



BAKER HEART RESEARCH INSTITUTE ANNUAL REPORT 2002



MISSION

Cardiovascular disease is the leading cause of death and disability worldwide and is responsible for over 40% of deaths in Australia each year. The risk factors for cardiovascular disease are highly prevalent in the Australian community with 80% of all adults having one of the following risk factors:

Smoking

Inactivity

High blood pressure

Overweight

Depression

Social isolation

At the Baker, our mission is to reduce death and disability from cardiovascular disease. We achieve this through activities ranging from research at the laboratory bench to clinical trials, patient care and education.

The major areas of research at the Baker are:

- *The risk factors and prevention of heart disease and stroke*
- *Coronary disease, heart attack and sudden coronary death*
- *Heart failure*

The Baker Heart Research Institute is funded from a diverse range of government and private sources. We remain grateful for the continuing support of the corporate sector, trusts, foundations and individual donations.

CONTENTS

ABOUT THE BAKER	4
PRESIDENT'S REPORT	5
DIRECTOR'S REPORT	7
RESEARCH STRUCTURE AND OVERVIEW	10
ORGANISATIONAL CHARTS	11-12
SCIENTIFIC OVERVIEW	
HEART – CARDIOLOGY DIVISION	13
BRAIN – CARDIOVASCULAR NEUROSCIENCE DIVISION	15
VESSELS – VASCULAR DIVISION	17
ABMU & CENTRE FOR CLINICAL RESEARCH EXCELLENCE	21
BAKER INTERNATIONALLY	25
TECHNICAL PLATFORMS & SUPPORT SERVICES	27
COMMERCIALISATION	28
BOARD OF MANAGEMENT	29
STAFF	30
FOUNDATION	34
SUPPORTERS	35
SUPPORTING THE BAKER	36
FINANCIAL REPORT	37-53
WHISTLEBLOWERS POLICY	54
PUBLICATIONS	55

ABOUT THE BAKER

The Baker Heart Research Institute is one of the world's leading medical research centres. With approximately 220 staff and students, the Baker operates Australia's most comprehensive cardiovascular research program.

The importance of the work at the Baker can't be underestimated. Heart disease is Australia's number one health problem. It is responsible for 40% of all deaths, and is the leading cause of death – ahead of cancer, HIV/AIDS, lung diseases and accidents.

The Baker has an international reputation for research excellence, and is committed to making a difference.

Baker scientists have discovered some of the medical knowledge we now take for granted. This includes:

- proving that mental stress and cigarette smoking create potentially harmful stimulation of the heart nerves
- proving that exercise can lower blood pressure
- developing non-invasive methods to assess the stiffness of arteries, to more accurately predict heart problems
- discovering the plasma protein, which led to the distinction between type 1 and type 2 diabetes, enabling more appropriate treatment; and not least
- establishing open-heart surgery in Australia, in partnership with the Alfred Hospital.

More detail on our research can be found on www.baker.edu.au



PRESIDENT'S REPORT 2002



The Baker had a very busy and highly successful year in 2002. It was a year of much change but also substantial consolidation. A new Baker is emerging in its new home, stronger and more productive than before.

Early in the year Gianni Gromo, a member of the Baker's International Scientific Advisory Board and the head of discovery and research at Hoffman-LaRoche, Basel visited the Baker to assess its scientific work and direction. Gianni issued an encouraging report on the Baker's science and urged us to work harder on our internal team building, our technology transfer and our external communications.

The leadership group at the Baker under the superb command of our new Director, Professor Garry Jennings, set out on these tasks with vigor.

By the end of 2002 many steps along the recommended path had been taken. The scientific

structure of the Baker was reconfigured under two key divisions, one directed towards coronary disease & vascular biology and the other to heart failure & molecular cardiology.

We also managed to attract Professor Mark Cooper's internationally renowned group of researchers to work at the Baker. Mark's team are best known for their excellent work in juvenile diabetes, which has significant links with heart disease. Thanks to the generosity of the Atherosclerosis Trust of the United Kingdom, the Wynn Department of Metabolic Cardiology was established and the Baker's own Associate Professor David Kaye was appointed as the inaugural Wynn Professor to head that department.

To further strengthen the Baker's operations, Mrs Erica Hughes joined as Chief Operating Officer in April 2002. Erica came to the Baker from the ANZ Bank where she had served as the head of

consumer banking. Erica has extensive experience in finance, marketing, change management and team building. She has quickly set about improving every aspect of the Baker's administration, adding greatly to the efficiency with which we can deliver our scientific resources and output.

In May 2002 the Alfred Medical Research and Education Precinct (which includes the Baker's new home) was officially opened by the Premier, Steve Bracks. This event was attended by the Health Minister, John Thwaites and a large number of VIP guests, friends of the Baker and the wider Baker family. Everyone expressed their enthusiasm for the Baker's new home and the important work which takes place within.

Not long after this Dr Alex Bobik was tragically caught up in the Moscow theatre siege. Alex has maintained strong scientific links with Russian cardiovascular scientists throughout his professional career. He regularly visits Russia where he has organised valuable scientific exchanges with Australia since the 1980's. We were all very relieved that Alex survived the siege, a most traumatic experience for him and his family but also for all of us who know and love him well. We are very happy that he has returned to the Baker family and is safely at home again in his lab!

Also during 2002 Clinical Trials Victoria was established with seed funding from science, technology and innovation infrastructure grants given by the Victorian Government. CTV is a joint venture between the Baker, CDCT, Melbourne Health, Neurosciences Victoria and our AMREP partners, the Alfred Hospital and Monash University. It will shortly establish a special purpose clinical trials facility in the East Tower of the AMREP precinct which will be used for the trialling of new drugs and therapies. We are very excited to have this additional resource for our clinical and scientific activities and know that it will reap great rewards for the Baker in future.

"We are very excited to have this additional resource for our clinical and scientific activities and know that it will reap great rewards for the Baker in future."

The Alfred Baker Medical Unit was again successful in attracting an award from the NHMRC as a Center of Clinical Research Excellence. These grants are especially significant to the Baker because they are directly aimed at converting medical research discoveries into actual clinical applications. The grant won by the ABMU will be used for training clinical researchers, developing further research databases and for the establishment of new research projects with practical clinical applications.

The Baker also managed to achieve 7 new project grants from NHMRC (a success rate twice the national average) and an important 5 year program grant which was awarded to Garry Jennings, Murray Esler, Tony Dart, Alex Bobik and David Kaye with a number of chief investigators all of whom are counted amongst the cream of the Baker's senior scientists. The Board is very proud of this endorsement of the Baker's medical research.

Also during the year the Baker changed its name and image slightly. The official name of the Baker remains the Baker Medical Research Institute in accordance with the terms of its governing legislation. But for marketing and external purposes we have adopted the "trade name" of the Baker Heart Research Institute and a new "double swoosh" heart logo. These changes are designed to identify the Baker's focus in the public mind directly with vital heart research. The new name and logo will offer a clearer focus and better brand recognition for our external marketing.

Along with all these activities, there were many other awards and achievements made in 2002. It is impossible in the space allotted here to mention all of the awards and achievements gained by Baker scientists, but special congratulations are due to:

- David Kaye and John Power for their achievements of NHMRC special interest grants;
- Julie Nigro for an Australian Vascular Biology Society Young Investigator Award;
- Ian Smith for leading Australia's successful bid to host the

International Peptide meeting in 2003;

- Geoff Head as an inaugural ambassador for Gippsland in recognition of his outstanding achievements as a Gippslander;
- Murray Esler who was elected a Fellow of the Australian Academy of Science and was awarded the 2002 Hartnett medal by the RSA;
- Markus Schlaich who was awarded an International Young Investigator's Award at the World Congress of Cardiology.

I want to thank especially the Baker's Director, Garry Jennings and its Chief Operating Officer, Erica Hughes for their huge efforts during the year. They are both towers of strength, with excellent long-term visions for the Baker's future. Best of all, they work very well together and with the Board. The Board is very grateful for the enormous contributions they both make to the health and well-being of the Baker and thereby to the wider community. The same is true of all the Baker's scientific and support staff who worked very hard throughout 2002 to help the Institute to settle into its new home and to remain a happy and productive environment for world class medical research.

I also thank my fellow Board members. They all gave unstintingly during 2002, particularly during my long absences in Sydney whilst I served as one of the Counsel Assisting the HIH Royal Commission. I am especially grateful to Vice-President, Gerry Johnston and the other members of the Board Executive Committee who tolerated my absences so often but nevertheless kept the Baker moving steadily forward and steering in the right direction.

Finally, hearty thanks are due to the wider Baker family of supporters, volunteers and donors. Without you the Baker would be poorer both morally and materially. Your work and generosity do make a difference to the health and well-being of others and will always be greatly appreciated.


Norman O'Bryan

DIRECTORS REPORT 2002



After one year in our new building there is a striking newness about the Baker that belies her venerable 76 years of contribution to medical research. Our mission is the same, however and put simply, it is to stop people suffering from heart disease, stroke and other vascular diseases.

"These will make eradication of heart disease, largely by prevention, a real and worthwhile aim for the present century."

Heart disease is on the increase worldwide and despite massive advances is set to become the greatest cause of death and disability, not only in the developed but also the developing world. We now have access to new knowledge and techniques, not just from the medical sciences but also mathematics, physics, computer and behavioural sciences. These will make eradication of heart disease, largely by prevention, a real and worthwhile aim for the present century.

The last year has been one of transition but a productive one scientifically. Readers interested in a comprehensive account of the scientific work of the Institute are referred to a separate companion volume to this Annual Report, the 2002 Scientific Research Report available on line at www.baker.edu.au. Scientific highlights have included the demonstration from some of our ABMU labs, particularly by Bronwyn Kingwell and Tony Dart, showing why and how stiffening of the arteries is bad for you. They have found some genetic determinants of undue stiffening as well as showing its reversibility with, amongst other things, simple lifestyle measures such as exercise and good nutrition.

A very important year of fundamental work from Wally Thomas and Ross Hannan has shown the detailed mechanism whereby angiotensin II causes the heart to grow when it is under the stress of conditions such as high blood pressure and heart failure. At present angiotensin effects in the heart, arteries, kidney and brain are blocked with ACE

inhibitors and angiotensin receptor blockers. These drugs are a keystone of modern therapy. Wally and Ross' work raises the possibility of targeting the bad things that angiotensin does in the body with blockers that leave good effects intact.

