PRIMA BIOMED

A Leader in Developing Immunocellular Therapeutics for Cancer

Analyst Briefing Document
09 December 2013

Investment Highlights

- ***CVac*** immunotherapy is a potential ***game changer*** in the treatment of ***ovarian cancer***:
  - Maintenance therapy for ovarian cancer after second line treatment is a significant unmet medical need
  - Favorable competitive environment for immunotherapy agents to treat ovarian cancer
- Randomized phase 2 identified a ***clear development path and target patient profile*** (n=20)
- Robust randomized phase 2 (n=210) ***proof of concept*** underway
- ***Orphan designation*** for ovarian cancer in the U.S. and Europe
- Prima has addressed ***production costs*** and manufacturing scale up globally
- Funded for 2+ years through ***multiple data catalysts***
- Significant ***non-dilutive grant support*** (Saxony Development Bank in Germany has provided nearly EUR 8 million in grants to support development)
- Potential for expanded cancer indications in large – pilot ***pancreatic cancer*** trial underway

Table of Contents

Table of Figures

Executive Summary. Prima Biomed is a company with a viable product candidate in CVac for second remission ovarian cancer – a product with minimal toxicity, good (but early phase) randomized trial data, focused in a market of significant medical need and little competition.

Given the robust revised clinical plan for ovarian cancer, the potential of treating additional mucin 1 overexpressing tumors such as pancreatic cancer, and Prima's current financial situation, there is significant upside to CVac.

CVac has demonstrated the ability to induce a robust T cell response specific for the mucin 1 cancer antigen. This is a significant milestone in the cancer vaccine space, as it presages clinical efficacy (e.g. survival). With the recent phase 2 data, Prima identified a relatively homogeneous patient population (second remission ovarian cancer) and now has a clear development pathway to bring CVac to market.

Most other ovarian cancer products in development are targeting different mechanisms than CVac and could even be beneficial in combination with CVac.

Prima holds a leadership position in the development of **personalized immunocellular therapeutics** – a space that should become increasingly exciting in the coming months and years as the cancer immunotherapy market starts maturing.

As Dr. Drew Pardoll, Director of the Cancer Immunology Program in Johns Hopkins University's Sidney Kimmel Comprehensive Cancer Center, was recently quoted talking about potential combinations of immunotherapies, "**. . . the real excitement lies with cancer vaccines. Their stock is very low right now, but they are going to come roaring back."**

It is becoming increasingly evident that cancer immunotherapy will work best in combinations – especially the checkpoint inhibitors (like PD-1 and PDL-1 inhibitors) in combination with T cell stimulating products like CVac. As the market realizes the magnitude increase in efficacy that checkpoint blockades combined with therapeutic vaccines will provide to patients, we see promising future for development of products such as CVac.

Company Background

Prima BioMed Ltd. was spun out in 2001 from the Austin Research Institute (since merged with the Burnet Institute) in Melbourne, Australia, a well-regarded immunology research center.

The focus on the company is developing personalized immunocellular therapeutics to treat cancer with a clinical stage program for CVac in ovarian and pancreatic cancers.

Over the past year, in conjunction with Mr. Lehman taking over as CEO, CVac entering later stage trials, and the listing of ADRs on NASDAQ, Prima has transitioned to a more US and European focused development stage company.

Prima operates in the US, Germany, and Australia, and has accomplished the challenging tasks of successfully transferring manufacturing technology and scaling up production cost effectively for autologous cell based products in three regions.

Prima's primary listing is in Australia on the ASX (ticker PRR) and has an ADR listed on NASDAQ in 2012 (ticker PBMD). Each 1 ADR = 30 ordinary shares.

Prima has licensed CVac rights to Neopharm Group in Israel and Palestine and retains rights in the rest of the world.

Management Team

Matthew Lehman, CEO, has been with Prima since 2010 – first as its Chief Operating Officer from February 2010 until his appointment as Chief Executive Officer in September 2012. Mr. Lehman has experience in clinical research, development programs, and obtaining drug approval. He has specific expertise in clinical development strategies, operations and in-outsourcing. From 2000 until 2010, Mr. Lehman was Chief Operating Officer for SPRI Clinical Trials in the US and Europe where he managed teams in all areas of clinical operations. Mr. Lehman is based in Redwood City, CA where the company's main US office is located.

Sharron Gargosky, PhD, Chief Technology Officer, has more than 17 years' experience in the biotechnology and pharmaceutical industries and was responsible for successful FDA approval for orphan drugs. She is leading the clinical development, as well as the scientific and technical advancement, of CVac. Dr Gargosky previously held the positions of Chief Scientific Officer at Pulse Health LLC and Chief Scientific Officer and Senior Vice-President of Corporate Development at Hyperion Therapeutics Inc. At Ucyclyd Pharma she managed the development and approval of orphan drug products in the metabolic, small molecule therapeutics and within Medics Pharmaceuticals, the successful BLA submission of Reloxin®. As Vice-President of Business Development for Diagnostic System Laboratories, she was responsible for business expansion through evaluation and implementation of new growth opportunities and patent portfolio management. Dr Gargosky did her postdoctoral fellowship at Stanford University in California and has a PhD in biochemistry from University of Adelaide in Australia.

Marc Voigt, CFO, has more than 14 years of experience in the corporate and biotechnology sectors. He joined Prima BioMed's management team in 2011. He started his career as a personal assistant to a member of the Executive Board of Allianz Insurance. Later, he worked for several years as an investment manager for a German biotech venture fund, investing in different biotech and medtech companies. Mr. Voigt has also worked for the German investment bank, net.IPO.AG, in business development and German securities offerings. In the biotech sector he held the positions of CFO/CBO at Revotar Biopharmaceuticals AG and Medical Enzymes AG. He has a Master's in Business Administration from the Freie University of Berlin.

Recent Performance

Clinical. Prima recently reported a 63-patient phase 2 (CAN-003 protocol) trial of CVac, a trial intended to inform continued development of CVac. The CAN-003 trial reported immune response and progression-free survival in two distinct patient groups – ovarian cancer patients in first remission and ovarian cancer patients in second remission. The immune response (i.e. T cell stimulation) was robust across the board. On PFS, CVac performed very well in the second remission patient population but did not improve PFS in the first remission patient population. We believe these results are influenced by two key factors: (a) the heterogeneity of the first remission ovarian cancer patient population and (b) the likelihood that CVac will have a bigger impact on overall survival like most cancer immunotherapies. These CAN-003 results give Prima a very clear direction in a well-defined patient group to move forward in development.

Prima had previously commenced a phase 2/3 trial (CAN-004 protocol) for first remission patients with PFS as the primary endpoint. In light of the CAN-003 results, it is evident this strategy would likely have yielded uninterpretable results without better specification of a target patient population. The suspension of this trial and the re-design of the program has resulted in short term disappointment in the market. We believe the updated plan (amended CAN-004 protocol for second remission patients) going forward makes good sense and is based on compelling data.

Corporate Development. Prima announced that it entered into a binding term sheet for the license of CVac rights to Neopharm Group in Israel and Palestine. Though a relatively small deal in a minor market, this is the first commercial deal for Prima and indicates the deal-making mentality of the company management. During the past year, Prima has made significant changes in the management team as it enters later phase development. The company has made visibility in the U.S. a priority in recent months; as a result the number of outstanding ADRs and NASDAQ trading volume has increased substantially.

Financial. Prima finished the last quarter (30 September 2012) with A$ 31 million in cash. Company guidance shows a cash burn of A$ 14 million for the current financial year (July 1, 2013 to June 30, 2014). At this rate, Prima has significant cash runway to achieve multiple data catalysts. Earlier in 2013, Prima raised about A$ 15 million from a rights offering and private placements in Australia. The company has not made a public offering in the U.S. to date.

Orphan designation & breakthrough designation. Prima has received orphan designation for ovarian cancer in both the U.S. and Europe. On approval, CVac would receive seven years (in the US) and ten years (in Europe) market exclusivity, as well as other benefits such as reduced regulatory fees. If Prima is able to confirm an OS benefit in second remission ovarian cancer patients and the CAN-003 results are confirmed in a larger trial, we believe that FDA breakthrough and/or fast track designation is a likely regulatory pathway given the dearth of approved drugs to treat ovarian cancer.

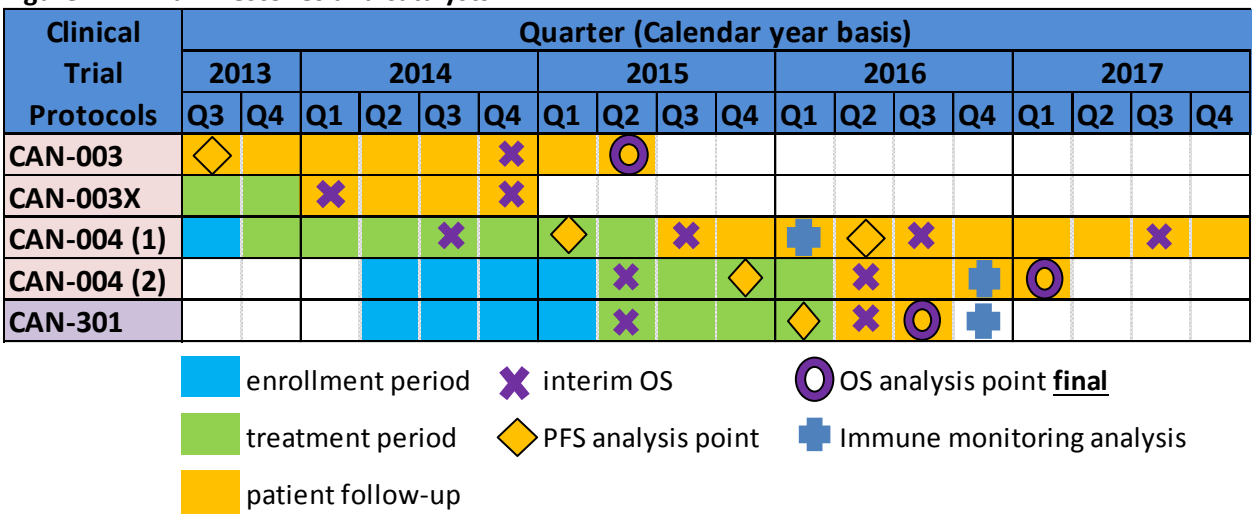
Personalized immunocellular therapeutics. In the scale up to prepare for CAN-004, Prima has accomplished a number of unique and valuable milestones in its ability to transfer manufacturing technology, to reliably produce a comparable product in 3 different facilities, to automate global logistics, standardize its cell collection processes, and meet manufacturing regulatory standards in a large number of potential markets including the US, Europe, and Asia-pacific.

