2001 President's Report

2001

The year 2001 was a very momentous and busy year for the Baker and the Baker family. Two events of particular significance occurred. In July, the Institute's much loved and long serving director, Professor John Funder, announced that he would retire upon the move into our new building. The second momentous event was the move itself, which occurred in late 2001. Each of these events caused some disruption to the settled life and activities of the Baker. But we seem to have survived both of them and the Board is confident, now that the dust has settled on the building site (and been brushed off the Institute's staff!) that the Baker has emerged as a stronger, more unified and far better resourced organisation. We are at the dawn of a new and exciting era in the Baker's history.

John Funder's contribution to the life and achievements of the Baker Institute over the last decade has been enormous. At valedictory dinners given for John late in 2001, many speeches of appreciation and thanks were given in his honour. John's contributions, not only to the affairs and direction of the Baker, but also to the wider faculties of Australian and international medical research, science and education, cannot be under-estimated. Virtually every Australian medical research scientist, most politicians, and all of those with any knowledge of the field (including even amateurs like me) have been beneficiaries of John's lifetime of effort on behalf of Australian science and medicine. The Baker will surely miss John, but we are grateful to have had his advice, care and attention for so long.

John Funder's successor is the ably qualified and equally well known and respected Professor Garry Jennings. Prior to his appointment as Director, Garry was the Deputy Director of the Baker, the Director of Cardiovascular Medical Services for the Bayside Network and Alfred Hospital and head of the Alfred Baker Medical Unit for many years. Garry's appointment as Director of the Baker strengthens and deepens the very important bonds that have always existed between the Baker, the Alfred and the Bayside Network and will ensure that the cardiovascular and clinical research foci of the Baker's work will continue to be at the forefront of our activities. Every local and international peer reviewer of the Baker whom the Board has consulted in the last decade has emphasised to us that our clinical research linkage with the Alfred is the most valuable competitive advantage we have over most other medical research institutes of similar scale and importance anywhere in the world. The Board is very conscious of the significance and value of this link and feels that, with Garry's appointment, even better results will be achieved from it in the future.

We are sorry that John Funder could not lead the troops on their storming of the citadel which the Baker now occupies overlooking Commercial Road and Fawkner Park. On the other hand, the building has John's francophile fingerprints all over it, right down to the magnificent "Chartres" stained glass East window and the Piet Mondrian colour scheme on its exterior. Despite the strict budgetary and efficiency constraints which were imposed in the building programme (necessary in any expenditure of public money for such a purpose), I am sure that all who visit will agree that the Baker is now housed in the finest research facility of its kind in Australia and one of the finest anywhere in the world. For this we have many people to thank, far too numerous for me to mention in this report.

President's Report

I do want to offer special thanks to all of the donors who gave so generously to the Capital Appeal which enabled the building to be completed and, in particular, to the trust which donated $33 million to enable the whole Alfred Medical Research and Education Precinct to be realised. At the same time, of course, an enormous amount of work was done by architects, planners, project managers and builders to create the Baker's new home and to all of them I express the gratitude of the Board and the Baker family. Coordination of the logistics for equipping the building and moving into it were undertaken by an internal Baker steering committee which included Michael MacKellar, Jan Strauss, Deb Ramsey, Tony Hendy, Garth Stewart, Ian Smith, Adrian O'Brien, Bryan Quinn and Annetta Conlan. For them and all the people who worked on the project, its successful completion was the culmination of many years of hard work.

It is wonderful to have state-of-the art facilities in which our scientists can pursue their vital work. But it is also essential that we provide the capital and recurrent funding necessary to ensure that the science can continue productively day to day and year to year. In this regard, the Federal Government's announcement, following the adoption of the Wills Review recommendations that it will substantially increase the resources available to medical research in Australia over the next five years has been most gratefully received. We are also hopeful that the Victorian Government will soon announce a substantial increase in the rate at which it supports the infrastructure necessary for the conduct of medical research in Victoria. In recent years the rate of this funding has fallen well behind our counterparts elsewhere in Australia. At a recent meeting of key Victorian Cabinet Ministers (including the Premier) and the Chairmen of the four largest medical research institutes in Victoria, we emphasised the unique role which Melbourne's elite medical research institutes play in developing the research and educational infrastructure of Victoria, which makes Melbourne a leading centre for such research in the world. Because knowledge is the most valuable resource of the twenty-first century, an investment in this sector of our economy is both sound and forward thinking.

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Recent changes to the basis of research funding through the National Health and Medical Research Council (including the gradual abolition of block funding for major institutes) mean that all medical researchers in Australia compete for every dollar of private and public funding which is available to pursue their work. This makes all the more important the efforts of those within and outside the Institute who assist us to raise funds. Special thanks are due to Bill Gurry, who has ably led the Baker Foundation and to Bobbie Renard and her wonderful team of Community Relations staff, who work so hard to keep the Baker's name known, recognised and respected in the wider community. We are also grateful to all of our supporters, large and small, who give so generously to assist the work of the Baker every year. Without you, much valuable work simply would not be done. All of your contributions are most gratefully received and carefully spent in the interests of the health of our whole community.

Bill Gurry recently announced his intention to retire from the Board and as inaugural Chair of the Baker Foundation. We will miss greatly Bill's wise counsel and sterling efforts on behalf of the Baker but wish him well in his near retirement (viticulture and the boards of a few public companies and similar activities will ensure that he is never entirely resting!). We also recently farewelled Peter Hughes, the Baker's first Chief Operating Officer who worked indefatigably during his year in office. Erica Hughes has recently joined the Baker as Chief Operating Officer and we are looking forward to Erica overseeing the operational functions of the Institute.

Our scientists were also very productive and energetic, despite all the other distractions around them in 2001. A substantial number of novel and important papers were published. These achievements are reported in greater depth elsewhere in this report. Exemplifying the wide-ranging talents of our scientists, Wally Thomas was created a Williamson Fellow under the Williamson Leadership Victoria program. Andrew Taylor won a Young Investigator Award from the European Society of Cardiology for his work on thrombosis. Sal Pepe won the Medal of Merit from the Institute of Cardiovascular Sciences in Winnipeg, Canada and Paul Nestel received CSIRO's first medal for Business Excellence for assisting in the establishment of Gropep Pty. Ltd., the first biotech spin off company from CSIRO. Our scientists contribute to our community in many and varied ways and we are justly proud of their efforts, both on and off the field.

Finally may I express particular thanks to my fellow Board members and to all those who have served on the many support Committees and in other ways, large and small, for the Institute during 2001. Long may you continue in your efforts on behalf of this great organisation.

Norman O'Bryan
PRESIDENT

16 April 2002

Director's Report

Director's Report

This new millennium year saw the Baker take on a new "front stalls" location in a new building with a new Director. This occurred in the midst of new funding processes initiated by the National Health and Medical Research Council (NHMRC). In case this was not interesting and exciting enough we embraced new technology, new research groups and a different organisational structure. We developed a new strategic plan leading to key staff appointments and by the end of the year were in the advanced stages of contemplation of a new name and logo. We have developed new relationships with academic and commercial partners in our academic precinct. It brings great credit to the resilience, creativity and resourcefulness of our staff that the Institute has come through this period with great optimism and conviction. The Institute is well positioned to make more than our fair contribution to knowledge, health and wealth of the community.

The retirement of our Director, John Funder, was perhaps the most dramatic of these events. Typically it was at a time of his choosing but the new building is a testament to his contribution to the Baker, to Australian science, and a very fine gift to assist the Institute in its new life. He left a platform from which the Institute will prosper and his personal support and guidance have made the accession as smooth as it could have been. It has been my privilege to work at the Institute under two predecessors, John Funder and Paul Korner. Although very different people, I learnt something from each of them every day and their support during this and many other transitions in the past has been paramount.

The stunning new Baker Institute is architecturally innovative thanks to Berry King and the team at Design Inc Melbourne Pty Ltd. They sought to show that medical researchers could and should work in an environment that is as inspiring and functional as anyone else's in the community. The design has already proven itself as a flexible and sympathetic layout for the conduct of science. It will undoubtedly be copied around the world, not just for its physical attributes but also the new model that it provides of the interface between scientific research and development. As well as our own scientists, we have those from a number of biotechnology companies bringing complementary skills, expertise, knowledge and networks to those of the Institute. The rent they pay is another attractive feature and will impact significantly on the financial pages of future annual reports.

Relocating has been a massive exercise led initially by Michael MacKellar and Jan Strauss but ultimately involving everyone. It is impossible to do justice to the contributions of our donors, supporters, staff and suppliers who brought this ambitious scheme to fruition but we are grateful to them all and undertake that the future work of the Institute will meet the high standards of the magnificent facility in which it is performed.

Our new strategic vision confirms the commitment of the Baker since the mid 1970s to focus on reducing death and disability from heart and vascular disease such as stroke. This is the biggest health problem in our community and around the world. It will become even more important in future as the average age of all communities increases and we face an epidemic of cardiovascular disease in certain disadvantaged groups in Australia and in developing countries. In this area the Baker has the greatest research critical mass in the country and an international reputation to enhance and maintain. To build upon this proud record of innovation and research in heart disease, we have been engaged in a major exercise taking into account the major health burdens in the community and our existing strengths and competitive advantages.

We plan to target specifically the two ends of the cardiovascular spectrum. On the one hand prevention of heart disease is our objective in consolidating the groups in the Institute that work on vascular biology, thrombosis, metabolism and risk factors. Diabetes has become particularly important in the community as a pre-disposing factor for cardiovascular disease. In the latter part of the year we therefore invited Professor Mark Cooper from the University of Melbourne, a highly regarded researcher and physician internationally in diabetes research to move his group to the Institute. The arrival of Mark's group and consolidation of our existing laboratories will round off a large, competitive team addressing one of the most important health problems in the world.

Director's Report

The other major target is one in which we have existing strengths, heart failure. The projections are that as heart disease is a disease of older people and we have an aging popultion heart failure will become an even more important health problem than it is now. The increase will have health expenditure implications that demand urgent breakthroughs in understanding and treatment. We have special expertise in the Institute and also the advantage of participation in the elite heart failure and cardiac transplant services of The Alfred. The latter provides access to both cutting edge therapies and heart failure patients and tissues. Laboratories working in molecular cardiology, physiology, as well as clinical heart failure research and cardiac surgery will work together to become a leading group making fundamental and practical contributions to control heart failure.

The impetus towards larger groupings in the Institute stems from many origins. Diabetes and heart failure are demanding problems and they need large groups to make significant impact. The Wills Report to the Federal Government and subsequent new NHMRC funding arrangements encourage critical mass. Biological problems have reached a level of sophistication that requires input from all sorts of disciplines within and outside the general biological sciences. The Baker's traditional strength in clinical research does encourage some channelling of science to particular ends. However our overriding philosophy is to seek scientific excellence, find the best people and give them enough resources to do important work. The groupings I have described could apply equally well to 'heart and arteries' or as 'prevention and cure'. They will not in any way constrain on the ideas and creativity of the scientists working within them. Each group has both basic and clinical research strengths to ensure that the capacity to pursue original findings into the clinical research arena is maintained. The Alfred and Baker Medical Unit, the clinical arm of the Institute is represented equally in the two divisions.

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As in any dynamic and progressive organisation there are always comings and goings. By the end of the year Jun-Ping Liu, Tim Cole, Craig Neylon and their laboratories had moved on to positions at either the University of Melbourne or Monash University. Michael Berndt, Rob Andrews and their group will go to the Department of Biochemistry at Monash University in 2002. Our Chief Operating Officer, Peter Hughes, who achieved major advances in the business functions of the Institute leaves in January 2002. We are grateful to them all and wish them every success.

There has been lots of good news in the midst of this tumultuous year. Technology moves very quickly in our business and we were most fortunate in our successful application for the Ramaciotti Foundation major grant which allowed us to introduce the world's best high-throughput, robotically-driven proteomics facility.

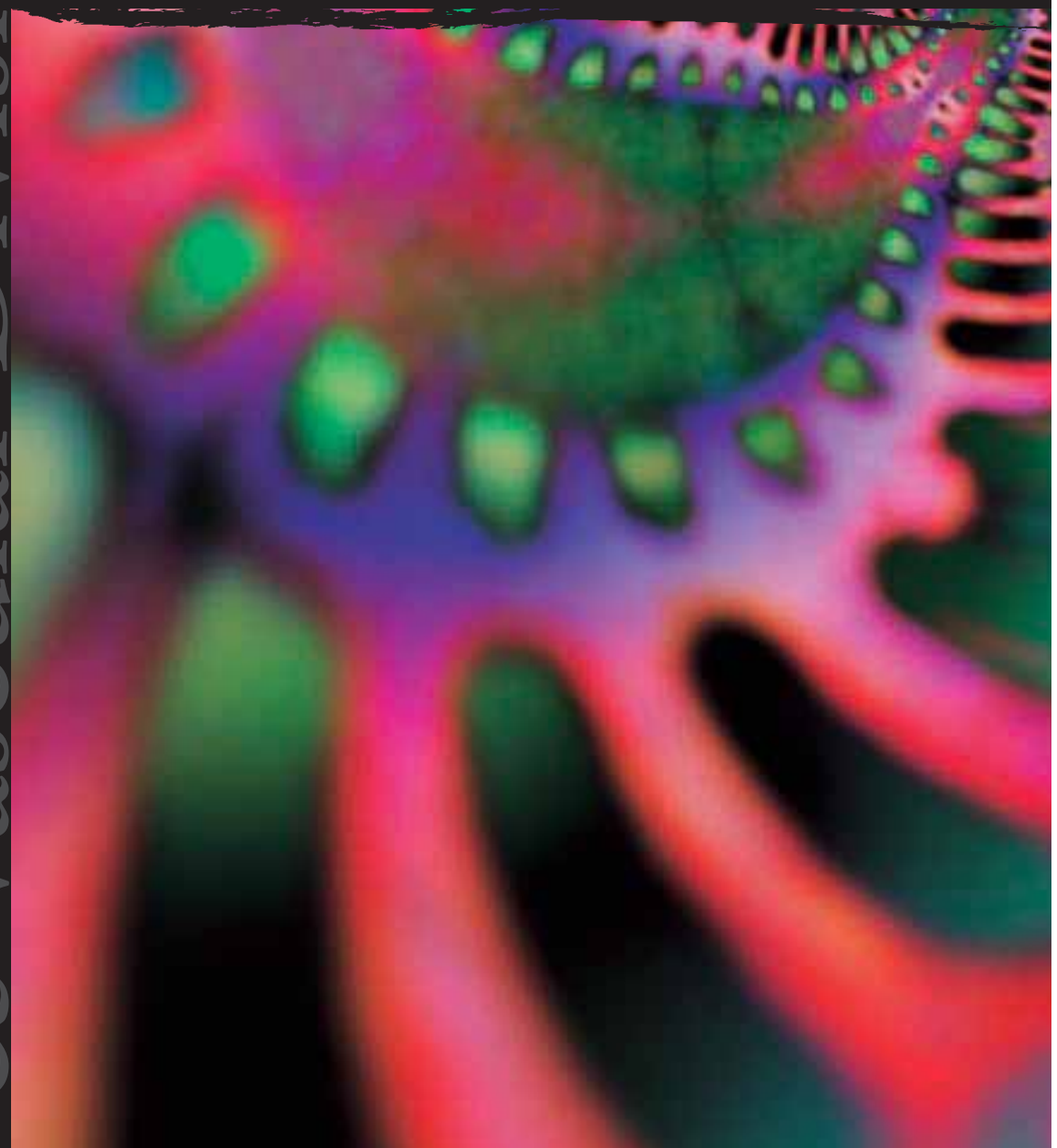
While we tend to blame globalisation for the increasing cost of scientific consumables and equipment, much of which must be sourced overseas, globalisation has also worked in our favour with a substantial amount of the capital for our new building being derived from US funds. At the end of the year we were in advanced stages of negotiation with Professor Victor Wynn of the Atherosclerosis Research Trust of the UK to establish a metabolic cardiology department at the Institute.