A trial coordinated at the Baker by Chris Reid on behalf of the High Blood Pressure Research Council of Australia (HBPRCA) has made a major contribution to our knowledge about how high blood pressure is best treated. Doctors choose medications from a range of options but there has been very little data to guide them as to which is the best choice to begin with. ANBP2 showed a small but significant advantage of ACE inhibitors over other forms of therapy. Although it was a massive financial and logistic effort involving 6,000 patients from all over Australia and the help of the 2,000 general practitioners, it was done much more cheaply than the massive US study, ALLHAT. Together the two studies have made quite a splash in the international scientific literature and the media. The results differed a little probably because our populations are different with a smaller proportion of African and Hispanic backgrounds in the Australian population. It does emphasise the obvious point that if we need results that are directly applicable to Australians then the research must be done here. ANBP2 is a model example of how organisations such as the Baker and HBPRCA can act as honest brokers in bringing together government, the pharmaceutical industry, the health system and the community to address a question of major importance, not only to health but also to the health budget.

"ANBP2 is a model example of how organisations such as the Baker and HBPRCA can act as honest brokers in bringing together government, the pharmaceutical industry, the health system and the community to address a question of major importance, not only to health but also to the budget."

Back in the laboratory Alex Bobik and Andrew Taylor did landmark studies that were recognised by a major award from the European Society of Cardiology. The studies provide clues as to how the common procedure of coronary artery angioplasty

and stent might be made to work better. David Kaye, Jaye Chin-Dusting and colleagues have been working on endothelial

dysfunction. This seems to be a common pathway through which all of the major common coronary risk factors like smoking, high cholesterol, high blood pressure or diabetes exert their adverse effects on arteries. They showed the endothelium (the inside lining of arteries) fails to perform its normal functions when processes that bring arginine into cells are disrupted. To do this they have had to develop new techniques of measuring arginine uptake and successfully translate their findings from isolated cells growing in culture to the healthy or diseased human circulation. This work has already proven important in people suffering from heart failure and David is working with commercial partners in testing potential new medicines that target the specific malfunction. Jaye deserves special commendation for her work in establishing the Bakers' Preclinical Testing Unit including a major new international initiative with Roche that will fund research projects and postdoctoral fellows.

Perhaps the major reason why heart disease, stroke and other vascular diseases have become rampant throughout the world in recent years has been our failure to control the increase in obesity and its metabolic consequences, particularly diabetes. Diabetes is much more than just a sugar problem and the mechanisms whereby it causes damage to the heart and arteries in the brain, kidneys and legs has become an important frontier in cardiovascular research. I was delighted, therefore, when the Board, with the support of the Baker Foundation, allowed us to bring Professor Mark Cooper and his group, who are world leaders in the study and treatment of diabetic complications affecting the kidney and arteries, to the Institute. Professor Cooper and his colleagues have a strong track record in taking clues derived from basic research into clinical

"I was delighted, therefore, when the Board, with the support of the Baker Foundation, allowed us to bring Professor Mark Cooper and his group, who are world leaders in the study and treatment of diabetic complications affecting the kidney and arteries, to the Institute."

practice, one of the things we like to think we do well at the Baker. The arrival of his group and its successful rapid integration into the Institute represents an extremely important strategic development, particularly when taken together with the establishment of the Wynn Department of Metabolic Cardiology to which I will refer below. It brings world class expertise on

diabetes and its complications together with the complementary skills in the Baker in vascular biology, physiology, molecular biology and proteomics, clinical research and trials.

Gavin Lambert, working with Murray Esler and colleagues, published a quirky but fascinating paper in The Lancet on the effect of seasons on health, particularly mood disorders. Using their unique techniques for studying activity of the sympathetic nervous system in human subjects they were able to explain some mechanisms that are probably involved.

Also out in the community many GPs in Australia have adopted a new program for risk factor prevention and control called Sentinel, developed by our Cardiovascular Diseases Prevention Unit. This program offers decision support for clinicians and the uptake and results so far suggest that it is filling a need, particularly in the secondary prevention area.

What else is new? To complement the major new strengths in diabetes research, brought about by the arrival of Professor Cooper and his group we have an exciting development in the establishment of the Wynn Domain and the Wynn Department of Metabolic Cardiology by virtue of a substantial grant from the Atherosclerosis Research Trust of the UK. David Kaye has been appointed Head of the Department after a comprehensive international search and at the time of writing is busily recruiting his new team, which will focus on metabolic aspects of cardiovascular disease. Also new has been the establishment of the Clive & Vera Ramaciotti Centre for Genomic and Proteomic Research. In the course of the year this high

throughput, robotic equipment has been commissioned by Ian Smith and numerous collaborations have started with other laboratories in the Institute and around Australia. Proteomics is an important new science following and complementary to the mapping of the human genome. This equipment is able to identify and quantitate the many proteins made by cells that are different in health and disease.

At the other end of the research spectrum the Baker has joined with Monash University and The Alfred in establishing a clinical trials centre, the Centre for Clinical Studies. This will include a new Phase I testing centre. Phase I is the very earliest stage of testing new drugs in humans. Seed money was provided by a grant from the Strategic Technology Initiative of the State Government of Victoria. The joint submission with other clinical trial networks in Melbourne establishes an umbrella group, Clinical Trials Victoria. By the end of 2003 our research precinct will have an interesting and powerful assembly of research institutes and university departments, biotechnology companies and a clinical trials facility that is probably unique in the world.

Staying alive financially is never far from the minds of an Institute Director, Chief Operating Officer and its Board and I am pleased to report good progress

on this front. As described in our previous report the National Health and Medical Research Council, our primary grant sponsor, has moved away from awarding Block Institute Grants of which we were a fortunate recipient for many years. Instead, over three years the Block Grant has become progressively contestable in the new Program and Project grant system. In this year's round we were successful with a Program Grant for Chief Investigators Alex Bobik, Tony Dart, Murray Esler, David Kaye and myself as well as a number

"By the end of 2003 our research precinct will have an interesting and powerful assembly of research institutes and university departments, biotechnology companies and a clinical trials facility that is probably unique in the world."

of other Principal Investigators. We also had a very satisfactory rate of success in the Project Grant applications, with a success rate about twice that of the national average of 23%. ABMU was successful in obtaining a renewal of its Centre of Clinical Excellence grant from the NHMRC, one of nine around the country. We are at the half way stage of the unbundling of the Block Grant, therefore we have recovered more in our NHMRC grant allocations than we have given up. Nevertheless the challenge to make ends meet will always be with us in medical research and a major objective in the coming years is to diversify not only our grant funding base but also commercialisation, trusts and foundations and general fundraising.

With so many changes going on in the internal and external environment good management will be a key success factor. We have been extraordinarily fortunate in having Erica Hughes join us as Chief Operating Officer. She brings expertise in management, finance, marketing and commercial skills at the high level of excellence we aspire to and expect in our scientific activities. She has assembled a very talented team to support the research of the Institute and established the foundation and marketing arms, the commercialisation unit and a wide range of new initiatives.

None of this can happen without the help of all our stakeholders and supporters. Our staff has responded magnificently to a time of great flux. We are grateful to granting bodies, governments, trusts, foundations, corporations and individuals who have provided the funds

that help keep us going and keep us at the advancing edge of medical research. In particular I am grateful to our Board of Management who have managed to find time in their busy lives to provide us with support, guidance and good governance.



Garry Jennings

RESEARCH STRUCTURE AND OVERVIEW

The scientific report that follows is presented in a way that reflects our evolving scientific structures, rather than a blow by blow account of the activities of over 20 individual laboratories as in the past.

The new structure involves the creation of three divisions, each with a mix of basic, molecular, physiological, translational and clinical skills with a view to being better positioned to tackle some of the major problems of contemporary cardiovascular science. The Cardiology Division, headed by David Kaye, includes our strong molecular cardiology groups and core facilities which provide large and small experimental animal models. The Vascular Division, headed by Mark Cooper includes a range of laboratories working on atherosclerosis and the prevention of diabetic complications. The division also includes strong molecular biology and clinical cardiology expertise. The largest group within the division is Mark's own Diabetes Complications group. Bronwyn Kingwell leads the Vascular Biology group which has strong links between its clinical and basic research arms. The Neuroscience Division is led by Murray Esler. This smaller group is built on the powerful clinical platform developed at the Institute in cardiovascular neurosciences with strengths in clinical biochemistry and physiology.

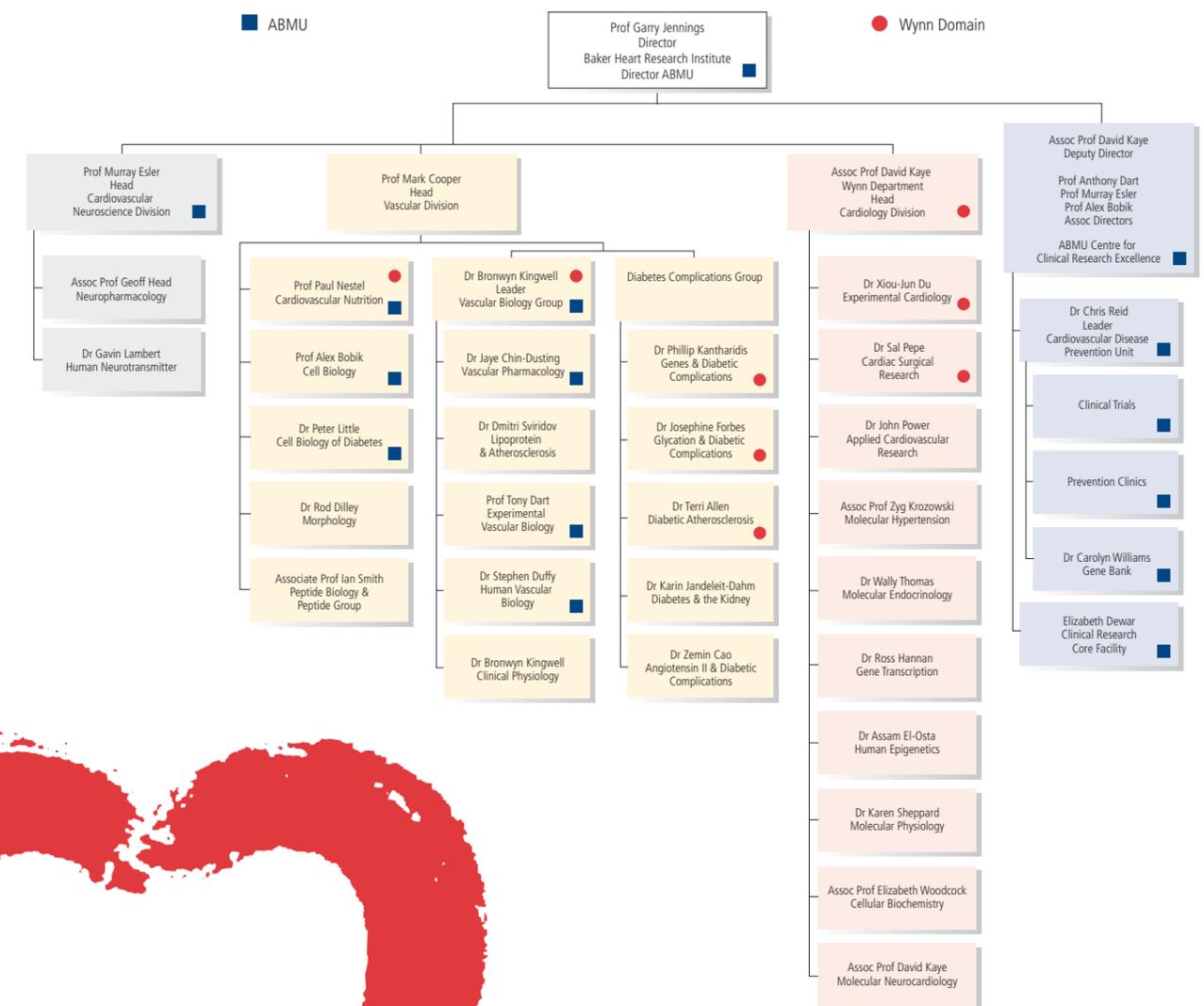
Although not strictly disease-based, each of these Divisions has a major health focus, which relates to important community needs. Heart failure is emerging as a major world problem arising in part from the general ageing of our communities.