Upcoming News

Milestones. Early 2014 is expected to provide a number of key operational milestones, namely the re-start of the amended CAN-004 trial in second remission ovarian cancer (n=210) as well as the start of the CAN-301 pilot pancreatic trial (n=40).

Data Catalysts. There are data catalysts coming over the coming years. Most importantly in 2014, overall survival data from the CAN-003 protocol should provide important validation for the revised CAN-004 (cohort in 2nd remission).

Figure 1. Prima milestones and catalysts



Source: company reports

CVac overview

Figure 2. CVac clinical trials – accomplished and planned objectives

Protocol	N	Patient population	Objectives (✓Accomplished)
CAN-001 *Phase 1*	10	Incurable adenocarcinoma (single arm)	✓ *Safety* ✓ *Feasibility* ✓ *Immune response*
CAN-002 *Phase 2*	28	Ovarian cancer with no other treatment options (single arm)	✓ *Safety* ✓ *Response by CA-125*
CAN-003 *Phase 2*	63	Ovarian cancer in 1st or 2nd remission (randomized & observation-only controlled)	✓ *Safety* ✓ *Immune response* ✓ *Progression-free survival* ○ Overall survival (est. 2014) ✓ *Manufacturing comparability*
CAN-003X	9	Extension of CAN-003 for patients who progressed	○ Longer-term safety ○ Case studies
CAN-004(1) *Phase 2*	76	Ovarian cancer patients in 1st remission (randomized & placebo-controlled)	○ Overall survival (primary) ○ Progression-free survival ○ Immune monitoring
CAN-004(2) *Phase 2*	210	Ovarian cancer patients in 2nd remission (randomized & observation-only controlled)	○ Overall survival (primary) ○ Progression-free survival ○ Immune monitoring
CAN-301 *Phase 2A*	40	Resected pancreatic cancer patients (single arm pilot)	○ Overall survival (primary) ○ Inform continued development

Early clinical history. A number of pilot trials (both in mice and humans) attempting to induce a mucin 1-specific T cell response were run at the Austin Research Institute prior to the Prima BioMed spinout. Different vaccine formulations and adjuvants were tested to come to the most consistently potent product. This evaluation process indicated that coupling mannan to the mucin 1 protein and pulsing this complex with dendritic cells ex vivo delivered the best cellular response (McKenzie 2003).

Figure 3. Immune responses generated in mice and in humans using mannan-mucin 1 (MUC1) with or without dendritic cells (DC)

Antigen Formulation	Cellular Responses	Humoral Responses
Mannan-MUC1 (mice)	+++	+
Mannan-MUC1 (humans)	+	+++
Mannan-MUC1-DC (mice)	+++	+
Mannan-MUC1-DC (humans)	+++	+
(+) weak	(++) strong	(+++) very strong

Source: McKenzie IF et al, Aspects of cancer immunotherapy, Immunol Cell Biol.2003 Feb;81(1):79-85. PMID: 12534951

CAN-001. For the phase 1 trial, 10 patients of different cancer types with no available curative therapy (i.e., either stage IV or with relapsed disease) were enrolled. CVac was prepared as a "fresh" product,

meaning that cells were harvested, prepared into CVac, and then re-injected for each dose. Trial patients underwent 3 such doses of CVac and, if the patient appeared to benefit, they could continue with further CVac doses.

CVac therapy led to strong T-cell IFNg Elispot responses and delayed-type hypersensitivity responses at injection sites in nine patients who completed treatments. Immune responses were sustained at 1 year in monitored patients. Antibody responses were seen in three patients only and were of low titer.

Side effects were grade 1 only.

Two patients with clearly progressive disease (ovarian and renal carcinoma) at entry were stable after initial therapy and went on to further leukapheresis and CVac therapy. These two patients each completed over 3 years of treatment.

Figure 4. Summary CAN-001 results

ID	Age/sex	HLA	Primary adenocarcinoma diagnosis	Best response	DTH*	Elispot	ELISA†
A01	58/F	DR2,4 A2 B38,62 C6	Fallopian tube	Progression	None	+	Not detected
A02	51/M	DR2,3 A2,24 B7,18 C5,7	Colon	Stable 7 mo§	Inj 3	+	Not detected
A03	62/M	DR2,4 A2,24 B7,60 C3,7	Lung	Progression	Inj 3	+	Not detected
A04	62/F	DR2,4 A24,29 B45,75 C4,6	Oesophagus	Progression	Inj 3	+	IgM, IgG
A05	56/M	DR1,2 A1,24 B7,35 C4,6	Renal cell	Progression	Inj 2, 3	+	Not detected
A07	51/F	DR1,3 A1,11 B8,35 C4,6	Breast	Progression	Inj 1, 3	+	Not detected
A08‡	69/M	DR2,7 A2 B7,50 C6,7	Renal cell	Stable 44 mo‡	Inj 2, 3	+	IgM, IgG
A09‡	64/F	DR1,3 A1,26 B8,27 C1,7	Ovary	Stable 43 mo‡	Inj 2, 3	+	IgG
A10	33/F	DR11 A1,25 B44,57 C5,6	Breast	Stable 8 mo§	Inj 2, 3	+	IgG
A11	70/M	DR2,11 A24,30 B51 C15	Renal cell	Progression	Inj 2, 3	+	Not detected

* DTH reactions at the dendritic cells intradermal injection sites are indicated following the specified injection times.
† Low titer (1/100) anti-VNTR serum antibody reactions or none detectable, measured within 6 months of treatment.
‡ Stable or slow progression at study entry and hence not considered to have clinical benefit from dendritic cell-MFP therapy.
§ Received ongoing dendritic cell-MFP treatment. Overall slow progression (A09) or stable with transient episode of resolving progression (A08).

Source: Loveland BE, et al, Mannan-MUC1-pulsed dendritic cell immunotherapy: a phase I trial in patients with adenocarcinoma, Clin Cancer Res. 2006 Feb 1;12(3 Pt 1):869-77, PMID: 16467101

Of interest from the CAN-001 protocol was the ovarian cancer patient who appeared to perform exceptionally well with CVac therapy. Her case study is presented below.

Patient A09, a 64-year-old woman, presented with stage III ovarian carcinoma 26 months before study entry, and underwent debulking surgery followed by six cycles of carboplatin and paclitaxel chemotherapy. Incurable recurrent disease, diagnosed by elevated serum CA125, occurred 20 months from presentation and she was treated with four cycles of carboplatin, with normalization of the CA125 level.

Study therapy with CVac commenced 3 months following completion of chemotherapy, at which time serum CA125 remained normal at 14 (normal <35) with no evaluable disease on computed tomography scanning (as typical of many ovarian cancer cases).

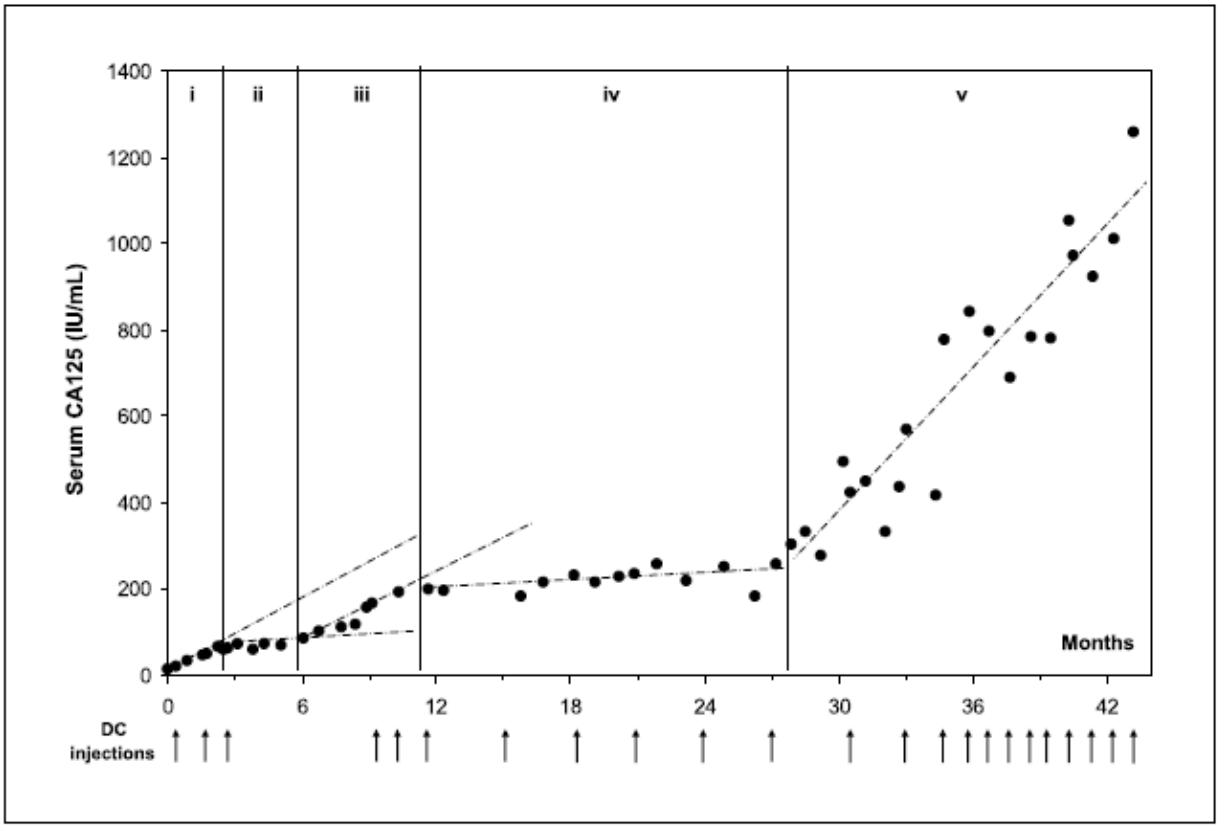
After 6 weeks on study, serum CA125 rose to 47 IU/mL (normal <35), then to 64 at 2 months from study entry, indicating definite progressive disease during the early stage of trial therapy. Following the second CVac injection, CA125 stabilized and remained in the range of 57 to 70 for 4 months, after which

the level again began to increase, increasing from 64 to 165 over 4 months (indicated as periods ii and iii in Fig. 5 using extrapolated trendlines).

In view of the observed 4-month stabilization, then increasing CA125 levels, approval for a protocol amendment was obtained from the Institutional Human Research Ethics Committee to allow for further therapy. This resulted in a delay of 7 months before restarting CVac injections, first at monthly intervals (three injections), then 3-monthly (using cryopreserved CVac for the first time). Serum CA125 on recommencing study therapy was 165, increasing to 191 during the next month.

