Appendix 4E for the Year Ended 30 June 2010

Results for announcement to the market

Current Reporting Period - Year Ended 30 June 2010
Previous Reporting Period - Year Ended 30 June 2009

Revenue from continuing operations	down	49.79%	to	$215,008
Loss after tax attributable to members	down	34.77%	to	($4,906,922)
Net loss for the period attributable to members	down	34.77%	to	($4,906,922)

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 2010	2.23
As at 30 June 2009	1.85

Record date for determining entitlements to the dividend, (in the case of a trust, distribution)	n/a

Explanation of the above information:

Prana Biotechnology Limited recorded revenue of A$215,008 for the year ended 30 June 2010 (2009: A$428,193), which is interest received on company bank accounts. The decrease in interest received is due to a reduction to cash on hand held.

Prana Biotechnology Limited has incurred a loss for the year of A$4,906,922 (2009: A$7,522,789). This loss has decreased due to a reduction in the payment of share based compensation during the current period and a decrease in expenditure on intellectual property and research and development.

Refer to the Directors' Report - Review of Operations for further information.

PRANA BIOTECHNOLOGY Limited

ACN 080 699 065

Appendix 4E
Preliminary Financial Report

for the year ended
30 June 2010

(and previous corresponding period: year ended 30 June 2009)

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 2010.

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
Mr Paul Marks	Non-Executive Independent Director (Appointed 14 January 2010)

REVIEW OF OPERATIONS

Key Events Summary

> In early August 2009, Prana received a Decision to Grant from the European Patent Office for its patent covering selected families of 8-Hydroxyquinoline compounds, including PBT2, Prana's lead clinical drug asset. The patent covers composition of matter claims for PBT2 and other 8-Hydroxyquinolines and their uses in various neurological diseases including Alzheimer's Disease and Huntington's Disease.

> Shortly after the announcement from the European Patent Office, the United States Patent and Trade Mark Office (USPTO) issued Prana with a Notice of Allowance that stated the USPTO's intention to grant the patent covering composition of matter claims for PBT2 and other 8-Hydroxyquinolines and pharmaceutical compositions containing these compounds. The Patent went to formal grant in November 2009.

> In November 2009, the company met with the U.S. FDA to discuss possible clinical trial options for PBT2 in Alzheimer's Disease and Huntington's disease, the meeting was conducted as part of the FDA Pre-Investigational New Drug (pre-IND) program and provided useful guidance on the data required to submit an IND application in the future for PBT2, in either Alzheimer's Disease or Huntington's Disease.

> In November 2009, a erratum to the July 2008 edition of The Lancet Neurology journal was published that corrected the original results of the Neuropsychological Test Battery (NTB) arising from the Phase IIa trial. Importantly, the corrected results were that in addition to two measures of executive function being significant, the overall executive function domain of the NTB comprising five cognitive tests, was significantly improved for those patients taking 250mg of PBT2 compared to patients on placebo. In April 2010, the company finalized its plans for a substantial and definitive Phase IIb trial of PBT2 in Alzheimer's Disease patients. The trial protocol being designed by an appointed protocol steering committee comprising key Alzheimer's Disease clinical trial investigators from the United States, Europe and Australia. The trial design centres on examining the cognitive and functional benefits that PBT2 could deliver to patients. Collectively, the corrected cognitive data from the Phase IIa together with this additional analysis provided strong evidence of the ability of PBT2 to improve cognitive executive function as measured by the NTB.

> In April 2010, an analysis of the responses of individual patients treated with PBT2 in the Phase IIa clinical trial was published in the Journal of Alzheimer's Disease. The analysis demonstrated that there was a significant probability that any patient that showed cognitive Executive Function improvement in the trial was being treated with 250mg of PBT2. Moreover 81% of patients on the 250mg dose of PBT2 responded better on the Executive Function of the NTB (Neuropsychological Test Battery) score than the best performing patient on placebo. Improvement in ADAS-cog a measure of memory and cognition was observed with patients treated with 250mg of PBT2, almost reaching statistical significance by twelve weeks of the Phase IIa trial.