New discoveries are needed in order for our health system to cope with the expected demand in the coming years. At the other end of the cardiovascular spectrum, the Vascular Division is addressing prevention. Not only in the more classical lines of investigation such as high blood pressure, cholesterol, nutrition and exercise but also the rising incidence of obesity and diabetes, both in Australia and most communities worldwide. The Neurosciences Division addresses the intriguing, complicated link between the nervous system and heart and vascular disease.

This structure overlays some of our pre-existing groups such as the Alfred Baker Medical Unit (ABMU), our important link between the Institute and The Alfred, particularly with Cardiovascular Medicine Services with which we are co-located in the Heart Centre. The Wynn Domain refers to groups located on the 3rd Floor of the Institute, recognising a nationally significant initiative of the Atherosclerosis Research Trust of the UK in support of the Department of Metabolic Cardiology headed by David Kaye.

The Associate Directors of the Institute, Alex Bobik, Mark Cooper, Tony Dart, Murray Esler and Ian Smith are also key figures in our organisational structure as they have Institute-wide portfolios and are responsible for maintaining cohesion and innovation across the Institute in basic science and technology (Ian Smith), clinical sciences (Tony Dart) and animal and other resources lying between the two (Alex Bobik).

SCIENTIFIC DIVISIONS & RESEARCH AFFILIATIONS

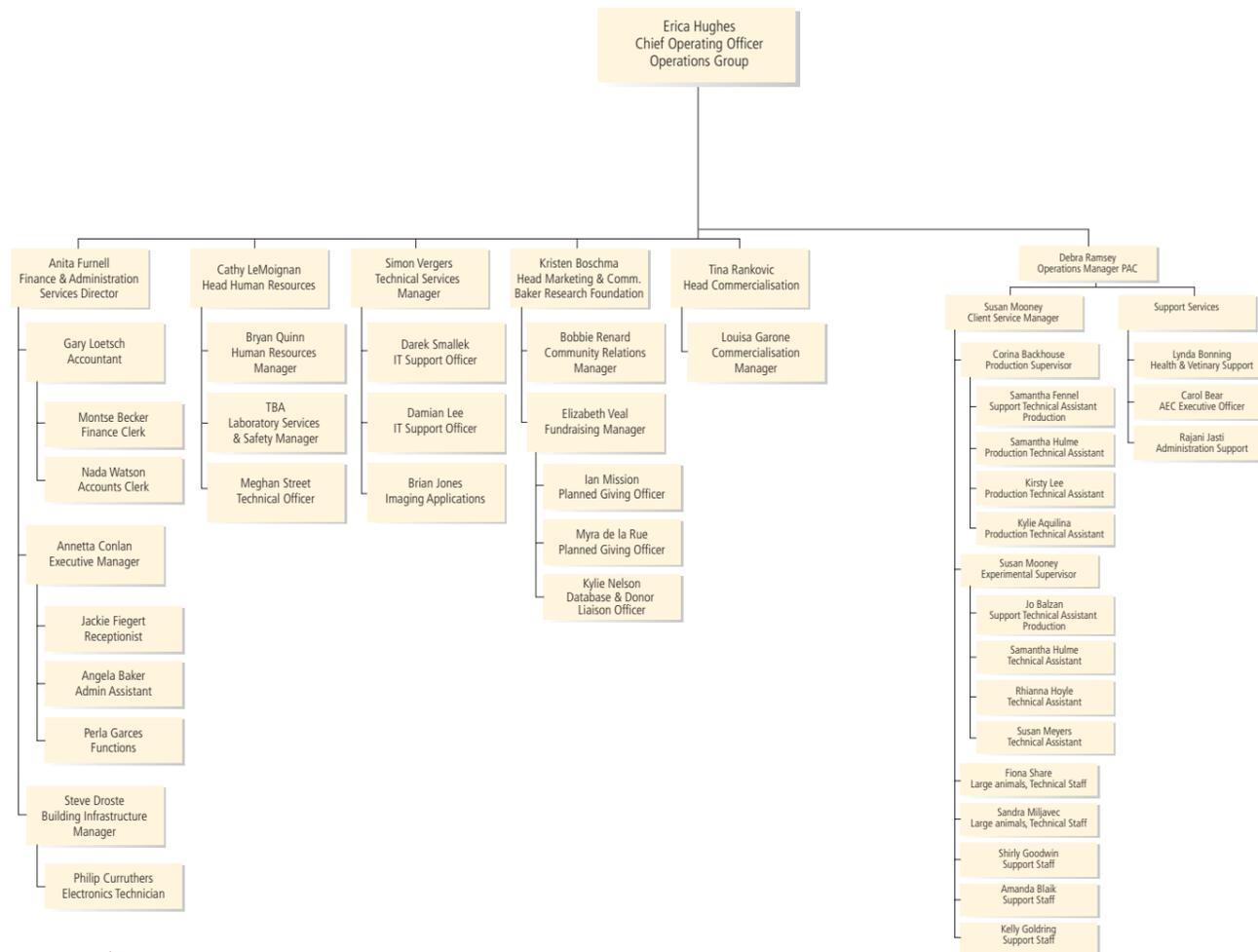


For more detailed description of the research at the Baker read the Scientific Research Report for 2002 which can be accessed at www.baker.edu.au

www.baker.edu.au

HEART

OPERATIONS GROUP STRUCTURE

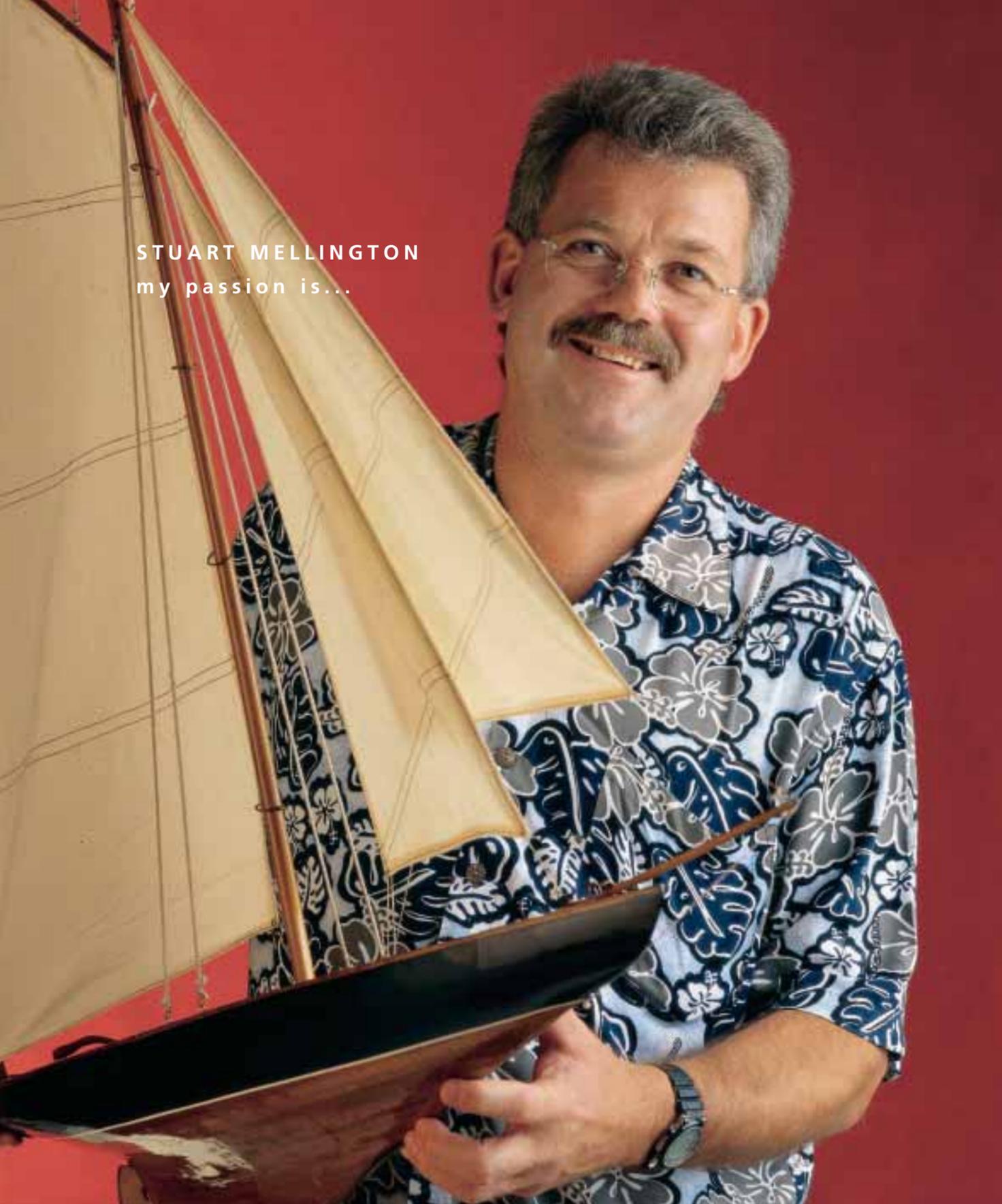


CARDIOLOGY DIVISION

The Cardiology Division focuses on key issues around the way in which the heart functions under normal and abnormal situations using a very comprehensive range of experimental methods from cells to patients awaiting heart transplant. The Cardiac Surgical Laboratory is investigating how to protect the heart during cardiac surgery and how to preserve the heart before transplantation. The molecular and biochemical groups have identified a number of novel mechanisms that determine how heart cells hypertrophy (grow) in response to certain clinically important stimuli and new signalling pathways that lead to arrhythmogenesis (irregular heart beats). The Molecular Physiology and Hypertension groups investigate the way in which steroid hormones influence the structure and function of the heart and lead to hypertension. Experimental Cardiology are using genetically engineered mouse models to study the development of heart failure. By deleting some genes or enhancing others it is hoped to discover new targets to which therapy could be directed. There is an extensive program in heart failure and its therapy in this division. The Molecular Neurocardiology Group has recently shown that a genetic defect in arginine (an amino acid) transport which is responsible for the formation of an