After the second of the additional injections at 11 months, the patient had largely stable disease for a period of 18 months, although with very slowly increasing CA125 levels which remained in the range of 182 to 258 IU/mL (Fig. X, period iv). From about 27 months, there was an inexorable increase in CA125 (to 1,256 IU/mL, 34 months from recommencing therapy and 43 months since study entry), interspersed with brief intervals where dendritic cell-MFP injections are associated with a transient reduction in serum CA125 (e.g., at 31, 35, and 40 months; Fig. X). Reversion to monthly injections at 35 months seemed to slow the CA125 increase for 5 months only. Through more than 3 years, there was no evaluable disease on CT scanning.

Figure 5. Ovarian cancer patient A09 – case study from CAN-001 trial. Plot of CA-125 over time with arrows indicating CVac injection points



Source: Loveland 2006

CAN-002. CAN-002 was a pilot trial in 28 ovarian cancer patients with incurable disease (as judged by the investigator) after prior platinum-based chemotherapy. Patients were required to have a 25% or

greater rise in CA125 within one month prior to treatment, indicating imminent or active disease progression. 68% of patients had received 3 or more previous lines of systemic therapy.

In this heavily pre-treated population with progressive disease, there were signs of anti-tumor response as defined by CA125.

21 patients were evaluable for clinical response, having remained on the study for 2 months and received 3 or more CVac injections. 4 of the patients demonstrated a response, i.e. a stable or declining CA125 from baseline (see Fig. 6). Upon post-study immunohistochemistry evaluation of tumor tissue, 2 of the patients' samples were negative for mucin 1. Of the 19 mucin 1-positive, evaluable patients, 4 patients (21%) had a confirmed response (confirmed CA125 testing 4 weeks apart) and 1 additional patient had an unconfirmed response.

Figure 6. Confirmed responders on CAN-002

Pt#	Prior therapy	CVac response	Duration	CT scan
228	3 chemo 1 immuno	Major (>50% decline in CA125)	12+ mo.	32% increase in sum of target lesions at 12 wk -> reduced to 21% at 42 wk; no change in non-measurable disease
219	1 chemo	Major (>50% decline in CA125)	10 mo.	No measurable disease
206	1 chemo	Minor (>25% decline in CA125)	7 mo.	No measurable disease; ascites at 18 wk; pleural effusion at 26 wk
202	4 chemo	Stabilization (+/- 25% in CA125)	6 mo.	Stable target lesions at 24 wk; progression of non-measurable disease

Source: Mitchell P et al, Durable clinical responses in patients with ovarian carcinoma treated on a Phase II trial of autologous dendritic cells (DC) pulsed with MUC1, ASCO poster 2007, Abstract #5515.

As may be expected with immunotherapy, there was a delayed response time from the start of CVac therapy of about 2 to 3 months.

CAN-003. The CAN-003 protocol is the first randomized, controlled, and multi-center trial of CVac. The study enrolled 63 patients at 18 centers (5 in Australia and 13 in the USA) in complete remission (CR), according to CT scan and CA125, after platinum-based first or second line chemotherapy.

The first 7 patients enrolled were all dosed with CVac, in accordance with the protocol design, to demonstrate manufacturing comparability between 2 GMP manufacturing sites.

56 patients were then randomized 1:1 to receive CVac treatment or to an observation-only control arm (current standard of care for patient in remission). Of the randomized patients, 46 were in first remission; 20 were in second remission.

Primary clinical endpoints: safety and progression-free survival (PFS). Secondary clinical endpoints: immune responses and overall survival (OS). Prima reported top-line data for safety, immune response, and PFS in September 2013.

CVac was well tolerated with no serious adverse events related to CVac treatment. Two incidences of severe but non-serious adverse events related to CVac included generalized widespread itch and headache. Other non-severe and non-serious adverse events related to CVac included transient injection site reactions (localized pain, erythema, redness, swelling, burning), fatigue, lethargy, and dizziness.

No humoral response was induced by CVac, which is consistent with earlier trials. A mucin 1-specific T cell response was observed by intracellular cytokine staining (ICS) of T cells from peripheral blood mononuclear cell collections (PBMCs). Figure 7 tabulates the maximum post-baseline response for CVac treated patients, for activity of 5 different cytokine from both CD4+ and CD8+ T cells. One star (*) indicates a statistically significant response at $p<0.10$ and two stars (**) indicates a statistically significant response at $p<0.05$.

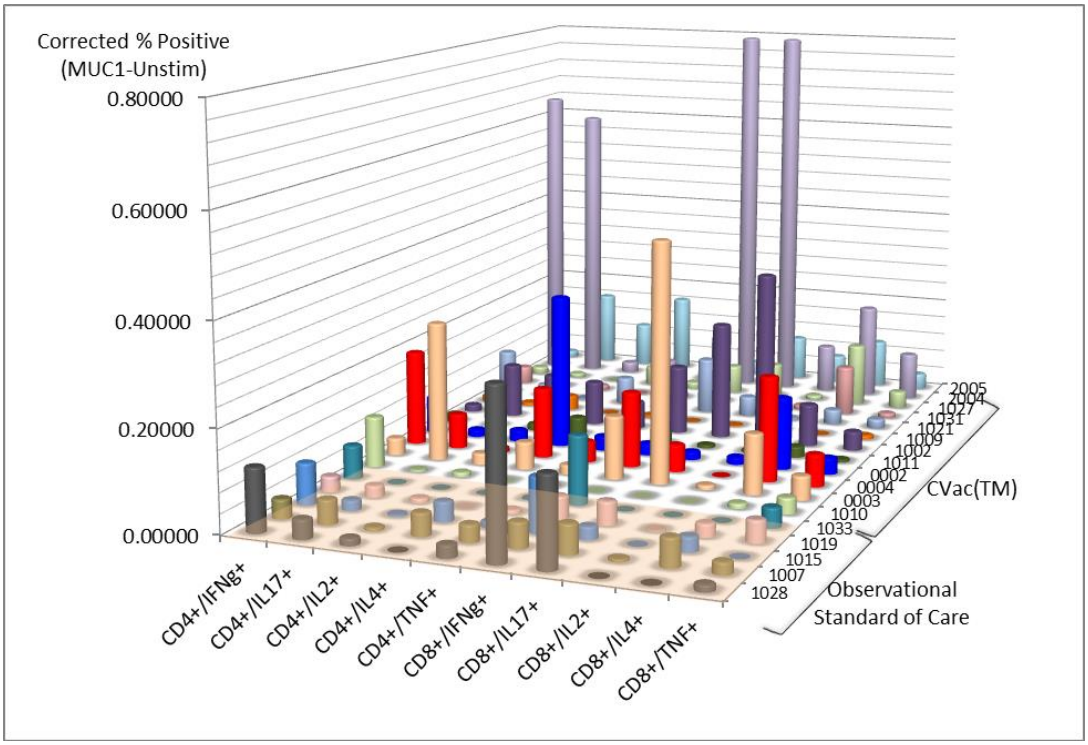
Figure 7. Summary of best T cell response for CVac treated patients on CAN-003
CR1 = first remission and CR2 = second remission.

	CD4 IL-2	CD4 IL-4	CD4 IFNg	CD4 TNFa	CD4 IL-17
CR1	*	*	*		
CR2	**	**	*		
CR1 + CR2					
	CD8 IL-2	**CD8 IL-4**	**CD8 IFNg**	**CD8 TNFa**	**CD8 IL-17**
CR1	*	**		**	
CR2		*		*	**
CR1 + CR2		*	*		*

Source: Goh et al, Clinical study of autologous dendritic cell therapy targeting mucin-1 (CVac) for treatment of ovarian cancer patients in first & second remission (CAN-003), ECC 2013 proffered paper oral presentation

When comparing the T cell activity of CVac patients versus the control group, it is visually clear that CVac induced generally higher levels of activity. Figure 8 presents cytokine activity on CD4+ and CD8+ T cells measured by ICS stimulated with mucin 1 peptides, after 56 weeks on study, after correction for baseline and unstimulated ICS assessments. There were 17 patients who stayed on study for ICS evaluations through 56 weeks.

Figure 8. T cell stimulation summary for 17 patients who completed 56 weeks on CAN-003, data corrected for baseline and unstimulated PBMC controls



Source: Goh et al, Study of autologous dendritic cell therapy targeting mucin 1 as a treatment for the maintenance of ovarian cancer patients in remission, ISCT poster 2013, Abstract# 241.

Progression free survival results indicated divergent trends for the first and second remission populations. Patient follow-up is continuing and updated PFS data will be available with the final Clinical Study report in early 2014; however, we believe the data presented in September and October 2013 will not substantially change given that most of the PFS events had already occurred.

In the first line setting, there was a significant amount of noise with a p value of 0.37. Though the Kaplan Meier estimates of PFS indicate that CVac patients progressed faster than the control group, we believe that this is likely due to random chance rather than an effect of intervention. In the first remission setting for ovarian cancer, there is a lot of heterogeneity in the enrolled patient population, especially regarding whether patients are platinum sensitive or platinum refractory. It is also worth noting that the control group on the CAN-003 trial experienced PFS times longer than the literature would indicate. We do not believe that the presented data indicate that CVac is not working (or that it is somehow harmful). Rather, we believe it indicates that we will need to wait for overall survival data to sort through a potential effect. We expect that we will also need to look back for platinum responsiveness and other key factors to better understand the CAN-003 first remission patient population and potential CVac effects. In any case, it is clear that developing an adjuvant or consolidation-type immunotherapy in this patient population would be challenging without stratifying for various chemotherapy response profiles.