> In light of the several drugs failing to hit cognitive endpoints in Phase II or Phase III Alzheimer's Disease trials during the year, and armed with evidence that PBT2 can improve cognition, the company issued a Therapeutic Strategy Paper on the Prana website. The paper outlines how such drugs purported to be based on the 'Amyloid Hypothesis' for Alzheimer's Disease have not targeted the actual gain of toxic function by amyloid unlike PBT2 to deliver patient benefit.

> In June 2010, Professor Ashley Bush, Prana's co-founder and a member of the company's R&D Advisory Board was invited to present at the Annual Meeting of the American Aging Association in Portland, Oregon. Professor Bush reported on the findings from animal models that PBT2 can be effective in reversing age-related cognitive decline by restoring zinc flow across the synapse (the gap between adjacent neurons). Alzheimer's Disease is believed to exaggerate the loss of normal zinc transport across the synapse due to the ability of amyloid plaques and oligomers in the synapses to trap zinc and copper.

> In July 2010, Prana's Head of Research, Assoc. Professor Robert Cherny presented a paper at the International Conference of Alzheimer's Disease (ICAD) in Honolulu, Hawaii entitled, "Novel molecular mechanisms for the neurotrophic and neuroprotective effects of PBT2 in Alzheimer's Disease and Huntington's Disease". Dr. Cherny presented new data showing that the effect of transporting zinc and copper to neurons results in the activation of important cell pathways that act to prevent neuronal death and promote neuronal function. In addition, Dr Cherny presented data linking the neuroprotective qualities of PBT2 to beneficial effects on survival, motor coordination and brain tissue preservation in an animal model of Huntington's Disease.

Drug Development and Research

PBT2 Clinical Development

In November 2009, Prana presented its pre-clinical and clinical information package to the FDA (U.S. Food and Drug Administration) in accordance with the Pre-Investigational New Drug (IND) Consultation Program. The meeting provided useful guidance on possible steps to take to open an IND Application with the FDA to undertake clinical trials in the United States in Alzheimer's Disease or Huntington's Disease. The meeting was productive and provided important information for the company to help form its regulatory strategy for the development of PBT2 in these neurological indications.

During early 2010, a Phase IIb trial protocol was developed and finalized under the guidance of an international protocol steering committee. The protocol provides for a substantial trial measuring the effects of PBT2 on cognition and functional abilities in patients with mild to moderate Alzheimer's Disease. In addition, the company has begun devising a protocol to test PBT2 in Phase II trial of PBT2 in patients in Huntington's Disease.

During 2000 and 2010, the company has successfully improved its manufacturing and product purification processes of the PBT2 drug substance. This has resulted in an increased efficiency in drug manufacture as evidenced from pilot runs that are now being implemented in large scale manufacturing campaigns to support PBT2's clinical development in Phase II trials.

PBT2 Research and Animal Modeling

Over the 2009/2010 fiscal year Prana scientists made further progress in revealing how PBT2 influences the complex web of neuronal biochemical signaling pathways which are responsible for switching on and off the production of receptors and growth factors in the brain. These signalling pathways have been found to be turned down in the absence of copper and zinc, which occurs in the aging and Alzheimer's brain. When these metals are replenished by the "chaperone" - like activity of PBT2, neurons exhibit improved function and plasticity (the ability to change shape and form new electrical connections). Our research studies indicate that in Alzheimer's disease this neuroprotective or restorative activity of PBT2 coupled with PBT2's ability to detoxify Abeta protein is responsible for the improvement in cognition observed in animal models and clinical studies to date.

