Biotech Daily

Friday May 10, 2013

Daily news on ASX-listed biotechnology companies

*** ASX, BIOTECH UP: ALLIED HEALTH UP 12.5%**
ATCOR, CIRCADIAN DOWN 6.25%

*** WEHI FINDS MODIFIED P53 PREVENTS CANCER FORMATION**

*** BIODIEM, US NIAID TAKE BDM-I TO PNEUMOCYSTOSIS MOUSE STUDY**

*** AUSBIOTECH'S AUSMEDTECH CONFERENCE OPENS MAY 15**

MARKET REPORT

The Australian stock market edged up 0.15 percent on Friday May 10, 2013, with the S&P ASX 200 up 7.7 points to 5,206.1 points.

Twenty of the Biotech Daily Top 40 stocks were up, 11 fell, five traded unchanged and four were untraded. All three Big Caps were up.

Allied Health was the best, up 0.4 cents or 12.5 percent to 3.6 cents with 10.5 million shares traded, followed by Living Cell up 11.1 percent to five cents with 241,250 shares traded and Avita up 10 percent to 11 cents with 1.6 million shares traded.

Phosphagenics climbed 8.7 percent; Benitec and Impedimed were up more than seven percent; Starpharma rose 6.3 percent; Nanosonics and Neuren were up more than five percent; Osprey and Tissue Therapies were up more than four percent; Sirtex and Universal Biosensors were up more than three percent; Bionomics, CSL, Medical Developments and Resmed rose more than two percent; Alchemia, Clinuvel and Cochlear were up one percent or more; with Acrux, Heartware and Mesoblast up by less than one percent.

Atcor and Circadian led the falls, down 6.25 percent to 7.5 cents and 22.5 cents, respectively, with 82,987 and 10,600 shares traded, respectively.

Phylogica and Psivida lost more than five percent; Patrys, Uscom and Viralytics fell four percent or more; Genetic Technologies shed 2.5 percent; with Prima, QRX and Reva down by less that one percent.

THE WALTER AND ELIZA HALL INSTITUTE FOR MEDICAL RESEARCH

The Walter and Eliza Hall Institute says its researchers have “upended” the understanding of how the p53 protein protects against cancer development.

The Institute said that more than half of human cancers carry defects in the gene for p53, and almost all other cancers, with a normal p53 gene, carry other defects that somehow impair the function of the p53 gene-expressed p53 protein.

The Institute said that inherited mutations in the p53 gene put people at a very high risk of developing a range of cancers.

WEHI said that the p53 protein’s functions were normally stimulated by potentially cancer-causing events, such as DNA damage from ultraviolet radiation, which was a cause of skin cancer, or the over-activity of cancer-causing genes.

The Institute said that Liz Valente, Dr Ana Janic and Prof Andreas Strasser from its Molecular Genetics of Cancer division had been dissecting the processes that were controlled by p53, to discover how the protein could suppress cancer development.

The research paper, entitled ‘p53 Efficiently Suppresses Tumor Development in the Complete Absence of Its Cell-Cycle Inhibitory and Proapoptotic Effectors p21, Puma and Noxa' were published online in the journal Cell Reports, which is available at: http://www.cell.com/cell-reports/fulltext/S2211-1247%2813%2900179-4.

Dr Janic said that many scientists believed that the most important processes activated by p53 to prevent cancer formation were stopping cells with DNA damage from dividing until the DNA could be repaired and making cells die if they had sustained irreparable genetic damage.

“Changes that make damaged cells become long-lived and divide uncontrollably are key features of cancer formation,” Dr Janic said.

“Because p53 can control cell survival and cell division, it was assumed that these two processes constituted the critical functions that p53 used to prevent cancer,” Dr Janic said. “The purpose of our research was to examine whether this assumption was correct.”

Ms Valente said the team compared cells that lacked p53 with cells in which p53 could not regulate cell survival and cell division.

“In the past 20 years it has become clear which proteins are activated by p53 to block cell division and promote cell death,” Ms Valente said.