important blood vessel molecule known as nitric oxide (NO) is defective in hypertension. Adding dietary arginine supplements would be an easy method to prevent hypertension and an early pilot study in patients is being planned. One of the team members, Markus Schlaich won an international prize at the World Congress of Cardiology for his work and presented his findings also at the American Heart Association meeting in November 2002. The Applied Cardiovascular Research group is focussed on the development of medical devices and techniques, which can be incorporated into clinical medicine. This includes the pioneering work on a new cardiac restraint device (a heart sock) implanted around the heart to prevent progressive dilation of the heart which leads to failure. Late in 2002 we welcomed Dr Assam El-Osta to the Cardiology Division from the Peter MacCallum Cancer Institute. At the MacCallum Institute he researched the reversal of chemotherapy resistance using gene therapy and discovered key information on the genetics involved in Fragile X inherited mental retardation. Now head of the Human Epigenetics Laboratory at the Baker, Dr El-Osta will bring his expertise on human genetics to the study of cardiovascular disease.

For more detail on our research please go to our website at www.baker.edu.au



STUART MELLINGTON
my passion is...

Cruising the bay while pursuing a passion for sailing isn't a great place or time for a heart attack. Luckily, Stuart was sailing with friends from his running club, friends who knew CPR. He was dead on arrival at the Alfred Hospital. Dead five times that night. Six years on, his heart with its new stent beats strongly, still with a passion for sailing – he's competed in the Sydney to Hobart. And now he has a passion for the Baker where he is participating in cholesterol-lowering drug trials. Every year, Stuart celebrates the anniversary of his coronary with a marathon run with the friends who helped save him. Here's to life!

BRAIN

CARDIOVASCULAR NEUROSCIENCE DIVISION

The brain-cardiovascular link is the primary focus of this division. We investigate this link both in humans and in experimental animals. Our research findings affirm the general importance of psychological mechanisms and mental stress in heart disease and high blood pressure causation.

The Specialist Medical Review Council has recently concluded that stress is one proven cause of hypertension. Experimental findings on laboratory mental stress (in humans) were in close agreement with these observations in patients, lending additional support.

In our animal research program we have been exploring how the brain and sympathetic nervous system contributes to blood pressure in conditions such as hypertension and also during emotional stress. We have made a number of advances including the discovery that angiotensin, a peptide normally associated with actions in the kidney to control fluid balance and in the brain to influence thirst, has other actions in the brainstem and is critical for the blood pressure rise associated with acute emotional stress.

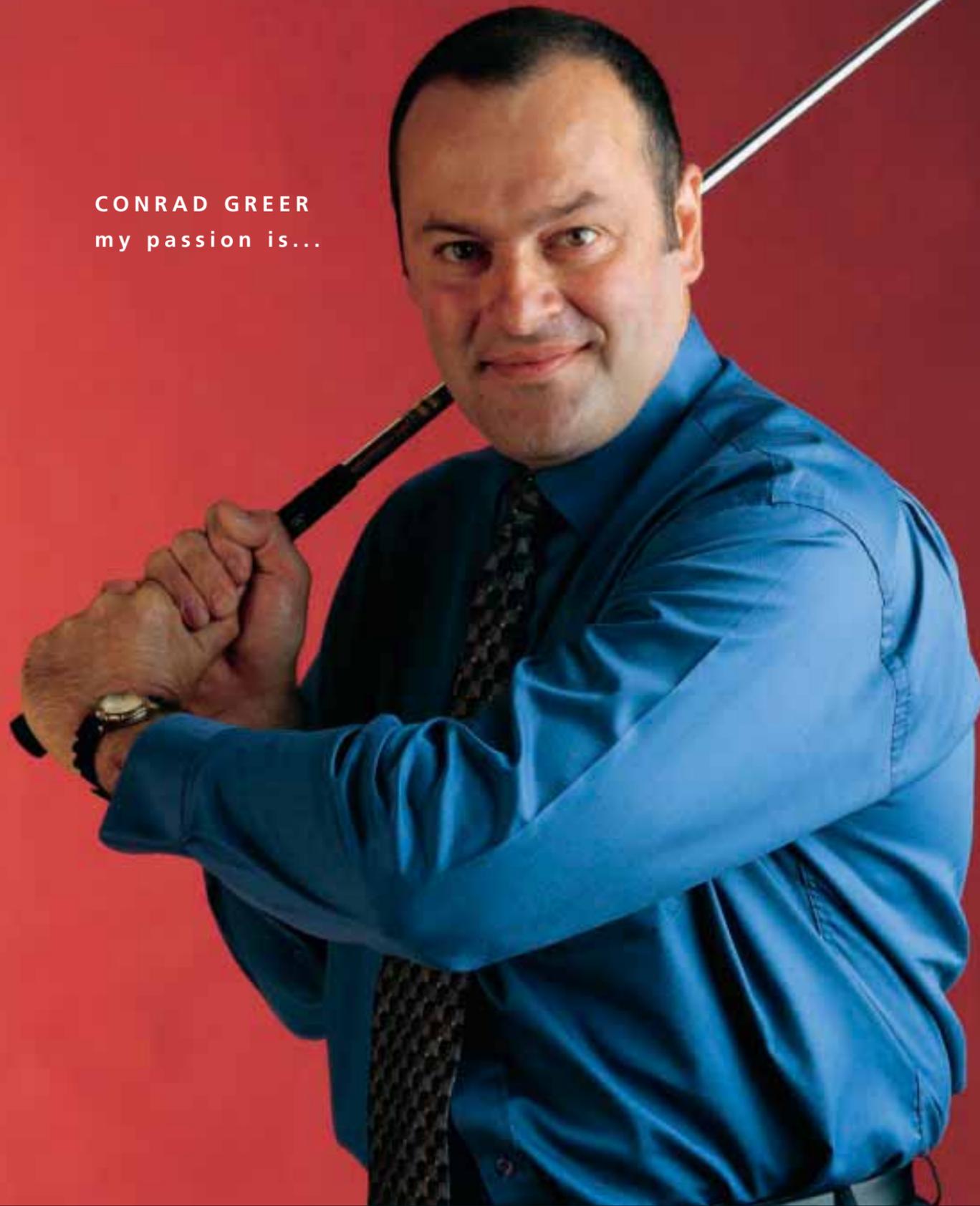
We are also actively investigating the ways in which depressive illness and panic disorder cause heart disease. Arising out of these studies, unique observations were made on a brain

chemical messenger, serotonin. Drawing on expertise in the placement of fine plastic tubes (cardiac catheters) in the blood vessels of the body, blood samples were taken from the internal jugular veins draining the brain, and measurements were made of the chemical messengers of the brain and their breakdown products (metabolites). As we have just reported in *The Lancet* and the *Journal of the American Association of Psychiatry*, there was a profound influence of the season, and sunlight, on release of serotonin in the brain (serotonin release is lowest in winter). This seasonal pattern of brain serotonin release was closely mirrored in the pattern of suicide incidence, pointing to an importance of brain serotonin in depression and suicide risk. These might seem to be surprising discoveries to be made by a heart research institute, but given our sharp research focus on the duality of mind and body, this is a natural finding for the Baker to make.

We also study how the central nervous system (CNS) controls the heart and circulation in the normal situation, and also during pathological conditions such as hypertension and heart failure. Our interests include the cardiovascular actions of neuromodulators, such as noradrenaline, serotonin and products of the renin-angiotensin system. We are also interested in the pharmacology and mechanism of action of centrally acting antihypertensive drugs.

For more detail on our research please go to our website at www.baker.edu.au

CONRAD GREER
my passion is...



A financial planner, Conrad was suddenly relocated to a bank branch adjoining the Baker. It was timely. With a family history of heart disease, including the early death of his mother, enrolling in a Baker research program for cholesterol-lowering drugs didn't require encouragement. It seemed natural. After all, he works to create futures worth living for. And to live his passion for golf. Out on the greens, the horizons stretch further. Now two of his sisters and his son also depend on the Baker. So it's almost like family.