Key publications from the laboratory of Prana Chief Scientific Advisor Professor Ashley Bush published in the first half of 2010 have provided further evidence of the link between age and disease-related defects in metal homeostasis. The first of these, in The Journal of Neuroscience, reported the strong link between the age-dependent loss of the ability of brain cells to pump zinc in and out and the accumulation of zinc in the amyloid plaques in Alzheimer's disease. In the second article, published in the top ranked journal Cell, the amyloid precursor protein, the molecule from which Abeta is generated, is shown to have a hitherto unknown function as the neuronal Ferroxidase, the purpose of which is to regulate the export of iron from brain cells. The emerging hypothesis is that age related defects in neuronal metal homeostasis may be exaggerated and accelerated in Alzheimer's disease.

In July 2010 Prana was invited to present at one of the "Hot Topics" sessions at the Alzheimer's Association International Conference on Alzheimer's disease in Hawaii. The theme of the talk presented by Prana's Head of Research, A/Prof Robert Cherny was the molecular basis of the neuroprotective properties of PBT2 in animal models of Alzheimer's disease, and for the first time in a scientific forum - Huntington's disease. The presentation demonstrated that the beneficial effects of PBT2 in animal models of both conditions may derive from stimulation of similar (metal responsive) intracellular signaling pathways. Prana's lead compound may have clinical application in both indications.

MPAC Pipeline Development

The growth of Prana's MPAC (Metal Protein Attenuating Compound) technology into various neurological disorders other than Alzheimer's Disease has been a key element of Prana's business plan to provide increased opportunity for product diversification. Prana's MPACs are brain penetrable and orally available neurologically active agents. To date, Prana's MPAC chemical library has yielded clinical development candidates in Parkinson's Disease and brain cancer.

Parkinson's Disease: During 2009/2010 we have continued in vivo testing of selected Parkinson's Disease drug candidates for their ability to preserve the target tissue in the brain that perishes in Parkinsons Disease, the substantia nigra. The substantia nigra produces dopamine, an important neurotransmitter that controls muscle movement. Several promising compounds were identified that preserved this tissue in two models of Parkinson's Disease, the 6-hydroxydopamine and the MPTP model. Moreover, work undertaken this year has shown that one drug in particular, PBT434 can significantly decrease the amount of a neuronal protein called α-synuclein that otherwise aggregates in Parkinson's Disease to form cellular inclusion bodies called Lewy Bodies, a hallmark of Parkinson's Disease. The neuroprotective qualities of PBT434 translated into significant improvement in motor function and motor coordination in the disease models. Prana's therapeutic strategy differs from others strategies by protecting the substantia nigra thus retaining motor function unlike other agents on the market which artificially supplement dopamine levels.

