ASX ANNOUNCEMENT

Business Update Conference Call Transcript

Sydney Australia, March 1, 2016: Benitec Biopharma (ASX: BLT; NASDAQ: BNTC; NASDAQ: BNTCW) is pleased to lodge its transcript for the conference call talking place at 8.30am AEDT on March 1, 2016.

Good morning everyone.

I am joined today by Board members Peter Francis, our Chairman, and John Chiplin.

We also have on this call David Suhy, our Chief Scientific Officer, Carl Stubbings, our Chief Business Officer and Georgina Kilfoil, our Chief Clinical Officer. We will take questions at the end of this update.

Let me get straight to the point.

When it comes to hep C, Benitec was in the TT-034 trial for two reasons.

- The first reason was to develop a new therapeutic for hep C. This objective became redundant because of competition.
- The second reason was to prove our platform technology and to inform other programs. This has been achieved through safety and hepatocyte transduction in the TT-034 trial.

As far as having the ability to develop a commercial product around the time we filed the IND for TT-034, Simeprevir and Solvaldi has just been granted commercialisation status for treatment of HCV in combination with pre-existing antiviral compounds. The cost of those combined treatments hovered at $100,000 USD or more.

Since that time a number of different drugs from Abbvie and BMS have been introduced into the market as well as Zepatier from Merck which has been priced at a 42% percent discount to Harvoni, Gilead's latest drug.

Many of these drugs have attained astounding cure rates that regularly achieve viral clearance at 95% or higher including typically hard to treat speciality populations in which similarly high cure rates were not largely anticipated.

Over the years, the average treatment times associated with reliable cures of the disease HCV drugs have been steadily reduced from 1 year, to 24 weeks to 12 weeks. Most recently, early stage data using a combination therapy with Regulus's compound and pre-existing HCV antiviral compounds have shown high cure rates after just four weeks of treatment. As treatment time significantly decreases, the competitive advantage of a one-time treatment like TT-034 decreases.

As a result of the competition from such highly effective antivirals, the ability to recruit patients into an early stage clinical trial has been difficult at best. Even with 4 sites and the engagement of a patient recruitment firm, patients were simply not available in appropriate numbers.

Simply put, the TT-034 program has not attracted meaningful interest from big pharma in spite of many meetings and negotiations.

We listen to our potential customers. Big pharma are not interested in TT-034 today. They are however, interested in hep B and we have more interest from big pharma in this program than we have ever had before on any program.

After consulting with Benitec senior management and assessing the cash runway of the company and the expenditure and resources to advance to the next stage of development for TT-034, the Board decided to conclude the TT-034 trial and use the money to support the development of our other pipeline programs, including HBV, AMD and OPMD.

I will now ask David Suhy to provide his thoughts to what has been accomplished in the HCV trial and provide a brief update on timelines and activity on our other pipeline programs.

Thank you Greg.

Initiation of clinical testing with TT-034 as a First-In-Man trial was a significant milestone for the company and represents the first time that ddRNAi has been administered directly into man in a non-withdrawable manner via viral vectors. From a regulatory standpoint, TT-034 has provided us with a "functional roadmap" with the US FDA as well as other international agencies to define the development path required to address safety questions and push ddRNAi-based therapeutics into the clinic.

The data collected thus far from the clinical study, as well as the data that remains to be collected from the patients, has been exceptionally valuable in establishing the safety of these products as well as define the relationship between the amount of drug administered versus the level of transduction and concomitant anti-HCV shRNA expression. In the nine patients dosed to date, the data presented has shown that TT-034 is safe and well tolerated, meeting the primary endpoint of the study. This result is thus predictive that our hepatitis B treatment, which has multiple components that mimic mechanistic aspects of TT-034, should also be safe in the clinic.

On viral load, based on pre-clinical animal experiments, we have always maintained that the first two dose cohorts in the clinical study were expected to be sub-therapeutic, meaning that large populations of liver hepatocytes may be untreated. We have also stated that we may or may not start seeing an impact of TT-034 by cohort 3. Finally, on the basis of the results from the non-human primate data; we postulated that the dose administered in cohort 4 might result in the expression anti-HCV shRNA levels high enough to have an impact on viral load. To clarify the status of this trial, we have dosed two subjects in cohort 4. One subject was dosed in November. The second subject in cohort 4 was dosed February 9th of this year. Thus, we are still accumulating a significant portion of the data from the 24 week follow-up period. As announced previously, we will not comment on viral load data until the results have been analysed and the database has been locked. We anticipate that the final results of the hepatitis C trial will be reported to the market in Q4 2016. Despite discontinuing further dosing, Benitec will continue to follow these patients which will provide valuable long-term data for our future programs.