VESSELS

VASCULAR DIVISION

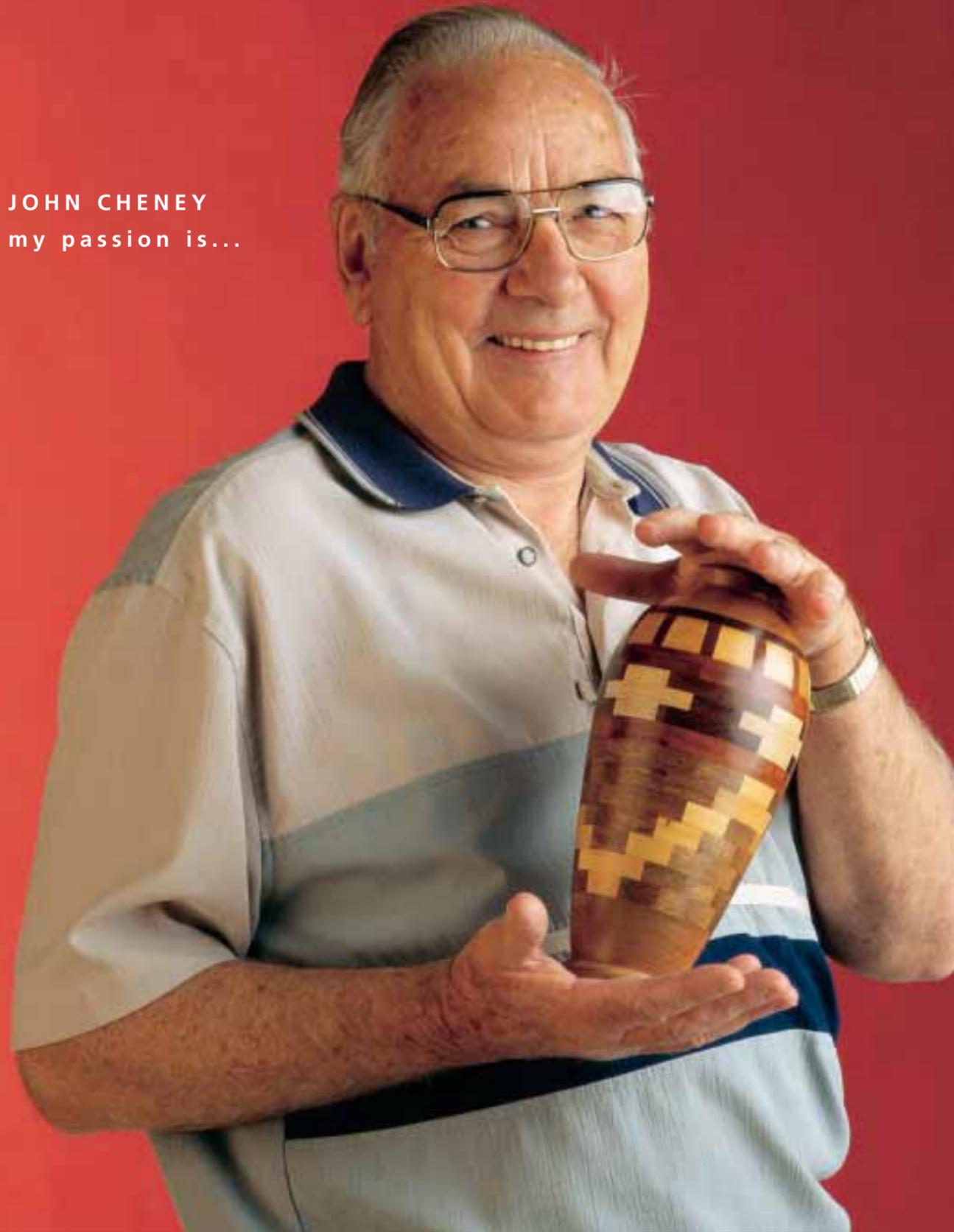
The Vascular Division is a diverse group of laboratories with three main subdivisions. All are engaged in the exploration and identification of the causes, processes, effects and new treatments of vascular disease. The Vascular Biology group undertakes research on atherosclerosis and vascular changes caused by genes, hormones, diet, exercise, ageing, high blood pressure and drugs.

Atherosclerosis is the formation of lipid fatty deposits in the vessel wall. When this occurs in the coronary vessels it leads to heart attacks; when it occurs in the brain it can lead to stroke. Deposition of cholesterol, the major component of the lipid plaques in the vessel, is a result of an imbalance between delivery of cholesterol to the vessels and removal of excess cholesterol from the vessels. Preventing the formation of cholesterol and therefore delivery of cholesterol to tissues has been the way that the group of drugs known as statins work to successfully lower cholesterol and prevent heart attacks. The Lipoprotein and Atherosclerosis laboratory is aimed at determining pathways involved in removal of cholesterol from the vessel wall and looking for ways of enhancing its removal. This would provide an alternative to statin therapy or an additional drug to treat atherosclerosis. Similarly the Cell Biology group are also trying to understand the development of the fatty lesions in blood vessels and the processes responsible for the progression of the lesions to the stage where they rupture and cause heart attack or stroke. At a clinical level, the Cardiovascular Nutrition group is focussing

on investigating nutrition and food related strategies that may contribute to cardiovascular health. Recently they showed in a large clinical study, that plant sterols which are available in foods, were safe and lowered cholesterol. They have also done a unique study in elite athletes which provides strong clues about the mechanisms through which physical activity raises blood high density lipoprotein (good cholesterol) levels. Also at a clinical level the Experimental and Human Vascular Biology group are looking at the relationship between lipids, the endothelium (vessel lining) and atherosclerosis.

The Clinical Physiology section has two major areas of interest. Diabetes is a major health problem, particularly late onset or Type II Diabetes. It is well known that exercise improves blood glucose control in diabetes and their laboratory has now unravelled the mechanism by which exercise improves glucose control. Understanding this means that one may be able to develop drugs that mimic the action of exercise. The second area that this laboratory is involved with is the compliance or stiffness of large arteries. As one ages, one's arteries get stiffer and it is now known that this is a risk factor for heart disease. In 2002 they demonstrated for the first time that this stiffness aggravates coronary artery disease leading to heart attacks. They have recently shown that there are certain genetic factors, particularly variations in genes coding for the structural components or building blocks of artery walls, that increase the risk of large artery stiffening leading to rises in systolic blood pressure and coronary artery disease.

JOHN CHENEY
my passion is...



When you volunteer for a health study you don't expect it to change your life. It was a study of Australians aged over 60. Any heart problems, the questionnaire asked. John hesitated. Was that occasional slight pain he felt after exercise worth a tick? What the hell, he ticked. A letter from the Baker: would you care to visit? An angiogram. A diagnosis. A quadruple bi-pass in the nick of time. To see the quality of his craft is to marvel: two hundred pieces of laminated wood perfectly meshed in a single vase. To hold one is to feel the very heart beat of his passion.

The third grouping in the division studies the cell biology of diabetes and its complications. This is headed by Professor Mark Cooper who recently transferred his large team of researchers from the University of Melbourne to the Baker to expand the program on diabetic complications. With the growing number of obese people and the lack of exercise, Type II diabetes which usually occurs in middle age and is not due to lack of insulin, is an important public health problem throughout the world. However most diabetics, both Type I and Type II, nowadays do not die from the metabolic abnormalities of diabetes but from cardiovascular and importantly atherosclerotic disease. The group have already published extensively on diabetic nephropathy which leads to kidney failure and devised treatment strategies to slow the progression of this diabetic renal disease. They are now focussing their skills and energies on trying to understand why diabetes leads to accelerated atherosclerosis and why 70% of diabetics die from cardiovascular disease, mainly heart attacks and strokes. For this, they have developed a new model of experimental diabetes associated atherosclerosis by inducing chemical diabetes in a genetically modified (apolipoprotein knock out) mouse that develops high cholesterol and spontaneous fatty streaks and plaques in the vessel.

The high sugar levels in diabetes leads to specific irreversible chemical reactions between the excess sugar and proteins such as haemoglobin and other structural proteins. This process is called advanced glycation. It prevents the ability of the human body to renew these proteins so that they accumulate in many

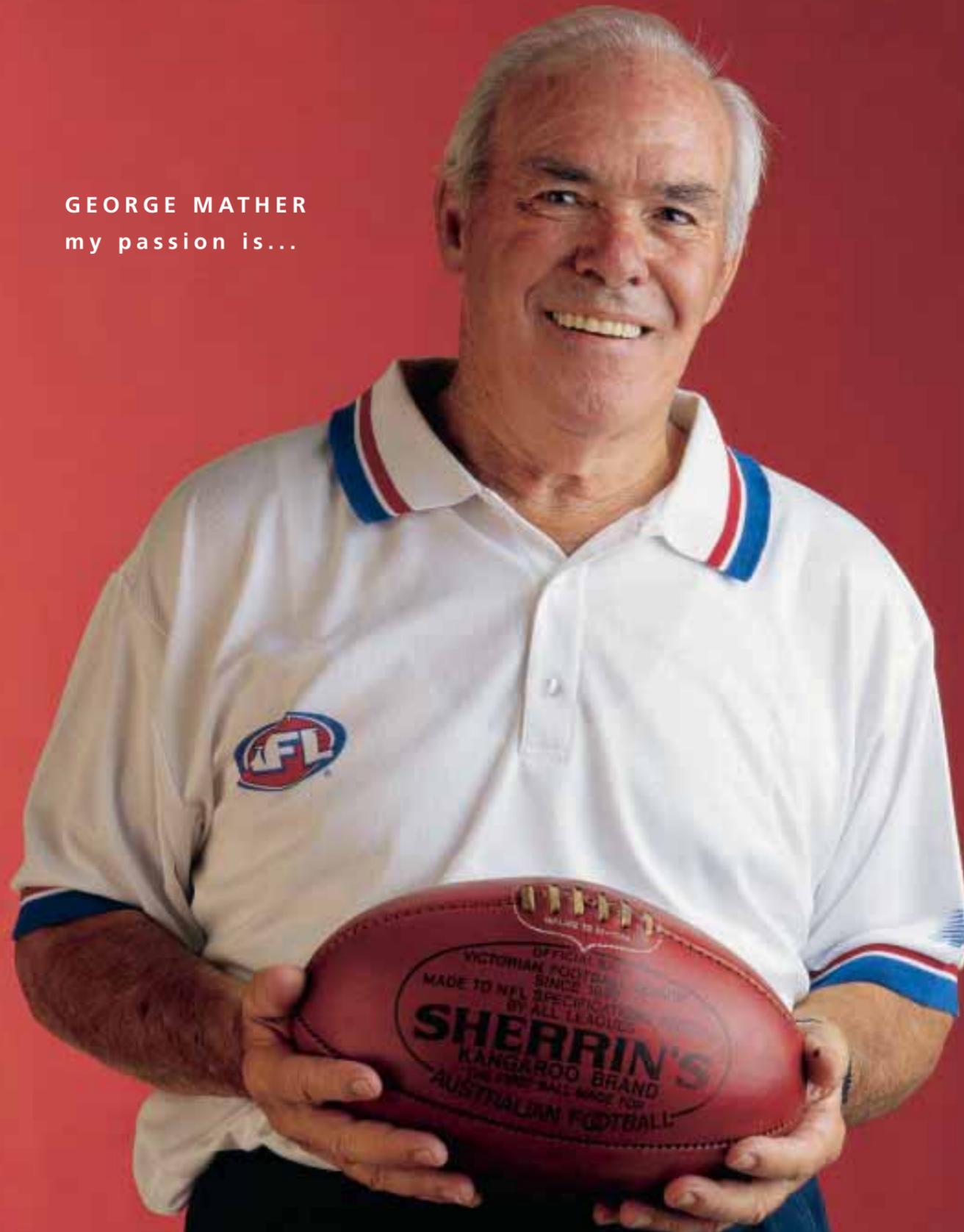
sites, causing disruption of normal tissue structure and function. Recently the Glycation and Complications group has shown that these molecules not only cause structural changes but lead to the production of oxygen-derived molecules, which are harmful. The group is now exploring ways of making these abnormal and sticky proteins dissolve.

This research into diabetes is part of a program funded by the Juvenile Diabetes Research Foundation International, the National Institutes of Health and the National Health & Medical Research Council of Australia.