Michael Berndt and his group were part of a successful program application in the first round of the revised NHMRC system and many other groups in the Institute were successful in a wide range of other grants. This year we have decided to give some detail of the research achievements of the Institute in a separate research report. Some of the highlights are listed in the President's report. For those not steeped in the technicalities of medical research this report shows a year with many good publications in high impact scientific journals, collaborations and support from around the world and plenty of promise for the future. I am most grateful to all those who have contributed.

Professor Gary Jennings
DIRECTOR

... the ABMU is the centre for clinical research at the Baker. A successful NHMRC Centre of Clinical Excellence in Hospital-based Research Grant to the ABMU has enabled translation of our findings to clinical practice...

Coronary Disease & Vascular Division



Coronary Disease and Vascular Division

Thrombosis Group
Hazel and Pip Appel Vascular Biology

Our research looks at the role of platelets in the formation of blood clots (thrombi) in arteries. Occlusive thrombi can result when platelets adhere after atherosclerotic plaque rupture or are activated by high shear stresses at sites of arterial stenosis. In either of these situations, the events are mediated by the platelet adhesion receptor – the GPIb-IX-V – complex, which binds von Willebrand factor (vWF).
Using protein engineering, we have gained detailed information of the regions and individual amino acid residues of GP1b involved in binding to vWF. We have also found that separate regions of GPIb-IX-V associate with cytosolic calmodulin, a novel interaction that may regulate aspects of GPIb-IX-V-dependent platelet activation.

In the inflammatory response, neutrophil rolling before adhesion and transmigration through the blood vessel wall is mediated by P-selectin expressed on activated endothelium and its counter-receptor on neutrophils, P-selectin glycoprotein ligand-1 (PSGL-1). We found that binding of P-selectin to neutrophils is lost when proteinases are released from neutrophil primary granules, such as by treating neutrophils with cathepsin G and elastase. This inactivation corresponded to loss of the N-terminal domain of PSGL-1.

Vascular, Lipoprotein & Metabolism Group
Cell Biology

Our research interests are directed toward understanding the biology of the cells in blood vessel walls and the heart, and the changes occurring in disease states. We studied the regulation of an enzyme system which produces damaging reactive oxygen species and found that cells from atherosclerotic lesions had greater activity of the enzyme than healthy tissue.

We have developed a rat model of left ventricular hypertrophy (LVH), a major risk factor for congestive heart failure in people with high blood pressure. The model is being used for gene therapy studies, initially to examine the effectiveness of inhibiting transforming growth factor-beta signalling in controlling fibrosis in hypertensive animals.

Rupture of atherosclerotic lesions is a pivotal event in unstable coronary syndromes. It results in localised thrombi and marked falls in blood flow beyond the rupture. It had been thought that material from the lesion caused physical obstruction of blood vessels to cause this decreased flow. However when we examined the changes after plaque rupture in a rabbit model that resembled the human disease situation, we found that extensive constriction of small vessels beyond the rupture was the cause. Therapy to prevent constriction of these small blood vessels is likely to improve reperfusion in acute coronary syndromes.

Clinical Physiology

We are investigating how large artery stiffness increases cardiovascular risk with a view to improving cardiovascular risk prediction, confirming that large artery stiffness is both a causative and a reversible risk factor and identifying therapeutic targets to lessen large artery stiffness.
Age, atherosclerosis and gender are important influences on large artery stiffness, ultimately via matrix composition as determined by matrix metalloproteinases (MMPs). We have shown that a common genetic variation in the matrix glycoprotein fibrillin-1, affected in Marfan syndrome, is associated with stiffer large

arteries. Heterozygosity for a common promoter polymorphism in another MMP, stromelysin-1, is associated with a reduction in age-related large artery stiffening.

In women, age-related increases in large artery stiffness were shown to be linked to hormonal changes and the menopause while in patients with isolated systolic hypertension, cholesterol lowering led to reduced arterial stiffness.

In studies of the vascular complications of type 2 diabetes, we have shown for the first time that nitric oxide (NO) is involved in contraction-mediated glucose uptake and may account for 78% of glucose uptake during exercise in patients with type 2 diabetes compared with 34% in healthy individuals. The contraction-mediated glucose uptake pathway at rest may therefore be stimulated by the provision of NO donors.

Vascular Pharmacology

A defective L-arginine - nitric oxide pathway may contribute to the impaired endothelium-dependent vasodilation seen in conditions such as congestive heart failure. We showed that angiotensin II (Ang II), a potent vasoconstrictor and growth hormone, stimulated the transport of L-arginine into human vascular endothelial cells, apparently after initial conversion to the degradation product, Ang IV.
Phytoestrogen metabolites were also shown to influence the nitric oxide system, in a macrophage cell line. Lipopolysaccharide/gamma interferon-activated, cytokine-induced nitric oxide overproduction in these cells was inhibited by genistein and other phytoestrogen metabolites, apparently through selective inhibition of iNOS protein expression.

Reduced effectiveness of the L-arginine -nitric oxide system has been implicated in cortisol-induced hypertension. A study in healthy males with normal blood pressure indicated that it was unlikely that abnormalities in the transport of L-arginine are associated with this action of cortisol.
Another area under investigation is the general vasodilation seen in patients with advanced cirrhosis of the liver. Tests of vascular responsiveness to the vasoactive peptide endothelin-1 showed increased forearm blood flow in patients, but decreased flow in healthy control subjects. After liver transplantation, the response to endothelin-1 was restored to normal.

H and L Hecht - Hormones and the Vasculature

Our research centres on defining the role of sex hormones on vascular function. Abnormal growth and death of vascular smooth muscle (VSM) cells contribute to the development of atherosclerosis. Using VSM from an estrogen-deficient mouse, we found that the addition of estrogen to the cell growth medium led to increased growth responses and decreased cell death.

Little is known about the cardiovascular actions of progesterone, despite the potential implications for cardiovascular health in pre- and post- menopausal women. We therefore studied the effects of 100 mg per day of micronised progesterone on cardiovascular risk factors and vascular function in healthy postmenopausal women not taking estrogens. Our results showed that the treatment had no cardiovascular effects.
Males produce low levels of estrogen from male sex hormone precursors, however the role of estrogen in men is unknown. We examined the effects on endothelial function and lipid levels in healthy young men of preventing estrogen production and showed that decreasing the estrogen level interfered with normal endothelial function.

In a study of the effects of DHEA (100 mg per day) on cognition and well-being – including anxiety and depression – in healthy post-menopausal women not taking estrogen, we found that DHEA is beneficial for memory and certain cognitive tasks.

Morphology

Our research examines how the growth of cardiovascular tissues is regulated under normal conditions and in disease states. In studying the thickening of heart and vessel walls in response to high blood pressure, we have found that a fibrinolytic system that removes blood clots and fibrous tissue components is activated in the heart and aorta of hypertensive mice.

Heparin is known to reduce proliferation and migration of smooth muscle cells – the cells from blood vessel walls that contribute most to artery disease. Heparin's high anticoagulant activity and large size make it an unsuitable candidate for chronic drug treatment, so we have tested a range of smaller molecules derived from heparin in a cell culture system and found them to have similar anti-growth activity to heparin without the anticoagulant side effects.

The arteries supplying the intestines develop thicker walls in rats with diabetes than in healthy animals, partly through increased activity of the system that regulates cell acidity. We tested whether these changes also occurred in other arteries, but found no difference in the acidity and growth of cells in the basilar arteries that supply the brain and are similar in size to mesenteric arteries.

Vascular Diabetes

The laboratory's research is centred on the biochemistry of proteoglycans (PGs) and their contribution to vascular disease, particularly as a complication of diabetes. Proteoglycans are large, negatively-charged molecules located within the walls of blood vessels where they attract and bind a variety of molecules such as low density lipoprotein (LDL), as well as controlling the water content.

We hypothesised that longer sugar chains attached to PGs cause stronger binding of LDL and thus increase the atherogenicity of the PGs. We have shown that the atherogenic growth factor, transforming growth factor-beta, elongates sugar chains on PGs and greatly increases the binding of LDL and also that fibrates such as gemfibrozil, used to treat high triglyceride levels, shorten the sugar chains.

A major finding has been the discovery in vascular smooth muscle cells of a biochemical pathway that has a marked influence on the properties of PGs.

People with diabetes accumulate advanced glycation end-products, or AGEs, in their blood due to the interaction of glucose at elevated levels with a variety of proteins. AGEs are associated with accelerated development of atherosclerosis. We tested a variety of AGEs on human vascular smooth muscle cells and found that they had no effect on any cellular responses.

Lipoprotein and Atherosclerosis

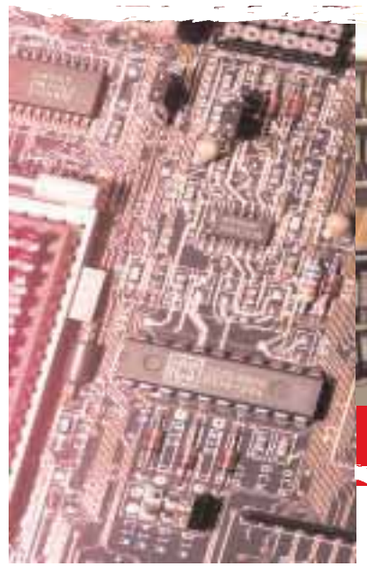
Continuing themes in our research have been the role of protein apoA-1 in determining the functions of high density lipoproteins (HDL) and the role of HDL in the process of cholesterol transport. We made a number of mutated forms of apoA-1 and found that regions covering amino acids 63 to 73 and 140 to 150 were important for cholesterol efflux from cells. We also studied the functional changes to apoA-1 of five natural, single amino acid mutations and found that three of them interfered with the activation of a crucial step in cholesterol transport.

We examined the role of the pre-beta1 form of HDL in cholesterol efflux. Pre-beta1-HDL-deficient plasma was as effective as normal plasma in promoting cholesterol efflux from human skin fibroblasts and THP-1 human macrophage cells. At equal concentrations of apoA-I, pre-beta1-HDL was less effective than whole plasma in promoting cholesterol efflux from fibroblasts but equally effective with THP-1 cells. Pre-beta1-HDL-deficient plasma supplemented with 16% pre-beta1-HDL was more active than whole plasma. Although not essential for cholesterol efflux, pre-beta1-HDL may be the first product of apoA-I lipidation during formation of HDL.