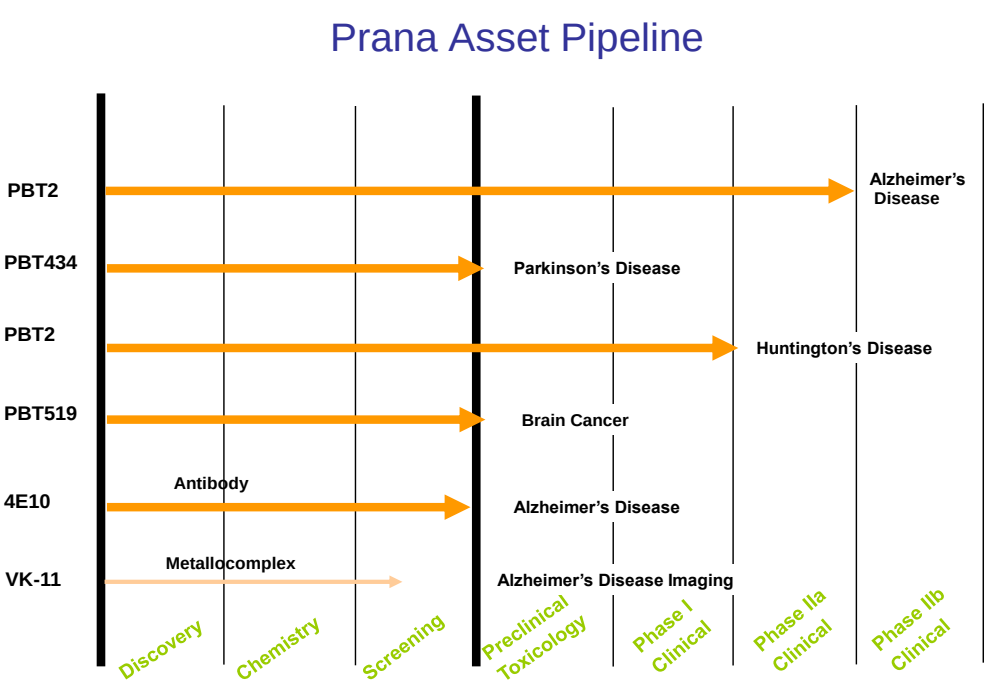
Brain Cancer: Prana had identified several MPAC compounds from its library which have demonstrated significant toxicity against brain cancer, specifically the highly malignant and most common form, glioblastoma multiforme. In work leading up to the end of 2009, several MPACs were tested in three mouse models of this glioma brain cancer, the C6, SMA560 and U87MG models with several compounds showing promising results. In vitro assays of the active compounds revealed that one compound; PBT519 was effective in killing human glioma cells, without affecting healthy neurons. The chemotherapeutic agent, temozolomide is currently the leading drug for use in high grade gliomas yet it only has modest effect on improving patient survival and is often associated with undesirable side effects. To examine the therapeutic potential of PBT519 the company began testing its efficacy in the presence or absence of temozolomide in two animal models of glioma, the SMA560 and U87MG. Previously the company reported preliminary findings which suggested that that the combination of PBT519 and temozolomide had improved tumour-reducing effects in animals injected with human glioma cells. These experiments have now been completed and demonstrate that PBT519 was effective in reducing glioma brain tumours. Moreover, when co administered with temozolmide, there was a synergistic effect in further reducing tumour volume compared to single compound treatment with PBT519 or temozolomide in both models of glioblastoma multiforme.

Alzheimer's Disease Immunotherapy

The science behind the MPAC platform also suggests that the oxidatively modified forms of the Abeta oligomers found in the AD brain, could be immunological targets for vaccine development. Prana undertaking validation of 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. Prana is currently in the process of scale 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 complementary drug discovery platform to the MPAC platform and may provide novel imaging agents which can reach the brain and specifically bind Abeta. Alternatively the compounds may be tested in the future for diseases outside the brain, such as diabetes, which do involve accumulation of amyloid-type protein aggregates.



Intellectual Property Developments:

Prana has maintained its intellectual property strategy of seeking broad 'composition of matter' claims and continuously improving the protection of its platform technology and drug assets. Over the last year Prana has received further approvals from international patent office's relating to its lead Alzheimer's Disease drug, PBT2.

> Four national phase patent cases protect Prana's core MPAC technology. The first case is directed to the 8-Hydroxyquinoline chemical class which covers PBT2 and other lead 8- Hydroxyquinoline compounds. The other three cases are directed to several 'Follow Up' next generation MPAC chemical classes, which comprise alternative MPAC scaffolds to the 8- Hydroxyquinoline chemical scaffold. These patent cases include claims to the MPAC compositions of matter and the uses of these compounds in numerous neurological disorders. All four cases have made further successful progress in their examination through the major international patent offices. In particular:-

(i) In November 2009, Prana received Grant from the United States Patent and Trade Mark Office for its key patent protecting the clinical drug asset PBT2. The United States patent, which is entitled, '8-Hydroxyquinoline derivatives,' covers the composition of matter of selected families of 8-Hydroxyquinoline compounds, including PBT2. In October 2009, Prana also received Grant from the Australian Patent and Trademark Office.

