6 July 2013

Dear Shareholders

I am writing this to flesh out the details, purpose and thinking behind the recent funding transaction.

The Deal

Back in May 2013 at our General Meeting, I flagged the need for the Company to raise up to $10 M in the short-term. This was to underwrite the passage of CS-6 into the clinic as well as supporting an ambitious drug discovery program. I also wanted a funding policy (now adopted by the Board) that saw the Company having at least 2 years' working capital for a basic level of activity, simply as a prudent measure.

Shareholders approved at that Meeting the issue of $10 M of shares at up to a 20% discount to market and with 1:2 attached warrants. At our stage of development and market capitalization, and given the general state of the current capital markets, those terms are fairly standard in today's market.

Happily, the terms that we were able to negotiate with Hudson Bay Capital are considerably more favorable to the Company than those May terms and reflect a mutual respect of each party's needs. The three key points of difference being:

- the bulk of the shares to be issued at 10% discount to market
- considerably fewer attached warrants than 1:2
- call on funds is discretionary.

More importantly, had we conducted a traditional share placement, we would have been locked into an issue price at today's levels. This transaction allows us to issue stock over time at prices that hopefully will reflect a positive news flow.

No-one enjoys raising funds at a low share price, and the idea of raising up to $5 M in funds through a series of convertible notes is, in my opinion, a sensible way to meet this problem. This way, we will be able to call upon the funds as we need them and in a way that achieves the best value for shareholders.

We have a considerable potential news flow coming up over the next 12 months. In this chicken-and-egg scenario, we need the funds to create the news flow in order to achieve market appreciation so that we can progressively raise the funds to support ongoing news flow. That was the challenge.

We also are pleased to have a sophisticated investment fund such as Hudson Bay Capital on our register, and given their investment record, we see them as a long-term shareholder. It also goes some way to helping raise our profile in the US.

Use of funds

We are going through exciting times. It's what happens when you are dealing with a new frontier in medicine, when almost every day seems to bring results that either confirm your predictions or throw up completely unexpected opportunities. That's our world at the moment and that's why it was so important to put in place a source of funding that would allow us to realize our technology's potential.

The centre-piece of our drug development program is bringing CS-6 through into the clinic. Our goal is a Phase 1a study in 1Q14, leading into a Phase 1b shortly thereafter. The two Phase 1b clinical indications at this stage remain late-stage glioblastoma multiforme (GBM) and late-stage ovarian cancer.

Apart from generating all the standard animal toxicity and efficacy data that goes with bringing a new drug into humans, we are doing a lot of studies in conjunction with various hospitals, universities and other biotech companies globally looking at the effect of CS-6 on both undifferentiated and differentiated GBM and ovarian cancer cells. These studies are intended to inform the design of the Phase 1b studies, one of which will be conducted in patients with late-stage GBM and the other in patients with late-stage ovarian cancer.

But that is just the start of the program. We are continuing with a Drug Discovery Program that is using CS-6 as the starting pharmacophore and building increasingly complex molecules of a type not previously envisaged. This program, even in its infancy, is uncovering molecules of significant anti-cancer potency that vary in their targets from that of CS-6. That is, as we modify the molecule, we appear to be altering the phenotype of the cancer cell target. And it is this discovery that leads us to believe that we have the potential to finally deliver on the long-awaited promise of personalized chemotherapy, or exposing a tumour biopsy to a panel of drugs active across a range of cancer phenotypes in order to identify the optimal treatment regime.

Latest developments

Finally, just to mention some recent refinements in our way of thinking.

One of those is the realization that we are sitting outside of the pack in the sense that we are not in the business of developing a drug or drugs that will push back the boundaries of chemotherapy in the incremental way that has characterized cancer drug development to date. Seeking a treatment that will extend life by just a matter of weeks or months, is not what we believe this technology is all about. The potent anti-cancer effect we are seeing across both undifferentiated cancer progenitor cells as well as differentiated cancer cells in different types of cancer increasingly is providing us with the confidence to believe that we are on the verge of a quantum leap forward in chemotherapy, with long-term remission across most forms of cancer a realistic possibility.

That realization is based on two key understandings of our technology platform.

The first concerns the way this family of drugs is working. We have come to realize recently that this is an entirely new class of anti-cancer drug that works by blocking the ability of the mitochondria within a cancer cell to produce ATP (adenosine triphosphate), the principal source of energy for any cell. About 90% of all of the energy that our body requires comes from ATP. Without ATP, the cancer cell quickly dies in a form of chemical asphyxiation.

The mitochondria are the nuclear power-plants of a cell. Within each individual mitochondrion, ATP is produced by a series of events reliant on the movement of electrons across membranes. As

the diagram below shows, the ultimate production of ATP is reliant on the movement of electrons across an internal membrane involving negatively-charged oxygen ($O^{=}$) atoms and positively-charged hydrogen (H^{+}) atoms. When that movement of electrons is blocked, ATP production is blocked.