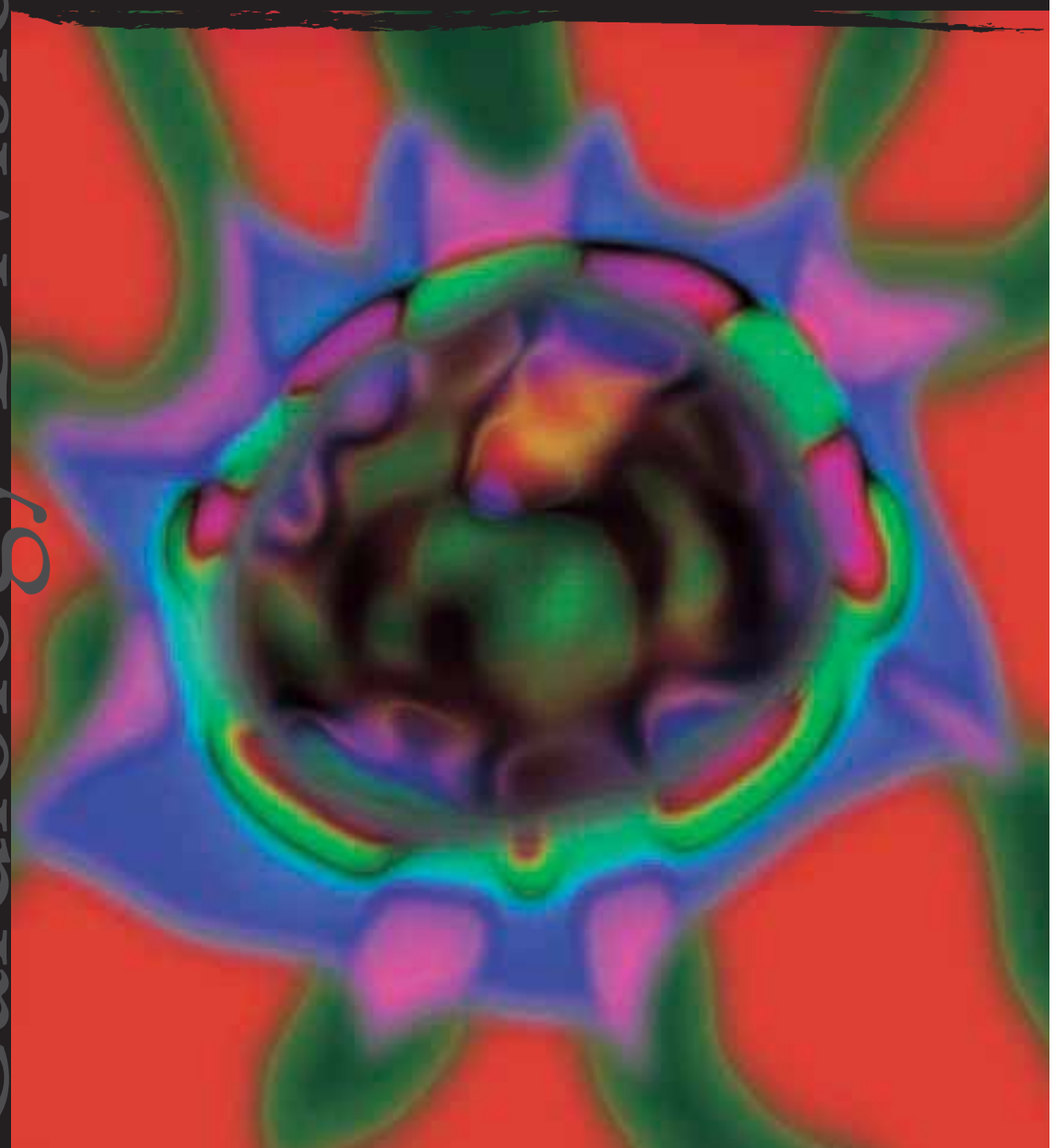
Cardiovascular Nutrition

The main objective of our research is to identify dietary components likely to prevent heart disease. We have tested the isoflavones biochanin B (genistein) and formononetin F (daidzein) for their effect on LDL levels and arterial function in a randomised, double-blind, placebo-controlled trial of 80 men and women. Only biochanin lowered plasma low density lipoprotein cholesterol significantly, and then, only in men. Importantly, pure isoflavones at a dose of 40 mg daily significantly improved central pulse wave velocity (a measure of arterial elasticity) and flow mediated dilatation.

In a second randomised, blinded, placebo-controlled, cross-over trial, we tested the absorption and excretion of several isoflavones, either conjugated as glycosides or as aglycones which are produced by the action of bowel microorganisms on the natural, conjugated isoflavones.We found similar absorption and excretion, irrespective of the nature or the conjugation status of several isoflavones.



Heart Failure & Molecular Cardiology Division



Heart Failure & Molecular Cardiology Division

Molecular Cardiology & Signalling Group
Cellular Biochemistry

Our research explores the functional importance of inositol phosphate signalling pathways in heart muscle, with a view to understanding how cardiac arrhythmias arise and how overgrowth (hypertrophy) of the heart, is initiated and progresses to heart failure.
We have identified in neonatal cardiomyocytes the different isoforms of phospholipase C (PLC) that mediate inositol phosphate responses to alpha1-adrenergic receptors, P2Y2-purinergic receptors and raised Ca^{2+}.
PLC beta1 selectively mediates responses to alpha1-receptor stimulation, while PLC beta3 mediates those to purinergic receptors. Calcium responses were mediated by both PLC beta1 and beta3.
To determine whether activation of Gq-coupled P2Y2-receptors stimulated hypertrophic signaling pathways, we measured the activities of the atrial natriuretic peptide and myosin light chain promoters in cardiomyocytes.The P2Y2 agonist, ATP, inhibited the activation of both promoters by other receptors, suggesting that ATP had multiple effects on growth signaling. With UTP as an alternative agonist at P2Y2-receptors, MLC activity was stimulated, and the size of cardiomyocytes was increased.
In related studies, we found that inositol polyphosphate 1'phosphatase (INPP) inhibited hypertrophic signalling without causing general damage or cell death, suggesting that the INPP substrate, inositol(1,4) bisphosphate, stimulates hypertrophic signalling.

Gene Transcription

The focus of our research is to understand how transcription of the mammalian ribosomal genes (rDNA) by RNA polymerase I is regulated. Transcription of rDNA is the rate-limiting step in the synthesis of functional ribosomes and therefore a likely influence on cellular growth.
We have begun to identify the regions of transcription factor UBF that account for its increased activity in promoting ribosome synthesis during the growth of cardiac cells. To test whether inhibition of UBF gene activity can lead to regression or prevention of hypertrophic heart disease, we are using gene therapy in a mouse model.
We have also shown that a protein called S6K, known to be involved in the regulation of growth, regulates rDNA transcription and are currently applying proteomic and biochemical approaches to identify its target in the nucleus of heart cells.
The initiation of left ventricular hypertrophy in essential hypertension, aortic stenosis and myocardial infarction correlates with altered expression of a receptor for the vasoactive peptide, angiotensin II. We have demonstrated that AngII promotes cardiac myocyte growth by a mechanism involving transactivation of the EGF receptor.

Molecular Physiology

Our research focuses on how the steroid hormones, mineralocorticoids and glucocorticoids, influence the physiology of the heart. We have shown that differential expression of glucocorticoid receptors and mineralocorticoid receptors occurs in cardiac myocytes and fibroblasts, with myocytes, but not fibroblasts, expressing the mineralocorticoid receptor. Only isoform 1 of 11beta-hydroxysteroid dehydrogenase (HSD) was present and its activity converted 11-dehydrocorticosterone into its transcriptionally active form in both types of cells. These data predict mineralocorticoid-induced fibrosis is unlikely to be a direct effect of mineralocorticoids on fibroblasts.
A second study was commenced to determine whether Serum- and Glucocorticoid- induced Kinase (SGK) had a role in cardiac physiology, and also to explore the interaction between corticosteroid signalling and protein kinase signalling and the potential impact on cardiac hypertrophy and fibrosis of stimulating these pathways.

Glucocorticoids, mineralocorticoids and the inactive glucocorticoid metabolite, 11-dehydro-corticosterone, caused transcriptional induction of SGK in cardiac myocytes and fibroblasts. Some agents which caused cardiomyocyte hypertrophy were also found to potentiate the induction by corticosteroid of SGK gene transcription, suggesting cross-talk between corticosteroid signalling and kinase signalling pathways.

Peptide Biology

The major aim of our research program is to gain a better understanding of the role played by vasoactive peptides in the regulation of cardiovascular function. Our research is directed toward the endopeptidases that generate and metabolise peptide signals, particularly EC 3.4.24.15 (EP24.15) and EC 3.4.25.16 (EP24.16), with a view to designing and characterising specific peptidase inhibitors as potential therapeutic agents.
We have shown that EP24.15 and EP24.16 are expressed in aortic endothelial cells in culture, and may participate in bradykinin metabolism in the circulation. By chemically modifying an existing inhibitor of EP24.15, cFP, we produced a new inhibitor, JA-2, with equal potency, but greater biological stability.
JA-2 represents a valuable tool for assessing the biological functions of this endopeptidase.
In conscious rabbits, JA-2 increased the depressor effects of bradykinin on mean arterial pressure, but had no effect on the hypertensive effects of angiotensins I and II. In a rat model, JA-2 also reduced the bradykinin-induced increase in the permeability of the blood brain barrier.
In seeking inhibitors of endopeptidase EP24.15, we synthesised a series of bradykinin analogues containing beta-amino acids – instead of the usual alpha-amino acids – and showed that a beta amino acid near the normal cleavage site prevented degradation of bradykinin by EP24.15.

Emily Stewart - Molecular Endocrinology

Our research examines the hormonal control of blood pressure, including regulation of the receptors for the vasoconstrictor, angiotensin II (AngII). In studying the interaction of arrestins with the angiotensin receptor, AT1, we have shown an association which was driven by AngII and dependent on specific phosphorylation in the carboxyl-terminal region of the receptor.
By comparing the properties of wild type and mutated AT1 receptors in response to angiotensin and substituted analogues, we had gained evidence for multiple states of functional AT1 receptors. We have now shown that separate contacts between the AngII peptide and the AT1A receptor induced distinct receptor conformations that preferentially affect receptor outcomes.
AngiotensinII may cause cardiac hypertrophy via the AT1 receptor on cardiomyocytes and through stimulating the release of growth factors from cardiac fibroblasts, however it is a difficult question to study because the cultured neonatal cardiomyocytes that comprise the current in vitro model express low levels of AT1 receptor.
We studied the effect of AngII in a modified in vitro model, that expressed normal levels of AT1A receptors. The use of this system showed that AngII directly promoted cardiac myocyte growth via AT1A receptors and that EGF receptor transactivated MAPK signalling was important in this process.

Myocardium & Heart Failure Group

Cardiac Surgical Research

Coenzyme Q10 (CoQ10) is essential for antioxidant function and mitochondrial energy production. We had previously shown that CoQ10 treatment of patients awaiting coronary artery bypass surgery produced major myocardial benefits. We have now completed a follow up which indicated that physical well-being scores based on a questionnaire were significantly better for CoQ10-treated patients, even 12 months after cardiac surgery.
We designed a study to evaluate the effects of ischemia and reperfusion on the turnover of heart proenkephalin and its main products, particularly during the cardioprotection produced by ischemic preconditioning. The study suggested that post-ischemic reperfusion caused a loss of enkephalins but that levels are preserved by ischemic preconditioning, in association with less cellular damage and better contractile performance.
Opioid peptides are protective in animal models of cardiac reperfusion injury. Using trabeculae isolated from right atria of humans undergoing coronary bypass surgery we showed that stimulation of the delta-opioid peptide receptor protects against post-hypoxic contractile dysfunction and reduces O_2 demand, suggesting that such stimulation increases energy efficiency. Also in human trabeculae, we showed that the Na^+/H^+ exchange inhibitor, HOE-642, which prevents post-ischemic Ca^{2+} overload in animal models, significantly improved post-ischemic recovery of developed force.

Experimental Cardiology

We have recently demonstrated that in transgenic mice that over-express the beta2 adrenergic receptor, females have considerably less functional and pathological abnormalities compared with males.
We have used a mouse model that lacks the heart specific intracellular calcium-binding protein, S100A, and found it to be important in the maintenance of cardiac function under conditions of acute and chronic stress.
We have observed that the hormone relaxin is expressed in the heart, and that male mice that lack this hormone develop cardiac diastolic dysfunction due to increased connective tissue between the cells. Further studies are in progress to examine the role of relaxin in cardiac diseases in relation to its activities on cardiomyocytes and on the extracellular matrix.
Cardiac rupture accounts for approximately 12% of in-hospital deaths in patients with acute myocardial infarction (AMI), but it is a poorly-researched phenomenon. Until recently, there has been no animal model of rupture available for research use. Our recent findings show that cardiac rupture occurs in mice with AMI, and that in many respects, it mimics human cardiac rupture.

Human Neurotransmitter

The broad area of our research is cardiovascular neuroscience, with projects on the neural aspects of high blood pressure, psychosomatic heart disease, heart failure and obesity and heart disease.
The cause of essential hypertension is often abnormal sympathetic nerve function. We are comparing sympathetic nerve density in vein biopsies from essential hypertensive patients and healthy volunteers and also studying levels of nerve growth factor. We have commenced a study to examine muscle sympathetic nerve activity and endothelium function in these two groups.
We have found that increasing the pressure in the thorax by a method widely used for obstructive sleep apnoea lowers the activity of sympathetic nerves to the heart in patients with heart failure. Disability in human heart failure is in part due to skeletal muscle dysfunction, with loss of tissue mass. We are currently studying aspects of this weight loss in the sheep paced heart failure model.
Panic disorder is now known to increase the risk of myocardial infarction and sudden death by 3- to 6- fold. People with depression are also at a greater risk of a heart attack.We have a number of studies under way to understand the neurochemical mechanisms underlying panic disorder and depression and how they relate to changes in cardiovascular health.

Molecular Hypertension

Our primary research interest is the role of steroid dehydrogenases in hypertension and heart disease.We identified in the heart a novel isoform of 17beta-hydroxysteroid dehydrogenase, 17betaHSDXI, that has conserved domains of the short chain alcohol dehydrogenase superfamily. It was also present in pancreas, kidney, liver, lung, adrenal gland and ovary. Results of studies on the regulation of 17betaHSDXI activity suggest a role in androgen metabolism during steroidogenesis.
To study further the role of 17betaHSDXI in the heart, we constructed a virus-based system for transferring the gene into heart cells grown in the laboratory in order to produce high levels of the enzyme in these cells.
We are also interested in how cholesterol metabolism affects its efflux from cells, which is thought to be the rate-limiting step of reverse cholesterol transport which removes excess cholesterol from tissues other than liver. Cholesterol efflux occurs by transfer of free cholesterol to lipid-free or -poor apolipoprotein A-I or by the transfer of membrane-associated cholesterol to lipidated apoA-I or mature high-density lipoprotein (HDL).
We found that the enzyme sterol 27-hydroxylase, which modifies cholesterol to make it more water-soluble, stimulated cholesterol efflux to both free apoA-1 and to HDL-containing human plasma.

Jo Guiliano - Molecular Neurocardiology

The research in this laboratory centres on the neurobiology of the sympathetic nervous system in the context of the failing heart and the role of the L-arginine/nitric oxide system. We have shown that high levels of catecholamine release are associated with poor outcome in heart failure patients and are now researching the cellular and molecular basis for these alterations in sympathetic nerve activity. This project has employed a broad suite of cellular and molecular techniques as well as studies in patients.
A detailed study of the secretion of NGF by cardiomyocytes will include exploration of whether NGF plays a therapeutic role in heart failure. These studies will examine the local and systemic delivery of NGF as well as the structure and function of the heart in transgenic mice that express high levels of NGF in the heart.
At the basic level, studies are aimed at determining how neurohormones and cytokines modulate L-arginine transport, and at assessing how cells regulate the location of the L-arginine transporter.

