



Appendix 4E Preliminary Financial Report

for the year ended
30 June 2009

(and previous corresponding period: year ended 30 June 2008)

In compliance with Listing Rule 4.3A

DIRECTORS' REPORT

Your Directors present their report on the consolidated entity consisting of Prana Biotechnology Limited and the entities it controlled at the end of, or during, the year ended 30 June 2009.

DIRECTORS

The following persons were Directors of Prana Biotechnology Limited during the whole of the financial year and up to the date of this report, unless stated otherwise:

Mr Geoffrey Kempler	Executive Chairman and Chief Executive Officer
Mr Brian Meltzer	Non-Executive Independent Director
Dr George Mihaly	Non-Executive Independent Director
Mr Peter Marks	Non-Executive Independent Director

REVIEW OF OPERATIONS

Key Events Summary

- > In July 2008, Professor Rudolph Tanzi, Co-Founding scientist of Prana, was appointed by the U.S. Senate Special Committee on Aging on the Future of Alzheimer's: Breakthroughs and Challenges, to testify on Alzheimer's Disease as a growing health care crisis. His testimony spoke to the available therapeutic approaches in Alzheimer's Disease, where PBT2 is differentiated to others by working to detoxify the Abeta protein in brain synapses as well as promoting the clearance of the toxic Abeta from the brain. PBT2 is Prana's lead drug candidate for the treatment of Alzheimer's Disease.
- > In late July 2008, the results of the Phase IIa trial in Alzheimer's Disease patients treated with Prana's PBT2 are published in The Lancet Neurology Journal. The published results described the significant decrease in Abeta protein levels in the cerebrospinal fluid of patients treated with PBT2 at the 250mg dose compared to patients on placebo. The published results also reported that at this same dose patients showed significant improvement in two measures of executive cognitive function compared with placebo, as assessed using the neuropsychological test battery (NTB).
- > During Prana's Annual General Meeting in November 2008, the company reported on the prospect of PBT2 being suitable for the treatment of Huntington's Disease as a second neurological therapeutic application. Prana had commissioned a report from two independent U.S. based clinical researches being key opinion leaders in the field of Huntington's Disease entitled, "The suitability and recommendations for the clinical development of PBT2 in Huntington's Disease". The report reviewed the pre-clinical animal modeling undertaken on PBT2 and the encouraging signs of cognitive effect and clinical safety observed with PBT2 from the Phase IIa Alzheimer's Disease trial. The authors of the report concluded that PBT2 was a suitable drug candidate for clinical development in Huntington's Disease.
- > Results from Prana's Parkinson's Disease program were presented at the Society for Neuroscience conference in Washington D.C. in November 2008. Several of Prana's Metal Protein Attenuating Compounds (MPACs) have demonstrated ability to protect the target brain tissue in Parkinson's Disease, the substantia nigra from cell death. In addition, animal modeling demonstrated that the preservation of the substantia nigra correlated with improved motor function in animals treated with Prana's Parkinson's Disease MPACs.
- > In March 2009, Professor Colin Masters, Director of the Mental Health Research Institute and co-founding scientist of Prana presented new research findings on PBT2 at the 9th International Conference on Alzheimer's and Parkinson's Disease in Prague. The data demonstrated that PBT2 could stop the toxicity of Abeta protein in synapses resulting in the preservation of brain cell synapses. Typically, in transgenic Alzheimer Disease mice there is a loss in the number of synapses leading to diminished neurotransmission and cognitive impairment.
- > In July 2009, Prana was invited to provide an update on the clinical findings to date with PBT2. Dr. Craig Ritchie, based at the Imperial College, London and Prana's European Clinical Advisor presented new results from the Phase IIa trial showing that there was a statistically significant improvement in the overall Executive Function z-score component of the NTB. Previously it had been reported that two of the five Executive Function tests in the NTB were significant but not that the overall composite measure of Executive Function was significant.
- > In July 2009, Prana presented at the International Conference of Alzheimer's Disease (ICAD) in Vienna that PBT2 was able to improve the cognition of transgenic Alzheimer's Disease mice as well as aged, cognitively impaired mice. This data was presented in the Hot Topics session and reported on PBT2's central ability to increase synaptic numbers and improve neurotransmission as a function of PBT2's ability to restore metal homeostasis in the brains of aged mice. Dr. Cherny, Prana's Head of Research explained that aberrant metal distribution in the aged brain is further exacerbated with Alzheimer's Disease.