(ii) In June 2010, the corresponding European patent also entitled '8-Hydroxyquinoline derivatives' protecting Prana's clinical drug asset PBT2 was placed on to the European Patent Registry after completion of its 9 month post-grant opposition period without challenge. The patent, also covers the composition of matter of selected families of 8-Hydroxyquinoline compounds, including PBT2, and the uses of such compounds for the treatment of neurological diseases, including Alzheimer's Disease and Huntington's Disease.

(iii) In February 2010, Prana received Grant from the United States Patent and Trade Mark Office for a sub-class of compounds within its Follow Up's patent entitled 'Neurologically active derivatives'. A further divisional case was also filed to seek protection of an additional sub-class of compounds. Also in February 2010, Prana received a Notice of Acceptance from the Australian Patent and Trademark Office for the corresponding Australian patent.

(iv) The second follow up case entitled 'Neurologically active compounds' is directed to alternative, selected MPAC scaffolds has applications granted in South Africa and Singapore. Both applications in Mexico and Russia were Accepted in July 2010.

(v) The third follow up case entitled 'Method of Treatment and prophylaxis and agetnts useful for same' is directed to novel MPAC scaffolds has had a South African patent Accepted in June 2010.

> A national phase patent family entitled 'Methods of treatment of Glioma Brain Tumour' directed to the use of MPAC compounds for the treatment of brain cancer has cases progressing in Australia, Canada, China, Europe, Japan and the USA.

> A patent application entitled 'Neurotoxic Oligomers' exclusively licensed from The General Hospital Corporation and relating to an immunotherapy treatment for Alzheimer's Disease continues to be successfully prosecuted in the major jurisdictions. Specific claims to preferred vaccine antigens for active immunotherapy treatment have been Granted in the United States and cases of broader scope have been Granted in Australia and New Zealand.

> An International (PCT) patent application entitled 'Compounds for Therapy and Diagnosis' has progressed to National phase in Australia, Canada, New Zealand, Europe, the United States and Japan. This case covers novel metallo-complex compounds that are designed to treat Alzheimer's Disease by binding to the metal binding site of ABeta in the brain. The case also covers the use of these metallo-complexes as imaging agents for Alzheimer's Disease.

> An Australian provisional patent application entitled 'Processes for the preparation of an 8-Hydroxy Quinoline derivative' has been re-filed to cover alternative synthetic routes to PBT2.

> An Australian provisional patent application has progressed to an International (PCT) application entitled 'Quinazolinone compounds' and covers novel chemical drug candidates for neurological conditions, particularly Parkinson's Disease.

This document contains some statements which are by their very nature forward looking or predictive. Such forwarding looking statements are by necessity at least partly based on assumptions about the results of future operations which are planned by the Company and other factors affecting the industry in which the Company conducts its business and markets generally. Such forward looking statements are not facts but rather represent only expectations, estimates and/or forecasts about the future and thereby need to be read bearing in mind the risks and uncertainties concerning future events generally. There are no guarantees about subjects dealt with in forward looking statements. Indeed, actual outcomes may differ substantially from that predicted due to a range of variable factors.

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

Mr Geoffrey Kempler
Executive Chairman and Chief Executive Officer
Melbourne
Dated 27 August 2010

CONSOLIDATED STATEMENT OF COMPREHENSIVE INCOME
FOR THE YEAR ENDED 30 JUNE 2010

	Note	Consolidated Entity 30 June 2010 $	30 June 2009 $
Revenue from ordinary activities		215,008	428,193
Intellectual property expenses		(431,082)	(1,107,534)
Auditor and accounting expenses		(168,909)	(129,998)
Research and development expenses	6	(87,992)	(2,215,358)
Personnel expenses		(3,087,234)	(3,832,804)
Depreciation expenses		(35,290)	(34,190)
Other expenses		(940,699)	(978,875)
Travel expenses		(234,555)	(195,251)
Public relations and marketing expenses		(130,090)	(222,679)
Foreign exchange gain (loss)		(6,079)	(6,723)
Gain (loss) on fair valuation of financial liabilities		-	772,430
Loss before income tax expense		(4,906,922)	(7,522,789)
Income Tax Expense		-	-
Loss for the period		(4,906,922)	(7,522,789)
Other comprehensive income		-	-
Total comprehensive income for the period		(4,906,922)	(7,522,789)
		Cents	**Cents**
Loss per share attributable to the ordinary equity holders of the Company:			
Basic loss per share	9	(2.16)	(3.72)
Diluted loss per share	9	(2.16)	(3.72)

The above consolidated statement of comprehensive income should be read in conjuction with the accompanying notes.