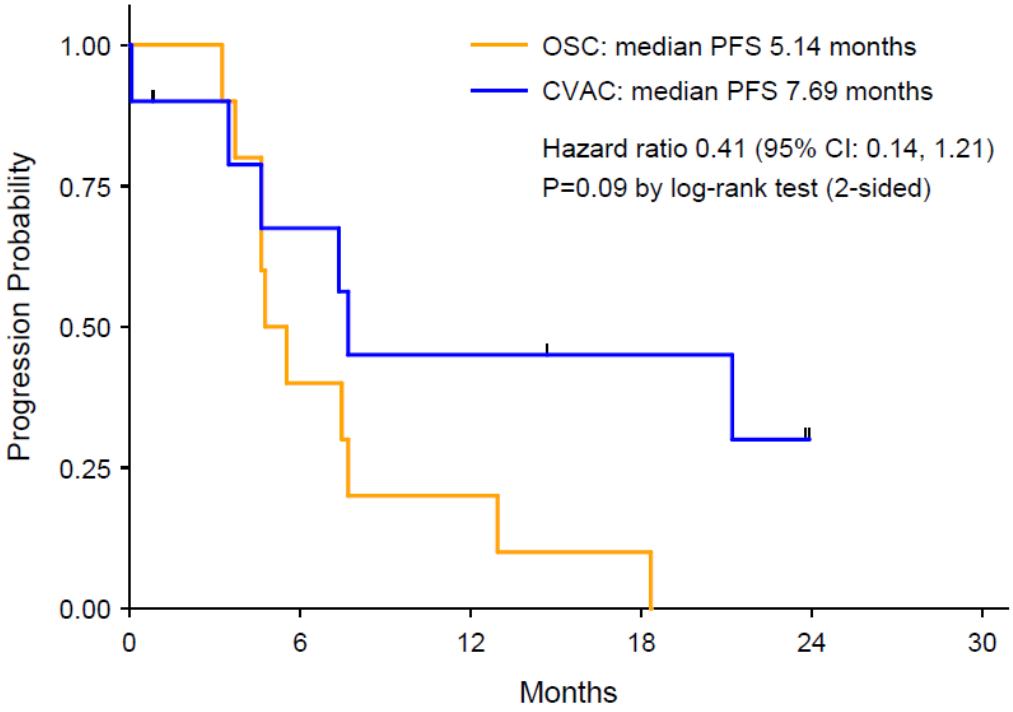
However, in the second line setting, where the cohort is far more consistent with platinum-responsive patient only, there was a strong clinical signal of a 50% increase in PFS for CVac treated patients. The

median PFS for CVac was 7.69 months versus a median PFS for the control group of 5.14 months. The observed hazard ratio was 0.41 at a p=0.09. This is a rather strong signal for only 20 patients.

What is also interesting to note is that the PFS curves in second remission patients have a very familiar shape in cancer immunotherapy. First, there is the classic delayed effect with no difference during the first 4-5 months of the study. Then there is a significant "long tail" indicating that the CVac patients who responded well stayed in remission for an extended time. Both of these phenomena have been well described in the cancer immunotherapy literature and are an indicative hallmark of a successful product.

We believe these data, especially when looking at immune response and PFS outcomes together, give us a high level of confidence that CVac can extend overall survival outcomes.

Figure 9. Kaplan Meier estimates of PFS for CAN-003 patients in second remission



PFS: progression-free survival OSC: observational standard of care

Source: Goh 2013 ISCT

CAN-003X. Prima allowed patients that progressed in the CAN-003 trial to enroll in the CAN-003X extension trial. Nine patients enrolled. We may expect some interesting individual case studies and some longer-term safety data from this trial.

CAN-004 (CR1). CAN-004 was initiated in 2011 with very ambitious goals. The original CAN-004 design was an 800-patient placebo-controlled trial of ovarian cancer patients in first remission with PFS as the primary endpoint. It was to include 3 GMP manufacturing sites in Australia, the USA, and Germany, about 50 blood cell collection sites, and 120 investigators in about a dozen countries globally. In light of

the CAN-003 data, it is evident this strategy would likely have yielded uninterpretable results without better specification of a target patient population. Prima suspended recruitment of first remission patients on this trial. As of September 2013, there were 76 patients randomized on the study. Prima has since changed the endpoint of this cohort to overall survival and will continue to monitor outcomes in this patient group.

Nonetheless, ***in the scale up to prepare for CAN-004, Prima has accomplished a number of unique and valuable milestones in its ability to transfer manufacturing technology, to reliably produce a comparable product in 3 different facilities, to automate global logistics, standardize its cell collection processes, and meet manufacturing regulatory standards in a large number of potential markets including the US, Europe, and Asia-pacific***. See further discussion of the value of Prima's cell manufacturing competency below.

CAN-004 (CR2). In November 2013, Prima came out with substantial and well-reasoned amendments to the CAN-004 protocol. Using the existing network built up for the previous program, Prima will enroll 210 ovarian cancer patients in second complete remission – the same population that has thus far responded well in the CAN-003 protocol. The trial will be a 1:1 randomized study of CVac versus an observation only control group, after completion of platinum-containing second line systemic therapy. Overall survival will be the primary endpoint with secondary endpoints including 1-year and 2-year survival rates, progression free survival, time-to-next-line-therapy (TTNT), and immune monitoring.

The study has been designed to obtain robust clinical "proof of concept," powered to observe a hazard ratio for OS of 0.67 with a p=0.10 (1-sided log rank test). Considering the CAN-003 data indicated a 0.41 hazard ratio, and that larger trials tend to have somewhat smaller effect sizes, we believe this is a well powered study to accomplish the development goals. Good CAN-004 data would open multiple options for Prima including potential breakthrough or fast-track designations with regulators. The CVac data package would put Prima is a good position to negotiate potential pharma partnerships for phase 3 and commercial.

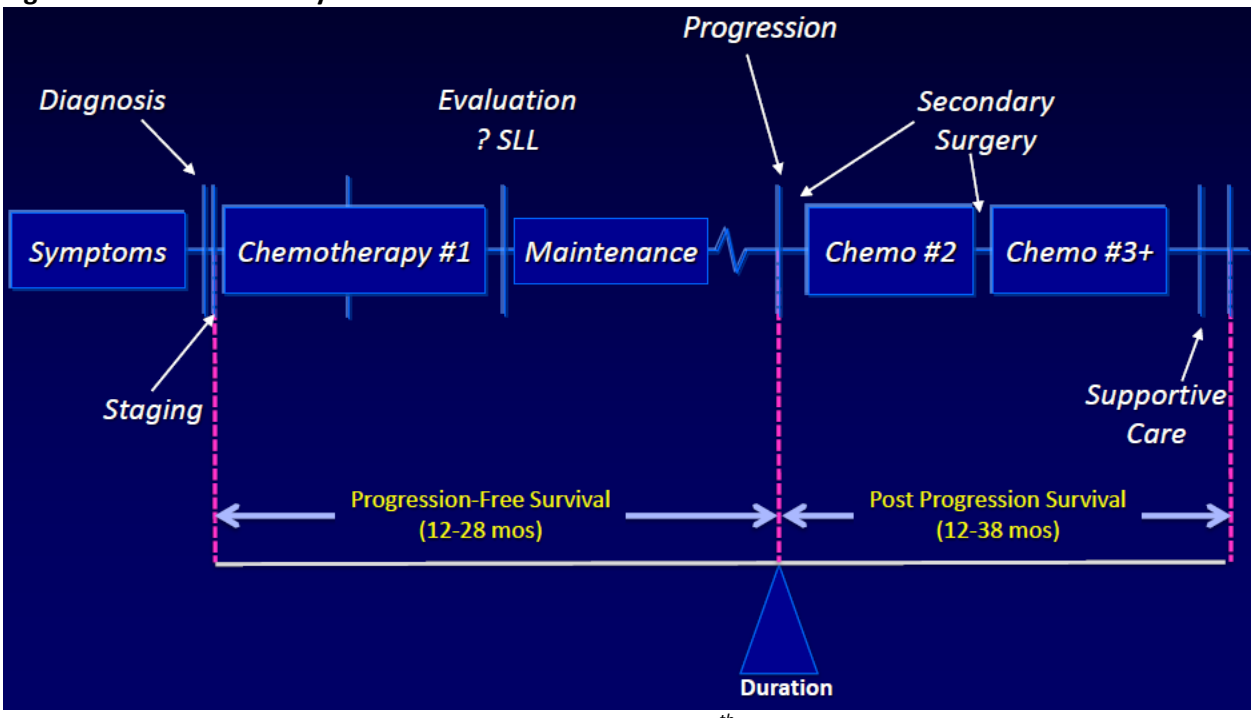
The amended CAN-004 trial in second remission is the highest priority for Prima. It is expected that recruitment will commence by the second quarter of 2014 and finish recruitment by the second quarter of 2015. There will be a number of interim data points throughout 2015 and final OS data would be expected by the end of 2016 according to expected median survival in the patient population.

CVac's place in treatment of ovarian cancer and new agents in development

Based on the CAN-003 randomized data, Prima has a clear pathway through clinical development and an important target patient profile that represents a major unmet medical need and with little competition.

The current general overview of the course of ovarian cancer is summarized in Figure 10.

Figure 10. "Natural History of Ovarian Cancer"



Source: Coleman R, 2013: Year in Review, presentation to 16th Annual Conference: Voices of Hope and Strength: Teal is Personal, available at http://www.ovariancancer.org/wp-content/uploads/2013/07/Coleman_OCNA_ovariancancer_update_2013.pdf

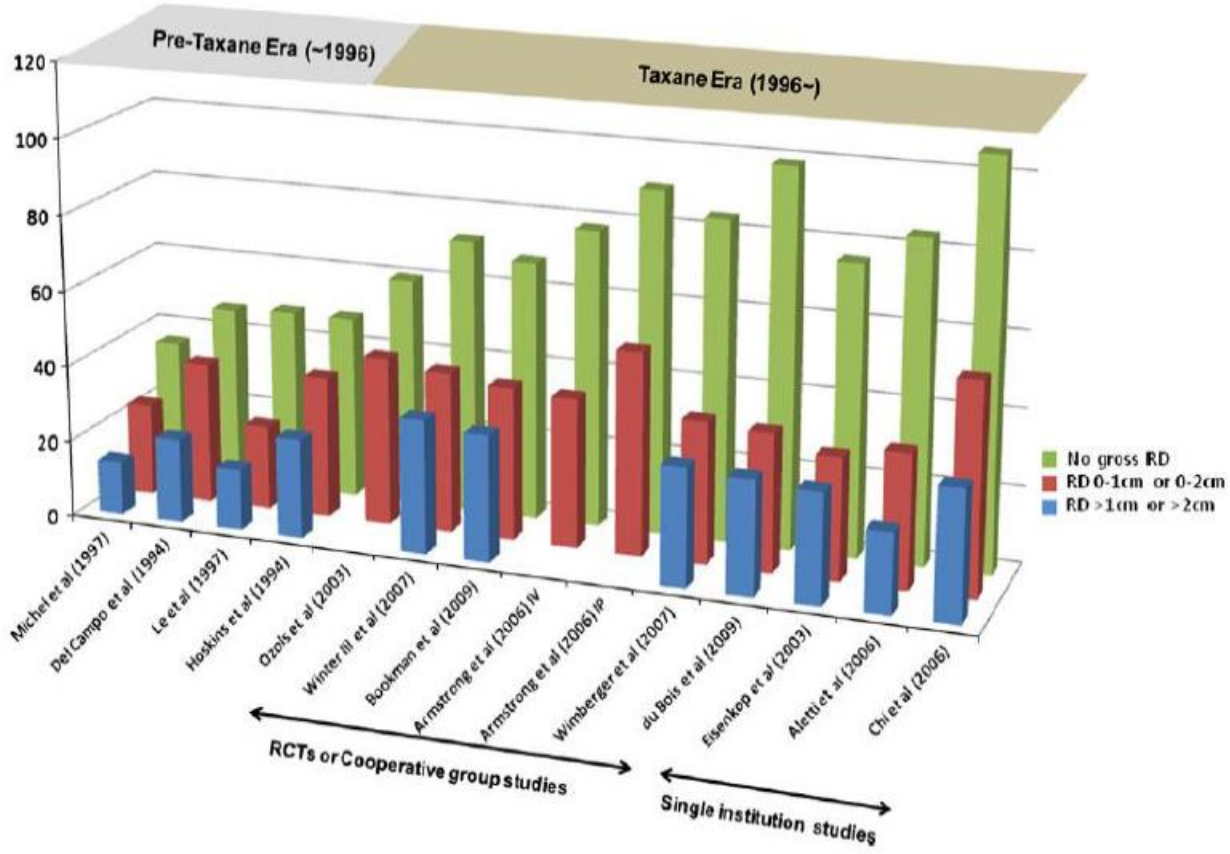
Treatment of Ovarian Cancer

The first step in treatment of ovarian cancer is surgery, during which the surgeon stages the cancer and attempts to remove as much of the tumor as possible (debulking surgery). FIGO stages are as follows:

