

**ASX ANNOUNCEMENT**  
**14 April 2008**

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**BIONOMICS PRESENTS BNC105 DATA  
AT INTERNATIONAL CANCER CONFERENCE**

**Key points:**

- New data describes the dual action of BNC105 which targets tumour blood vessels and tumour cells.
- BNC105 shown to shut down tumour blood vessels in mouse models of human lung, brain and head and neck cancers, bringing the number of tumour types sensitive to BNC105 in animal tests to six.
- Low doses of BNC105 in combination with FDA-approved anti-cancer drug Avastin® prolonged tumour blood vessel shutdown and enhanced anti-tumour effect.
- New data demonstrates BNC105 is rapidly cleared from normal tissues but gets “locked in” the tumour where it exerts potent cytotoxic effects directly on cancer cells.

**Adelaide, Australia and San Diego, CA:** Bionomics Limited (ASX: BNO, US OTC: BMICY) today presented new data at the 2008 American Association for Cancer Research (AACR) Annual Meeting in San Diego, California further demonstrating that the Company’s anti-cancer compound BNC105 acts as a vascular disrupting agent (VDA) in multiple models of human cancer.

The data confirmed that BNC105 is a VDA with a dual mode of action in a number of cancers, causing both the disruption of blood vessels and the inhibition of tumour growth in animal models of human lung and brain cancers. Additional data presented confirmed the activity of BNC105 against breast, colon and prostate tumours.

Following a single intravenous BNC105 injection, tumours showed evidence of significant disruption of tumour blood vessels in addition to death of the cancer cells within the solid tumour mass.

The data also showed that low doses of BNC105 in combination with FDA-approved drug Avastin® (bevacizumab) resulted in an enhanced anti-cancer effect on cancer blood vessels and tumours. In addition, the combined treatment extended the duration of tumour blood vessel shutdown from at least 24 hours to 5 days.

Bionomics’ Director of Cancer Research Dr Tina Lavranos commented, “Our data clearly show that BNC105 can cause almost complete disruption of tumour blood vessels in all six tumour types we have investigated so far. We are very excited by these findings since they suggest that our drug may have therapeutic benefit in many different types of cancer.”

Bionomics’ Chief Executive Officer, Dr Deborah Rathjen said, “The new preclinical data on BNC105 provides strong evidence to confirm its anti-cancer activity. Through its vascular disruptive action, BNC105 can block multiple cancer indications and can enhance the efficacy of a commercial product that acts by a different mechanism.”

“BNC105 has the added advantage of being cleared from all healthy tissues after 24 hours while being retained at high levels in the tumour to achieve maximum anti-cancer results,” she added.

Avastin® was recently approved by the FDA for the treatment of breast cancer based on its ability to stop tumour growth by preventing the formation of new blood vessels. This is a similar outcome to that of BNC105 treatment of mouse models of human tumours, but achieved by a different mechanism, as Bionomics’ new results on the enhanced effects of the two agents in combination show.

BNC105 is currently in clinical trial in patients with advanced cancer at three Melbourne cancer centres: the Peter MacCallum Cancer Centre, The Western Hospital and the Royal Melbourne Hospital.

Some of the data referred to in this announcement is shown in the Appendix (see below). The full poster presented at AACR can be viewed on Bionomics' website at [www.bionomics.com.au](http://www.bionomics.com.au)

**FOR FURTHER INFORMATION PLEASE CONTACT:**

**Bionomics Limited**

Dr Deborah Rathjen  
CEO & Managing Director  
+618 8354 6101 / 0418 160 425  
[drathjen@bionomics.com.au](mailto:drathjen@bionomics.com.au)

**Media Enquiries**

Philippa Harris  
Buchan Consulting  
+612 9237 2800 / 0408 465 800  
[pharris@bcg.com.au](mailto:pharris@bcg.com.au)