We now refer to this class of anti-cancer drug as Mitochondrial Electron Transfer Inhibitors (METIs). [*Don't bother Googling this term, because it hasn't existed until now. We have had to invent the term to describe this entirely novel mechanism of action.*]

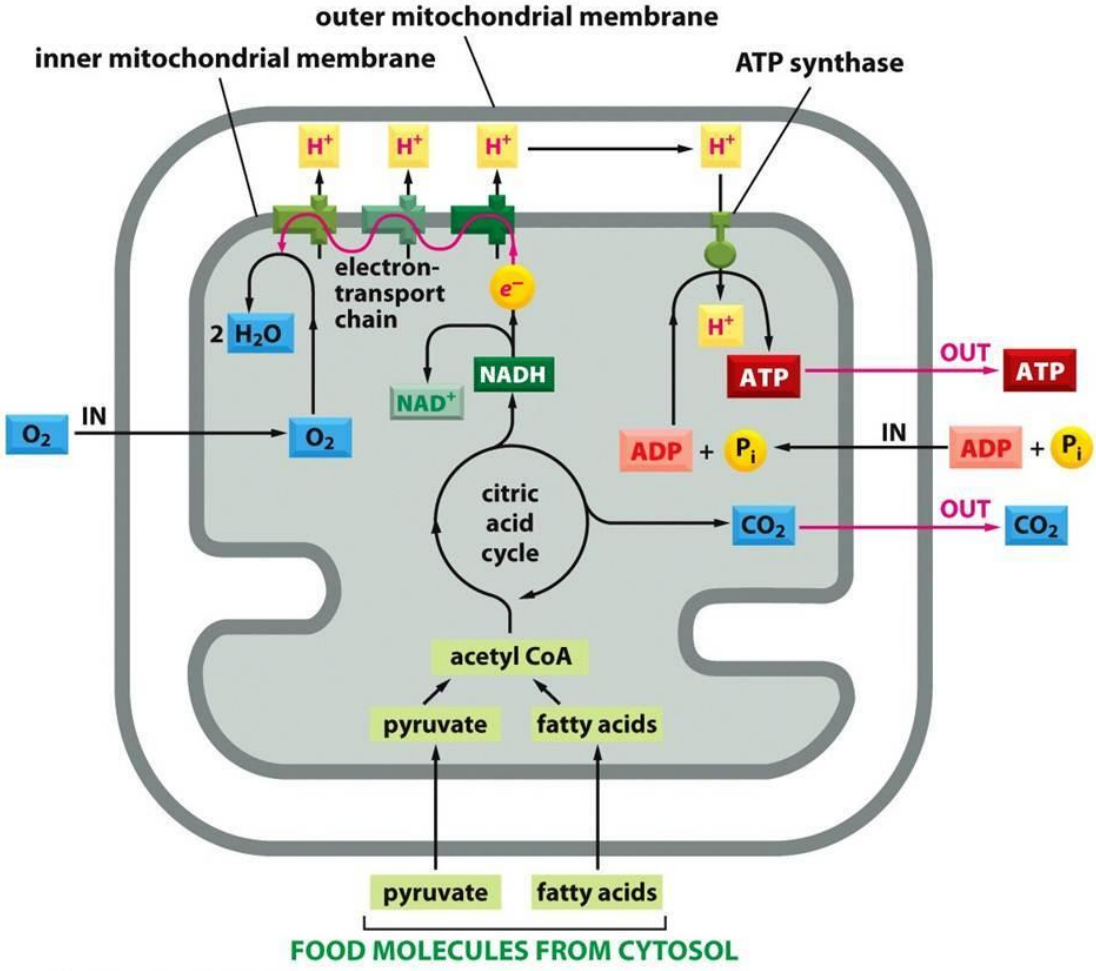

Figure 14-7 Essential Cell Biology 3/e (© Garland Science 2010)

The electron transport chain inside the mitochondrion is our target. What makes this finding particularly exciting is that the rest of the biotech/pharma world is focusing almost solely on the development of anti-cancer drugs directed at specific receptors or specific signaling pathways within a cancer cell. And with each month comes news of a new 'hot' target that excites the scientific and investment markets – PARP, Brf, FAK, Wnt, polo-like kinase 1 and P13K/mTOR are the current hotties.

We don't share the excitement…in fact, we find this a curious rationale, given the complexity of a cell's signaling structure and the well-established ability of a cancer cell to find an alternative pathway around any blockage. The harsh biological reality is that there is no one single signaling pathway that is so vital to a cell's survival that its inhibition will cause the cell to die. And this is why all 'designer drugs' produced over the past 2 decades that target specific receptors or signaling proteins have never delivered anything more than modest increases in patient survival.

The exception to this rule is the mitochondrial electron transfer system. In our view this is the cancer cell's Achilles Heel. Without ATP, the full complexity of the cell's signaling network comes to a grinding halt. And without an alternative energy pathway to call on, the cancer cell quickly dies.

That is the basis of the ability of CS-6 to work across both undifferentiated and differentiated cancer cells, and across all forms of cancer.

The second refinement in our way of thinking has to do with why CS-6 and its fellow drugs are so much more potent than anything we have worked with before. We have come to understand that this is related to a change in the *electrical signature* of the molecule. By this we mean the ability of key parts of the molecule to exchange electrons. By increasing the strength by which a molecule such as CS-6 can donate and receive electrons, we have significantly changed the dynamics with which CS-6 is interacting with target molecules. Two obvious outcomes of this change are more potent triggering of the target in the cancer cell, and greater resistance to glucuronide conjugation in the body with resulting greater bio-availability ('Stealth' technology).

Conclusion

We firmly believe that we are on the verge of changing forever how we treat cancer. Not just any one form of cancer and not just with the goal of prolonging life by weeks or months. Increasingly it is appearing that the Novogen technology platform is capable of delivering drugs that target both differentiated and undifferentiated cancer cells, something that is essential if a patient is to be given any chance of truly being able to eliminate a cancer. This recent funding arrangement is the next step in providing us with the ability to move to a key point in that process …. the ability to take the drug(s) into the clinic.