Neuropharmacology

We study how the central nervous system controls the heart and circulation. Our interests include the mechanism of action of centrally acting antihypertensive agents and the cardiovascular actions of neurotransmitters – noradrenaline, serotonin and dopamine – and the renin-angiotensin system.
In a study of men and women to determine the rate of change in blood pressure and heart rate in the transition from sleep to activity and vice versa, we found that the pattern of higher day values and lower night values was similar for males and females. The morning increase in heart rate was more rapid in women than men.
We have found that the surge in blood pressure during the arousal period in hypertensive rats could be markedly reduced by the centrally acting antihypertensive drug, rilmenidine, but was not affected by an ACE inhibitor. Rilmenidine treatment of renal hypertensive rabbits normalised blood pressure and cardiovascular hypertrophy, restored baroreflex sensitivity and reduced blood pressure variability.
By injecting agents into specific sites of the brain in conscious rabbits (to avoid interference from anaesthetics), we have found that central angiotensin II receptors mediate components of the response to environmental stress by the sympathetic nervous system.

Associated laboratories and clinics

Cardiovascular Disease Prevention

The CVD Prevention Unit is coordinating several national and international clinical trials, some of which are described. ANBP2 is a large clinical outcome trial comparing two types of treatment for high blood pressure: ACE inhibition versus diuretic-based regimens. More than 6,000 subjects have been monitored by more than 2,000 GPs over five years for heart attacks, strokes and other non-fatal outcomes.
We have been appointed by the Victorian Division of the Australian Society of Cardio-thoracic Surgeons as the centre for Data Management and Analysis for a project to identify key performance indicators for cardiac surgical outcomes. We have developed a standard database for implementation at each public hospital in Victoria.
We are the national coordinating centre for the ONTARGET trial of an angiotensin II antagonist, telmisartan, versus an angiotensin converting enzyme inhibitor, ramipril, versus a combination of these two. The study aims to recruit 4,000 Australian patients, of a total of 28,000 world-wide, and follow their cardiovascular outcomes over five years.
In a systematic review of studies that have examined the effects of left ventricular hypertrophy (LVH) on cardiovascular outcomes, we have found that LVH is associated with a two-fold increase in risk for all cause mortality and a 2.3-fold increase in risk for non-fatal and fatal cardiovascular events.

Risk Reduction Clinic

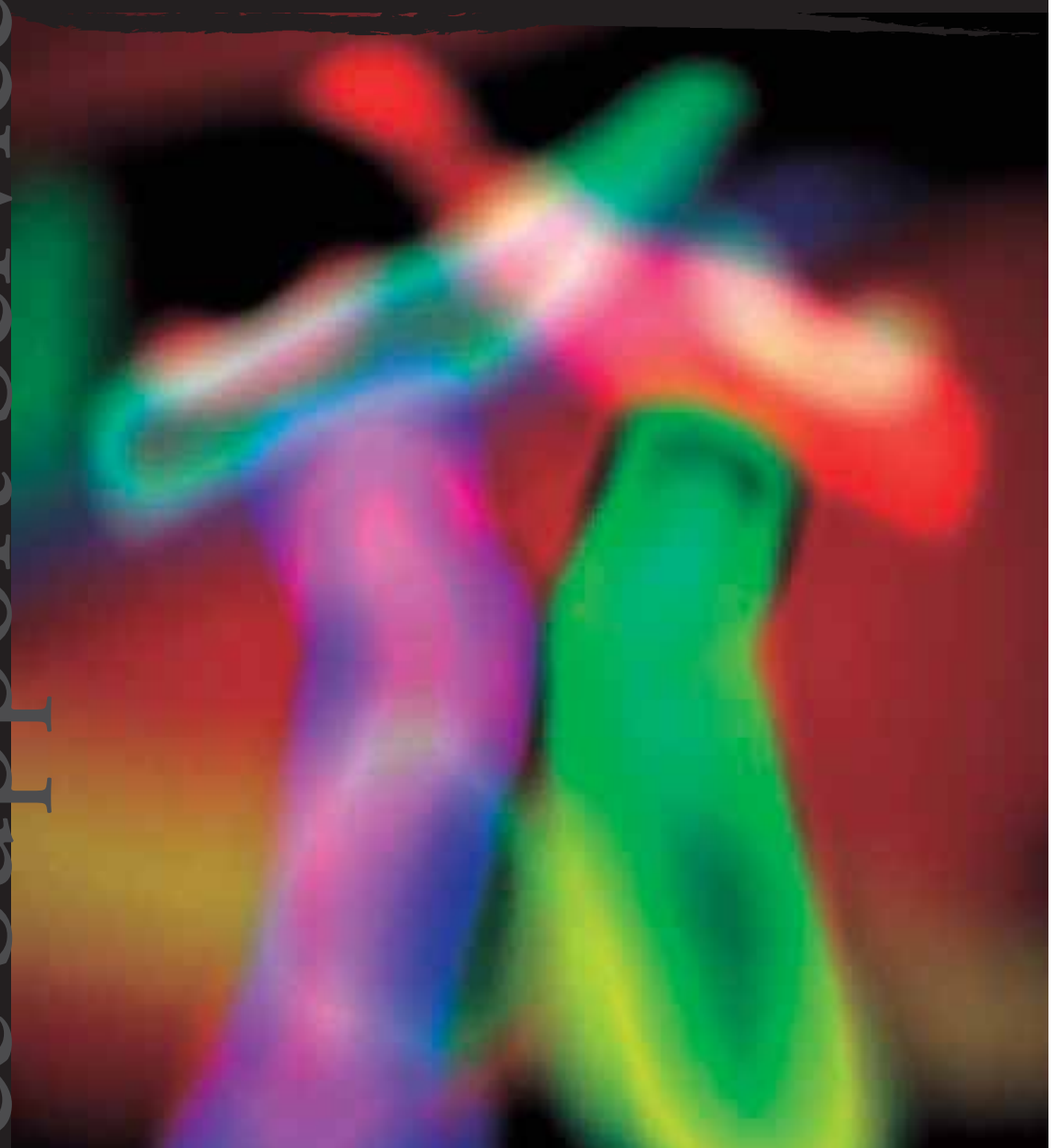
The Risk Reduction Clinic performs free screening to members of the community for risk factors related to diseases of the heart and circulation. The approach to screening is to apply simple and cost-effective tests, linked to lifestyle, that are of proven usefulness. We measure cholesterol and triglycerides and obtain information from a lifestyle questionnaire. Where necessary, the initial contact with the Risk Clinic may be followed up by medical intervention.
A close link exists between the Risk Clinic and the ABMU research interests in prevention of cardiovascular disease, nutrition and exercise. Staff at the Risk Clinic are involved in acquiring samples for the Gene Bank and a broad range of research studies in addition to their critical role of recruiting subjects for ABMU studies.
The clinic provides a base for the Menopause Clinic and also for nutrition studies performed by the Cardiovascular Nutrition Laboratory.

Alfred Baker Medical Unit

The ABMU is the centre for clinical research at the Baker. A successful NHMRC Centre of Clinical Excellence in Hospital-based Research Grant to the ABMU has enabled translation of our findings to clinical practice.
Specific research findings are reported elsewhere in individual laboratory reports from Cardiovascular Nutrition, Cell Biology, Clinical Physiology, Experimental Cardiology, Human Neurotransmitter Research, Molecular Neurocardiology and Vascular Pharmacology.
The ABMU provides specific research platforms for these activities, such as clinical laboratories for invasive and non-invasive testing, clinical databases, clinical trial networks and a gene bank.

Technology Platforms & Support Services

...The Gene Bank is a valuable resource to researchers investigating the genetic basis of cardiovascular disease and drug therapy...



Alfred & Baker Gene Bank

The Alfred & Baker Gene Bank was officially launched in 2001. It aims to collect 10,000 DNA samples from volunteers attending The Alfred, the Baker Risk Clinic and the Alfred Baker Medical Unit. DNA is isolated from blood and stored indefinitely at minus 80°C. Personal and clinical information is also taken and maintained in a limited access database strictly under the control of the Human Research Ethics Committee. The Gene Bank is a valuable resource to researchers investigating the genetic basis of cardiovascular disease and drug therapy.

We are studying the association between arterial properties and homocysteine levels in a group of hypertensive subjects after dietary folate supplementation. We hypothesise that folate supplementation will decrease the risk of cardiovascular disease by improving arterial properties and reducing homocysteine levels.
We have commenced a study to determine the effect of two common polymorphisms of the beta2-adrenoceptor gene on the currently unexplained variation in beta-blocker responsiveness found in heart failure patients.
The only difference in the baseline characteristics of good and poor responders is the frequency of the position 27 genotype of the receptor gene.

Proteomics

Proteomics is the analysis of proteins. It is important to basic research for understanding aspects of cellular function. In more applied research, it can also be used to identify drug targets, to screen potential lead compounds and to identify protein markers for human conditions such as heart disease, infectious diseases, neurological disorders and various cancers.

The Baker has recently set up a new proteomics facility that represents a major commitment to advanced technology. The facility complements the Institute's existing capabilities for molecular biology and protein analysis and, coupled with the its world class standing in heart disease research, distinguishes the Baker as the only research centre in Australia – and among very few in the world – able to bring a broad-based, multidisciplinary approach to clinical research programs.

The technology behind proteomics employs an automated system to resolve individual proteins from complex mixtures. Proteins are separated, the pattern is analysed and the protein(s) of interest isolated and identified following mass spectrometry and comparison with protein and gene databases.
The Baker facility provides proteomic analysis for AMREP, the Monash Institutes of Health and others performing basic research who stand to benefit from this type of analysis.

Precinct Animal Centre

The Precinct Animal Centre (PAC) is a purpose-built facility for breeding and housing laboratory animals for use in medical research. The environmental conditions within the PAC ensure the highest standards of animal welfare and meet the varying needs of the Baker's researchers as well as those of other precinct partners. Animals rooms were designed for flexibility in terms of the species that can be accommodated. The maintenance of good health in the production animals is assisted by their physical separation from experimental animals in the PAC. The temperature and humidity throughout the facility are controlled electronically.

For researchers, the PAC offers a number of advantages. The flexible spaces can be readily adapted to projects using infectious or non-infectious animal models under various levels of biocontainment.

In parallel with building and commissioning the PAC, considerable resources have been directed toward the Web-based System for Ethics Monitoring and Control of Animals (SEMCA). This innovative system, developed at the Baker, signifies a major advancement in the process of using animals for research purposes – from the initial application by researchers to the Animal Ethics Committee to the ordering and issue of animals by PAC staff. Legislative and reporting requirements for the AEC, the National Health and Medical Research Council and the Victorian Bureau of Animal Welfare are all covered by SEMCA.

Information Services Group

The year 2001 saw many challenges for the Information Services Group.

- A complete review of all software and the implementation of the network management tool ZenWorks to monitor licensing and remote PC management.

- The development and roll-out of a new Standard Operating Environment together with the installation of a robust firewall and virus detection program have significantly improved security and reduced the number of incidences of computer viruses.

- The relocation of IT services to the new building required the disconnection and reconnection of 300 personal computers, servers and printers and the installation a new telephone system. The new telephone system is integrated with the network so that the telephone and network systems can be simultaneously updated.

- The development of a new laboratory animal management system to assist with streamlining the animal ethics process was also undertaken. This Baker specific software has, with the Baker's joint venture developers, commercial potential for utilisation by other similar facilities.

Library

The end of 2001 saw the close of one era and the beginning of a new one for the way information is supplied to aid research at the Baker. When the Institute was founded in 1926, the first volumes of a highly specialised collection began to be amassed. In 1954 a formal library was established, thanks to the support of the Rouse Family, and the collection was gradually expanded.

In the mid 1990's, some of the journals so important to Baker research started to become available through the Internet and the Baker library was quick to take advantage of this trend. Easily searchable material, at the scientist's desktop, is now the face of our library. The most important part of this change from print to electronic, was the immediacy of access to the latest work in our field in national and international publications. By 2001 the Baker Library subscribed to over 110 journal titles and took the majority of these in both print and on-line formats. The library receives more than 1000 requests per year for specific research materials (with a 99% success rate in sourcing what is required) and collates and makes available more than 200 Baker scientific publications each year..

In December 2001, the libraries of the Alfred Hospital, Monash Medical School at Alfred, and the Macfarlane Burnet Institute, merged with the Baker Institute Library to form the Ian Potter Library. With a pleasant modern environment in which to study, Baker staff will benefit in many ways from this new library and the specialist medical resources it brings together.

Imaging

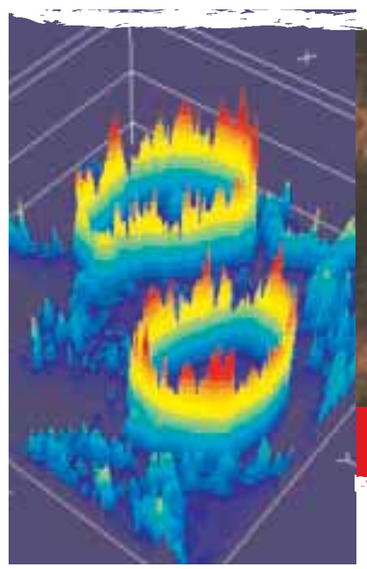
The Baker's imaging facilities fall into two primary categories, general imaging and imaging for scientific experiments.

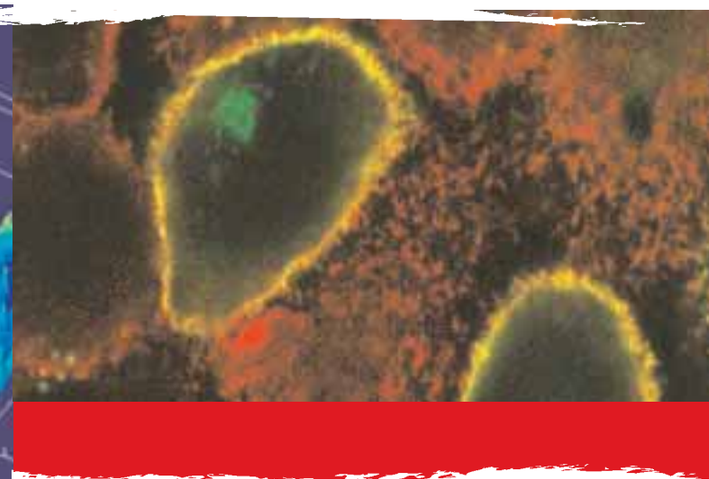
General imaging uses digital or film cameras to record the working environment and special events of scientists and administrative staff. Whether digital or film based, the images are digitised and edited to suit different output requirements such as reports, scientific poster displays, external promotional displays and external/internal Web based documents.