“We were able to remove all of these proteins, called p21, Puma and Noxa, from cells, to completely disable the ability of p53 to stop cell division and trigger cell death,” Ms Valente said.

“To our surprise, p53 could still prevent cancer formation, even without being able to make cells die or stop dividing after DNA damage,” Ms Valente said.

Prof Strasser said the team’s discovery had upended the understanding of how p53 functions.

“When p53’s cancer-suppressing function was first discovered, it was important to understand how this protein functioned,” Prof Strasser said.

“Many scientists had concluded that regulation of cell death and division were the key roles of p53,” Prof Strasser said.

“Our findings have re-opened the question of how p53 functions,” Prof Strasser said.

“My suspicion is that it is not one protein but several with very many critical functions that work together to prevent cancer formation by coordinating the proper repair of damaged DNA, rather than stopping cells from dividing or killing them,” Prof Strasser said.

“Further research to decipher how these processes are integrated will be an important step towards understanding the tumor-suppressing function of p53 function [which] …in turn, may then be exploited to develop improved cancer therapies.”

BIODIEM

Biodiem says its novel antimicrobial BDM-I has progressed to preclinical mouse studies to assess its potential as a treatment for the fungal disease, pneumocystosis.

Biodiem said that two new studies would be conducted under the US National Institute of Allergy and Infectious Diseases preclinical services program, which had earlier assessed BDM-I's effectiveness in-vitro against 70 different strains of opportunistic or hospital-acquired fungi with significant medical need for better treatments (BD: Feb 1, 2013).

Biodiem has also said that BDM-I would enter pre-clinical testing with the US Army to assess its potential as a biological weapons counter-measure (BD: Feb 14, 2013).

Today, Biodiem said that the in-vitro anti-fungal data was "encouraging and, in particular, showed BDM-I to have significant activity against infection caused by the organism, Pneumocystis and its several species.

The company said that Pneumocystis was a difficult-to-treat opportunistic, yeast –like, parasitic fungus which could cause a serious pneumonia infection in the aged and infants and those who are ill or have a weakened immune system, for example those who have had cancer treatment or organ transplants.

Biodiem said that the new study will involve National Institute of Allergy and Infectious Diseases (NIAID) Animal Models of Infectious Disease Program, where a NIAID-funded contractor will conduct studies in a mouse model of Pneumocystis infection to assess both optimal dose and overall effectiveness of BDM-I as a novel treatment.

Biodiem chief executive Julie Phillips said that the move to studies in an animal model of Pneumocystis infection was "another important step in BDM-I's development pathway towards use in difficult-to-treat infections".

"This is where new treatments are urgently needed," Ms Phillips said.

Biodiem said that Pneumocystis was "very problematic" for cancer and HIV patients, causing pneumonia which was a major cause of death for patients who do not have preventative treatment.

The company said that in the US, the incidence of pneumocystis pneumonia was estimated to be nine percent among hospitalized HIV/AIDS patients and one percent among solid organ transplant recipients, but in immuno-compromised patients, the mortality rate ranged from five percent to 40 percent in those who received treatment and approaching 100 percent without therapy.

Biodiem fell 0.1 cents or three percent to 3.2 cents.

AUSBIOTECH

Ausbiotech says its Ausmedtech conference will be opened in Melbourne, next week, by Victoria's Minister for Technology Gordon Rich-Phillips.

The Ausbiotech program said that the medical technologies conference on May 15 and 16 would be preceded by a Victoria Government briefing on initiatives for the sector.

Topics to be discussed include 'Engaging with multinantional medical technology companies', 'The future direction of in-vitro diagnostics', '3D printing of living cells and biomedical devices', 'TGA's ambitious program of regulatory reform - is it right for Australia' and 'Reimbursement or bust?'

Former University of Melbourne vice-chancellor and chairman of Bionic Vision Australia Prof David Pennington will present the conference dinner address.

For more information go to: http://www.ausmedtech.com.au.