Dear Shareholders,

It has been a pleasure coming onboard as Invion’s CEO, meeting with shareholders in Australia and having a chance to share our vision for the future of the company. This newsletter is an extension of that, giving shareholders we haven’t met yet a chance to review our plans and assets, and learn more about why we believe Invion is one of the most exciting new opportunities in biotechnology on the market.

There is a lot of work ahead in 2013. We have just announced initiation of our phase II trial of INV102 in asthma, which is being funded by a US$4.4 million grant from the National Institutes of Health (NIH).

The NIH are the primary agency of the United States government responsible for biomedical and health-related research, and are funding the trial under our Investigational New Drug (IND) application through a cooperative agreement grant to Baylor University, with additional sites at Duke University and Washington University.

Securing the NIH grant was a powerful boost to the company, in terms of non-dilutive capital support, the underpinning INV102 data gathered in clinical trials conducted so far, and the validation of our trial design.

The asthma trial will provide further valuable data around INV102’s effect on ‘airway hyperresponsiveness’ (the likeliness of the airways to tighten or spasm), which is a crucial part of assessing the drug’s potential as a treatment for chronic obstructive lung diseases.

The trial will also provide valuable information around the precise mechanisms through which INV102 works. As you may know, to date, two phase II clinical trials of INV102 have been completed which demonstrated acceptable safety as well as dose-related reduction of airway hyper-responsiveness.

We will soon also be announcing patient dosing in our phase II trial assessing INV102 as an aid to smoking cessation in patients with chronic bronchitis. Invion believes INV102’s action will significantly reduce “smoker’s cough”, a symptom of the smoking cessation period which causes many smokers to relapse.

Research to date suggests INV102 will tackle smoker’s cough by restoring the inflamed airways responsible for producing the mucus that causes the cough. A therapy which can reduce or eliminate smokers cough could be life changing for patients.

Being able to commence two new phase II trials for an asset that has already generated positive phase II data is an enviable position for Invion to be in, and part of the rationale for the company’s creation. We believe we have identified an opportunity to take an existing drug (INV102) into new indications and generate considerable value from this clearly mapped repurposing. Our Chief Medical Officer Dr Mitchell Glass speaks more on this in an article later in this newsletter.

Rounding out our portfolio is our INV103 (ala-Cpn10) asset, which has considerable preclinical support for its efficacy as a modulator of the immune system. This drug holds potential for use in treating diseases where the immune system has drifted from normal functioning, such as the common autoimmune disease, SLE (lupus).

In recent weeks the company met with the Pulmonary, Allergy and Rheumatology Products Division of the FDA’s Center for Drug Evaluation and Research for its pre-IND meeting for INV103 (ala-Cpn10), and we are now moving forward with our clinical development plan.

I look forward to continuing to share Invion’s progress with you, and send my best wishes for 2013.

Yours faithfully,

Dr William Garner, CEO.
INV102, also known as nadolol, is a beta-adrenergic inverse agonist (BAIA) that has been used in more than eight million people for the treatment of high blood pressure, migraine and chest pain. Invion is repurposing INV102 and targeting it to the treatment of chronic inflammatory diseases of the lungs, including asthma and chronic bronchitis.

INV102 belongs to a family of drugs known as beta blockers. Beta blockers are so named because they block beta adrenergic receptors in cells, which are involved in muscular action in the body.

Beta blockers fall into two categories: antagonists, and inverse agonists. Fourteen of the seventeen beta blockers currently on the market are antagonists: they block agonist stimulation of the receptor but do not inactivate spontaneously active receptors.

INV102 is one of the three BAIA. This subset of beta blockers block agonist stimulation of the receptor and also inactivate intracellular inflammatory events.

LONG-TERM EXPOSURE TO BAIA LIKE INV102 DECREASE SENSITIVITY OF THE AIRWAYS TO AGENTS THAT WOULD NORMAL CONSTRUCTION.

Of the three BAIA, INV102 demonstrates the best inverse agonist activity in the airways. Invion believes that INV102 is the most effective inverse agonist for pulmonary indications.

Preclinical studies in mouse models of obstructive lung disease demonstrated that chronic treatment with INV102 resulted in healing of the airway epithelium (a beta-receptor rich surface layer of cells essential for normal lung function).

Two proof-of-concept phase II clinical trials in mild asthmatics demonstrated that 9 to 10 weeks of treatment produced a dose-dependent decrease in airway hyper-responsiveness (the likeliness of the airways to tighten or spasm) that achieved clinically significant improvement.

These findings contributed to the NIH's decision to fund Invion's upcoming phase II trial of INV102 in asthma patients.

The repurposing and use INV102 in inflammatory lung disease follows the medical, regulatory and commercial precedent of the use of beta-inverse agonists in congestive heart failure.

The diagram below outlines the precedent laid down for INV102 by the drug carvedilol, branded as COREG® by GlaxoSmithKline. Carvedilol made modest sales revenues of approximately $40 million as a beta blocker, but for decades was labelled against (contraindicated) use in patients with chronic heart failure.

Researchers explored how the effect of carvedilol changed when it was administered first at very low, then slowly increasing dosages. The surprising finding was that it reduced mortality in patients with chronic heart failure.

This new indication for the drug led to new regulatory approvals and a relaunch that saw carvedilol reach sales of more than US$1 billion a year.

In summary, certain beta blockers, i.e. beta-adrenergic inverse agonists (or BAIA), that are initially detrimental when used in the short-term are now considered beneficial in the treatment of disease when used chronically.

This is the rationale behind Invion's development of INV102, which is currently contraindicated for short-term administration in asthma because of an association with worsened spasming of the airways.