I will now provide more background on our programs and their timelines starting with **HBV**. First of all, I think it is important to spend a brief minute in order to differentiate the HBV competitive space from HCV. Although the names of the viruses sound similar, these are completely different. At the most basic level HCV is an RNA virus while HBV is a DNA virus. The current paradigm for HCV of using NUC inhibitors to treat the disease is simply not working for HBV. The current HBV drugs are, at best, able to induce a loss of the HBV S antigen at low double digit percentages (i.e. low teens). Most of the studies demonstrate HBsAg loss at high single digit percentages. The corresponding rates of HbsAg seroconversion, a key viral parameter to assess viral clearance, are even less.

Thus, HBV necessitates a lifelong commitment to monitoring disease status and concomitant therapy for most infected individuals. From a competitive standpoint, there is a considerably larger window to develop a therapeutic regimen superior to the current standard of care. For TT-034 to have been competitive, we would have had to achieve cure rates close to 100% in patients. As a result, Pharma companies are eager to find novel therapeutic modalities to treat HBV and reach out to partners such as Benitec.

We understand that following the recent TT-034 announcement, that many of you are concerned with the impact on the HBV program, but let me assure you that we feel confident in regards to its progress. Because many components of TT-034 are mimicked in our HBV candidate, BB-HB-331, we believe that the clinical results that have been presented publicly to date substantially de-risk that the HBV program in terms of safety and delivery. We also believe that the TT-034 data that we have in hand may decrease the timeline required for the development of BB-HB-331. For instance, if we were to maintain the same AAV8 viral capsid for delivery, the relationship between the dose administered and the level of liver transduction, a measure of how much drug gets into those tissues would be expected to be nearly identical. Likewise, we would expect no change in biodistribution of the drug into non-hepatic tissues. Finally, because the safety profile established in the HCV trial, we would hope that we would be able to make a compelling case to start the HBV trial at drug concentration levels closer to the anticipated therapeutic dose.

To be clear, we will also carefully analyse data from the TT-034 trial in order to incorporate beneficial technological changes that may further enhance activity of BB-HB-331. As one example, the ability to generate significantly more potent triggers of RNA interference against target sequences has improved substantially over the last few years since the time that the design of the TT-034 construct was “locked down” for clinical testing.

For BB-HB-331**,** we presented a strong package of *in vitro* data to the market in December 2015. We anticipate our *in vivo* data to be released shortly and covers results from BB-HB-331 treatment of a chimeric mouse model in which engrafted human hepatocytes can sustain active HBV replication. As Greg stated, we have more interest from big pharma in this program than we have ever had before on any program. It is important to stress that it in addition to the novelty of a ddRNAi therapeutic approach, these partners are keenly interested in the TT-034 clinical data because, as mentioned previously, it significantly de-risks the key aspects of safety and delivery.

I’d like to assure everyone that the advances being made on AMD and OPMD are equally as exciting. In our ocular program, we are focusing our efforts on developing a

ddRNAi therapeutic against Age-Related Macular Degeneration. **AMD** is a disease associated with the growth of blood vessels into the retina that rapidly leads to significantly impaired vision, and if left untreated, blindness. We have developed a series of ddRNAi constructs that silence the disease causing genes associated with neovascularization.

We have spent much of the last year engaged in a collaboration with 4D Molecular Therapeutics to use directed evolution screening techniques in order to identify novel viral vectors that are capable of delivering our ddRNAi constructs into retinal cells following intravitreal injection, a commercially viable route of administration. It is important to note that our collaboration with 4D includes an exclusive license for use of their vectors to deliver therapeutic payloads that treat ocular diseases by RNA interference. This license not only includes RNAi-mediated knockdown of disease causing genes but also covers therapeutic strategies that use vectors to silence expression of a mutant gene and concomitantly express a wildtype form of the same gene to restore normal protein function.