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**About Bionomics Limited**

Bionomics (ASX: BNO) discovers and develops innovative therapeutics for cancer and diseases of the central nervous system. Bionomics has small molecule product development programs in the areas of cancer, anxiety, epilepsy and multiple sclerosis. Bionomics' most advanced program, BNC105 for the treatment of cancer, is based upon the identification of a novel compound that potently and selectively restricts blood flow within tumours. Bionomics' discovery and development activities are driven by its three technology platforms: Angene®, the company's angiogenesis target and drug discovery platform, incorporates a variety of genomics tools to identify and validate novel angiogenesis targets. MultiCore® is Bionomics' proprietary, diversity orientated chemistry platform for the discovery of small molecule drugs. ionX® is a set of novel technologies for the identification of drugs targeting ion channels for diseases of the central nervous system.

For more information about Bionomics, visit [www.bionomics.com.au](http://www.bionomics.com.au)

**About BNC105**

BNC105 is a new type of drug called a Vascular Disruption Agent (VDA) that acts to rapidly shut down the blood supply within a tumour. It thereby "starves" the tumour of the oxygen and nutrients it needs to survive.

VDAs (Vascular Disruption Agents) have significant clinical potential in the treatment of cancer, as they may potentially be applied across a variety of cancer types, including colon, lung, and breast cancers. The market potential for VDAs has been estimated at approximately US\$5 billion annually (ASInsights, 2003).

**About Avastin®**

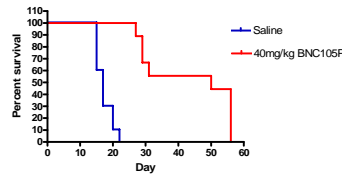
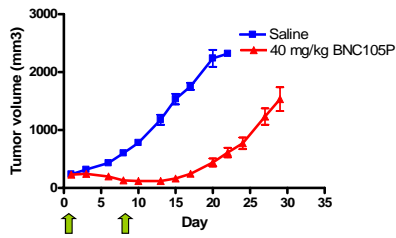
Avastin®, by leading biotechnology company, Genentech, is the first U.S. Food and Drug Administration (FDA) approved therapy designed to inhibit angiogenesis, the process by which new blood vessels develop and carry vital nutrients to a tumour. Avastin® is approved, in combination with intravenous 5-fluorouracil-based (5-FU) chemotherapy, for first- or second-line treatment of patients with metastatic carcinoma of the colon or rectum and in combination with carboplatin and paclitaxel for the first-line treatment of patients with unresectable, locally advanced, recurrent or metastatic non-squamous non-small cell lung cancer (NSCLC).

## Appendix

### BNC105 TUMOR GROWTH INHIBITION and SURVIVAL IN DIFFERENT CANCER TYPES

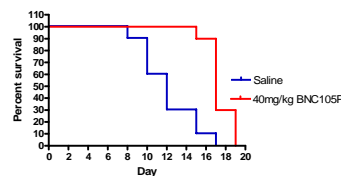
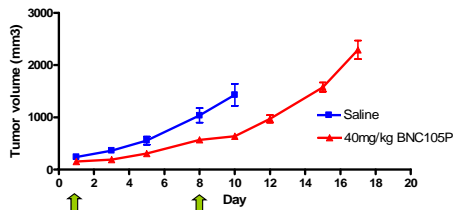
#### Calu-6 (lung cancer tumor model)

Arrows (↑) indicate dosing days.