An important group in the Vascular Division is the Peptide Biology Laboratory which not only undertakes research but runs the Clive and Vera Ramaciotti Proteomics and Genomics Research Facility for the Institute. Although the gene and human genome project has received the most publicity and popular press, the importance of genes is that they code for proteins, the structural elements of the body. The major aim of the Peptide Biology Laboratory is to better understand the role of peptides and proteins in the regulation of cardiovascular function. They are especially interested in enzymes (peptidases) that generate or break down peptide substances that act on the heart and blood vessel. Knowing the processes enables one to more specifically design pharmaceutical compounds that will inhibit them and lead to restoring the balance. The group has recently developed and validated a novel platform technology to generate simply and quickly, specific inhibitors of these enzymes.

For more detail on our research please go to our website at www.baker.edu.au

GEORGE MATHER
my passion is...



A footy boundary umpire for 20 years, George gave it away to enjoy retirement with his wife. They even volunteered for a health research program together. While waiting for his wife to complete her form a researcher got talking to him. Any chest pain? Well, now that you mention it. Within 3 days there was a letter from the Baker to call. One and a half blocked arteries. He's been a patient for 10 years. The pains have gone. In fact, when the AFL asked him recently to return as a umpire's trainer, he said yes. You can't run from your passion.

ALFRED BAKER MEDICAL UNIT (ABMU) AND CENTRE FOR CLINICAL RESEARCH EXCELLENCE

Centre for Clinical Research Excellence and the Alfred Baker Medical Unit (ABMU)

The ABMU is a collaborative research unit between the Baker and the Alfred that has been established for over 50 years. The unit provides a unique smooth interface between medical research and clinical research. It is the bridge between "bench top to bed side". This division conducts the preclinical and clinical trials of therapies developed in this and other Baker divisions, as well as those commissioned from outside. The newly formed and funded Centre of Clinical Research Excellence (CCRE) is a clinical research division that operates from within the ABMU.

The Baker was the first Australian World Health Organisation Collaborating Centre for Research and Training in Cardiovascular Diseases.

Cardiovascular Disease Prevention Unit

The Cardiovascular Disease Prevention Unit (CVDPU) is headed by Dr Chris Reid. This highly active centre is engaged in both domestic and overseas heart disease prevention projects.

Within the ABMU, the CVDPU runs the Gene Bank and the Risk Reduction Clinic.

The CVDPU also coordinates, conducts and analyses major statewide, national and international clinical trials:

- The ANBP2 (Australian National Blood Pressure) study was a joint venture between the Commonwealth Government, the pharmaceutical industry and the High Blood Pressure Research Council of Australia, which compared two types of treatment for high blood pressure – ACE inhibitors versus

diuretics. More than half the hypertensive patients enrolled in ANBP2 have also taken part in sub-studies, including the importance of left ventricular hypertrophy and ambulatory blood pressure monitoring in the management of hypertension. The study was successfully completed after seven years and the results have recently been published in the prestigious medical publication The New England Journal of Medicine.

- Other large clinical trials conducted, or still in progress, coordinated nationally from the Baker include three international studies, OPERA, ON TARGET and OCTAVE.
- The CVDPU has been appointed by the Australian Society of Cardiothoracic Surgeons as a Data Management and Analysis centre for a project to identify key performance indicators for cardiac surgical outcomes.

The Risk Reduction Clinic

The Risk Reduction Clinic is one way in which our expertise in reducing the risk of heart disease is made directly available to the community. The service is free of charge and is conducted by highly trained clinical nursing and technical staff.

The staff at the Risk Reduction Clinic are involved in a broad range of research studies, including collecting samples for the Alfred & Baker Gene Bank, in addition to the critical role of recruiting subjects for ABMU studies.

Recently, the Clinic has studied the genetic causes of hypertension and audited secondary prevention measures for heart attack and cardiac surgery patients. Research continues into better methods of defining risk in healthy subjects.

BOBBIE RENARD
my passion is...



Life is a journey. Once she was a nurse. Now she nurses community relations for the Baker. Fund raising and friend raising. Increasing the number of post doctoral fellowships has been a keen goal and one that makes her work a pleasure. She considers the passion of Baker scientists for their research, another joy of the job. Just don't ask Bobbie about the software. Ask Rupert, her son because he's a programmer who's going places. And her other passion.

WHO Collaborating Centre for Research and Training in Cardiovascular Disease

The Baker is a World Health Organisation (WHO) Collaborating Centre for Research and Training in Cardiovascular Disease.

The appointment by the WHO to the Baker, was the first of its kind in Australia. Currently, the Baker has two overseas projects with the WHO, one in Vietnam and one in Mongolia.

The occurrence of heart disease in these and many Asian countries has escalated in the past few years mainly due to the erosion of traditional lifestyles with the increasing pervasion of Western influences. The joint WHO and Baker projects involve assessing the prevalence of heart disease in these countries and providing medical research training. This will enable the provision of better heart disease prevention, treatment and education in these regional countries.

Alfred & Baker Gene Bank

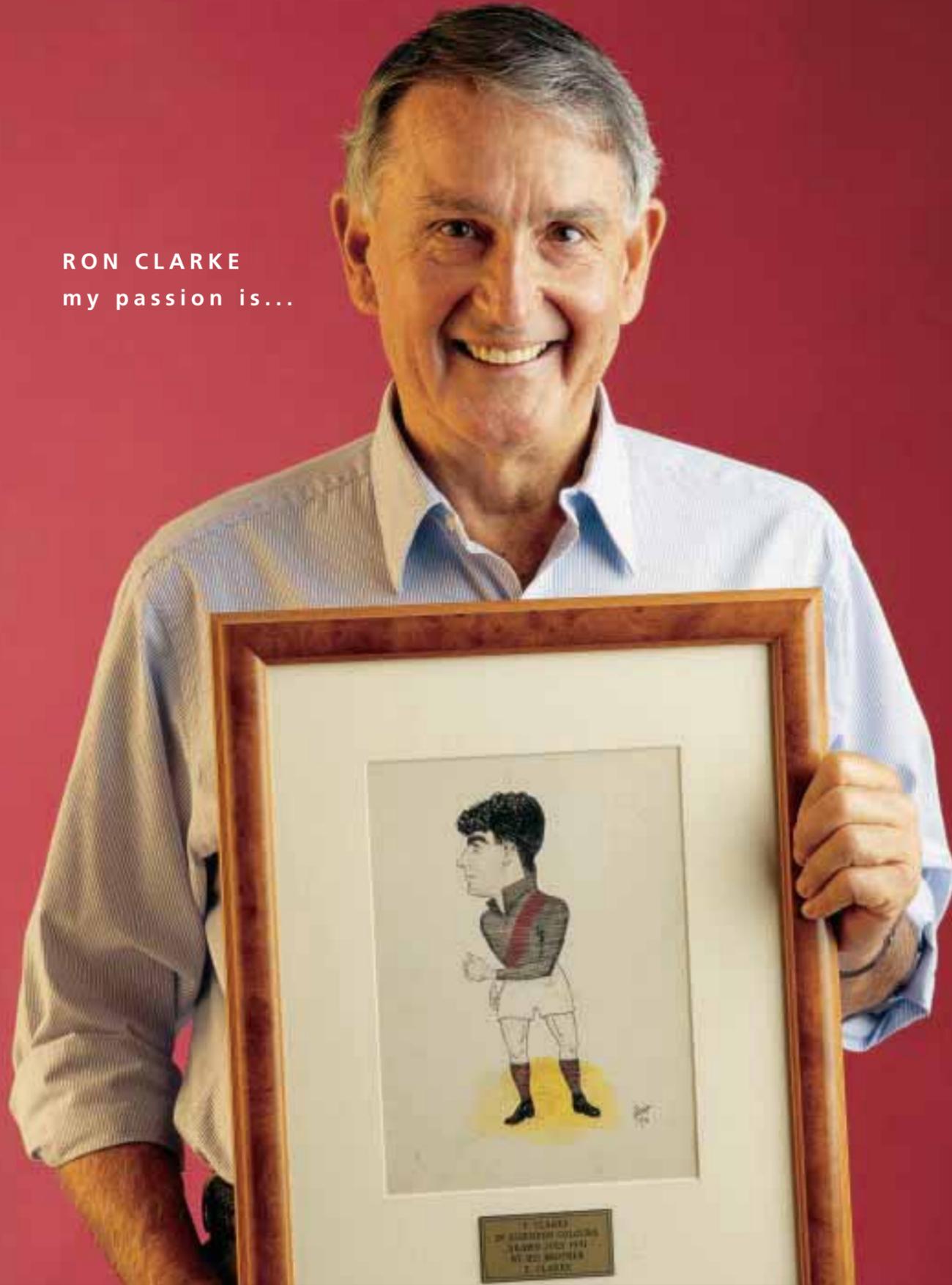
The Alfred and Baker Gene Bank is an important research initiative of the Baker Heart Research Institute. The aim of the Gene Bank is to collect small samples of blood or tissue in order to study the genetic determinants of cardiovascular disorders. This research may lead to important new discoveries in drug treatment and prevention of heart attack and stroke.

The Gene Bank relies on blood and tissue donations from healthy volunteer subjects in addition to people who have already had a heart attack, stroke or have high blood pressure, high cholesterol or other risk factors for cardiovascular disease. Currently over 2000 volunteers have provided samples for the Gene Bank and it is well on the way to becoming an important resource for the discovery of new ways to prevent heart disease.

If you would like further information please call:

(03) 9276 2000

RON CLARKE
my passion is...



With a father and brother who were VFL legends, sport was always a passion. Running 150 miles a week was normal. It carried him far. To the Olympics of '56, '64, and then Mexico City in '68 where, at the end of the 10,000 metres, he collapsed. In the altitude, his lungs felt like fire. But his heart, though permanently weakened, would not give out. The Baker has been there for him. It's been a marathon, including surgery to replace a valve. Olympic sprints are no longer on his agenda but the race certainly isn't over. Not by 150 miles.