Drug Development and Research

PBT2 Clinical Development

In late July 2008, the key findings from the Phase IIa trial in Alzheimer's Disease were published in The Lancet Neurology Journal. In patients diagnosed with mild Alzheimer's Disease, those treated with PBT2 on a 250mg per day single dose showed a significant decrease in the Alzheimer's Disease target protein, Abeta, compared to patients on placebo. In addition, we reported a significant improvement in two measures of cognitive Executive Function as assessed using the neuropsychological test battery (NTB) compared with placebo.

In July 2009, the company presented new data at ICAD that an erratum was being prepared for the Lancet Neurology Journal to state that in addition to the previously reported significant findings in two measures of cognitive Executive Function, the overall composite Executive Function z-score assessed across the five component Executive Function tests using was also statistically significant. Performance on cognitive measures of Executive Function has been found in the literature to correlate well with functional measures that are a person's ability to plan, coordinate and execute daily activities. At ICAD, the company also reported on results of a post hoc analysis of the Phase IIa data. The analysis of individual patient responses in the trial, showed that 41% of those receiving PBT2 demonstrated substantial cognitive improvement compared to only 4% of those on placebo, thus re-confirming the promising cognitive effects of PBT2.

During 2008 and 2009, the company has undertaken process development in the production and purification of the PBT2 drug substance. This has enabled Prana to identify a more efficient manufacturing process for prospective scale up manufacturing activities.

PBT2 Research and Animal Modeling

Over the 2008/2009 fiscal year, Prana has been investigating the ability of PBT2 to inhibit the toxic effects of Abeta protein in the synapses of the Alzheimer's Disease brain. Previously our researchers have demonstrated that PBT2 is able to prevent the formation of toxic Abeta oligomers that otherwise accumulate in the synapses, impairing neurotransmission and cognitive function. More recently, we have shown that being able to prevent oligomer formation may be responsible for the observed ability of PBT2 to prevent the loss of synapses and also increase synaptic density in transgenic Alzheimer's Disease mice. The beneficial effect of PBT2 at the synapses is also accompanied by the restoration of the levels of chemical neurotransmitter receptors that are important for normal neurotransmission. This work was presented at the 9th International Conference on Alzheimer's and Parkinson's Disease in Prague, March 2009. This work was supported by the publication in April, 2009 of a paper by Deshpande et al based at the University of California, Irvine which showed Prana's proof of concept MPAC, PBT1, was able to prevent the accumulation of Abeta oligomers at brain synapses enabling normal synaptic function. PBT2 like PBT1, is a zinc binding agent and is able to capture the zinc released by synapses during normal neurotransmission and return the zinc to the synapses. Thus preventing the zinc associated formation of toxic oligomers in synapses.

At ICAD July 2009, the above role of PBT2 in the detoxification of the synapse was presented together with data that PBT2 was also able to improve cognition in cognitively impaired aged mice. This important finding supports the notion that in the aged brain, normal zinc transport mechanisms that influence neurotransmission become impaired resulting in decreased cognitive function. Prana's science suggests that in Alzheimer's Disease the build up of zinc associated Abeta oligomers in the synapses is not only toxic to synapses, but also exacerbates zinc dependent neurotransmission which becomes impaired with age. These mechanistic findings establish PBT2 as an Alzheimer's Disease agent with a differentiated approach to disease modification which targets synaptic toxicity to confer cognitive benefit.

MPAC Pipeline Development

Screening of the MPAC chemical library has yielded very promising opportunities for selected MPACs in neurological disorders other than Alzheimer's Disease. Based on positive in vitro and in vivo modeling of candidate compounds, our medicinal chemistry team has created a series of drug candidates in Parkinson's Disease and brain cancer.

Parkinson's Disease: Of the Parkinson's Disease drug candidates, we have extensively profiled several compounds for their ability to reach the target tissue, being the substantia nigra in the brain and to preserve it from toxins which mimic the slow death of this tissue that results in lowered dopamine production and motor dysfunction in patients. These candidate compounds offer a differentiated therapeutic strategy as they do not artificially supplement dopamine levels as most dopaminergic agents on the market, rather our compounds preserve the substantia nigra from cell death such that normal levels of dopamine are maintained to retain motor function. This strategy appears successful with several agents being able to reduce the loss of motor function in animal models. The company will be positioned to select its lead Parkinson's Disease drug to commence pre-clinical development this year.