N=8/group

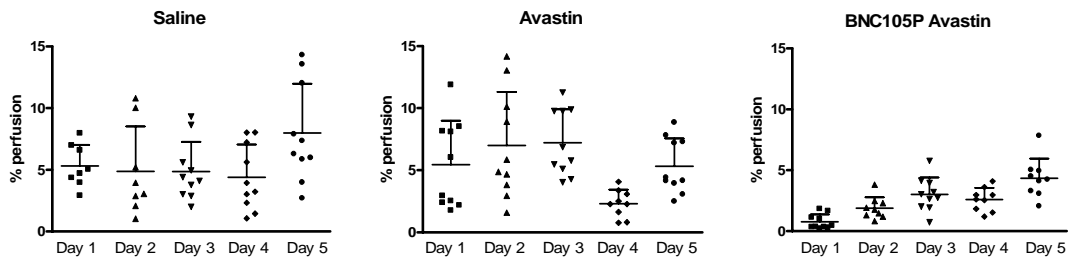
#### U87-MG (brain cancer tumor model)



N=10/group

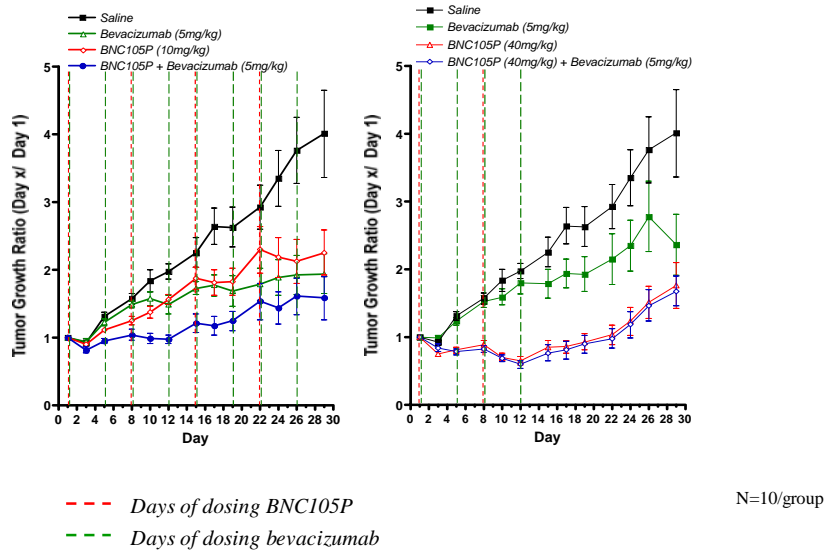
The effects of a single administration of BNC105P on disrupting tumor vasculature prompted us to evaluate the effect of BNC105P treatment on the growth of different tumor types. Human solid tumors were generated in Balb/c nu/nu mice through subcutaneous injection of the human cancer cell lines representing lung (Calu-6), prostate (DU145), brain (U87-MG), breast (MDA-MB-231) and colon (Colo205) cancer. Mice bearing such tumors were treated with BNC105P at 10mg/kg (Colo205; DU145) or 40mg/kg (Calu-6; U87-MG; MDA-MB-231) per dose. Arrows indicate dosing days. Significant inhibition of tumor growth was seen in all cases. Higher responding tumors were lung and breast with 20% of breast tumors treated being cleared after four doses. The graphs above show representative data derived from analyses of lung and brain solid tumors. Tumor growth and animal survival were investigated. Clearly, BNC105 treatment suppresses tumor growth and significantly improves animal survival.