Scientific imaging at the Baker falls into two categories; microscope imaging and non-microscope imaging. Both imaging modes use digital detectors (generally cameras) to produce images. These images are then qualitatively and quantitatively analysed or measured. The images and analysis results are used to test scientific hypotheses in experiments and also to present the scientific findings at conferences and seminars and in journal publications.

Scientific imaging requires specialised digital cameras that are designed and calibrated to perform optimally under a range of experimental conditions, often at the limits of current digital camera technology. These digital cameras need specialised software to run them and to analyse the images. The common link between general and scientific imaging at the Baker is the use of computers and software to view and store the images.

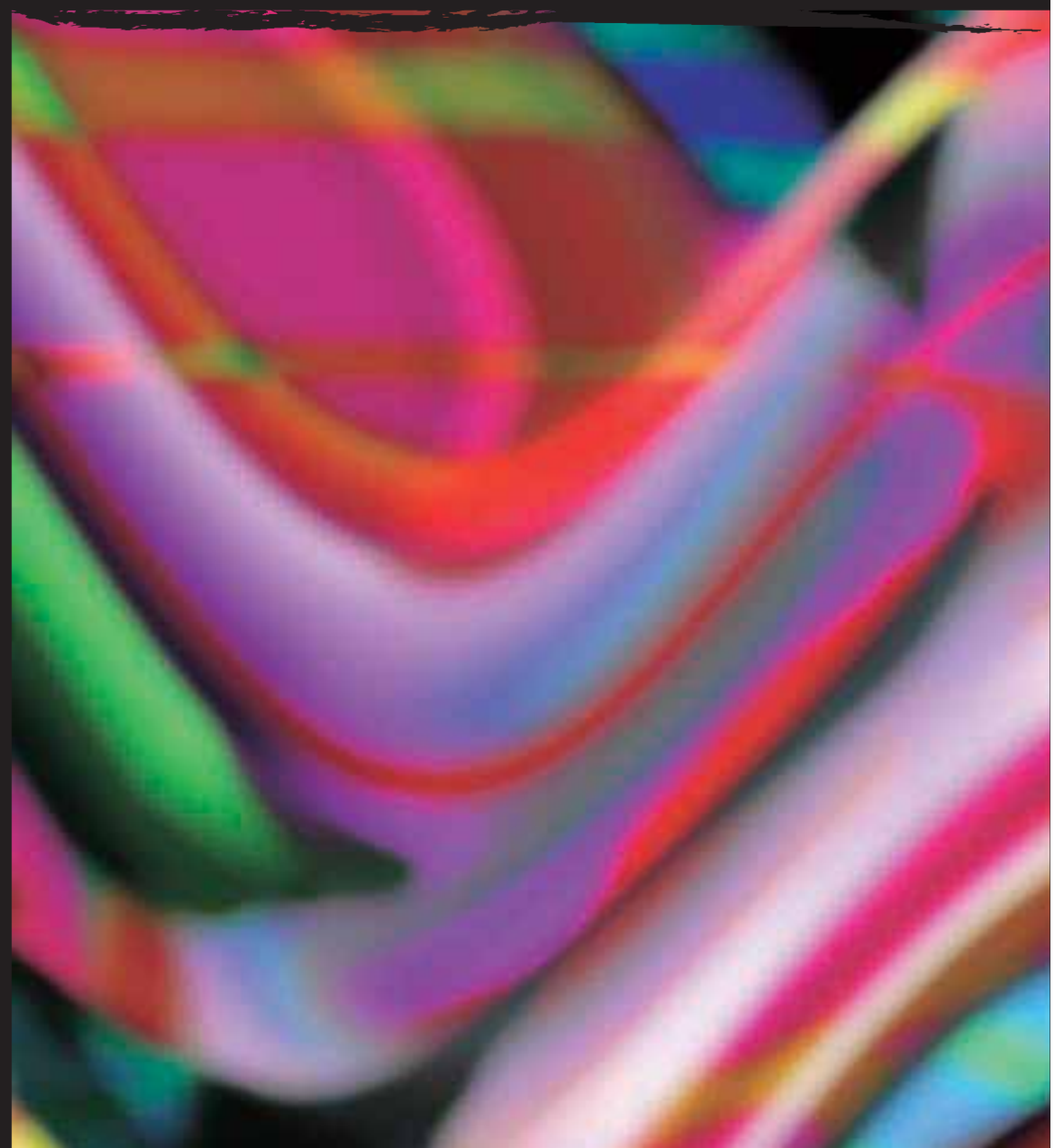
The Digital Imaging Laboratory coordinates the purchase and use of all digital imaging equipment and software for both general and scientific imaging needs. The Morphology Lab houses the microscopes that have specialised cameras and software attached to them.





Commercialisation

...Australia has a long tradition of excellence in biomedical research and its scientists have been at the leading edge of a number of important breakthroughs that have fundamentally changed clinical practice around the world...



Commercialisation

Australia has a long tradition of excellence in biomedical research and its scientists have been at the leading edge of a number of important breakthroughs that have fundamentally changed clinical practice around the world. For some time now, there has been a drive to capture the most innovative science and, through commercial exploitation, provide a return on research investment. This return may take the form of increased employment, local and national wealth creation or new treatments for the community.

Australia now has a relatively small but burgeoning biotechnology industry supported by Government incentives, by venture capital groups, private individuals and by a growing group of specialist support functions.
The Baker Medical Research Institute and its partners at the Alfred Medical Research and Education Precinct form the nucleus of a new biotechnology cluster that is developing on the site. Self-supporting clusters of technology based industries and institutes are a model that has been adopted throughout the world and has led to the development of significant commercial activity and success. These clusters are important for attracting the most skilled individuals, for sharing ideas, for developing economies of scale and for attracting support industries. The Baker now houses seven biotechnology based companies that are bringing a change to the way we do research and a rich interchange of ideas and opportunities.
The protection of our intellectual property has been an important focus and in the last 12 months, we have seen our intellectual property portfolio increase by over 50%.

Our links with the commercial world have continued to develop, with existing alliances growing in scope and new alliances being forged. A number of agreements were entered into over the year ranging from clinical evaluation of new cardiovascular treatments through to evaluation of experimental compounds in tissue baths. These alliances are important for the Baker and provide us with access to new technologies and exposure to the world outside the Institute. Importantly, they help to provide revenue for our research activities.

In addition to the activities of intellectual property management and commercial agreements, the Baker is a founding member of Biocomm Pty Ltd. This company was established by the Victorian Government to assist with the capture and commercialisation of research from its member institutes.

The Baker now receives in excess of $3 million dollars per annum through a variety of commercial activities across a wide range of disciplines. Commercial research collaborations were undertaken with the following groups during the year.

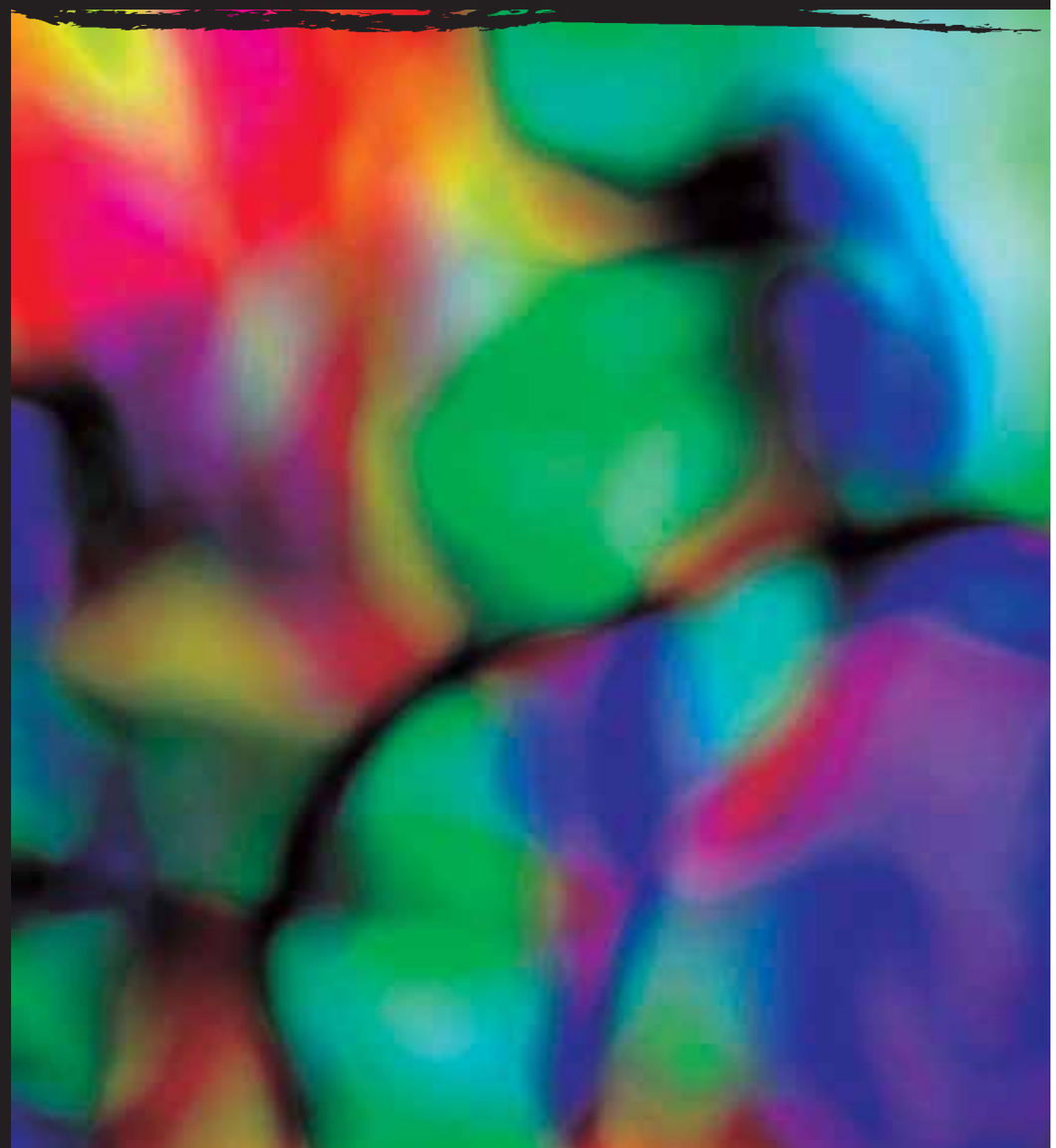
Commercial collaborations 2001

COMPANY	ACTIVITY
Amrad Operations Pty Ltd	Applications of LIF in cardiac disease
Xenome Limited	Noradrenaline transporter research
Novogen	Application of novel compounds in models of restenosis
Centre for Molecular Biology & Medicine	New approach to the management of hyperlipidaemia
Merck Sharp & Dohme	Cardiovascular disease risk
Novogen	Evaluation of novel compounds
Compumedics	Collaborative research agreement
Roche	Investigations of novel compound
Unida	Effects of new compounds on human blood vessels
Mimotopes	Development of new molecules to improve vascular health
Promics Pty Ltd	New complement inhibitors
Cytopia Pty Ltd	New approach to atherosclerosis
Servier	Evaluation of cardiovascular effects of new therapies
Pharmacia	Collaborative research

Intellectual Property Management 2001.

TITLE
Method of analysis of circadian rhythms
CYP27A1 in the treatment of atherosclerosis
Telomerase inhibitory peptides and uses thereof
An isoform of 17beta hydroxysteroid dehydrogenase and uses therof
Inhibitors of C-ABL as anti-atherosclerotic agents
Novel inhibitors of arginine transport
ACE inhibition and NO production in heart failure

... Singapore is an important gateway to the region and while it has an impressive track record in the area of basic research it foresees a need to build capacity in clinical and translational research experience...



International

Baker Internationally

Baker Singapore

The Baker Institute has a valuable repertoire of expertise in unravelling the causes and risks of heart disease and also in developing treatments. Now more than ever the Baker influence is extending beyond Australian shores. With the current worldwide explosion in the incidence of heart disease, particularly in developing countries, the Baker has a particular responsibility in the Asia Pacific regions. The most recent focus has been the establishment and operation of the Singapore Baker Medical Unit at the National Heart Centre.
Singapore is an important gateway to the region and while it has an impressive track record in the area of basic research it foresees a need to build capacity in clinical and translational research experience.
The Director Professor Garry Jennings, Chief Operating Officer, Dr Peter Hughes and Board Member, Philip Munz were accompanied by the Executive Director of Victoria's Science and Technology Initiative, Dr Jane Niall, and Stan Yakatan, State Biotechnology Adviser and Chair of Biocomm International, in a high level delegation to Singapore in late 2001. They met many of the key people from health, biotechnology, academia and government, including Philip Yeo, the chair of the National Science and Technology Board.
Research opportunities include access to a different population of patients with heart disease to enable comparative studies and we are establishing a good platform from which the Baker and the National Heart Centre can extend their research elsewhere in the region.
The Baker has been asked to submit a business development plan. In the first instance we plan to increase our Singapore laboratories from one to four using the local grants system. We will maintain a presence at the senior staff level whose brief is to establish the Baker Institute as a significant presence in Singapore and the region.
The seed to establish a structured collaborative venture between the Baker Institute and Singapore was planted many years ago when Singapore cardiologist, Professor Yean Leng Lim was a registrar and resident at the Alfred Baker Medical Unit. Professor Yean Leng Lim has had an outstanding career as a clinician and educator. He is now the Director of the National Heart Centre in Singapore and guides national policy.
He has founded a Medical School in China and brought the latest advances in cardiology and heart surgery to regional centres in China, often working with our own surgeon, Associate Professor Donald Esmore.