BAKER INTERNATIONALLY

Medical research is an international affair and we only ever consider measuring ourselves against international benchmarks and standards. Information travels around the world very quickly and our scientists are in day to day contact with collaborators and competitors, scientific journals and publishers. Nevertheless, attendance at international meetings, visiting laboratories, exchanging scientists and negotiating contracts still demand a physical presence and a fair degree of travel. This year Baker staff members were present and actively participating in a broad range of conferences around the world. Many were asked to give key lectures. These include:

- World Congress of Cardiology. Sir Kempson Maddox lecture, Sydney
- The International Society of Hypertension, Prague
- Cardiovascular Conference Future Forum, Monte Carlo
- Asian Pacific Society of Vascular Biology and Atherosclerosis, Cebu (Philippines)
- XIth International Vascular Biology Meeting, Karuizawa, Japan
- International Diabetes Federation Western Pacific Region 5th Congress, Beijing China
- European Atherosclerosis Society 43rd Annual Scientific meeting, Salzburg, Austria
- Gordon Conference, Cardiac excitation contraction coupling mechanisms, New London, Connecticut, USA
- International Society for Heart & Lung Transplantation Scientific Meeting, Cambridge, U.K
- Scientific Conference on Advances in the Molecular and Cellular Mechanisms of Heart Failure, USA
- European Society of Cardiology, Berlin, Germany
- Angiotensin Gordon Conference, Italy
- Wellcome Trust International Fellows meeting, London

- European College of Cardiology, Oslo, Norway
- American College of Cardiology, Atlanta, USA
- American Heart Association, Scientific Sessions, Chicago, USA
- Federation American Societies of Experimental Biology (FASEB) Summer Conference, Colorado
- French Society of Hypertension, Paris, France.
- 5th International Congress of Endocrinology, Bristol, United Kingdom
- 4th International Symposium on Podocyte Biology, Niigata, Japan
- Keystone Symposia on Inflammatory Paradigms and the Vasculature II, Colorado, USA
- 3rd Annual Conference on Arteriosclerosis, Thrombosis and Vascular Biology by AHA, Salt Lake City, UT, USA.

In return many eminent scientists visited the Baker.

These included:

- Kip Guy (University of California, San Francisco)
- Frans Leenen (Ottawa Heart Institute, Canada)
- Xinheng Feng, MD (Beijing)
- Geng Hua (Columbia University New York)
- Jonas Berquist (Uppsala, Sweden)
- Elena V Lukoshkova (National Cardiology Research Center, Moscow, Russia)
- Jan-Pierre Montani (University of Fribourg, Switzerland)
- Natalia Kalinina (Cardiology Research Centre, Moscow, Russia)
- Riccardo Candido (Trieste Italy)
- Gurong Ma (China)
- Alberto Zanchetti (Milan)
- Xiao-Li Zhang (China)

Our International Scientific Advisory Board, a panel of eminent people from different disciplines around the world, have willingly provided us with assessments and advice over the years. In 2002 a member of this Board, Dr Gianni Gromo, Head of Drug Discovery at Hoffman La-Roche visited the Institute for a week. His report formed a significant contribution to the development of overall Institute strategies.

Our exchange programs continue to flourish. One of the most successful has been the exchange with Russia, coordinated by Alex Bobik on behalf of the Australian Government. This exchange has brought many collaborative projects and some uniquely talented scientists to the Institute. The exchange was associated with our most dramatic and unwelcome event of the year when Alex was held captive in a siege in a Moscow theatre. Typically he remains committed to our international exchange program.

Another program with the National Institutes of Health (NIH) of the United States continues. We had meetings with Dr Claude Lenfant, Director of the NIH on the evolution of this program, at the end of last year.

In the meantime our laboratory at the National Heart Centre in Singapore is conducting research in collaboration with local clinicians and scientists. This program has particularly involved Jaye Chin-Dusting and Bronwyn Kingwell. We hope to see it expand in future with the involvement of other laboratories.

We have had a longstanding relationship with the Peking University Institute of Cardiovascular Sciences in Beijing. In September Jaye



Picture taken by Dr Xiou-Jun Du on a recent research study visit to China.

Chin-Dusting, Ruchong Ou and Garry Jennings visited the Institute and other commercial and academic institutes in China.

By the end of 2002 we had numerous scientists from Russia, Switzerland, Italy, China, USA and Sweden working in the Institute on sabbaticals, training or exchange programs.

As a WHO collaborating centre for research and training in Cardiovascular Disease we have a commitment to improving health and health systems in developing countries. This year Chris Reid visited Mongolia to provide expertise in developing a national hypertension program. He also visited Vietnam to develop a program of cardiovascular disease monitoring.

TECHNICAL PLATFORMS & SUPPORT SERVICES

Core Facilities

The various Baker core facilities are crucial to the success of the scientific research programs running within the Institute. Access to these different facilities allows a very efficient and cost effective mechanism by which Institute scientists can quickly access essential scientific support for their research. Highlights of the year in 2002 were:

- The installation and commissioning of the new Clive & Vera Ramaciotti Proteomics and Genomics Research Facility. The actual installation required a team of special engineers from Sweden and took the best part of a week to complete. This is state-of-the-art facility, which is directed by Associate Professor Ian Smith, will be continuously upgraded.
- The second important support service that became fully operational in 2002 was the Precinct Animal Centre under the leadership of Debbie Ramsey. This is a purpose built facility for breeding and housing laboratory animals used for medical research. The environmental conditions within the PAC ensures the highest standards of animal welfare and meets the varying needs of the Baker researchers as well as those of other Precinct partners. Furthermore they enable the Baker to maintain the highest standards and meet all the regulatory requirements. The PAC offers a number of advantages. The rooms were designed for flexibility in terms of the species that can be accommodated, and also readily adapts to projects using infectious or non infectious animal models under various levels of biocontainment.

- The most recent addition to support services has been the purchase and installation of a state of the art multi-photon confocal microscope. This adds to the already extensive imaging facilities available for researchers at the Baker. These include the most up to date scientific imaging of microscopy and other technical techniques, as well as imaging facilities for reports, scientific poster displays and promotional displays.
- The newly established Ian Potter Library, which serves all the members of the AMREP partnership and combines the collection and services of the former Baker Institute library, Alfred Hospital library, Monash Medical School Library and the Burnet Institute Library was officially opened on 27 March 2002. The opening was attended by representatives from the Ian Potter Foundation and the respective AMREP partners. As Master of Ceremonies the Baker's Director, Professor Garry Jennings spoke of the importance of a strong library and information service to support biomedical research and clinical practice. The library also received a plaque honouring the contribution of the Rouse Family to the former Baker Institute Library over the last 75 years.
- The other AMREP shared facility that became available in 2002 was the Educational Centre situated between the Ian Potter Library and the new Baker building. This consists of large seminar rooms, class rooms and smaller meeting rooms. It is a flexible arrangement, which has excellent audio visual equipment available. A total of 84 Baker research seminars and other meetings were held in the Centre during the year, many involving invited international speakers.

COMMERCIALISATION

Overview

Commercialisation has been defined as “the process of transforming ideas, knowledge and inventions into greater wealth for individuals, businesses and/or society at large. The wealth comes in many forms: new products, services and business opportunities, which meet the public’s needs, as well as possible benefits for research institutions.”¹

The Baker Heart Research Institute leverages its niche cardiovascular position in the market to facilitate the commercialisation process. Commercialisation activities are on the rise, and the Baker’s objective is to further increase the current level of commercialisation activity.

Commercialisation Outcomes 2002

The Baker Heart Research Institute has identified four key types of commercial revenue: intellectual property commercialisation, contract research services (including pre-clinical and clinical research), industry funded research projects and commercialisation grants, which provide pre-seed funding to demonstrate “proof of concept” for initial research.

In 2002, contract research services contributed 70% of commercial revenue, while industry funded research contributed 23% of revenue. The remaining 7% of revenue was derived from miscellaneous sources.

Key Milestones 2002

- Pre-Clinical contract research services formally launched by Sir Gustav Nossal
- Associate Professor David Kaye and Mimotopes Pty Ltd awarded a Biotechnology Innovation Fund (BIF) grant from AusIndustry to continue their collaborative research into the function of blood vessels
- Board approval given to establish a dedicated Commercialisation Unit
- Head of Commercialisation (Tina Rankovic) and Commercialisation Manager (Louisa Garone) appointed
- Commercialisation business model developed and approved

Intellectual Property

The Baker Heart Research Institute has a small, but growing patent portfolio. In the future, our aim is to achieve best practice in commercialisation, particularly with respect to the development of our intellectual property. The existing patent portfolio will be proactively managed to capitalise on its latent potential. Proposed enhancements of the intellectual property policy and researcher education are designed to increase the number of invention disclosures. Higher rates of disclosures will inevitably lead to a higher rate of patents filed and licences executed. Further developments of patents with commercial partners may then result in returns on research investment. Key performance indicators to measure and monitor commercialisation outcomes will be systematically implemented.

Future Directions 2003

In 2003, strong emphasis will be placed on the further expansion of commercial activities. This will be achieved by developing the Baker’s intellectual property, introducing new product portfolios and entering new segments, in addition to increasing existing services. Direct indicators of success will include revenue growth, creation of new jobs and a contribution to the overall economic development of Australia.

BOARD OF MANAGEMENT



From left:

Mr Norman O’Byrne – SC BA, LLB, BCL (President), *Mrs Carol Schwartz*, *Mr Alan Stockdale* – The Hon BA, LLB
Professor Garry Jennings – MD, MBBS, FRCP, FRACP, *Professor Nicholas Saunders* – MD, FRACP, FRCP, FAIM
Dr Gerard P Johnston – BSc, PhD (Vice President), *Dr Peter G Habersberger* – AM RFD, MBBS, FRACP, *Professor Daine Alcorn* BSc (Hons), Ph
Mr Rob Stewart – LLB (Hons) BComs, MBA (Harv), *Mr Philip Munz* – LLB (Hons)
Absent: *Professor Richard Smallwood* – AO MD, FRACP, FRCP, FACP(Hon), *Dr Michael Walsh* MBBS (Hons), BHA, FRACMA, MPA (Harv)
Other Board Members: (by invitation) *Erica Hughes* Chief Operating Officer
(by invitation) *Anita Furnell* Finance & Administration Services Director

STAFF

SCIENTIFIC STAFF

CARDIOLOGY DIVISION

HEAD

David M. Kaye MBBS, PhD (Monash) FRACP, FACC

Cardiac Surgical Laboratory

HEAD

Salvatore Pepe PhD(Adelaide), BScHons(Flinders), PGrad Dip Health Counsel(Sth.Aust)

CLINICAL HEAD

Franklin L. Rosenfeldt MBBS, MD (Adelaide), FRCSE, FRACS

SENIOR SCIENTIFIC

Deahne Quick BScHons (Deakin)
 Freya Sheeran BA, BScHons (Monash)
 Christine Egan DipAppScVet
 Yvonne Rowley RN
 Robyn Ascham RN

PROFESSIONAL & TECHNICAL

Donald S. Esmore MBBS (Melbourne), FRCSE, FRACS
 Bruce B. Davis MBBS (Melbourne), FRACS, FACS
 Karen Hansen
 Sandra Hayes
 Silvana Marasco MBBS, MS (Monash), FRACS
 Justin Negri MBBS (Monash), FRACS
 Takahiro Oto MD, PhD (Okayama)
 Adrian Pick MBBS, MD (Melb), FRACS
 Marc Rabinov MBBS, PhD (Monash), FRCS, FRACS
 Michael Rowland MBBS (Melbourne), FRACS
 Robert F. Salamonsen MBChB, MD (Otago), FFICANZCA
 James Anderson MSc, CClin Perf
 Robyn McEgan RN, CClin Perf
 Mark Mennen BScN, CClin Perf
 Arthur Prevolos BSc, CClin Perf
 Kate Kingsford-Smith BSc, Grad Dip Clin Res, CClin Perf