Studies to date on escalating dosages of INV102 in airway disease have provided positive results, echoing the precedent of carvedilol. This is a key target Invion will make significant steps in exploring in the year ahead.

INV102: Compelling evidence and precedent drive strategy.

Medical, regulatory and commercial precedent for INV102 strategy

Precedent: Carvedilol in Chronic Heart Failure

TO STANDARD OF CARE
After careful titration Carvedilol reduced mortality in all classes of chronic heart failure. First in class: peak annual sales $1.5 BILLION (2010)

FROM
• CONTRAINDICATED Warning against Beta adrenergic inverse agonist use in chronic heart failure for > 25 years.
• Carvedilol annual sales: (1998) $40m

Invion target: Nadolol (INV102) in Chronic Obstructive Pulmonary Disease (COPD)

TO STANDARD OF CARE
After careful titration Nadolol (INV102) targeted to reduce airflow obstruction due to damaged airways.
Target: First in class

FROM
• CONTRAINDICATED Warning against Beta adrenergic inverse agonist use in COPD for > 25 years.
• Nadolol current sales: $ nominal (generic)
Beta-blockers like INV102 are currently contraindicated in chronic obstructive lung diseases because short-term administration may be associated with worsening bronchospasm. However following the precedent of carvedilol as a treatment for chronic heart failure, also once contraindicated, it is hypothesized that carefully titrated, longer-term dosing of INV102 may provide a safe and effective therapy for the treatment of diseases like asthma and chronic bronchitis, said Dr Glass.

The smoking cessation drug market was estimated at $2.4 billion in 2012. Tobacco is a known or probable cause of at least 25 diseases, including lung and other cancers, heart disease, stroke, emphysema and other chronic lung diseases, and people who smoke have higher rates of wound infection following surgical procedures.

Dr Glass served as Chief Scientific Officer of the University City Science Center in Philadelphia, the largest and oldest urban research park in the United States. He was Vice President and Director at SmithKline Beecham where he worked on the beta blocker, carvedilol (COREG®), and later Chief Medical Officer and Vice President of Clinical Development and Regulatory Affairs of AtheroGenics, Inc. (AGIX).

At AtheroGenics, Dr Glass led product development from IND to initiation of phase 3 for the anti-inflammatory drug AGI1067 and was a member of the IPO team.

INV102’s potential caught Dr Glass’ attention, and he believes the data demonstrates its ability to restore normal structure and function to inflamed airways holds the key to the drug’s successful registration for several therapies.

“Beta-blockers like INV102 are currently contraindicated in chronic obstructive lung diseases because short-term administration may be associated with worsening bronchospasm.

“About one in two regular smokers dies of a smoking related disease, losing on average 16 years of life. This is a significant global health problem and a huge commercial space.

A therapy which can reduce or eliminate smokers cough, a key barrier to quitting smoking, could be life changing for patients,” said Dr Glass.

As Invion returns to the clinic for a series of phase II trials for INV102, Dr Glass’ experience and strong networks in the United States will be an invaluable aid to the company’s clinical planning, design and execution.
Q&A: Smoking cessation clinical trial

What is the name of the trial?
The formal title of the "Smoking Cessation" trial is Phase II, Double Blind, Randomized, Placebo-Controlled Study of INV102 versus placebo in facilitating smoking cessation in subjects with chronic bronchitis and increased cough and sputum (mucus). The Protocol Number is INVS001.

How many patients will be enrolled in the study?
There will be up to 120 patients in this study.

How long is the study?
Each patient will be enrolled in the study for 14 weeks, this includes a six week follow-up period at the end of study dosing.

Where is the study being conducted?
The study is being carried out at a single site in Delaware, United States. The Principal Investigator is Albert A. Rizzo, M.D., immediate past-chair of the National Board of the American Lung Association.

What are you aiming to find out in the study?
The primary objective of the study is smoking abstinence at end of active dosing. Secondary objectives include assessing the tolerability of INV102 in patients who are attempting to quit smoking, the safety and effectiveness of titration up and down, to assess the duration of cessation and any symptoms, including cough.

Why do you expect the study to work?
First, we never expect a study to work but we have excellent data to support our thinking. INV102 has already shown a consistent response in previously conducted Phase II clinical trials. Second, you can see in the diagram on the previous page the effect INV102 has on the airways in our animal model. The picture on the left is of healthy lung tissue with ciliated epithelial cells (stained green), which help remove foreign particles, viruses and bacteria from the airway and keep the airway and its thin mucus lining moist. The middle picture shows you what inflamed epithelial cells look like, whether from a smoker or a patient with asthma, chronic bronchitis or cystic fibrosis. These are goblet cells stuffed full of (stained red) mucus. This extra mucus impacts the free flow of mucus and clearance of infection. This mucus becomes sticky and acts like a "petri dish" that promotes infection. When the animal is treated with a beta-blocker that is NOT an inverse agonist, there is no change from the middle picture. On the far right, we see the airway from a mouse that was treated like the mouse in the middle panel, but was also given INV102 for 28 days. Treatment has virtually restored the airway to normal. So, if we achieve the same effect in the airways of patients who smoke, they should have an easier time quitting and for those undergoing surgery, a lower rate of infection.

Contact details:
Dr William Garner, Managing Director and CEO, Invion Limited
2/120 Bluestone Circuit, Seventeen Mile Rocks, QLD, 4073.

Invion’s pipeline

Invion is developing INV102 to address major market opportunities in chronic inflammatory conditions of the lungs. The upcoming phase II clinical trials using orally administered INV102 are in the area of asthma and aiding smoking cessation. A separate program investigating inhaled INV102 is aimed to target chronic bronchitis and cystic fibrosis.

The INV103 (ala-Cpn10) program is supported by preclinical and clinical data.

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