International Visiting Scientists to the Baker

Dr Chris Brown, University of Calgary, Calgary, Canada
Dr Joan Heller Brown, University of California, San Diego, USA
Professor Mikael Elam, Gothenburg University, Gothenburg, Sweden
Dr Genevieve Escher, Berne, Switzerland
Dr Gabrielle Gallon, University of Toulouse, Toulouse, France
Dr Xavier Gonzales, Vice President Spiration Inc, Redmond WA, USA.
Ms Sarah Hamilton, Dartmouth College, USA
Dr Ben Janssen, University of Limburg, Limburg, The Netherlands
Dr Natalia Kalinina, Institute of Experimental Cardiology, Moscow, Russia
Dr Alexandr Kapustin, Moscow State University, Moscow, Russia
Dr David Kass, Johns Hopkins University, Baltimore, USA.
Prof Jose Lopez, Baylor College of Medicine, USA
Dr EV Lukoshkova, National Cardiology Research Center, Moscow, Russia
Mr Timothy Matthews, University of New Mexico, Alburquerque, USA
Prof Chris Munsch, Leeds Royal Infirmary, Leeds, UK
Dr David Reuter, Cardiac Dimensions Inc, Kirkland, USA
Dr Simon Slight, Columbia, USA
Dr Nobuyo Tsunoda, Josai University, Japan
Dr Huy Than, Boston Scientific, San Jose, USA
Dr Mark Tuner, St Mary Hospital, London, UK
Professor Irv Zucker, University of Nebraska, Nebraska, USA

Overseas Conferences where the Baker was represented

XVIIIth Congress of the International Society on Thrombosis and Haemostasis, Paris, France
17th Wilhelm Bernard's Workshop for the Nucleus, Arcachon, France
3rd France Australia Meeting on Hypertension, Porticcio, France
Fourth International Workshop on Structure and Function of Large Arteries, Paris, France
IIIrd Franco-Australian Meeting on Hypertension, Corsica, France
Second International Symposium on Angiotensin Receptor Blockers, Monte Carlo
Conference on Functional Foods, Paris, France
Conference on Health Aspects of Phytosterols, Stresa, Italy
11th Scientific Meeting of the European Society of Hypertension, Milan, Italy
Satellite Symposium of the 11th European Meeting on Hypertension, Verbania, Italy
Symposium on Adrenergic Mechanisms in Heart Failure, Lubeck, Germany
3rd Cardiovascular-Pharmacology Symposium, Lubeck, Germany
2nd Internation Society of Obesity Meeting, Berlin, Germany
European Cardiology Conference, Stockholm Sweden
7th WHO-ISH Blood Pressure Collaboration Meeting, Cambridge, UK
British Electrophoresis Society, York, UK
Future Forum (Cardiovascular Conference), London, UK
Satellite Meeting to XVII World Congress of the International Society for Heart Research, Banff, Canada
International Society for Heart Research meeting, Winnipeg, Canada
Annual meeting of the North American Association for the Study of Obesity, Quebec City, Canada
9th Congress of the International Association of Biomedical Gerontology, Vancouver, Canada
American Heart Association, Anaheim, USA
American College of Cardiology, Orlando, USA
American Heart Association Meeting, Anaheim, USA
Heart Failure Society of America Meeting, Washington, USA
Canada American Society of Hematology Meeting, Orlando, USA
Drugs Affecting Lipid Metabolism, New York, USA
American Society for Human Genetics, San Diego, USA
Aldosterone Conference, Denver, USA
Endocrine Society Meeting, Denver, USA
4th International/17th American Peptide Symposium Peptide meeting, 2001 San Diego California, USA
Molecular Pharmacology Gordon Research Conference, Ventura, California, USA
American Society of Hypertension Meeting, San Francisco, USA
Bio 2001, San Diego, California, USA
2nd Annual Conference on Arteriosclerosis, Thrombosis and Vascular Biology, Washington DC, USA
Angiotensin Gordon Research Conference Ventura, California, USA
4th International Symposium on Vasoactive Peptides, Belo Horizonte, Brazil
International Meeting on Presynaptic Receptors, Madeira
1st World Heart Federation Global Conference on Cardiovascular Clinical Trials, Hong Kong
2nd Asian Pacific Congress on Hypertension, Thailand
International Catecholamine Symposium, Kyoto, Japan
24th Annal Scientific Meeting of the Japanese Society of Hypertension, Osaka, Japan
5th International Society of Preventive Cardiology, Osaka, Japan
The 2nd Vascular Biology Meeting, Singapore
Conference on Heart Protection Study, Singapore
Korean Peptide Society Meeting, Seoul, Korea
Korean Atherosclerosis Society Scientific Meeting, Seoul Korea
15th Bienniel Asian Congress on Thoracic and Cardiovascular Surgery, Mumbai, India
Satellite meeting of the 2001 International Congress of Physiological Sciences – Cardiorenal Control in Health and Disease, Queenstown, New Zealand
34th IUPS Congress in Christchurch, New Zealand
A Satellite of the 34th IUPS Congress 2001 in Queenstown New Zealand
International Telemetry User Group in Queenstown, New Zealand
Cardiac Society of Australia and New Zealand, Auckland, New Zealand
49th Annual Scientific Meeting of the Cardiac Society of Australia & New Zealand, Auckland, New Zealand

Board of Management

Norman O'Bryan SC
BA, LLB, BCL

President,
Baker Board of Management,
Barrister-at-Law

Dr Gerard P Johnston
BSC, PHD

Vice President,
Baker Board of Management
Trustee Baker Foundation

Prof Garry Jennings
MD, MBBS, FRCP, FRACP, FAHA

Director,
Baker Medical Research Institute

Prof Daine Alcorn
BSc (HONS), PhD

Department of Anatomy & Cell Biology University of Melbourne

Ross E Baker
BSC (HONS) MBA ASIA

Hon Treasurer
Baker Board of Management,
Managing Director
Australian Foundation
Investment Co

William P Gurry AO
LLB

Chairman
Baker Research Foundation

Dr Peter G. Habersberger AM
RFD, MB, BS, FRACP

Visiting Cardiologist,
Alfred Hospital
Assistant Surgeon General,
Australian Defence Forces - Navy

Philip Munz
LLB (HONS)

Group Executive Chairman,
GSA Group Pty Ltd

Prof Nicholas Saunders
MD, FRACP, FRCP, FAIM

Dean Faculty of Medicine,
Nursing & Health Sciences
Monash University

Professor Richard Smallwood AO
MD, FRACP, FRCP, FACP (HON)

Chief Medical Officer
Department Health & Aging,
NHMRC Representative

Rob Stewart
LLB (HONS), BCom, MBA (HARV)

Company Director
& Management Consultant

Dr Michael Walsh
MBBS (HONS), BHA, FRACMA, MPA (Harv)

Chief Executive
Bayside Health

Patron Sir Laurence Muir VRD, LLB, FSIA, FIAM
Patron of the Institute and former President of the Board of Management

Financial Report

Board Members' Report

FOR THE YEAR ENDED 31 DECEMBER 2001

The Board of Management present their report together with the financial statements of the Institute for the year ended 31 December, 2001 and the auditors' report thereon.

Board Members

The following persons were Board Members of the Institute during the whole of the financial year up to the date of this report:

Mr N O'Bryan SC, President
Dr G P Johnston Vice-President
Mr R E Barker Hon. Treasurer
Professor D Alcorn
Dr P G Habersberger AM
Mr P Munz
Professor N Saunders
Professor R Smallwood AO
Mr R Stewart
Dr M Walsh

Professor J W Funder AO was a Board Member from the beginning of the financial year up to until his resignation on 31 October 2001. Professor G L Jenninngs was appointed Institute Director on 1 November 2001.

Mr W Gurry AO has resigned on 6 March 2002 during the period after financial year end, up to the date of this report.

Principal Activities

The principal activities of the Institute are medical research into the basic causes of cardiovascular disease, to use this knowledge to help prevent heart and vascular disease in the community, and to improve medical and surgical treatment. No significant change in the nature of these activities occurred during the year.

Operating Result

The financial result from research activities was a deficit of $2,488,928 (2000: deficit $1,507,828). After allowing for the deficit from the Capital Fund which arises from the cost of the new Institute, the consolidated result for the year was a deficit of $13,006,505 (2000: surplus $13,389,846). Income tax is not applicable.

Review of Operations

A review of the operations of the Institute during the year has been included in the President's and Director's report. The Intitute's activities continued to be dedicated to medical research into the basic causes of cardiovascular disease. The Institute is a body corporate under an Act of Parliament and has no share capital.

Likely Developments

The Institute does not expect any significant changes to its operations in the coming year.

Environmental Regulations

The Institute complies with the Environmental Protection Act in respect of its operations.

Insurance of Officers

During the financial year, the Baker Medical Research Institute paid a premium of $6,116 to insure certain officers and board members of the Institute.

The liabilities insured include costs and expenses that may be incurred in defending civil or criminal proceedings that may be brought against the officers in their capacity as officers of the Institute.

State of Affairs

During November and December, The Baker, its staff and thousands of pieces of laboratory equipment computers, files and publications were moved to the new Baker Building. During this period, the Baker was operational across two buildings complete with interlinking telephone, computer systems, supply and all other functions.

In the first half of 2002, on completion of the the extension of the Burnett Institute building, we look forward to the official opening by the Premier of Victoria.

Events Subsequent to Balance Date

There has not arisen in the interval between the end of the financial year and the date of this report any item, transaction or event of a material and unusual nature likely, in the opinion of the Board of Management of the Institute, to affect significantly the operations of the Institute, the results of those operations or state of affairs of the Institute in subsequent financial years.

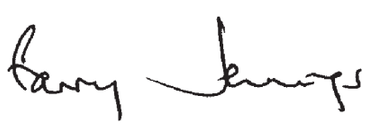
Board Members Benefits

Since the end of the previous financial year, other than Mr R E Barker who is a shareholder of a firm of Stockbrokers which has received, or become entitled to receive, fees for services rendered to the Institute on normal commercial terms, no Board member has received or has become entitled to receive any benefit, by reason of a contract made by the Institute or a related corporation with any Board Member or with a firm of which a Board Member is a member or with an entity in which any Board Member has a substantial financial interest.

Dated at Melbourne this 18th day of April 2002

Signed in accordance with a resolution of the Board of Management

Norman O'Bryan SC
PRESIDENT



Garry Jennings
DIRECTOR

Baker Medical Research Institute
Statement of Financial Performance Year ended 31 December 2001

	NOTE	2001 $	2000 $
Revenue from ordinary activities	3	**28,447,013**	47,792,705
Expenses for building works		**(25,228,178)**	(19,781,593)
Employee benefits expense		**(11,488,939)**	(9,931,555)
Laboratory consumables used		**(1,673,071)**	(1,850,064)
Depreciation and amortisation expenses		**(902,548)**	(599,171)
Borrowing costs expense		**(27,969)**	(20,396)
Other expenses from ordinary activities		**(2,132,813)**	(2,220,080)
(Deficit) / Surplus from ordinary activities before income tax expense	4	**(13,006,505)**	13,389,846
Income tax expense	2(k)	0	0
Consolidated (Deficit) / Surplus from ordinary activities after income tax expense		**(13,006,505)**	13,389,846
Total changes in funds other than those resulting from transactions with owners as owners		**(13,006,505)**	13,389,846

The above statement of financial performance should be read in conjunction with the accompanying notes

Baker Medical Research Institute
Statement of Financial Position as at 31 December 2001

	NOTE	2001 $	2000 $
Assets			
Current Assets			
Term Deposits	9	**0**	15,604,598
Receivables		**5,361,503**	2,536,996
Inventories	2(h)	**0**	55,143
Prepayments		**109,839**	277,022
Accrued Interest		**0**	30,451
Total Current Assets		**5,471,342**	18,504,210
Non - Current Assets			
Plant & Equipment	2(e), 11	**4,083,997**	2,913,234
Investments (at cost)	10	**6,902,197**	8,330,634
Total Non - Current Assets		**10,986,194**	11,243,868
Total Assets		**16,457,536**	29,748,078
Liabilities			
Current Liabilities			
Interest Bearing Liabilities	2(f), 14	**1,307,833**	3,081,487
Payables		**1,838,234**	390,811
Prepaid Grants	12	**91,509**	0
Provisions	13	**1,141,490**	1,043,629
Total Current Liabilities		**4,379,066**	4,515,927
Non - Current Liabilities			
Interest Bearing Liabilities	2(f), 15	**96,720**	136,790
Provisions	16	**173,039**	280,145
Total Non - Current Liabilities		**269,759**	416,935
Total Liabilities		**4,648,825**	4,932,862
Net Assets		**11,808,711**	24,815,216
Funds			
Accumulated Funds			
Operating Fund	5	**(8,378,475)**	(5,889,547)
Capital Fund	6	**17,887,164**	28,528,987
Specific Purpose Fund	7	**298,534**	174,288
Asset Revaluation Reserve - 1/1/93		**2,001,488**	2,001,488
Total Funds	8	**11,808,711**	24,815,216

The above statement of financial position should be read in conjunction with the accompanying notes

Baker Medical Research Institute
Statement of Cash Flows for the year ended 31 December 2001

	NOTE	2001 $	2000 $
Cash Flows from Consolidated Activities			
Receipts from Granting Bodies		**6,380,274**	3,651,958
Donations and Bequests		**6,586,615**	7,037,473
Receipts for Building Works		**10,516,036**	28,917,049
Payments to Suppliers & Employees (inc. GST)		**(39,273,196)**	(34,532,583)
Dividends Received		**400,966**	402,947
Interest Received		**320,311**	736,274
General Income		**468,300**	244,987
Net Cash Inflow from Consolidated Activities	19b	**(14,600,694)**	6,458,105
Cash Flows from Investing Activities			
Payment for Investment Securities		**(1,291,017)**	(3,048,330)
Proceeds from sale of Investment Securities		**4,159,974**	3,346,401
Payment for Property, Plant & Equipment		**(2,080,598)**	(1,077,851)
Net Cash Outflow from Investing Activities		**788,359**	(779,780)
Cash Flows from financing activities			
Principal Repayments under finance leases		**(35,505)**	(47,593)
Net Cash Outflow from financing activities		**(35,505)**	(47,593)
Net Cash Increase in cash held		**(13,847,840)**	5,630,732
Cash at beginning of the financial year		**12,583,317**	6,950,159
Effects of exchange rate changes on cash held in foreign currencies		**(3,239)**	2,426
Cash at the end of the financial year	19(a)	**(1,267,762)**	12,583,317

The above statement of cash flows should be read in conjunction with the accompanying notes

1. Incorporation

The Thomas Baker, Alice Baker and Eleanor Shaw Medical Research Institute was incorporated as the "Baker Medical Research Institute' ("the Institute") under the Baker Medical Research Act 1980.