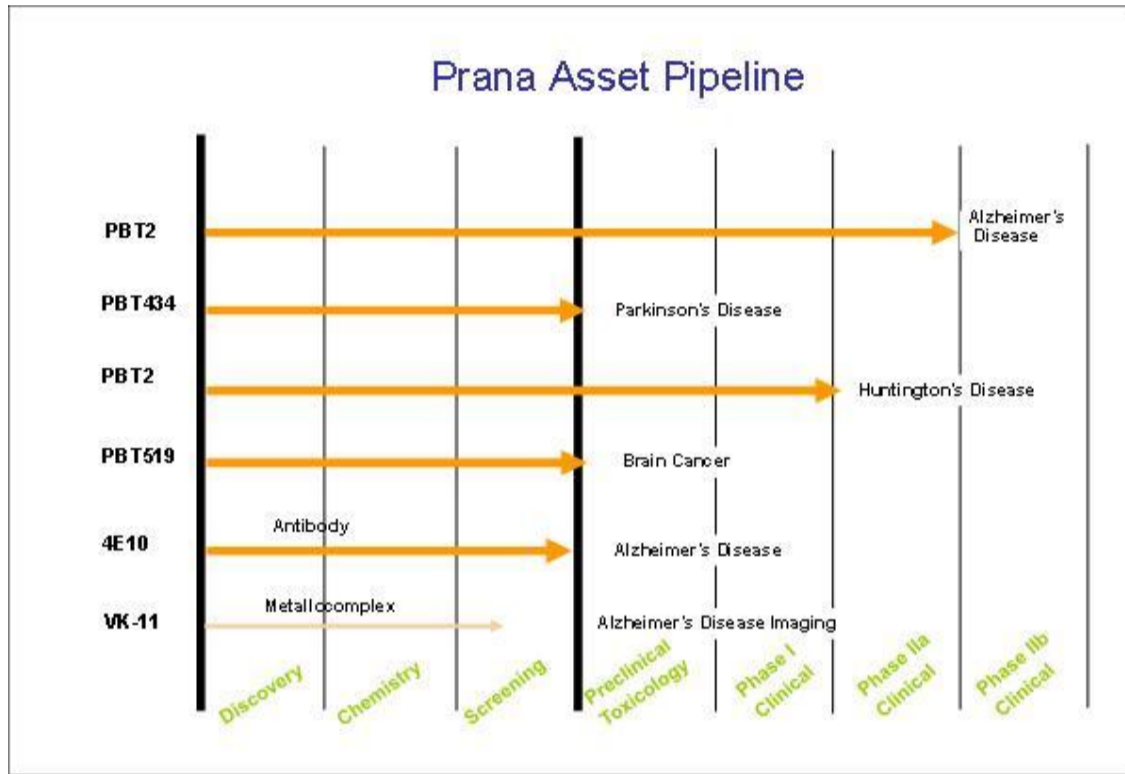
Brain Cancer: Prana had identified several MPAC compounds from its library which have demonstrated significant toxicity against brain cancer with one compound in particular, remaining un toxic to normal neurons. The company undertook further animal modeling with this agent during 2009 with recent results showing that the compound was as effective as the top chemotherapy agent on the market, temozolomide. In addition, animals treated with both our lead brain cancer agent and temozolomide showed added toxicity directed to human brain cancer cells (glioblastoma multiforme) in animal models. Prana is looking to further the development of this compound.

Alzheimer's Disease Immunotherapy

The science behind the MPAC platform also suggests that the oxidatively modified forms of the Abeta oligomers found in the Alzheimer's Disease brain, could be immunological targets for vaccine development. Prana is attempting to validate this selective immunological strategy and will conduct mouse passive vaccine trials with its selective monoclonal antibody which targets a proprietary pathological Abeta target epitope but not the normal, endogenous Abeta. Currently, Prana is scaling up production and purification of the antibody to conduct proof of concept mouse trials. These trials will determine the ability of the antibody to effect improve neuron functioning and cognition.