- Stage I: Cancer is restricted to one or both ovaries
- Stage II: Cancer is in one or both ovaries and has involved other pelvic organs such as the fallopian tubes and uterus
- Stage III: Cancer involves one or both ovaries and has spread beyond the pelvis to the lining of the abdomen or the lymph nodes
- Stage IV: Distant metastases – cancer has spread to organs outside the peritoneal cavity such as the lungs and liver

The success of initial debulking surgery remains the single most important prognostic factor for ovarian cancer outcomes, as has been confirmed in numerous studies over the years (see Fig. 11)

Figure 11. Summary of median survival outcomes depending on residual disease (RD) after primary debulking surgery in ovarian cancer



Source: Chang and Bristow, Evolution of surgical treatment paradigms for advanced-stage ovarian cancer: redefining 'optimal' residual disease, Gynecol Oncol 125 (2012): 483-492, PMID: 22366151

After surgery, patients with Stage I disease may receive no further treatment but are closely monitored.

Patients with Stage II – IV disease may receive chemotherapy. The most commonly used regimen for front-line treatment is six cycles of a platinum-taxane combination therapy, typically carboplatin-paclitaxel. These drugs may be delivered by intravenous (iv) or intraperitoneal (ip) administration; ip administration has greater efficacy but also far greater toxicity. Response rates to initial chemotherapy are in the 70%-80% range but eventually most patients relapse.

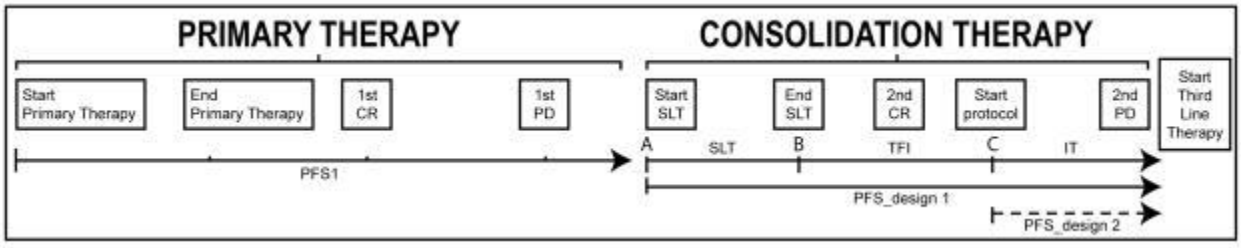
Several new agents have been tested in the front-line setting in recent years, with little success in changing overall survival outcomes. Bevacizumab (Avastin®) has demonstrated moderate improvement in PFS (Burger 2011, Perin 2011) in two large phase 3 trials. Nintedanib and trebananib are currently in phase 3 trials in the front line setting as well. To date, no clinically meaningful OS improvement has yet to be identified with new agents. Most improvements to survival in the first line setting have been a result of improved surgical techniques and improving dosing regimens for platinum and taxane based chemotherapy.

Further treatment after front line therapy depends on the duration of response since prognosis for response to further therapy is strongly correlated with platinum-free interval after initial therapy. Patients with disease that recurs greater than six months after the initial platinum-taxane regimen are defined as platinum-sensitive and are retreated with the same platinum-based regimen. Patients who relapse in less than six months are defined as platinum-resistant and usually receive single agent therapy with drugs such as Doxil (pegylated liposomal doxorubicin), Hycamtin (topotecan), Gemzar (gemcitabine), etoposide, cyclophosphamide and a long list of others. There is no consensus on the best regimen for treatment of platinum-resistant or platinum-refractory patients because the impact on overall survival is not demonstrably different with any of the available choices.

Second-line therapy and second remission maintenance (Consolidation)

Figure 12. Typical management course for ovarian cancer consolidation therapy

Consolidation treatment in Ovarian Cancer received in Second Line Setting following primary recurrence: definition of treatment intervals. Abbreviations: CR (complete response); PD (progressive disease); SLT (second-line therapy); TFI (Treatment-free interval); IT (investigational therapy).



Source: Iasonos A et al, Identifying Clinical Improvement in Consolidation Single-arm Phase II Trials in Ovarian Cancer Patients in Second or Greater Clinical Remission, Int J Gynecol Cancer. 2012 January; 22(1): 63–69

As discussed above, most patients respond well to initial surgery and chemotherapy with 70-80% of patients achieving a complete remission. However, rates of relapse are also very high. The literature suggests that most patients will have a first progression-free interval (PFS1) of 12-28 months.

After relapse, about 80-85% of platinum sensitive patients will go onto second-line therapy (SLT), also known as consolidation therapy. The progression free interval after SLT (PFS2) is most often shorter than PFS1. In fact, only about 9-16% of patients will experience a longer PFS2 than PFS1 (Sabbatini 2010).

The expected median PFS2 of patients that undergo SLT is about 8-12 months (as measured from the start of SLT (i.e. from point A in Figure 12 above). The post-SLT overall survival generally ranges from 12-38 months.

Due to the greater consistency of the SLT and consolidation patient populations, and the generally shorter timelines to PFS and OS data points, the area has attracted increasing interest for the development of new drugs and biologics.

Agents in development for consolidation therapy

There are four general classes of new agents under investigation in the ovarian cancer consolidation space. An overview of the classes and selected therapies in clinical trials are summarized below:

Angiogenesis inhibitors: bevacizumab (Roche), trebananib (Amgen), nintedanib (Boehringer Ingleheim), cediranib (AstraZeneca), sorafinib (Onyx/Amgen), and pazopanib (GlaxoSmithKline)

PARP inhibitors: olaparib (AstraZeneca), veliparib (AbbVie), niraparib (TESARO), rucaparib (Clovis), BMN-673 (BioMarin)

Folate receptor targeting agents: vintafolide (Merck), farletuzumab (Eisai)

Immunotherapy: CVac (Prima), polyvalent KLH-linked vaccine with OPT-821 adjuvant (Memorial Sloan Kettering), VTX-2337 (VentiRx), as well as University of Pennsylvania's adoptive T cell and DC vaccines. Very early investigation has started into the potential of anti-PD1 and anti-PDL1 agents in ovarian cancer treatment.

Several of these agents under development have shown promising activity in different subsets of the SLT and/or consolidation therapy setting. In particular, bevacizumab, olaparib, and trebananib have recently reported positive PFS outcomes for platinum-sensitive recurrent ovarian cancer patients. However, none of these agents have demonstrated a clinically and statistically significant impact on overall survival to date; mature OS data from ongoing trials may be expected in the coming 1-2 years.

Longer term, Prima expects that, given complementary mechanisms of action, CVac could potentially work very well in combination with angiogenesis and PARP inhibitors.

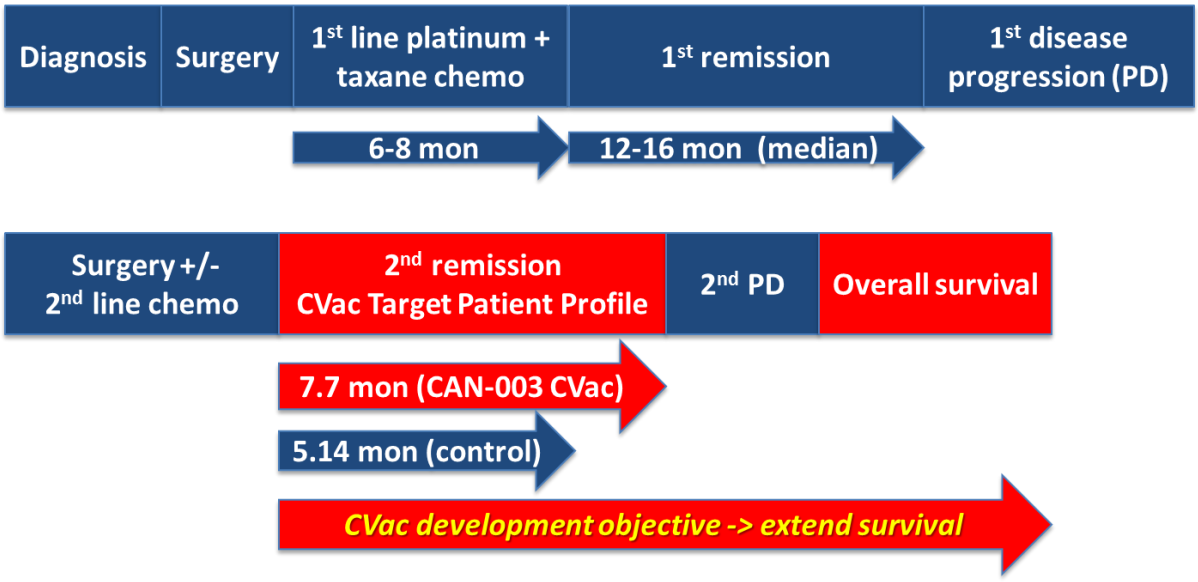
Looking very specifically at trials in the same setting as CVac – i.e. after completion of platinum-containing SLT, there are only two publications that indicate potential efficacy with as strong a signal as seen in the CAN-003 data:

One is a report from Sabbatini (2009) of pooled data from six phase 1 trials of KLH-linked vaccines (which would indicate validation of the CVac approach due to a similar mechanism of action). The median PFS of CVac treated patients and the Sabbatini vaccine data are practically the same.