2. Summary of Significant Accounting Policies

This general purpose financial report has been prepared in accordance with Accounting Standards, other authoritative pronouncements of the Australian Accounting Standards, Urgent Issues Group Consensus Views and the Corporations Law.

Set out hereunder are the significant accounting policies adopted by the Institute in the preparation of its accounts for the year ended 31 December 2001. These policies have been consistently applied unless otherwise indicated.

(a) Accrual basis
The accrual basis of accounting has been used with revenues and expenses being recognised as they are incurred, and brought to account in the period to which they relate.

(b) Historical cost
The financial statements have been prepared on a historical cost basis and except where stated do not take into account current valuations of non-current assets.

(c) Fund accounting
The Institute operates on a fund accounting basis and maintains three funds; Operating, Specific Purpose and Capital Funds. The work of the Institute is financed from grants, investment income and donations of both general and specific natures. Income of a specific nature is used in accordance with the terms of any relevant convenants. The amount of grants received for specific purposes during the year but unspent at year end, will be generally expended in the next financial year. The Institute's capital fund comprises the capital donations, bequests and receipts from fundraising activities carried forward.

(d) Principles of consolidation
The Institute's accounts have been prepared on a consolidated basis. All inter-fund transactions have been eliminated on consolidation.

(e) Depreciation of property, plant and equipment
Depreciation is calculated on a straight line basis to write off the net cost or revalued amount of each item of property, plant and equipment (excluding building) over its expected useful life to the Institute. The following estimated useful lives are used in the calculation of depreciation:
- plant and equipment (5-20 years)
- furniture and fittings (5 years).

Profits and losses on the disposal of plant and equipment are taken into account in determining the result for the year.

(f) Leased Assets
A distinction is made between finance leases, which effectively transfer from the lessor to the lessee substantially all the risks and benefits incident to ownership of leased non-current assets, and operating leases under which the lessor effectively retains substantially all such risks and benefits.

Assets acquired under finance leases are included as property, plant and equipment in the balance sheet. Finance leases effectively transfer from the lessor to the lessee substantially all the risks and benefits incidential to ownership of the leased property. Where assets are aquired by means of finance leases, the present value of the minimum lease payments is recognised as an asset at the beginning of the lease term and amortised on a straight line basis over the expected useful life of the asset. A corresponding liability is also established and each lease payment is allocated between the liability and finance charge.

Operating lease payments are charged to the Statement of Financial Performance in periods in which they are incurred, as this represents the pattern of benefits derived from the leased assets.

(g) Land and building
The Institute has adopted the policy that capital expenditure incurred in respect of the planned new building is written off against income during the year. The Baker's new Medical Research Institute is not included as an asset in the accounts as the Institute does not have title to the property.

(h) Inventories
During 2001, the Institute entered into a shared service agreement for the Alfred Hospital to manage all supply functions on its behalf. Under this arrangement the Institute no longer carries a store of consumable supplies.

(i) Cash
For purposes of the statement of cash flows, cash includes deposits at call which are readily convertible to cash on hand and are subject to an insignificant risk of changes in value, net of outstanding bank overdrafts.

(j) Investments
Interests in listed and unlisted securities are brought to account at cost and dividend income is recognised in the profit and loss account when receivable.

(k) Tax status
The income of the Institute is exempt from income tax pursuant to the provisions of section 50-5 of the Income Tax Assessment Act 1997. The Institute is also exempt from other government levies such as payroll tax and sales tax but not fringe benefits tax.

(l) Employee entitlements
(i) *Wages and salaries and annual leave*
Liabilities for wages and salaries and annual leave are recognised, and are measured as the amount unpaid at the reporting date at current pay rates in respect of employees' services up to that date.

(ii) *Long service leave*
A liability for long service leave is recognised, and is measured as the present value of expected future payments to be made in respect of services provided by employees up to the reporting date. Consideration is given to expected future wage and salary levels, experience of employee departures and periods of service. Expected future payments are discounted using interest rates on national government guaranteed securities with terms to maturity that match, as closely as possible, the estimated future cash outflows.

(m) Foreign exchange transactions
The Institute maintains a bank account in the USA for the purpose of receiving donations and for the purchase of equipment and supplies. Foreign currency transactions are initially translated into Australian currency at the rate of exchange at date of the transaction. Amounts receivable or payable in foreign currency at balance date are translated to Australian currency at exchange rates at balance date. Exchange gains and losses are brought to account in determining the operating surplus or deficit for the year.

(n) Trade and Other Creditors
These amounts represent liabilities for goods and services provided to the Institute prior to the end of the financial year and which are unpaid. The amounts are unsecured and are usually paid within 30 days of recognition.

(o) Receivables
All trade debtors are recognised at the amounts receivable as they are due for settlement. Collectibility of trade debtors is reviewed on an ongoing basis. Debts which are known to be uncollectible are written off.

(p) Borrowing costs
Borrowing costs are recognised as expenses in the period in which they are incurred. Borrowing costs include:
- interest on bank overdrafts and short-term and long-term borrowings
- amortisation of discounts or premiums relating to borrowings
- amortisation of ancilliary costs incurred in connection with the arrangement of borrowings
- finance lease charges, and
- certain exchange differences arising from foreign currency borrowings

(q) Revenue Recognition

Amounts disclosed as revenue are net of returns, trade allowances and duties and taxes paid. Revenue is recognised for the major business activities as follows:

(i) *Grant income*
Recognised when due and payable under terms and conditions of an agreement.

(ii) *Interest revenue*
Recognised when received

(iii) *Investment revenue*
Recognised on sale of investments

(r) Comparative figures

Where necessary comparative figures have been adjusted to conform with changes in presentation in the current year.

3. Revenue from Ordinary Activities

	2001 $	2000 $
Revenue from operating activities		
Government and Statutory Bodies	**6,297,503**	6,202,009
Baker Foundation	**1,150,000**	1,150,000
Revenue from outside the operating activities		
Fundraising, Corporate & Private Support	**5,847,571**	6,787,503
Capital Works Campaign	**12,540,080**	30,783,651
Dividends Received / Receivable	**390,553**	389,923
Interest Received / Receivable	**289,859**	670,868
Foreign exchange gain	**0**	2,426
Proceeds from sale of non-current assets	**1,457,935**	1,466,398
General Income	**473,512**	339,927
Total Income	**28,447,013**	47,792,705

4. Operating Surplus / Deficit

The deficit from ordinary activities before income tax expense includes the following specific net gains and expenses:

Net gains		
Net gain on disposal		
Motor vehicles	**17,414**	27,725
Investments	**1,440,521**	1,438,673
Foreign exchange gain	**0**	2,426
Expenses		
Borrowing costs		
Interest and finance charges paid/payable	**60,235**	67,989
Foreign exchange loss	**3,239**	0
	63,474	67,989
Less: Amount capitalised	**(35,505)**	(47,593)
Borrowing costs expensed	**27,969**	20,396
Depreciation - Plant and Equipment	**853,440**	531,481
Amortisation - Motor Vehicles under finance lease	**49,108**	67,690
Write down of inventories to net realisable value	**55,143**	22,018
Employee Entitlements	**62,808**	282,169
Rental expense relating to operating leases	**500,843**	429,502

5. Operating Fund

Balance at beginning of year	**(5,889,547)**	(4,381,719)
Deficit for year	**(2,488,928)**	(1,507,828)
Balance at end of year	**(8,378,475)**	(5,889,547)

6. Capital Fund

The Institute's Capital fund comprises donations, bequests and receipts from fundraising activities. Each year the Board allocates a proportion of these funds to supplement the research operations of the Institute. The Fund also incorporates grants and contributions received towards the cost of the new Institute building and the associated interest earned thereon.
Funds received in respect of the new Medical Research Institute, but not outlaid at 31 December 2001, are carried forward. The current balance is:

	2001 $	2000 $
Balance at beginning of year	**28,528,987**	13,604,570
Surplus for year	**(10,641,823)**	14,924,417
Balance at end of year	**17,887,164**	28,528,987

7. Specific Purpose Fund

Specific purpose funds comprise funds provided to the Institute for special purposes other than through normal fund-raising activities. The funds are used in accordance with the wishes of donors. Institute accounting records are kept so as to identify expenditure charged against income of these funds. All such income and expenditure is incorporated in the Statement of Financial Performance. The current fund balance is:

Balance at beginning of year	**174,288**	201,031
Deficit for year	**124,246**	(26,743)
Balance at end of year	**298,534**	174,288

8. Fund Balances

Balance at 1 January 2001	**24,815,216**	11,425,370
Surplus / (Deficit) for year -		
Operating Fund	**(2,488,928)**	(1,507,828)
Capital Fund	**(10,641,823)**	14,924,417
Specific Purpose Fund	**124,246**	(26,743)
	(13,006,505)	13,389,846
Balance at 31 December 2001	**11,808,711**	24,815,216

9. Current Assets – Term Deposits

Term deposits	**0**	15,604,598
Total Term Deposits	**0**	15,604,598

10. Non-Current – Investments (at cost)

Shares and Debentures	**6,902,197**	8,330,634
Total Non - Current Investments	**6,902,197**	8,330,634

The Institute's investments are shown at cost. As at the 31 December 2001 the market value of the Institute's non-current investments was $8,865,007 (2000: $11,206,292).

11. Non-Current Assets – Property, Plant and Equipment

Prior to the move to the new Baker building, a management review was undertaken of the plant and equipment across the Institute. As a consequence of this review an estimated $2,504,551 of plant and equipment was written off. This plant and equipment have been fully depreciated to nil residual value thus financial effect of this change in the current financial year was a decrease in plant and equipment by $2,504,551 with a corresponding decrease in accumulated depreciation.

Plant and Equipment (at cost or Board's Valuation)	**6,235,514**	6,659,467
Less: Accumulated depreciation	**(2,258,433)**	(3,909,544)
	3,977,081	2,749,923

	2001 $	2000 $
Motor Vehicles under finance leases	**200,262**	275,213
Less: Accumulated amortisation	**(93,346)**	(111,902)
	106,916	163,311
Total Plant and Equipment	**4,083,997**	2,913,234

Reconciliations of the carrying amounts of each class of property, plant and equipment at the beginning and end of the current financial year are set out below.

	PLANT & EQUIPMENT	MOTOR VEHICLES	TOTAL
Gross Carrying Value			
Carrying amount at 1 January 2001	6,659,467	275,213	**6,934,680**
Additions at cost	2,080,598	0	**2,080,598**
Disposals	(2,504,551)	(74,951)	**(2,579,502)**
Balance at 31 December 2001	6,235,514	200,262	**6,435,776**
Accumulated Depreciation			
Balance at 1 January 2001	(3,909,544)	(111,902)	**(4,021,446)**
Depreciation expense	(853,440)	(49,108)	**(902,548)**
Disposals	2,504,551	67,664	**2,572,215**
Balance at 31 December 2001	(2,258,433)	(93,346)	**(2,351,779)**
Net Book Value			
As at 1 January 2001	2,749,923	163,311	**2,913,234**
As at 31 December 2001	3,977,081	106,916	**4,083,997**

12. Current Liabilities – Prepaid Grant

Prepaid Grant represents the monthly grant received from the State Government for infrastructure support.

Prepaid Grants	**91,509**	0

13. Current Liabilities – Provisions

Annual Leave	**542,584**	528,355
Long Service Leave	**598,906**	515,274
Total Current Provisions	**1,141,490**	1,043,629

14. Current Liabilities – Interest Bearing Liabilities

Bank Overdraft	**1,267,762**	3,021,281
Lease liability	**40,071**	60,206
Total Current Interest Bearing Liabilities	**1,307,833**	3,081,487

15. Non-Current Liabilities – Interest Bearing Liabilities

Lease liability	**96,720**	136,790
Total Non-Current Interest Bearing Liabilities	**96,720**	136,790

16. Non-Current Liabilities - Provisions

Long Service Leave	**173,039**	208,092
Deferred Maintenance	**0**	72,053
Total Non - Current Provisions	**173,039**	280,145

17. Lease Commitments

Finance Lease Commitments

Finance Lease Commitments are payable as follows:

	2001 $	2000 $
Not later than 1 year	**50,262**	76,292
Later than 1 year and not later than 5 years	**112,418**	162,927
Minimum lease payments	**162,680**	239,219
Less: Future lease charges	**(25,889)**	(42,223)
Provided for in accounts	**136,791**	196,996
Representing lease liabilities:		
Current lease liability	**40,071**	60,206
Non-current liability	**96,720**	136,790
	136,791	196,996

Operating Lease Commitments

Commitments for minimum lease payments in relation to non-cancellable operating leases are payable as follows:

Not later than 1 year	**81,819**	143,167
Later than 1 year and not later than 5 years	**172,961**	286,334
	254,780	429,501

18. Related Parties

(a) The names of each person who held office as a Board Member of the Baker Medical Research Institute during the financial year ended 31 December 2001 are:

Mr N O'Bryan SC
Mrs M Ross (resigned 28/3/01)
Prof J W Funder (resigned 31/10/01)
Mr R E Barker
Prof R Smallwood AO
Dr P G Habersberger
Prof D Alcorn (from 5/2/01)
Dr M Walsh (appointed 24/4/01)
Mr. P. Munz
Prof P Darvall (resigned 28/3/01)
Dr G P Johnston
Prof N Saunders (from 28/2/01)
Mr W P Gurry
Prof G Jennings (from 1/11/01)
Mr R Stewart (from 17/2/01)
Prof S Holdsworth (resigned 16/2/01)
Mr P C Barnett (resigned 26/2/01)

(b) Other than Mr R E Barker who is a shareholder of a firm of Stockbrokers which has received, or become entitled to receive for services rendered to the Institute on normal commercial terms, no Board Member has received or has become become entitled to receive any benefit, by reason of a contract made by the Institute or a related corporation with any Board Member or with a firm of which a Board Member is a member or with an entity in which any Board Member has a substantial financial interest.