### BEVACIZUMAB (AVASTIN®) COMPLEMENTS THE ACTIVITY OF BNC105



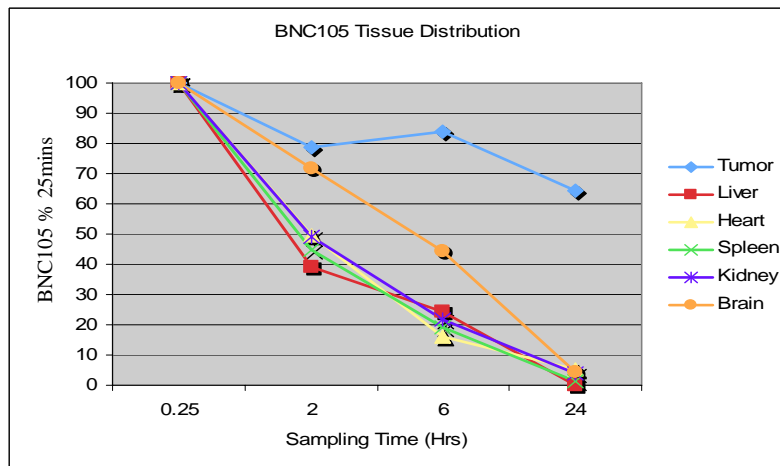
We reasoned that a drug that targets angiogenesis would synergise very well with BNC105 by inhibiting re-vascularization. Bevacizumab is a well characterised anti-angiogenic drug. The effects of combining a single dose of BNC105P with a single dose of bevacizumab on tumor vasculature were evaluated in mice bearing breast tumors. Animals were treated with a single intravenous dose of 10mg/kg BNC105P and a single intraperitoneal dose of 5mg/kg bevacizumab or either drug alone. Animal groups were sacrificed every day for a period of 5 days post treatment. The tumors were recovered and analyzed for vascular disruption. As seen previously, BNC105 caused a tumor vascular shutdown effect that lasted at least 24 hrs. The inclusion of bevacizumab clearly inhibited tumor re-vascularization extending the period of vascular shutdown to day 5.

## BNC105 & BEVACIZUMAB (AVASTIN®) EXHIBIT ADDITIVE ACTIVITY ON TUMOR GROWTH INHIBITION



The observation of complementing mechanisms of action between BNC105 and bevacizumab in extending the duration of tumor vascular shutdown prompted us to investigate the consequences of combining these two agents on tumor growth. We explored potential synergisms when BNC105P is used at 10mg/kg (minimum dose of maximum VDA effect) and at 40mg/kg (dose that achieves 20% tumor clearance). Our results demonstrate additive effects between BNC105P at 10mg/kg and bevacizumab in inhibiting the growth of solid breast tumors. This additive effect was particularly prominent at the 2-week time point ( $p < 0.05$ ; ANOVA with Tukey's Multiple Comparison Test). At the higher dose of 40mg/kg, BNC105P clearly acts as a stand-alone therapy and the co-administration of bevacizumab did not appear to add benefit.

## BNC105 TISSUE DISTRIBUTION - TUMOR "LOCK-IN" EFFECT



The distribution of BNC105 following intravenous administration of 10 mg/mL BNC105P was evaluated in mice ( $n=4$ /group) bearing breast tumors. It was hypothesized that BNC105 induced vascular shutdown in the tumor resulted in its trapping within the tumor enabling it to exert direct cytotoxic activity on tumor cells. This distribution study showed that the levels of BNC105 are significantly reduced in all healthy tissues 24 hrs after dosing (to  $\leq 5\%$ ) yet they remain high in the tumor (64%) over this period. This is consistent with the "lock-in" of drug in the tumor expected after vascular shutdown (98% shutdown was expected at this dose).

## **Factors Affecting Future Performance**

*This announcement contains "forward-looking" statements within the meaning of the United States' Private Securities Litigation Reform Act of 1995. Any statements contained in this press release that relate to prospective events or developments, including, without limitation, statements made regarding BNC105, BNC210 and its' drug development programs are deemed to be forward-looking statements. Words such as "believes," "anticipates," "plans," "expects," "projects," "forecasts," "will" and similar expressions are intended to identify forward-looking statements. There are a number of important factors that could cause actual results or events to differ materially from those indicated by these forward looking statements, including risks related to our available funds or existing funding arrangements, a further downturn in our customers' markets, our failure to introduce new products or technologies in a timely manner, regulatory changes, risks related to our international operations, our inability to integrate acquired businesses and technologies into our existing business and to our competitive advantages, as well as other factors. Subject to the requirements of any applicable legislation or the listing rules of any stock exchange on which our securities are quoted, we disclaim any intention or obligation to update any forward-looking statements as a result of developments occurring after the date of this announcement.*