The other trial (Kaye 2012) that reported a similar median PFS improvement in the same patient population was an 84-patient trial of vimodegib (median PFS 7.5 months compared to 5.6 months on placebo). However, there was significant toxicity associate with the product and a large number of study dropouts. Genentech decided not to pursue development.

CVac's target patient profile & the CR2 ovarian cancer market

Figure 13. CVac's place in ovarian cancer management



Source: company reports

Figure 13 illustrates the target patient profile for CVac within the ovarian cancer space. The incidence of ovarian cancer in Australia & "major markets" (USA, Japan, UK, Germany, France, Italy, Spain) was approximately 61,283 annually (2010) and expected to grow approximately 2% per year.

Of those patients, about 70-75% diagnosed in late stage, or about 44,400 per annum. 70-80% of those platinum sensitive and will achieve remission, or about 33,300 per annum.

According to market research from Global Data's 2011 study of the paclitaxel market for ovarian cancer, it is estimated that about 80-85% of the platinum sensitive patients will then relapse and undergo second-line therapy. This gives CVac an approximate addressable market of more than 25,000 patients annually in the major markets.

Pancreatic cancer

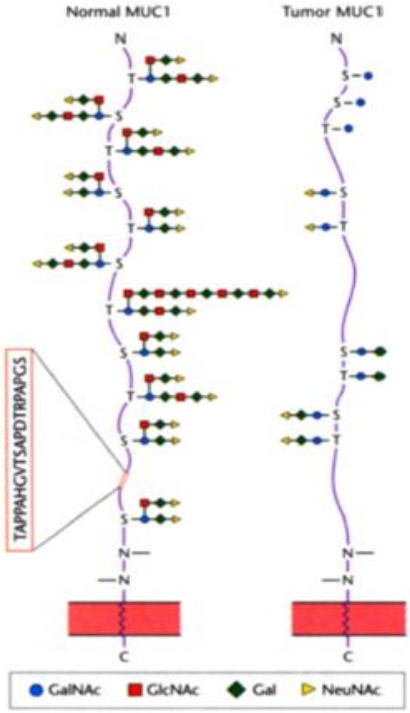
Prima has also recently announced plans to commence a pilot trial in resected pancreatic cancer (protocol CAN-301). This trial will include 40 patients in Europe in a single-arm design. The goal of the trial will be to assess the feasibility of CVac therapy in this patient population, overall survival, progression-free survival, and other endpoints. The data from CAN-301 will be used to make a go/no-go decision on continued development in pancreatic cancer.

The literature strongly supports immunotherapeutic approaches to treating pancreatic cancer in a maintenance setting after resection. More than 80% of these patients are expected to have mucin 1 overexpressing cancer cells which is the target of CVac.

The annual incidence of pancreatic cancer in the "major markets" (USA, Japan, UK, Germany, France, Italy, Spain) is about 99,000 per annum. Of those, about 20% (20,000 per annum) are suitable for surgical resection. About 80-95% of those who survive surgery may be expected to benefit from CVac treatment, giving an estimated addressable market of about 17,000 patients annually.

Mucin 1 background

Figure 14. Aberrant glycosylation of mucin 1expressed on tumor cells



Mucin 1 is a member of a membrane-bound class of proteins that form a protective mucous gel on the apical surface of normal cells of the reproductive, respiratory and digestive organs. Mucin 1 has three domains: a cytoplasmic domain (inside the cell), transmembrane domain and extracellular domain (outside the cell). Its function is generally to hydrate and act as a protective barrier against microbes.

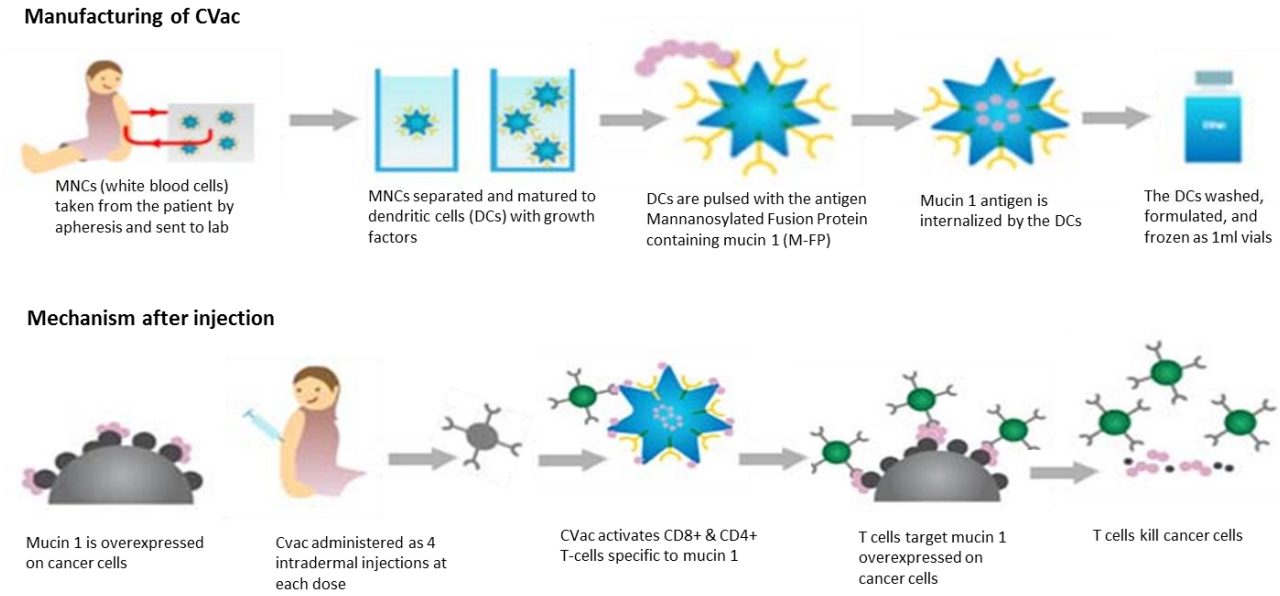
In many types of malignancies, such as ovarian, pancreatic, breast, and lung tumors, Mucin 1 is abnormally over-expressed and is present throughout the cell surface, rather than just on the apical surface. This overexpression appears to inhibit cell-to-cell adhesion, and also correlates with tumor invasiveness and propensity to metastasize. Mucin 1 on tumor cells also demonstrated aberrant glycosylation patterns, which makes this cancer mucin 1 highly distinct. Hence, mucin 1 has attracted interest as a potential target for immunotherapy.

The mucin 1 protein has a 20-amino acid sequence which occurs as tandem repeats as many as 125 times in a single molecule. This region, known as VNTR (variable number of tandem repeats), is highly immunogenic and has been tested in immunotherapy.

Personalized immune cell manufacturing

CVac is produced by collecting autologous mononuclear cells (MNCs) from the patient by apheresis, enriching and maturing the cell population to maturing dendritic cells and then pulsing the cells with mannosylated human fusion protein containing mucin 1. The cells are then washed, concentrated, and formulated as frozen 1ml vials for injection.

Figure 15. CVac production and mechanism



Prima has invested significantly in its manufacturing operational platform. We believe that the Provenge® story has highlighted the importance of developing a well characterized product and managing the costs of goods appropriately.

The key differentiating factors for Prima's manufacturing platform can be summarized as follows:

1. Supply and logistics: too often overlooked in the cell therapy space, the costs associated with cell harvest, cell shipping, and the management of the logistics can be a substantial burden on the cost of production. Prima has addressed the issues successfully with a number of automated tools to maximize efficiency.
2. Product processing: Prima now has experience with manufacturing CVac in three GMP facilities in three global regions with demonstrated comparability. We believe this is a feat no one else has accomplished with a cell based product like CVac and demonstrates the Prima team's competence when it comes time to scale up the process.
3. Quality control: also often overlooked, the bioassays that characterize the CVac product are of critical importance to regulators and to potential pharma partners. These assays help make CVac an identifiable "product" and not just a "process." This gives Prima flexibility to incorporate process changes as it optimizes its manufacturing at a commercial scale.
4. Formulation and stability: CVac is formulated as frozen 1ml vials for injection and these vials are stable for 18+ months (stability studies ongoing). This allows Prima to obtain multiple doses from one cell collection and manufacturing process. The small frozen product is then much easier to ship with fewer time constraints.