19. Notes to the Statement of Cash Flows

(a) For the purpose of the statement of cash flows, cash includes cash on hand and in the bank and investments in the money market instruments, net of outstanding bank overdrafts. The Institute has an unsecured overdraft facility of $330,000 in place with Westpac Banking Corporation in relation to its ongoing research operations.

Cash at the end of the financial year as shown in the statement of cash flows is reconciled to the related items in the balance sheet as follows:

	2001 $	2000 $
Interest bearing liabilities	(1,267,762)	(3,021,281)
Term Deposits	0	15,604,598
Total	(1,267,762)	12,583,317

(b) Reconciliation of Operating Deficit after income tax to net cash from consolidated activities

Operating (Deficit) / Surplus from Consolidated Activities	(13,006,505)	13,389,846
Effects of exchange rate changes on cash held in foreign currencies	3,239	(2,426)
Depreciation and Amortisation	902,548	599,171
(Profit) on sale of non-current assets	(1,457,934)	(1,466,398)
Changes in net assets and liabilities		
(Increase) / Decrease in debtors	(2,824,507)	(1,598,824)
Decrease in inventories	55,143	22,018
Decrease / (Increase) in prepayments	167,183	(139,244)
Decrease / (Increase) in accrued interest	30,451	64,938
Increase / (Decrease) in creditors	1,447,423	(2,131,036)
Increase / (Decrease) in prepaid grants	91,509	(2,562,109)
(Decrease) / Increase in provisions	(9,244)	282,169
Net cash from consolidated activities	(14,600,694)	6,458,105

(c) Non-cash financing activities

Motor Vehicles

During the year the Institute provided motor vehicles for staff under salary sacrifice arrangements with a value of $200,261 by means of finance leases. These acquisitions are not reflected in the statement of cash flows.

20. Financial Instruments

(a) Significant Accounting Policies

Details of the significant accounting policies and methods adopted, including the criteria for recognition, the basis of measurement and the basis on which revenues and expenses are recognised, in respect of each class of financial asset, financial liability and equity instruments are disclosed in note 1 to the accounts.

(b) Significant Terms, Conditions and Objectives of Derivative Financial Instruments

The Institute does not enter into or trade complex derivative financial instruments.

(c) Credit Risk

The Institute does not have any significant credit risk exposure to any single counterparty or any group of counterparties having similar characteristics. The carrying amount of financial assets recorded in the consolidated balance sheet, net of any provision for losses, represents the Institute's maximum exposure to credit risk.

(d) Net Fair Value

The net fair value of the Institute's financial assets and financial liabilities is not materially different to their carrying amount in the financial statements, other than non-current investments. The net fair value of non-current investments is disclosed in note 9 to the accounts.

(e) Interest Rate Risk

The following table details the Institute's exposure to interest rate risk and the effective weighted average interest rates by maturity on financial instruments at balance date.

31 December 2001	VARIABLE INTEREST RATE	LESS THAN 1 YEAR $	1 TO 5 YEARS $	MORE THAN 5 YEARS $	NON - INTEREST BEARING	TOTAL
Financial Assets						
Receivables					5,361,503	**5,361,503**
Investments	6,902,197					**6,902,197**
Total Financial Assets	6,902,197				5,361,503	**12,263,700**
Weighted average interest rate	3.48%					
Financial Liabilities						
Bank Overdraft	1,267,762					**1,267,762**
Payables					1,838,234	**1,838,234**
Lease liabilities			40,071	96,720		**136,791**
Total Financial Liabilities	1,267,762		40,071	96,720	1,838,234	**3,242,787**
Weighted average interest rate	9.24%		20.28%	13.96%		
Net Financial Assets /(Liabilities)	5,634,435		(40,071)	(96,720)	3,523,269	**9,020,913**

31 December 2001	VARIABLE INTEREST RATE	LESS THAN 1 YEAR $	1 TO 5 YEARS $	MORE THAN 5 YEARS $	NON - INTEREST BEARING	TOTAL
Financial Assets						
Receivables					2,536,996	**2,536,996**
Fixed Interest Securities		15,604,598				**15,604,598**
Investments	8,330,634					**8,330,634**
Total Financial Assets	8,330,634	15,604,598			2,536,996	**26,472,228**
Average interest rate	3.44%	6.26%				
Financial Liabilities						
Bank Overdraft	3,021,281					**3,021,281**
Payables					390,811	**390,811**
Lease liabilities			60,206	136,790		**196,996**
Total Financial Liabilities	3,021,281		60,206	136,790	390,811	**3,609,088**
Average interest rate	8.86%		21.08%	16.04%		
Net Financial Assets /(Liabilities)	5,309,353	15,604,598	(60,206)	(136,790)	2,146,185	**22,863,140**

21. Capital Commitments

As at 31 December 2001, capital expenditure contracted for, in respect of completion of the building, at balance date but not provided for in the accounts of the Institute, is payable:

	2001 $	2000 $
Not later than 1 year	209,834	31,669,961
Total Capital Commitments	209,834	31,669,961

22. Superannuation

The Institute operates an accumulation type superannuation plan under which all employees are entitled on retirement, disability or death. Employees contribute to the plan at various percentages of their salaries. The Institute also contributes to the plan at rates related to employee contributions and pursuant to an award set down under a national wage case. Funds are available to satisfy all benefits that have been vested under the plan in the event of termination of the plan or voluntary or compulsory termination of employment of each employee.

23. Auditors Remuneration

Amounts received or due and receivable by the auditors of the Institute for:		
- audit of the financial report	17,700	17,000

24. Segment Information

The Institute operates in the medical research industry in the geographical area of Australia.

25. Reconciliation of Net Assets / (Liabilities) to Net Assets

	NOTE	2001 $	2000 $
Net financial assets as above	20	**9,020,913**	22,863,140
Non-financial assets and liabilities:			
Other assets		**109,839**	362,616
Property, plant and equipment	11	**4,083,997**	2,913,234
Other liabilities	12	**(91,509)**	-
Provisions	13, 16	**(1,314,529)**	(1,323,774)
Net Assets per Balance Sheet		**11,808,711**	24,815,216

Baker Medical Research Institute
Board Members' Declaration

The Board Members declare that the financial statements and notes set out on pages 33 to 44:

(a) comply with Accounting Standards, the Corporations Regulations and other mandatory professional reporting requirements; and

(b) give a true and fair view of the Institute's financial position as at 31 December 2001 and of its performance, as represented by the results of its operations and its cash flows, for the financial year ended on that date.

In the Board Members' opinion:

(a) the financial statements and notes are in accordance with the Corporations Law; and

(b) there are reasonable grounds to believe that the Institute will be able to pay its debts as and when they become due and payable.

This declaration is made in accordance with a resolution of the Board of Management.

For and on behalf of the Board

Norman O'Bryan SC
PRESIDENT

Garry Jennings
DIRECTOR

Melbourne
18th April 2002

Independent Audit Report
to the Members of the Baker Medical Research Institute

Scope

We have audited the financial report of the Baker Medical Research Institute (the Institute) for the financial year ended 31 December 2001 as set out on pages to . The Institute's Board Members are responsible for the financial report. We have conducted an independent audit of the financial statements in order to express an opinion on them to the members of the Institute.

Our audit has been conducted in accordance with Australian Auditing Standards to provide reasonable assurance as to whether the financial statements are free of material misstatement. Our procedures included examination, on a test basis, of evidence supporting the amounts and other disclosures in the financial statements, and the evaluation of accounting policies and significant accounting estimates. These procedures have been undertaken to form an opinion as to whether, in all material respects, the financial report is presented fairly in accordance with the Accounting Standards, other mandatory professional reporting requirements, and the Corporations Act 2001 in Australia so as to present a view which is consistent with our understanding of the Institute's financial position, and performance as represented by the results of its operations and its cash flows.

The audit opinion expressed in this report has been formed on the above basis.

Qualification

As stated in note 2(g) to the accounts, the Institute has written off to expense certain capital expenditures incurred on a planned new building, currently under construction, which we understand is going to be subject to a long term sublease to the Institute and other related parties. This is a departure from Accounting Standard AASB 1021 'Depreciation' which requires recognition of an asset with physical substance which is expected to be used during more than one financial year.

In our opinion, costs amounting to $25,228,178 (2000 -$19,745,601) should have been recognised as capital works in progress. Had this been done, non-current assets would be $36,214,372 (2000 - $30,989,469), total assets would be $41,685,714 (2000 - $49,493,679), consolidated surplus after income tax would be $12,221,673 (2000 – 33,135,447), capital funds would be $43,115,342 (2000 - $34,670,018) and accumulated funds would be $37,036,889 (2000 - $44,560,817).

Audit Opinion

In our opinion, except for the effects on the finacial report of the matter referred to in the qualification paragraph, the financial report of the Institute is in accordance with:

(a) the Corporations Act 2001, including;
  (i) giving a true and fair view of the Instute's financial position as at 31 December 2001 and of its performance for the year ended on that date; and
  (ii) complying with Accounting Standards and the Corporations Regulations 2001; and
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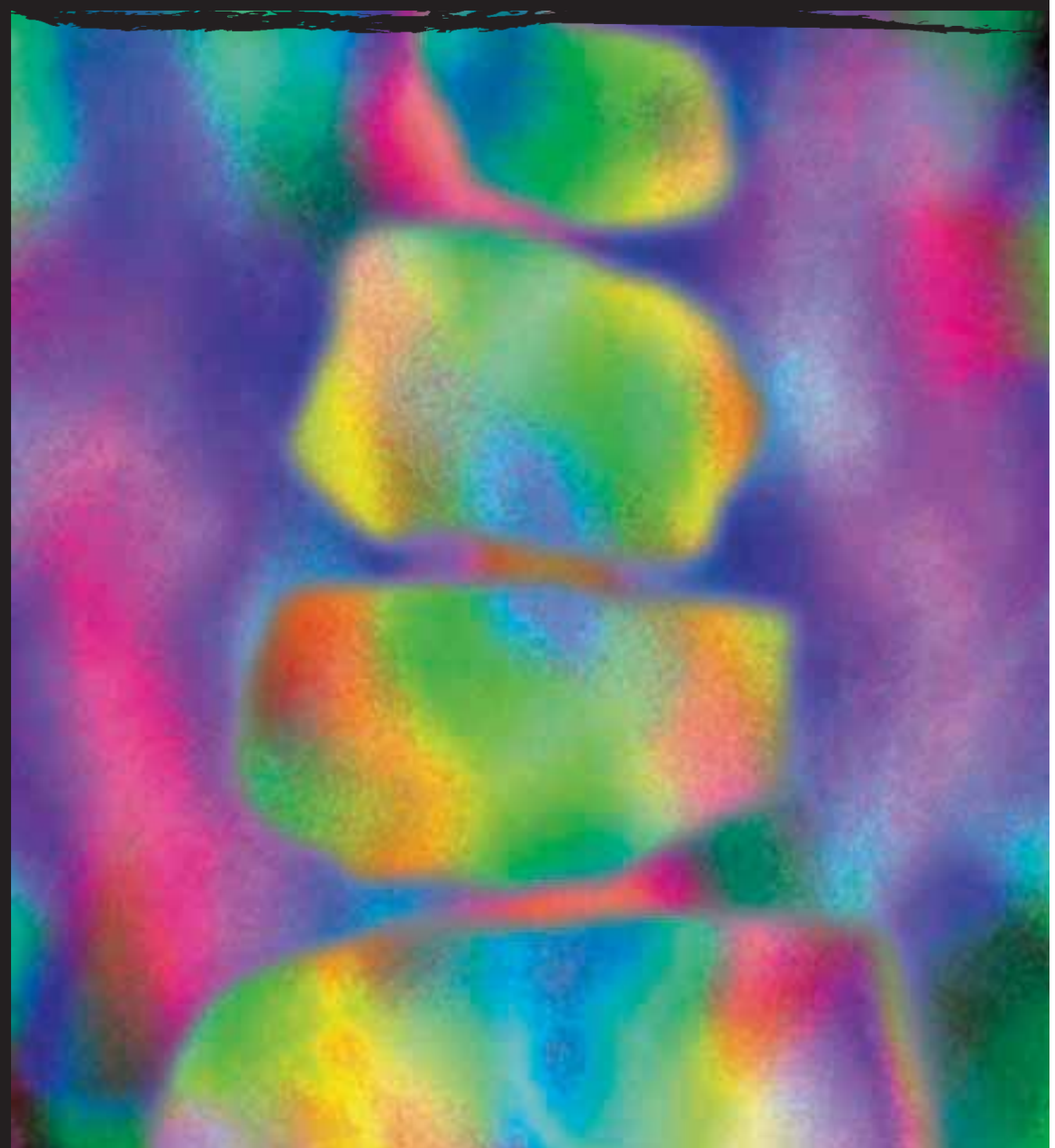

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Foundation

The Baker Research Foundation was established during 2001 to build upon the considerable support provided over the years by the corporate and community sectors. The Foundation will serve to channel additional research funds to the laboratories while also raising the general awareness of the valuable work being done by Baker scientists.

We were delighted to have Bill Gurry, AO, a member of the Baker Board of Management, as the inaugural Chairman of the Foundations and Judie Denham-Brennan as our first Foundation Executive Officer. The specific role of the Foundation within the Institute is to coordinate the community relations, communications, marketing and fundraising activities.

During the year a number of special events took place including a successful function at Moonee Valley, sponsored by the Bensons Group of Companies and organised by the Special Events Committee chaired by Sue Calwell; the Wine Lover's Dinner held at and supported by the Savage Club, organised by Bobbie Renard, Brian Leask and Ian Loftus; and the Baker Golf Classic sponsored by Minter Ellison and organised by Michael MacKellar.

The move into the new building at the end of 2001 signified the culmination of the Building Capital Campaign and we would like to thank all our supporters in this endeavour over the years. In particular, we would like to acknowledge the efforts and commitment of the Capital Campaign Taskforce. One of the building's most stunning architectural features is the atrium, which was assisted by, and dedicated to, Mr George and Gita Smorgon.

In the following pages the Institute gratefully acknowledges our many supporters whose generosity, has significantly contributed to the quality of our work.

To all our donors

We gratefully acknowledge the significant support of many donors who have made smaller but equally valuable contributions to our work, some over many years. We wish also to acknowledge those people who have chosen to send a donation to the Baker in memory of loved ones.
Thank you.

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