Amyloid targeting Metallocomplexes

New chemical entities have been generated by Prana scientists that can bind to, and block the metal binding site of Abeta, preventing Abeta from forming toxic aggregates and fibrils. These anti-amyloid 'metallo-compounds' represent a second and complimentary drug discovery platform to the MPAC platform and may provide novel imaging agents which can reach the brain and specifically bind Abeta. If successful, this strategy will provide a novel means of imaging Abeta protein build up in the brain to help in the diagnosis and treatment of Alzheimer's Disease patients. Currently, several agents are being tested for their ability to penetrate the brain.



This report is made in accordance with a resolution of Directors.

Mr Geoffrey Kempler
Executive Chairman and Chief Executive Officer
Melbourne
Dated 26 August 2009

Appendix 4E for the Year Ended 30 June 2009

Results for announcement to the market

Current Reporting Period - Year Ended 30 June 2009
 Previous Reporting Period - Year Ended 30 June 2008

Revenue from continuing operations	down	12.78%	to	\$428,193
Loss after tax attributable to members	down	44.52%	to	(\$7,522,789)
Net loss for the period attributable to members	down	44.52%	to	(\$7,522,789)

Dividends (distribution)	Amount per Security	Franked Amount per Security
Final dividend	n/a	n/a
Previous corresponding period	n/a	n/a

Net Tangible Asset per Security (cents per security)		
As at 30 June 2009	1.85	
As at 30 June 2008	4.89	
Loss per share attributable to the ordinary equity holders of the company, from overall operations (Basic and diluted)		
As at 30 June 2009	3.72	
As at 30 June 2008	7.76	
Record date for determining entitlements to the dividend, (in the case of a trust, distribution)		n/a
Explanation of the above information: Refer to the Directors' Report - Review of Operations.		

CONSOLIDATED INCOME STATEMENT FOR THE YEAR ENDED 30 JUNE 2009

	Notes	30 June 2009 \$	Consolidated Entity	30 June 2008 \$
Revenue from continuing operations		428,193		490,943
Other income		-		170
Intellectual property expenses		(1,107,534)		(469,428)
Auditor and accounting expenses		(129,998)		(331,950)
Research and development expenses		(2,215,358)		(5,757,168)
Personnel expenses		(3,832,804)		(5,350,189)
Depreciation expenses		(34,190)		(25,349)
Other expenses		(978,875)		(975,404)
Travel expenses		(195,251)		(146,651)
Public relations and marketing expenses		(222,679)		(141,337)
Foreign exchange Gain/(Loss)		(6,723)		(402,886)
Gain/(Loss) on fair valuation of financial liabilities		772,430		(451,429)
LOSS BEFORE INCOME TAX		(7,522,789)		(13,560,678)
INCOME TAX EXPENSE		-		-
LOSS FOR THE PERIOD		(7,522,789)		(13,560,678)
		Cents		Cents
Loss per share attributable to the ordinary equity holders of the company, from overall operations				
Basic loss per share	9	(3.72)		(7.76)
Diluted loss per share	9	(3.72)		(7.76)

The above Income Statement should be read in conjunction with the accompanying notes.

CONSOLIDATED BALANCE SHEET AS AT 30 JUNE 2009

	Note	30 June 2009 \$	Consolidated Entity	30 June 2008 \$
CURRENT ASSETS				
Cash and cash equivalents		4,304,977		11,219,035
Trade and other receivables		526		120,641
Other assets		185,433		254,325
TOTAL CURRENT ASSETS		4,490,936		11,594,001
NON-CURRENT ASSETS				
Plant and equipment		71,150		69,148
Other assets		35,164		35,164
TOTAL NON-CURRENT ASSETS		106,314		104,312
TOTAL ASSETS		4,597,250		11,698,313
CURRENT LIABILITIES				
Trade and other payables		604,142		849,113
Other financial liabilities	6	-		772,430
Provisions		194,903		121,082
TOTAL CURRENT LIABILITIES		799,045		1,742,625
NON-CURRENT LIABILITIES				
Provisions		48,389		89,361
TOTAL NON-CURRENT LIABILITIES		48,389		89,361
TOTAL LIABILITIES		847,434		1,831,986
NET ASSETS		3,749,816		9,866,327
EQUITY				
Issued and unissued capital	7	70,188,989		69,842,303
Reserves	8	7,127,332		6,067,740
Accumulated losses		(73,566,505)		(66,043,716)
TOTAL EQUITY		3,749,816		9,866,327

The above Balance Sheet should be read in conjunction with the accompanying notes.