Figure 16. Prima's approach to CVac manufacturing optimization

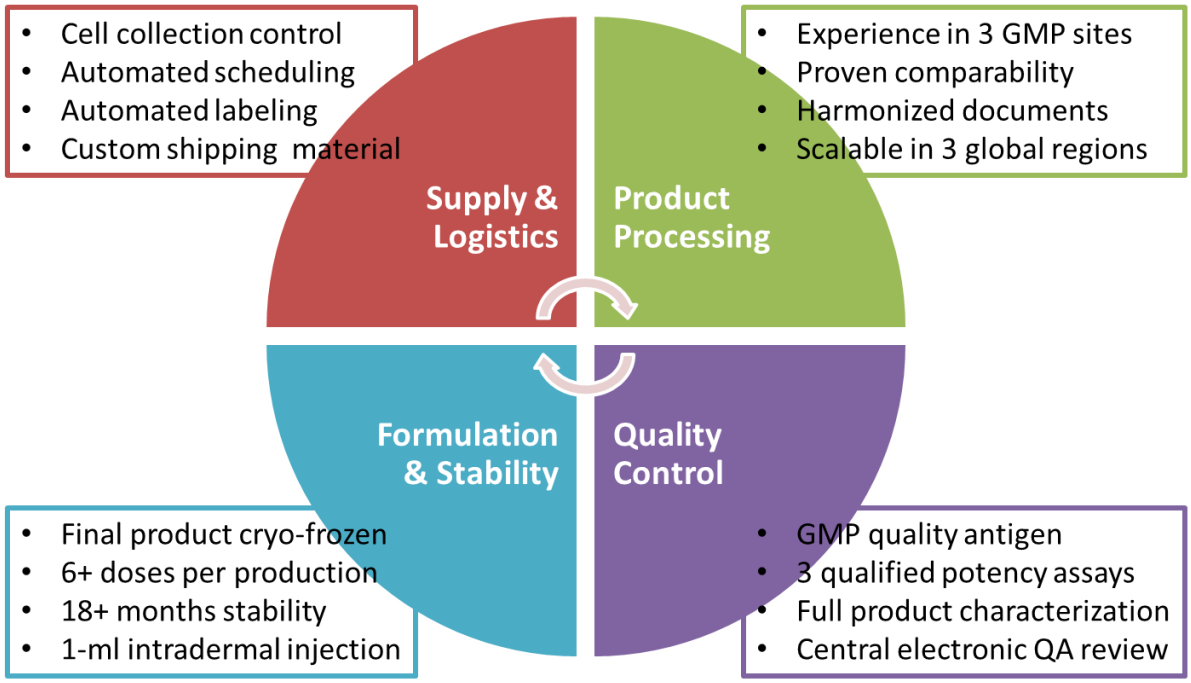
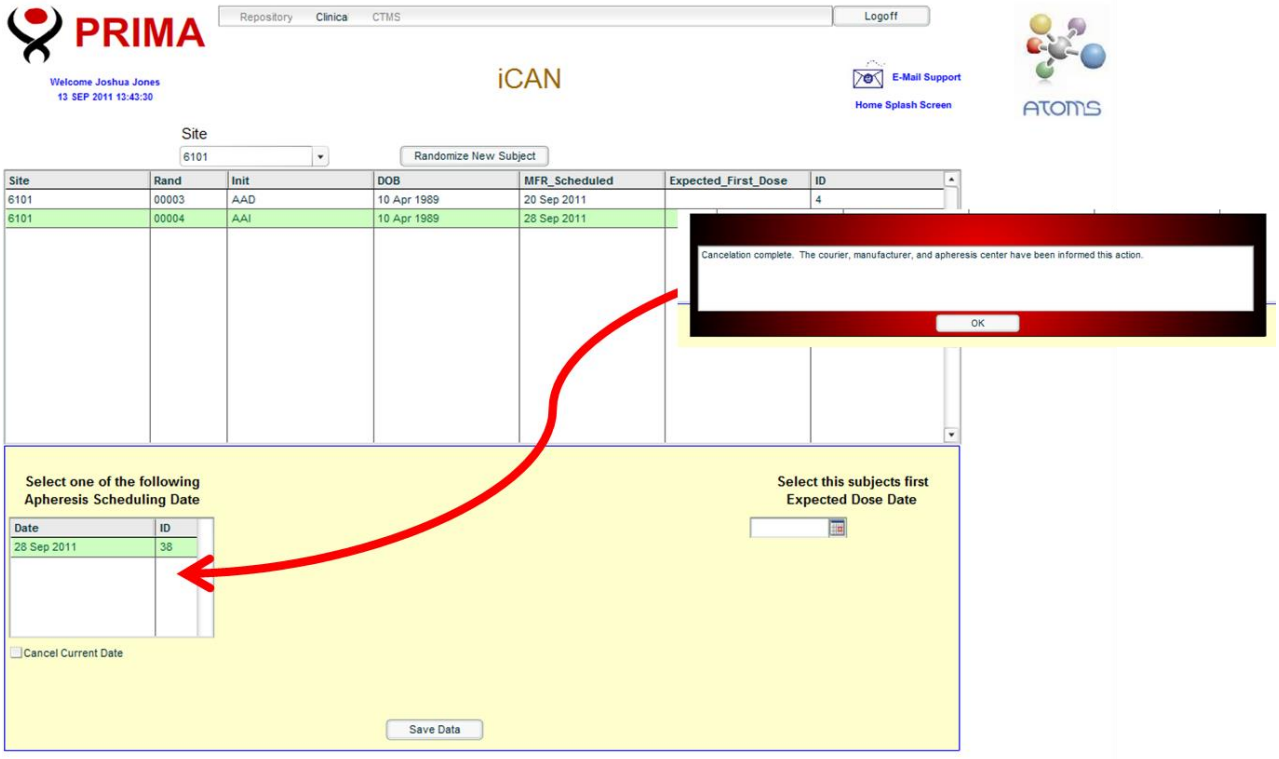


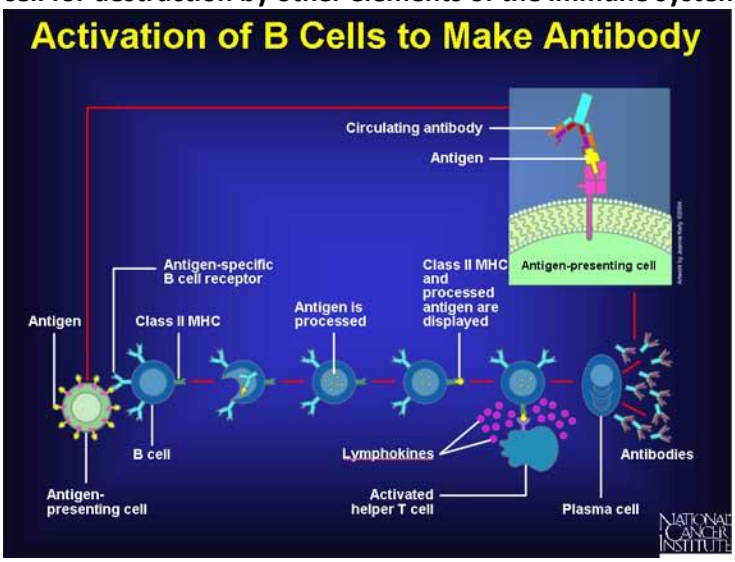
Figure 17. Screen shot of Prima's web-based automated logistics software



Dendritic cell immunotherapy therapy background

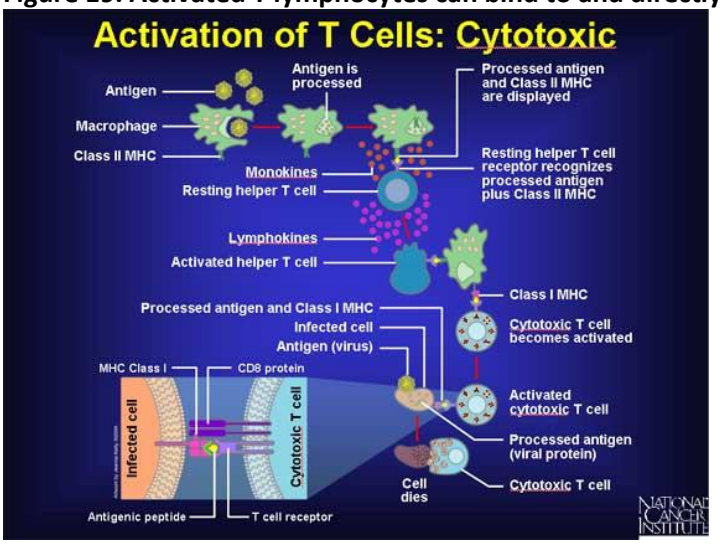
The concept of vaccination is based on training the immune system to recognize a pathogen by presenting a whole organism (killed or weakened bacteria or viruses) or a surface antigen from the organism. The antigen could be a protein, a carbohydrate (a structure composed of sugars) or a carbohydrate-protein or carbohydrate-fat complex that is presented as a linear sequence or as a three dimensional structure. The immune system responds to the antigen by expanding and refining its repertoire of antibody-producing B cells or cytotoxic T cells that can now recognize and neutralize the organism. It then stores plasma cells which retain a memory of the antigen, so that when the same or similar organisms or antigens are next encountered, it can rapidly ramp up an antibody-based or cellular response and thus eliminate these organisms.

Figure 18. B cells cannot kill directly – instead, they make antibodies which flag a pathogen or foreign cell for destruction by other elements of the immune system



Source: National Cancer Institute

Figure 19. Activated T lymphocytes can bind to and directly destroy cells expressing a foreign antigen



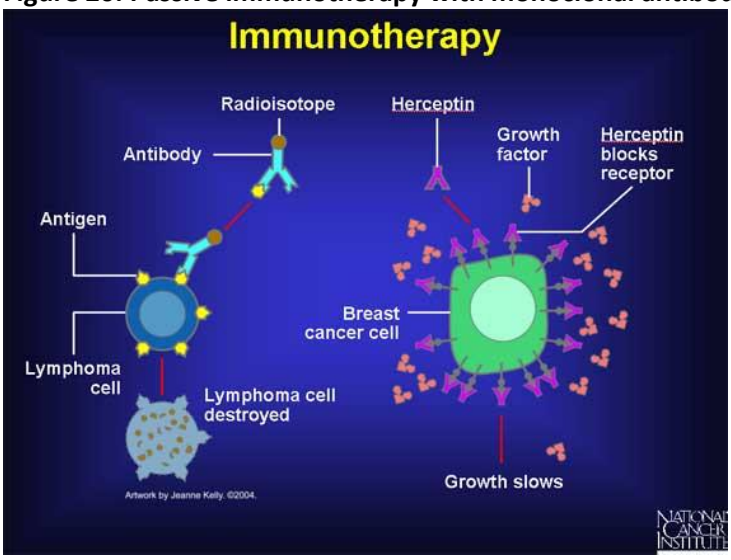
Source: National Cancer Institute

This generally works well with organisms such as bacteria and viruses, unless they have the ability to mutate or change their surface antigens. Scientists have attempted to extend this concept to tumor cells by defining tumor-specific or tumor-associated antigens as potential targets for immunotherapy.

Some antigens, such as EGFR and Her-2, that are overexpressed on tumor cells are also expressed, albeit at lower levels, on normal tissues. Since these antigens are not "foreign", it is difficult to train the patient's immune system to treat them as invaders. In such cases, monoclonal antibodies generated in mice are administered to patients to treat these cancers. This is referred to as passive immunotherapy, whereas when an individual is treated merely with the antigen so they generate their own immune response, it is called active immunotherapy.

Although external antibodies target and bind to tumor cells, they cannot directly kill them, but instead must recruit other elements of the immune system to act as the "executioners". Certain FDA-approved and marketed antibodies are known to do this, a good example being Rituxan for the treatment of non-Hodgkin's lymphoma. However, not all antibodies are able to engage the cell killing mechanism of the immune system, so they are sometimes tagged with a radioisotope to improve their ability to kill tumors, eg. Zevalin.

