration.

AVE8062 was licensed from Ajinomoto in July of 2001 by Aventis Pharma Ltd (Sanofi-Aventis) and currently undergoing Phase I clinical studies in Europe and the United States for patients with solid tumours, although no details have been published to date¹.

⁴ Clinical Cancer Research Vol. 11, 6615-6624, September 15, 2005

Oxi4503

Oxi4503 is the pro-drug of the potent tubulin-binding agent CA1 and behaves in a similar manner to CA4P. However, the preclinical evaluation of Oxi4503 shows that not only is it a much more potent agent than CA4P, but it can also induce tumour growth delays and regressions when used as a single agent. This enhanced activity was unexpected based on the *in vitro* data for tubulin-binding and for the inhibition of cell proliferation for the active parent drug, CA1. As regressions are observed, it indicates that Oxi4503, in addition to the vascular effects, is also directly attacking the remaining viable cells at the rim of the tumour mass. One possible explanation is that it is metabolised *in vivo* to the cytotoxic, o-quinone. Supportive evidence has shown that CA1 is metabolised by tumour tissue to an agent that covalently binds to the cellular contents of the tumour.

Recent positive preclinical reports on Oxi4503 have prompted its further evaluation as a VDA. Oxi4503 is currently undergoing Phase I clinical studies for patients with advanced cancer in England (sponsored by Oxigene)¹.

Other Compounds In Development

Newer microtubule-disrupting agents undergoing preclinical testing that are active at or near the colchicine binding site include a series of chromenes developed by Maxim Pharmaceuticals (lead compound MX-116407) and a diketopiperazine, NPI-2358, developed by Nereus Pharmaceuticals.

Anxiety, Multiple Sclerosis and Epilepsy Investigational Drugs

The company has an emerging preclinical pipeline of drugs broadly targeting neurological conditions. The most advanced is BNC-210 targeting anxiety disorders. This compound is in preclinical development. Two other compounds in discovery development are Kv1.3 Blockers targeting multiple sclerosis and GABA-A antagonists targeting Epilepsy.

BNC-210 for Anxiety Disorders

Mouse Studies had demonstrated that BNC210 could be a good drug candidate targeting anxiety and depression. No side effects were observed and this experimental drug demonstrated changes in the behaviour of mice that indicate the compound has potentially clinically useful properties. The three mouse studies that were all positive were the marble burying test, the light/dark test and the elevated plus maze test. Anxiety in mice tested was diminished.

The results were statistically significant. The result also indicated that BNC210 had a milder impact in behaviour than the marketed anti-anxiety drug Diazepam (Valium®). This is positive as 1) diazepam can result in overly exerted behavioural changes and 2) low-dose-dependence. Diazepam is a core medicine in the WHO "Essential Drugs List", and required for a basic health care system. Diazepam

is used to treat a wide range of conditions such as anxiety, insomnia and seizures.

BNC-210 was identified as Bionomics' top compound exhibiting potent activity in various mouse models. The compound also has good physicochemical properties and acceptable stability in microsome incubations. BNC-210 showed similar levels of anxiolytic efficacy to known anti-anxiety drugs, but importantly it was non-sedating in the mouse Modified Open Field model.

There is an approximately 1,000 fold difference between the dose at which BNC-210 causes suppression of anxiety and the dose at which it induces sedation. BNC-210 also has other desirable properties: it is orally active with a plasma half-life in excess of seven hours, which means that it may be able to be used once a day to relieve the symptoms of anxiety.

Kv1.3 Blockers for Multiple Sclerosis

Kv1.3 is a key regulator of the effector-memory T-Cells of the immune system that are the key mediators of disease in MS. Kv1.3 is expressed in a limited range of tissues, lowering the likelihood of side-effects.

The compound has good potency (better than 25nM) and specificity for Kv1.3 over other ion channels. Efficacy has been demonstrated in animal models of MS.

GABA-A Agonists for Epilepsy

The company is developing new generation of epilepsy treatments based upon modulators of the GABA-A receptor.

Directors

Dr Peter Jonson BComm (Hons), MA (Hons), PhD, FAID, FASSA (Chairman) became Chairman of Bionomics in November 2004. Dr Jonson began his career with the Reserve Bank of Australia where he became an internationally-recognised economist and influential policy adviser. He subsequently gained extensive experience at senior levels of the international financial services industry, serving as Chief Executive Officer of Norwich Union's Australian business and Managing Director and then Chairman of ANZ Funds Management. Dr Jonson has also previously chaired the Federal Government's Biotechnology Centre of Excellence Expert Panel and the Major National Research Facilities Committee, both of which were set up to advise Federal Ministers on major strategic and investment decisions affecting the biotechnology sector. Currently he is a director of Village Roadshow Ltd, Pro Medicus Ltd, Australian Aerospace and Defence Innovations Ltd and Metal Storm Ltd, and is the Chairman of the Australian Institute for Commercialisation and the Federal Government's CRC Committee.