CONSOLIDATED STATEMENT OF CHANGES IN EQUITY FOR THE YEAR ENDED 30 JUNE 2009

Consolidated Entity

	Issued and Unissued Capital \$	Reserves \$	Accumulated Losses \$	Total Equity \$
Balance at 1 July 2007	53,988,412	4,106,821	(52,483,038)	5,612,195
Shares issued, net of costs	14,005,650	-	-	14,005,650
Options exercised	408,936	(408,936)	-	-
Options issued	1,439,305	2,512,988	-	3,952,293
Net (Loss) for the period	-	-	(13,560,678)	(13,560,678)
Options forfeited	-	(143,133)	-	(143,133)
Balance at 30 June 2008	69,842,303	6,067,740	(66,043,716)	9,866,327
Shares issued, net of costs	128,932	-	-	128,932
Options exercised	217,754	(217,754)	-	-
Options issued	-	1,277,346	-	1,277,346
Net (Loss) for the period	-	-	(7,522,789)	(7,522,789)
Options forfeited	-	-	-	-
Balance at 30 June 2009	70,188,989	7,127,332	(73,566,505)	3,749,816

The above Statement of Changes in Equity should be read in conjunction with the accompanying notes.

CONSOLIDATED CASH FLOW STATEMENT FOR THE YEAR ENDED 30 JUNE 2009

	Note	30 June 2009 \$	Consolidated Entity	30 June 2008 \$
CASH FLOWS FROM OPERATING ACTIVITIES				
Payments to suppliers and employees		(7,511,372)		(9,766,851)
Interest received		517,198		375,461
		<hr/>		<hr/>
NET CASH OUTFLOW FROM OPERATING ACTIVITIES	11	(6,994,174)		(9,391,390)
		<hr/>		<hr/>
CASH FLOWS FROM INVESTING ACTIVITIES				
Payments for purchases of plant and equipment		(36,192)		(46,606)
Payment for rental security deposits		-		(35,164)
		<hr/>		<hr/>
NET CASH OUTFLOW FROM INVESTING ACTIVITIES		(36,192)		(81,770)
		<hr/>		<hr/>
CASH FLOWS FROM FINANCING ACTIVITIES				
Proceeds from issues of securities		114,000		14,297,620
Capital raising costs		(13,193)		(580,372)
		<hr/>		<hr/>
NET CASH INFLOW FROM FINANCING ACTIVITIES		100,807		13,717,248
		<hr/>		<hr/>
NET INCREASE/(DECREASE) IN CASH AND CASH EQUIVALENTS		(6,929,559)		4,244,088
		<hr/>		<hr/>
Cash and cash equivalents at the beginning of the year		11,219,035		7,409,256
Effects of exchange rate changes on cash and cash equivalents		15,501		(434,309)
		<hr/>		<hr/>
CASH AND CASH EQUIVALENTS AT THE END OF THE YEAR		4,304,977		11,219,035
		<hr/> <hr/>		<hr/> <hr/>

The above Cash Flow Statement should be read in conjunction with the accompanying notes.

NOTES TO THE FINANCIAL STATEMENTS

Note 1. Basis of Preparation

The company's preliminary financial report does not include all the notes of the type normally included in an annual financial report. The preliminary financial report has been prepared in accordance with the recognition and measurement requirements, but not all disclosure requirements, of Australian Accounting Standards and Interpretations and the Corporations Act 2001. Australian Accounting Standards include Australian equivalents to International Financial Reporting Standards.

Significant accounting policies adopted in preparation of the preliminary financial report are consistent with those adopted by the Company in preparation of the 30 June 2008 financial report and the 31 December 2008 half year financial report.

The preliminary financial report is presented in Australian dollars.