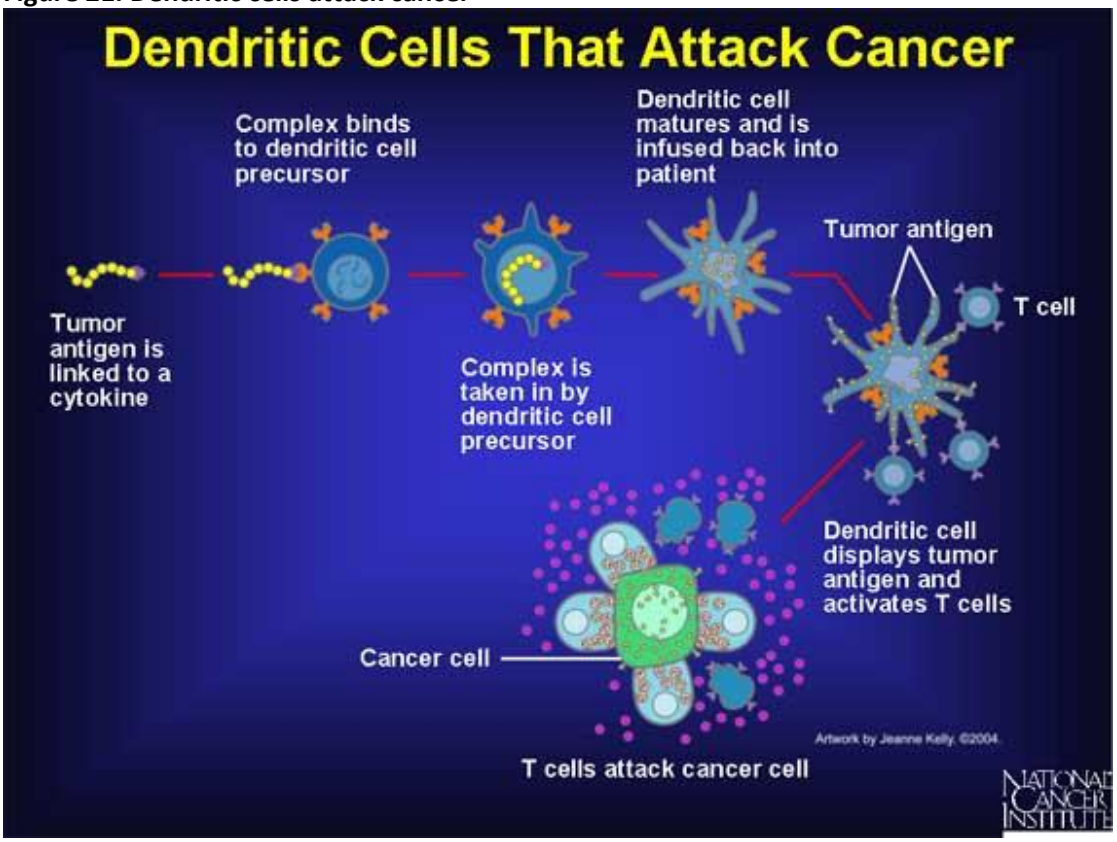
Figure 20. Passive immunotherapy with monoclonal antibodies



Source: National Cancer Institute

In recent years, scientists have begun to focus on ways to activate the cellular arm of the immune system as a means to treat cancer. One method that has been explored is to collect a patient's dendritic cells and expose these to tumor antigens in culture, in the presence of cytokines. When these activated dendritic cells are re-administered to the patient, they are able to activate cytotoxic T lymphocytes to attack the tumor. This approach is utilized by Dendreon (DNDN) in the manufacture of Provenge, and by companies like Prima BioMed and Immunocellular Therapeutics (IMUC). These products have demonstrated good clinical success and highly consistent stimulation of cytotoxic T cells in patients.

Figure 21. Dendritic cells attack cancer



Source: National Cancer Institute

Considerations for cancer vaccines

Complex mixtures or purified antigens

Vaccines may consist of a mixture of components derived from whole cells/cell extracts or may consist of a purified antigen (or antigens).

The disadvantage of cell based products like CVac is the difficulty of ensuring the consistency and potency of whole cells or cell extracts.

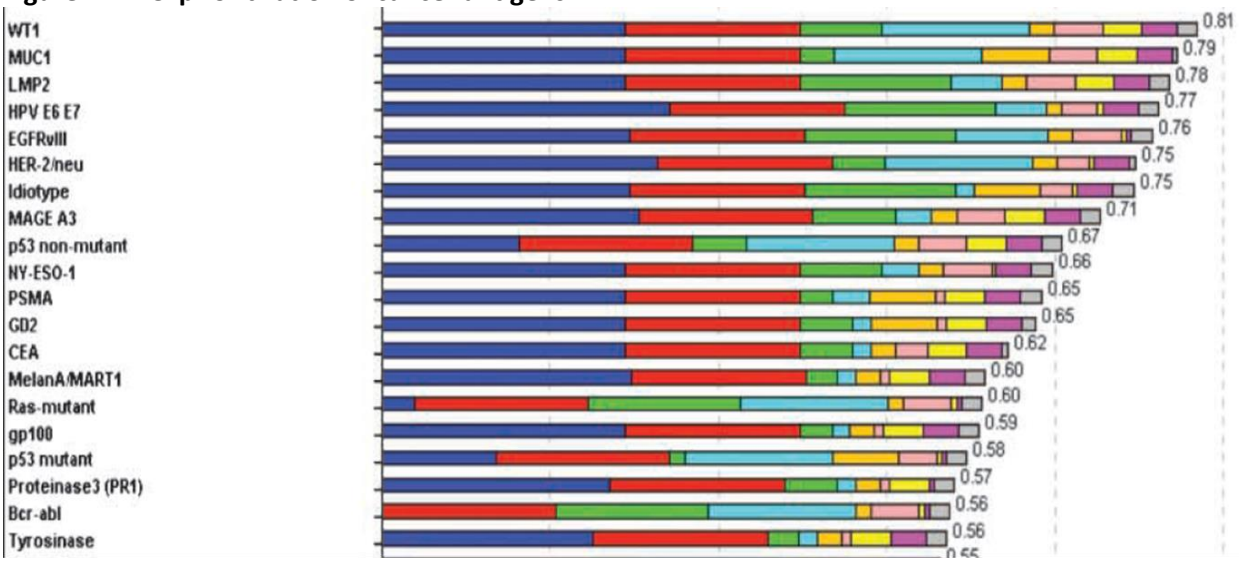
However, a purified antigen alone may not be able to elicit a strong immune response and often require adjuvants, carrier proteins, or additional cytokines. The clinical data in these type of cancer vaccines has not been as consistently impressive as with the cell based approaches.

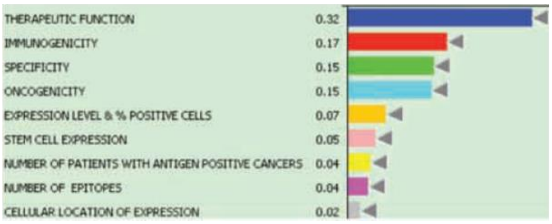
Antigen selection, immunogenicity, and cross reactions

A vaccine must elicit an immune response, either an antibody response or a cellular response. The strength of the immune response is determined by the immunogenicity of the antigen. The ideal antigen would elicit a strong but focused response. However, when the immune response elicited by the antigen also targets normal tissues expressing similar antigens, this is referred to as cross reactivity. Cross reactivity may result in severe side effects, including potential autoimmune reactions.

In 2009, the National Cancer Institute sponsored a pilot project to identify and prioritize potential cancer antigens for cancer vaccine approaches. The ranking are based on the therapeutic function of the antigen, immunogenicity, the specificity to cancer cells, and the oncogenicity of the target, among other factors.

Figure 22. NCI prioritization of cancer antigens





Cheever, et al. The Prioritization of Cancer Antigens: A National Cancer Institute Pilot Project for the Acceleration of Translational Research. *Clin Cancer Res* 2009; 15:5323-5337

Checkpoint blockades

One potential limitation to the effectiveness of cancer vaccines has been the cancer cells' ability to "camouflage" themselves and to mutate to evade the human immune system. Several agents called "checkpoint blockades" have been developed to remove tumor camouflage mechanisms to better allow T cells to infiltrate and kill cancer cells. The first product in this class to market was ipilimumab or Yervoy® from Bristol Myers Squibb. The next products in this class to make it to market appear to be the PD-1 and PDL-1 inhibitors, several of which are in clinical trials. The blockbuster potential of the checkpoint inhibitors has been discussed at length elsewhere and has been well understood and properly valued by the market.

Though the scientific community is increasingly excited, we believe the potential synergy between checkpoint blockades and antigen-specific T cell stimulating products like CVac has not been well understood by the market. The evidence in clinical trials is growing that both approaches in combination are likely to yield clinically meaningful results and longer term control of cancer.

As Dr. Drew Pardoll, Director of the Cancer Immunology Program in Johns Hopkins University's Sidney Kimmel Comprehensive Cancer Center, was recently quoted talking about potential combinations of immunotherapies, "**. . . the real excitement lies with cancer vaccines. Their stock is very low right now, but they are going to come roaring back."**

Prima intellectual property

Prima BioMed has licensed key patent families from the Burnett Institute in Melbourne, Australia, for the process of conjugating an antigen to oxidized mannan for use as a vaccine as well as the process for generating antigen-pulsed dendritic cells.

Prima BioMed has developed important and proprietary bioassays for the characterization of CVac after manufacturing. We believe that the most important intellectual property assets in this space, as is the case with many biologicals, will be around the protection of identity and potency assays used to characterize a product. Prima is well advanced in this regard.

Finances

In line with the Australian regulations Prima BioMed reports financials according to Australian accounting standards (AASB), which is consistently with International Financial Reporting Standards (IFRS).

All numbers are in Australian dollars and the financial year runs from July 1st through June 30th.

More details of the company's finances are obtainable through the company's annual reports and filings to the Australian Securities Exchange at www.asx.com.au

Figure 23. Prima balance sheet

	30 June 2013	30 June 2012	30 June 2011
Cash and cash equivalents	22,023,143	16,991,716	45,918,552
Total current assets	31,808,299	40,902,984	57,062,802
Total non-current assets	1,005,999	709,687	577,859
Total assets	**32,814,298**	**41,216,671**	**57,640,661**
Total current liabilities	3,560,132	4,444,472	2,537,091
Total non-current liabilities	5,748	10,328	4,440
Total liabilities	3,565,880	4,454,800	2,541,531
Total equity	**29,248,418**	**37,157,871**	**55,099,130**

Figure 24. Income Statement

Other Income	30 June 2013	30 June 2012	30 June 2011
Grant income	1,648,725	1,494,253	-
Interest income	939,056	2,682,548	1,033,316
Total other income	**4,005,394**	**4,202,567**	**1,033,316**
Expenses			
R&D and IP	14,005,259	15,118,816	9,531,163
Administrative	4,851,195	5,977,619	5,600,988
Loss before income tax expense for the year	-15,138,798	-19,940,960	-21,081,167
Loss after income tax expense for the year	-15,225,671	-19,940,960	-21,081,167
Total comprehensive loss for the year	-15,261,003	-20,058,195	-21,081,167

Cash position and guidance

Cash, cash equivalents, and term deposit as of September 30, 2013 were approximately A$31.37 million as compared to a little above $30 million at the end of the previous quarter.

Projected loss for the total financial year (July 1, 2013 to June 30, 2014) is approximately A$14 million.