CONSOLIDATED STATEMENT OF FINANCIAL POSITION
AS AT 30 JUNE 2010

	Note	Consolidated Entity 30 June 2010 $	30 June 2009 $
ASSETS			
CURRENT ASSETS			
Cash and cash equivalents		5,227,298	4,304,977
Trade and other receivables		825	526
Other current assets		1,479,603	185,433
TOTAL CURRENT ASSETS		6,707,726	4,490,936
NON-CURRENT ASSETS			
Plant and equipment		58,527	71,150
Other non-current assets		35,164	35,164
TOTAL NON-CURRENT ASSETS		93,691	106,314
TOTAL ASSETS		6,801,417	4,597,250
LIABILITIES			
CURRENT LIABILITIES			
Trade and other payables		1,244,417	604,142
Provisions		256,074	194,903
TOTAL CURRENT LIABILITIES		1,500,491	799,045
NON-CURRENT LIABILITIES			
Provisions		71,610	48,389
TOTAL NON-CURRENT LIABILITIES		71,610	48,389
TOTAL LIABILITIES		1,572,101	847,434
NET ASSETS		5,229,316	3,749,816
EQUITY			
Issued and unissued capital	7	75,120,164	70,188,989
Reserves	8	8,582,579	7,127,332
Accumulated losses		(78,473,427)	(73,566,505)
TOTAL EQUITY		5,229,316	3,749,816

The above consolidated statement of financial position should be read in conjuction with the accompanying notes.

CONSOLIDATED STATEMENT OF CHANGES IN EQUITY
FOR THE YEAR ENDED 30 JUNE 2010

	Consolidated Entity			
	Issued and Unissued Capital	Reserve	Accumulated Losses	Total
	$	$	$	$
Balance at 30 June 2008	**69,842,303**	**6,067,740**	**(66,043,716)**	**9,866,327**
Transactions with owners in their capacity as owners:				
Shares issued gross of costs	142,125	-	-	142,125
Options exercised	217,754	(217,754)	-	-
Options issued	-	760,913	-	760,913
Transaction costs	(13,193)	-	-	(13,193)
Share options - value of share option scheme	-	516,433	-	516,433
	346,686	1,059,592	-	1,406,278
Loss for the year	-	-	(7,522,789)	(7,522,789)
Total comprehensive income for the year	**-**	**-**	**(7,522,789)**	**(7,522,789)**
Balance at 30 June 2009	**70,188,989**	**7,127,332**	**(73,566,505)**	**3,749,816**
Transactions with owners in their capacity as owners:				
Shares issued gross of costs	5,185,124	-	-	5,185,124
Options exercised	90,107	(90,107)	-	-
Options issued	-	1,330,403	-	1,330,403
Transaction costs	(344,056)	-	-	(344,056)
Share options - value of share option scheme	-	214,951	-	214,951
	4,931,175	1,455,247	-	6,386,422
Loss for the year	-	-	(4,906,922)	(4,906,922)
Total comprehensive income for the year	**-**	**-**	**(4,906,922)**	**(4,906,922)**
Balance at 30 June 2010	**75,120,164**	**8,582,579**	**(78,473,427)**	**5,229,316**