One of the major limitations of most ocular gene therapy applications is that many use a highly complex surgical technique called subretinal injection for delivery into the eye. In this procedure, a needle bent with a 90 degree angle is inserted into the eye and used to carefully tease apart retinal cell layers to create a pocket in which to inject the gene therapy agent. This is an intensive medical procedure that requires an exceptionally skilled surgeon. In our collaboration with 4D, we are developing AAV capsids which can efficiently transduce cells within the retina following an office friendly, intravitreal injection which is a route of administration no different than Lucentis or Eylea, used as the current standard of care for treating AMD. Yet, these drugs require intravitreal injection once a month or once every other month. It is our intention to develop a gene therapy-based ddRNAi approach that will only require a single injection to stop the progression of this disease.

We are nearing the completion of the first phase of the collaboration with 4D Molecular Therapeutics. The bioinformatics analysis from the screening suggests the identification of several promising candidates with the desired properties. Once we have verified that these vectors possess the required biodistribution following intravitreal injection, we will progress our clinical AMD constructs into laser induced models of neovascularization in non human primates. We anticipate completing the initial AAV capsid selection in Q2 and have POC concept studies completed in Q4 2016. In addition to using these capsids to deliver ddRNAi cassettes to treat AMD, we also envision the application of this powerful delivery technology to rapidly move into other ocular-based therapeutic indications that have a genetic basis of disease.

Oculopharyngeal muscular dystrophy (OPMD) is a unique program that has strategic advantages in the fact that it is an orphan disease, meaning that there is significantly less competitive pressure through competing drugs and programs. Because it is a small market indication, it also means that we may have the ability to take this program through to commercialization ourselves. In August 2015, Benitec signed an extension to the Collaboration Agreement with Royal Holloway University of London which continues to enable us to work with world class scientists who have initiated the primary work in the program to identify a genetic basis for this disease. As part of this collaboration, we are also working with researchers at the Institute de Myologie in Paris, a centre of expertise for muscular based disorder that has access to a large proportion of OPMD patients in France. The clinical vector being developed uses a silence and replace approach. Part of the vector uses ddRNAi to knockdown the mutant form of the PABPN1 gene and a separate portion of the construct encodes for a healthy wildtype version of the same gene in order to restore function. We aim to complete preclinical proof of concept studies by Q4 of 2016.

Thankyou David

I will now pass over to Peter Francis

I appreciate the opportunity to be on this call today and I would be pleased to answer any questions you may have later in this call.

I know there is strong shareholder interest in the appointment of a new CEO and I am pleased to advise that we have several candidates who have had multiple interviews and, all going well; we expect to make an announcement as soon as possible. In the meantime, we have a strong executive team that is focused on achieving our goals.

We want to ensure we have a leader with the right skills set and this will naturally take some time

In the meantime, we have a strong executive team that is focused on achieving our goals.

I will now let Greg wrap up our briefing on this call.

Thank you Peter

Like all companies, Benitec needs to adapt and evolve in the long-term best interests of the company, and we have prioritized our programs to where there is the most value to Benitec. We continue to believe in the potential of ddRNAi and its novel capabilities for broad application in human therapeutics.

We will continue to develop a unique combination of gene therapy with our novel gene silencing technology, in-house, in collaboration with partners in addition to out-licensing the technology to parties interested in developing it independently.

Partnering remains a crucial part of our strategy. We are in discussions with more pharma companies on all of our programs than ever before and we will update the market as material developments occur.

Hep B, in particular, and AMD and OPMD have attracted strong interest from potential pharmaceutical company partners. I would also note that, as a result of the decision to discontinue hepatitis C, we expect operational costs to reduce and we anticipate our current cash will enable us to achieve our key milestones.

We will remain listed in Australia where we can access the R&D government tax credits. In addition we have our NASDAQ listing. In the future we will see a greater emphasis on the US markets, and advancing collaborations with global pharmaceutical companies, and a greater representation of our team in the US.

In summary, the decision to focus on hepatitis B and other programs comes down to the much stronger interest we have received from pharma companies and investors, and our assessment of the commercial landscape.

We remain committed to developing our ddRNAi technology to one day change the way we treat human disease and cure patients. I would like to thank all of our investors for their continued support.

At this point, I will ask the operator for questions.