Note 2. Going Concern

The consolidated entity is a development stage medical biotechnology company and as such expects to be utilising cash until its research activities have become marketable. As at 30 June 2009, the consolidated entity incurred an operating loss of A\$7,522,789 (2008 loss: A\$13,560,678). As at year end, the consolidated entity's net assets stood at A\$3,749,816 (2008: A\$9,866,327). The consolidated entity's cash position has decreased to A\$4,304,977 from A\$11,219,035 at 30 June 2008.

The Directors believe that the going concern basis of preparation is appropriate given the following reasons:

- > We plan to increase company funding during the 2009/2010 financial year through a capital raising and/or through licensing of our assets, but if this is unsuccessful or not sufficient, the company has the ability to scale down its operations sufficiently to remain a going concern and keep certain programs going including our research and discovery programs in Parkinson's Disease and vaccine program in Alzheimer's Disease. The company has the ability to further scale down operations if required to maintain IP and search for collaboration opportunities should the need arise.
- > At this time, the Directors are of the opinion that no asset is likely to be realised for an amount less than the amount at which it is recorded in the Balance Sheet at 30 June 2009. Accordingly, no adjustments have been made to the financial report relating to the recoverability and classification of the asset carrying amounts or the classification of liabilities that might be necessary should the consolidated entity not continue as a going concern.

Note 3. Dividends

The company resolved not to declare any dividends in the period ended 30 June 2009.

Note 4. Segment Information

The company's activities are predominately within Australia and cover research into Alzheimer's Disease and other major age-related degenerative disorders.

Note 5. Contingent Liabilities

There has been no change in contingent liabilities since the last annual reporting date.

The Company's former Chief Executive Officer, who also served as a Director, has threatened to initiate a claim against the Company arising from his alleged inability to freely transfer shares underlying certain unexercised ADR options previously granted to him pursuant to the Company's 2004 ADS Plan. The Company believes, based on the explicit terms of the 2004 ADS Plan and applicable law and regulations, his allegations to be without merit and it intends to vigorously defend any such claim if formally asserted.

The company has disclaimed the liability and is defending the action. It is not practical to estimate the potential effect of the claim but legal advice indicates that any liability that may arise in the unlikely event the claim is successful may be significant. No actions or other legal proceedings in respect of this case have been filed.

Otherwise, the Group is not involved in any legal or arbitration proceedings nor, so far as Directors are aware, are such proceedings pending or threatened against the consolidated entity.

Note 6. Financial Liabilities

	30 June 2009		30 June 2008	
<u>Current liabilities</u>	No.	\$	No.	\$
Warrants over ADRs (1 ADR = 10 ordinary shares)	-	-	3,000,000	772,430

Following a meeting of shareholders on 1 June 2004, the Company issued 4 million ADRs (1 ADR = 10 ordinary shares) and 3 million warrants to US investors. The US investors acquired the ADRs at a price of USD 5.00 per ADR with a 3 for 4 attaching warrant. The issue raised USD 20 million (AUD 28.9 million) before costs. The warrants are convertible to ADRs on or before 4 June 2009 at an exercise price of USD 8.00 per warrant.

The warrants expired without being exercised on 4 June 2009.

Under AASB 132 paragraph 11, the warrants associated with this transaction were required to be classified as a Financial Liability, as opposed to Issued Capital, as a result of the warrants being exercisable in a foreign currency, that is a currency different to the functional currency of the Company.

During 2005 the International Financial Reporting Interpretations Committee ("IFRIC") noted that based on the existing wording of IAS 32 (the International Financial Reporting Standards equivalent to AASB 132), any contract entered into by an entity to exchange a fixed number of its own equity instruments for a fixed amount of cash that is denominated in a foreign currency is a Financial Liability and not an equity instrument. The IFRIC discussed and questioned whether this was the appropriate and intended outcome of the standard, and consequently submitted a proposal to the International Accounting Standards Board ("IASB") to amend IAS 32. As the IASB declined to make such an amendment to the standard, the IFRIC conclusion that instruments as described above should be classified as Financial Liabilities continues to stand.

At each reporting date the Financial Liability representing the warrants were required to be revalued to fair value with the movement in the fair value recorded in the Income Statement.