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 2010

	Note	Consolidated Entity 30 June 2010 $	30 June 2009 $
CASH FLOWS RELATED TO OPERATING ACTIVITIES			
Payments to suppliers and employees		(4,923,648)	(7,511,372)
Interest received		214,709	517,198
NET OPERATING CASH FLOWS	11	(4,708,939)	(6,994,174)
CASH FLOWS RELATED TO INVESTING ACTIVITIES			
Payments for purchases of plant and equipment		(22,667)	(36,192)
NET INVESTING CASH FLOWS		(22,667)	(36,192)
CASH FLOWS RELATED TO FINANCING ACTIVITIES			
Proceeds from issues of securities		6,000,000	114,000
Transaction costs relating to equity issuances		(344,056)	(13,193)
NET FINANCING CASH FLOWS		5,655,944	100,807
NET INCREASE (DECREASE) IN CASH AND CASH EQUIVALENTS		924,338	(6,929,559)
Cash and cash equivalents at the beginning of the year		4,304,977	11,219,035
Effects of exchange rate changes on cash and cash equivalents		(2,017)	15,501
CASH AND CASH EQUIVALENTS AT THE END OF THE YEAR		5,227,298	4,304,977

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

NOTES TO THE FINANCIAL STATEMENTS

Note 1. Basis of Preparation
These general purpose financial statements for the year ended 30 June 2010 have been prepared in accordance with Australian Accounting Standards, other authoritative pronouncements of the Australian Accounting Standards Board, Urgent Issues Group Interpretations and the *Corporations Act 2001.*

The company's prelimiary financial statements comply with International Financial Reporting Standards ("IFRS") as issued by the International Accounting Standards Board ("IASB") and Australian equivalents to International Financial Reporting Standards ("A-IFRS").

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 2009 financial report and the 31 December 2009 half year financial report.

Effective as of 1 July 2009, the Company adopted AASB 8 Operating Segments, which replaces AASB 114 Segment Reporting. The new standard requires a 'management approach', under which segment information is presented on the same basis as that used for internal reporting purposes. This has had no effect on the consolidated entity as one reporting segment is still deemed applicable given that the Company is not a complex operation. The Company reports segment information in a manner that is consistent with the internal reporting provided to the chief operating decision maker. The chief operating decision maker, who is responsible for allocationg resources and assessing performance of the operating segments, has been identified as the Company's steering committee that makes strategic decisions.

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 2010, the consolidated entity incurred an operating loss of A$4,906,922 (2009 loss: A$7,522,789). As at year end, the consolidated entity's net assets stood at A$5,229,316 (2009: A$3,749,816). The consolidated entity's cash position has increased to A$5,227,298 from A$4,304,977 at 30 June 2009.

There remains significant uncertainty of the Company’s ability to continue as a going concern for a further 12 months from the date of signing the financial report and, therefore, whether the Company will realize its assets and extinguish its liabilities in the normal course of business and at the amounts stated in the financial report. However, the Directors believe that the going concern basis of preparation is appropriate given the funding expected from the following sources:

> Since inception, the consolidated entity has been able to raise funds to pursue its research programs, raising in excess of $85m through the issue of equity and warrants, before costs. In the past twelve months, the consolidated entity has demonstrated that it can raise capital by raising A$6,000,000 through the issue of equity, before costs. The Directors believe that there is an expectation that they can raise additional funding to enable the consolidated entity to continue to pursue the current business objectives and at the General Meeting held on 17 August 2010, received shareholder approval to issue 225,000,000 new ordinary shares to raise approximately A$27M, dependant on the final issue price.

> Given the significant uncertainty of capital markets, other sources of funding to support the current business objectives are being pursued in parallel. Including potential joint venture arrangements, merger, acquisition and other means of leveraging resources from potential partners to continue the business objectives of the consolidated entity over the next twelve months.

At this time, the Directors are of the opinion that no asset is likely to be realized for an amount less than the amount at which it is recorded in the Statement of Financial Position at 30 June 2010. Therefore, 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 2010.

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
A contingent liability which was reported by the company in its last annual report, relating to a past employee matter, is no longer considered material.