Dr Deborah Rathjen BSc (Hons), PhD (CEO and Managing Director) has been biotech executive of almost 20 years, Dr Rathjen joined Bionomics in June 2000 from Peptech Limited, where she was Manager of Business Development and Licensing. Dr Rathjen was a co-inventor of

Peptech's TNF technology and leader of Peptech's successful defence of its key TNF patents against a legal challenge by BASF, providing Peptech with a strong commercial basis for licensing negotiations with BASF, Centocor and other companies with anti-TNF products. Dr Rathjen has experience in research and product development, intellectual property protection, business development and licensing and has licensed a range of products including drug delivery technologies, diagnostics, new therapeutics and cosmetic products. She is an expert in the field of cell biology with specific expertise in infection, inflammation and cancer. Dr Rathjen was until recently Deputy Chair of the Federal Government's Australian Biotechnology Advisory Council, and is a current member of the Prime Minister's Science Engineering and Innovation Council and the Industry Research and Development Board. In 2004 Dr Rathjen was awarded the AusBiotech President's Medal for her significant contribution to the Australian biotechnology industry and in 2006 received a Distinguished Alumni Award from Flinders University.

Dr George Jessup MB, BS, MBIomedEng, MBA (Non-Executive Director) is the co-founder and Managing Director of Start-up Australia, a venture capital company which is active in investing in biotechnology companies. Under his leadership, Start-up Australia has raised \$55 million and has been an early stage investor in more than ten biotechnology companies. He has been in the venture capital industry for more than ten years. Dr Jessup has extensive experience in senior management roles in the medical device and pharmaceutical industries, including a role in the US as Global Director of Medical Affairs of the Medical Device Division of an international pharmaceutical company (American Cyanamid, later acquired by Wyeth). Dr Jessup is a medical graduate and has worked in various clinical and research positions at a number of Sydney teaching hospitals including Prince of Wales, Prince Henry and Royal North Shore Hospitals.

Mr Trevor Tappenden ACA, FAICD (Non-Executive Director) commenced a career as a non-executive director in 2003 after a career with Ernst & Young spanning 30 years. He holds directorships in various private, Government and not for profit organisations. Mr Tappenden was a partner of Ernst & Young from June 1982 to March 2003, during which time he held a variety of positions including Managing Partner of the Melbourne Office 1997 – 1999, Member of the Firm's Board of Partners 1998 – 2001, Head of the Victorian Government Services Group 2001 – 2003 and National Director of the Entrepreneurial Services Division 1993 – 1997.

Financials

For the Half Year ending December 2007, Bionomics had a net operating cash outflow of \$2.3m. Preclinical development costs for BNC-105 increased expenditure, as the FY06a operational cash outflow was \$3.75m. The company had \$7.0m cash at the end of December 2006 and

the additional \$10m raised on the 10th June 2007 places the company in a good position to reach the next revaluation trigger, being Phase I/IIa clinical trials. The company is estimated to have cash of about \$13m.

In FY06, revenues from license agreements and diagnostic royalties were \$2.26m and grants were \$2.30m. The biggest expense was R&D at \$6.81m. The HY07 revenue total was \$1.8m, with the largest expense being R&D at \$3.0m.

Assuming current grant income and commercial revenue remains at similar levels to 05/06 the company has the funds to take BNC-105 through Phase I/IIa clinical trials and the anxiety and epilepsy compounds into preclinical development. The table below summarises the forecast financials.

Forecast Financials

	FY05a	FY06a	FY07e	FY08e
Revenue (\$m)	2.9	4.2	4.5	5.0
EBITDA (\$m)	(4.1)	(4.6)	(4.9)	(5.8)
D&A (\$m)	(0.5)	(1.0)	(1.0)	(1.0)
EBIT (\$m)	(4.6)	(5.6)	(5.9)	(6.9)
NPBT (\$m)	(4.6)	(5.6)	(5.8)	(6.8)
NPAT (\$m)	(4.6)	(5.4)	(5.8)	(6.8)
Basic EPS (¢)	(4.1)	(3.5)	(2.6)	(3.1)
Diluted EPS (¢)	(3.6)	(2.6)	(2.1)	(2.5)
Dividend per share (¢)	0.0	0.0	0.0	0.0
Dividend Yield (%)	0.0	0.0	0.0	0.0
EBITDA Multiple (x)	(6.5)	(9.2)	(17.6)	(14.7)
EBIT Multiple (x)	(5.7)	(7.6)	(14.6)	(12.5)
PE Ratio (x)	(5.7)	(7.9)	(14.8)	(12.6)

Model Assumptions

- 1) The number of shares and options as of June 2007 remains stable.
- 2) There are no new license agreements or royalty revenues.
- 3) Bionomics is a speculative investment and these forecast financials should be seen as indicative only.

Recommendation

Bionomics has focused on developing unique compounds with modes-of-action and clinical performance superior to the compounds in the market or undergoing clinical trials.

The most notable compound, BNC-105, is demonstrating qualities that could make it a highly successful compound, if it can successfully complete human clinical trials.

We recommend Bionomics as a SPECULATIVE BUY, to the high risk / high reward investor who is knowledgeable and accepting of the inherent risks in investing in biotechnology companies.

The logo for Intersuisse Investment Research is located at the top of the page. It features a dark blue rectangular box on the left containing the word "INTERSUISSE" in white, uppercase letters. To the right of this box, the words "INVESTMENT" and "RESEARCH" are stacked vertically in a large, white, serif font. The background of the entire header area is a dark blue gradient with faint, light blue circular patterns and a grid of white numbers, resembling a financial data table or a stock market display.

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Intersuisse Corporate Pty Ltd was the lead broker for the \$10m capital raising announced in June 2007, for which Intersuisse Corporate Pty Ltd received fees from Bionomics for its services.

Prepared by *Darren J. Grubb PhD MBA GDipAppFin*