Note 7. Issued and Unissued Capital

	Note	No.	30 June 2009 \$	No.	30 June 2008 \$
Fully Paid Ordinary Shares	(a)	202,710,473	67,487,345	201,800,240	67,140,659
Options over Fully Paid Ordinary Shares	(b)	14,279,133	2,701,644	14,279,133	2,701,644
Total Issued and Unissued Capital			70,188,989		69,842,303

(a) Fully paid ordinary shares

At the beginning of the year	201,800,240	67,140,659	151,517,978	52,726,073
Shares issued	93,750	142,125	48,888,699	14,586,026
Shares issued on exercise of options	816,483	217,754	1,393,563	408,936
Transaction costs relating to share issues	-	(13,193)	-	(580,376)
At the end of the year	202,710,473	67,487,345	201,800,240	67,140,659

(b) Options over fully paid ordinary shares

At the beginning of the year	14,279,133	2,701,644	4,352,893	1,262,339
Options issued as part of capital raising	-	-	9,926,240	1,439,305
At the end of the year	14,279,133	2,701,644	14,279,133	2,701,644

Note 8. Reserves - Share Based Payments

	No.	30 June 2009 \$	No.	30 June 2008 \$
Options over Fully Paid Ordinary Shares	13,335,167	5,158,335	11,051,832	4,098,743
Options over ADRs	380,000	1,515,434	380,000	1,515,434
Options over Warrants	-	453,563	320,000	453,563
Total Share Based Payments	13,715,167	7,127,332	11,751,832	6,067,740

During the year ended 30 June 2009, the following movements in options over fully paid ordinary shares and options over warrants occurred:

Options

- * Issue of 769,818 options to employees
- * Issue of 2,330,000 options to consultants
- * Exercise of 119,641 options by employees
- * Exercise of 696,842 options by consultants

Warrants

- * 320,000 warrants expired on 4 June 2009, held by US investors

Note 9. Loss per Share

	30 June 2009	30 June 2008
Basic loss per share (cents)	(3.72)	(7.76)
Diluted loss per share (cents)	(3.72)	(7.76)
	\$	\$
a) Net loss used in the calculation of basic and diluted loss per share	(7,522,789)	(13,560,678)
	No.	No.
b) Weighted average number of ordinary shares outstanding during the period used in the calculation of basic and diluted loss per share	202,357,885	174,714,146

Options that are considered to be potential ordinary shares are excluded from the weighted average number of ordinary shares used in the calculation of basic loss per share. Where dilutive, potential ordinary shares are included in the calculation of diluted loss per share. All the options on issue do not have the effect to dilute the loss per share. Therefore they have been excluded from the calculation of diluted loss per share.

Note 10. Net Tangible Assets

	30 June 2009	30 June 2008
Net Tangible Assets	\$3,749,816	\$9,866,327
No. of Shares	202,710,473	201,800,240
Net Tangible Assets (cents)	1.85	4.89

Note 11. Cash Flow Reconciliation

	30 June 2009	30 June 2008
	\$	\$
(a) Reconciliation of Cash Flow from Operating Activities with Net Loss after Income Tax Expense for the Year		
	(7,522,789)	(13,560,678)
Add back depreciation expense	34,190	25,349
Add back (gain)/loss on fair value of financial liabilities	(772,430)	451,429
Add back share based payments expense	1,305,471	4,097,562
Loss on sale of plant & equipment	-	-
(Increase)/Decrease in accounts receivable	120,115	(24,142)
(Increase)/Decrease in other current assets	68,892	(85,786)
Increase/(Decrease) in provisions	32,849	83,063
Increase/(Decrease) in accounts payable	(244,971)	(812,496)
Add back foreign exchange	(15,501)	434,309
Net cash flow used in operating activities	<u>(6,994,174)</u>	<u>(9,391,390)</u>

(b) Reconciliation of cash and cash equivalents

Cash and cash equivalents at the end of the financial year as shown in the Cash Flow Statement is reconciled to items in the Balance Sheet as follows:

Cash and cash equivalents	4,304,977	11,219,035
---------------------------	-----------	------------

Note 12. Events Subsequent to Reporting Date

No matters or circumstances have arisen since the end of the reporting period, not otherwise disclosed in this report, which significantly affected or may significantly affect the operations of the consolidated entity, the result of those operations or the state of affairs of the consolidated entity in subsequent financial years.

Note 13. Audit

These accounts are currently in the process of being audited. An Annual Report containing the audit report shall be provided in due course.