There are no contingent assets or liabilities at the date of this report. The consolidated entity is not involved in any legal or arbitration proceedings and, so far as the Directors are aware, no such proceedings are pending or threatened against the company.

Note 6. Research and Development

For the year ended 30 June 2010, the Company incurred research and development expenses of $2,340,377. Such expenses were offset by cash that the Company received or is receivable, due to an adjustment under a research and development contract, resulting in the line item of research and development expenses for such period being $87,992.

Note 7. Issued and Unissued Capital

	Note	30 June 2010 No.	30 June 2010 $	30 June 2009 No.	30 June 2009 $
Fully Paid Ordinary Shares	(a)	234,045,871	72,418,520	202,710,473	67,487,345
Options over Fully Paid Ordinary Shares	(b)	-	2,701,644	14,279,133	2,701,644
Total Issued and Unissued Capital			75,120,164		70,188,989
(a) Fully paid ordinary shares					
At the beginning of the year		202,710,473	67,487,345	201,800,240	67,140,659
Shares issued		30,915,000	5,185,124	93,750	142,125
Shares issued on exercise of options		420,398	90,107	816,483	217,754
Transaction costs relating to share issues		-	(344,056)	-	(13,193)
At the end of the year		234,045,871	72,418,520	202,710,473	67,487,345
(b) Options over fully paid ordinary shares					
At the beginning of the year		14,279,133	2,701,644	14,279,133	2,701,644
Expired options, unexercised		(14,279,133)	-	-	-
At the end of the year		-	2,701,644	14,279,133	2,701,644

Note 8. Reserves - Share Based Payments

	30 June 2010 No.	30 June 2010 $	30 June 2009 No.	30 June 2009 $
Options over Fully Paid Ordinary Shares*	26,419,378	6,613,582	13,335,167	5,158,335
Options over ADRs	380,000	1,515,434	380,000	1,515,434
Options over Warrants	-	453,563	-	453,563
Total Share Based Payments	26,799,378	8,582,579	13,715,167	7,127,332

*Subsequent to the year end, 4,677,500 options expired unexercised.

During the year ended 30 June 2010, the following movements in options to purchase fully paid ordinary shares occurred:
Options
* Issue of 10,000,000 options to an investor
* Issue of 4,640,000 options to consultants
* Issue of 1,064,609 options to employees
* Exercise of 260,398 options by employees
* Exercise of 160,000 options by consultants

Note 9. Loss per Share

	30 June 2010	30 June 2009
Basic loss per share (cents)	(2.16)	(3.72)
Diluted loss per share (cents)	(2.16)	(3.72)
	$	$
a) Net loss used in the calculation of basic and diluted loss per share	(4,906,922)	(7,522,789)
	No.	No.
b) Weighted average number of ordinary shares outstanding during the period used in the calculation of basic and diluted loss per share	227,527,388	202,357,885

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 2010	30 June 2009
Net Tangible Assets	$5,229,316	$3,749,816
No. of Shares	234,045,871	202,710,473
Net Tangible Assets (cents)	2.23	1.85

Note 11. Cash Flow Reconciliation

	30 June 2010	30 June 2009
	$	$
(a) Reconciliation of Cash Flow from Operating Activities with Net Loss after Income Tax Expense for the Year		
	(4,906,922)	(7,522,789)
Add back depreciation expense	35,290	34,190
Add back (gain)/loss on fair value of financial liabilities	-	(772,430)
Add back share based payments expense	730,478	1,305,471
(Increase)/Decrease in accounts receivable	(299)	120,115
(Increase)/Decrease in other current assets	(1,294,170)	68,892
Increase/(Decrease) in provisions	84,392	32,849
Increase/(Decrease) in accounts payable	640,275	(244,971)
Add back foreign exchange	2,017	(15,501)
Net cash flow used in operating activities	(4,708,939)	(6,994,174)
(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	5,227,298	4,304,977

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.