VISITING SCIENTISTS

Rachel Denver BScHons. Dept. of Medicine, Monash University, Alfred Hospital
 Lloyd Einsiedel MBBS, PhD. Infectious Diseases Unit, Alfred Hospital

Cellular Biochemistry Laboratory

HEAD

Elizabeth A. Woodcock BSc Hons (Queensland) PhD (Macquarie)

PROFESSIONAL & TECHNICAL

Jane Arthur BSc Hons PhD (Melbourne)
 James Morris BSc Hons (Bath), PhD (Cambridge)
 Bronwyn Kenney Dip BiolSc (Swinburne)

Experimental Cardiology Laboratory

HEADS

Xiou-Jun Du MBBS (Chongqing) M.Med (Xian), PhD (Edinburgh)
 Anthony M Dart BA, BMBCh Dphil (Oxford), FRACP

SENIOR SCIENTIFIC

Helen Kiriazis PhD (Monash)

PROFESSIONAL AND TECHNICAL

Elodie Hotchkin BscHons (Melbourne)
 Xiaoming Gao MBBS (Xinjiang)

VISITING SCIENTIST

Xinheng Feng MD (Beijing)

Gene Transcription Laboratory

HEAD

Ross D. Hannan BSc Hons PhD (Tasmania)

SENIOR SCIENTIFIC STAFF

Yves Brandenburger PhD (Geneva)

PROFESSIONAL & TECHNICAL

Kerith Sharkey BSc Hons 1
 Anna Jenkins BSc
 Affrica Jenkins BSc Hons 1

Molecular Endocrinology Laboratory

HEAD

Walter G. Thomas BSc Hons, PhD (Queensland)

SENIOR SCIENTIFIC

Hongwei Qian PhD (WVU)
 Diem Dinh (Peter Doherty Fellow)

PROFESSIONAL & TECHNICAL

Thao Pham
 Luisa Pipolo AssDipAppSc (Swinburne)

VISITING SCIENTISTS

James Ziogas Department of Pharmacology (University of Melbourne)

Molecular Hypertension Laboratory

HEAD

Zygmunt Krozowski PhD (Sydney)

SENIOR SCIENTIFIC

Zhonglin Chai PhD (Monash)
 Genevieve Escher PhD (Berne, Switzerland)

PROFESSIONAL AND TECHNICAL STAFF

Ling Guo PhD (Tongji, China)
 Amanda Mitchelson BSc Hons (La Trobe)
 Varuni Obeyesekere BSc Hons (Monash)
 Michelle Cinel Cert Vet Nursing, AssDipAppSci (Animal Tech)
 Carla Duarte BSc Hons (La Trobe)

VISITING SCIENTIST

Genevieve Escher PhD (Berne Switzerland)

Molecular Neurocardiology Laboratory

HEAD

David M Kaye MBBS, PhD (Monash) FRACP FACC

SENIOR SCIENTIFIC

Rebecca Ritchie BSc Hons (Adelaide) PhD
 Wei-Zheng Zhang MSc (Melbourne) PhD

PROFESSIONAL & TECHNICAL

Samara Finch BSc Hons (Melbourne)
 Sara Gruskin BSc (Murdoch), PGDipSc (WA)
 Paul Horton
 Tanneale Marshall BSc Hons (Monash)
 Belinda Smirk BSc MedSci Hons (La Trobe)
 Tonya Stokes BVSc Hons (Melbourne)
 Anh Cao BSc Hons
 Joanne Harwood

Molecular Physiology Laboratory

HEAD

Karen Sheppard BSc Hons, PhD (Monash)

TECHNICAL STAFF

Marissa Boyd DipHealthSc, DipMedSc (RMIT)

Applied Cardiovascular Research Laboratory

HEAD

John Power BVSc PhD

PROFESSIONAL & TECHNICAL STAFF

Paul Horton
 Mariko Kramer BSc
 Tonya Stokes

VISITING SCIENTISTS

Geng Hua Columbia University New York
 Ed Shapland Director of Research, Acorn Cardiovascular

Human Epigenetics Laboratory

HEAD

Assam El-Osta BSc Hons (Melbourne) PhD

CARDIOVASCULAR NEUROSCIENCE DIVISION

HEAD

Murray Esler BMedSci, MBBS (Melbourne), PhD (ANU)

Human Neurotransmitter Laboratory

HEAD

Gavin Lambert PhD (Monash)

SENIOR SCIENTIFIC

Jacqueline Hastings BSc PhD (Deakin)
 Elisabeth Lambert PhD (France)
 Markus Schlaich MD (Germany)

PROFESSIONAL & TECHNICAL

Flora Socratous BSc (LaTrobe)

VISITING SCIENTISTS

Jonas Berquist (Uppsala, Sweden)

Neuropharmacology Laboratory

HEAD

Geoffrey A. Head BSc Hons (Melbourne), PhD (Monash)

SENIOR SCIENTIFIC

Dmitry N. Mayorov BSc Hons (Moscow), PhD (Moscow)

PROFESSIONAL & TECHNICAL

Sandra L. Burke BSc Hons (Syd), MSc (Mon)
 Lara Sim BSc (Mon), Dip Anim Tech (Box Hill)
 Luisa La Greca BBiol Sci (LaTrobe)
 Robert D Matteo Bsc Hons (Melbourne), PhD (Melbourne)

VISITING SCIENTISTS

Elena V. Lukoshkova National Cardiology Research Center, Moscow, Russia
 Jan-Pierre Montani University of Fribourg, Switzerland

VASCULAR DIVISION

HEAD

Mark Cooper MBBS, FRACP, PhD (Melbourne)

Cardiovascular Nutrition Laboratory

HEAD

Paul Nestel AO MD (Sydney) FTSE FRACP FAHA

PROFESSIONAL & TECHNICAL

Lawrence Schneider MB BS (Cape Town) part-time
 Marja Cehun BEd (LaTrobe) RN
 Andriana Fassaloukis BSc Hons 1 (RMIT)

Cell Biology Laboratory

HEAD

Alexander Bobik Bpharm (Vic) MSc, PhD (Sydney)

SENIOR SCIENTIFIC

Alex Agrotis BSc Hons, PhD (Monash)

PROFESSIONAL & TECHNICAL

Peter Kanellakis BSc (Monash)
 Gina Kostolias BSc Hons (LaTrobe)
 Giovanna DiVitto BSc Hons (Melbourne)

VISITING SCIENTISTS

Natalia Kalinina Cardiology Research Centre (Moscow, Russia)
 Yukimasa Igawa Dept of Immunology & Pathology (Monash University)

Cell Biology of Diabetes Laboratory

HEAD

Peter J Little BPharm, MSc (Syd), PhD (Syd), ASIA

PROFESSIONAL & TECHNICAL

Melanie Ivey

Peptide Biology Laboratory

HEAD

Ian Smith PhD (Monash)

SCIENTIFIC

Rebecca Lew PhD (Virginia)
 Mark Lanigan BSc (Swinburne), PhD (Melbourne)

POST DOC

Mike Yarski PhD

PROFESSIONAL & TECHNICAL

Shane Reeve AssDipAppSci
 Cath Hamilton

JDF Centre for Diabetic Complications (Melbourne)

HEAD

Mark Cooper MBBS, FRACP, PhD (Melbourne)

ADMINISTRATIVE

Ms Laurel Ring

SCIENTIFIC

Terri Allen PhD (Melbourne)
 Zemin Cao MBBS, China, MD (Melbourne)
 Josephine Forbes BSc, PhD (Melbourne)
 Karin Jandeleit-Dahm MD (Aachen), PhD (Hannover)
 Phillip Kantarides BSc Hons, PhD (Melbourne)

PROFESSIONAL & TECHNICAL

Maryanne Arnstein
 Wendy Cao
 Gavin Langmaid
 David Long BSc Hons (Melbourne)
 Vicki Thallas BSc
 Chris Tikellis BSc Hons (Monash)
 Craig Smith BSc Hons (Melbourne)

VISITING SCIENTISTS

Markus Lasilla BSc, MSc, PhD (Finland)
 Riccardo Candido MBBS (Italy)
 Guorong Ma MD, Nephrology (China)
 Xiao Li Zhang MD, Endocrinology (China)

Clinical Physiology Laboratory

HEAD

Bronwyn Kingwell BScHons, PhD (Melbourne)

PROFESSIONAL & TECHNICAL

Melissa Formosa BSc (VUT)
 Brian Drew BSc Hons (Deakin University)

Vascular Pharmacology Laboratory

HEAD
Jaye Chin-Dusting BSc Hons, PhD (Monash)

SENIOR SCIENTIFIC
Ru Chong Ou MBBS MD (Kunming, China)

PROFESSIONAL & TECHNICAL SUPPORT
Ann-Maree Jefferis BSc (Melbourne)
Margaret Vincent AssDipAppSci (RMIT)
Emma Jones BSc Hons (Monash)
Jenny Starr

Lipoproteins and Atherosclerosis Laboratory

HEAD
Dmitri Sviridov PhD (Moscow)

PROFESSIONAL & TECHNICAL
Anh Hoang BSc (Melbourne)
Ying Fu MSc (LaTrobe)
Kally Theodore B.Sc (Monash)

Experimental Vascular Biology Laboratory

HEAD
Anthony Dart BA, DPhil, BMBCh (Oxford), MRCP, FRCP

SENIOR SCIENTIFIC
Elodie Percy BScHons (Melbourne)

Human Vascular Biology Laboratory

HEAD
Stephen Duffy MB, BS Hons, PhD, FRACP, MRCP

PROFESSIONAL & TECHNICAL
Jessica Ziolkowski BSc (Hons)

Morphology Laboratory

HEAD
Rodney Dilley PhD

PROFESSIONAL & TECHNICAL
Natalie Kvalheim BSc (RMIT)

ABMU CENTRE FOR CLINICAL RESEARCH EXCELLENCE

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

DEPUTY DIRECTOR
David M. Kaye MBBS, PhD (Monash) FRACP, FACC

ASSOC DIRECTORS
Anthony Dart BA, DPhil, BMBCh (Oxford) FRCP
Murray Esler BmedSci, MBBS (Melbourne) PhD (Monash)
Alexander Bobik Bpharm (Vic) MSc, PhD (Sydney)

Cardiovascular Disease Prevention Unit

HEAD
Christopher Reid BA (Qld) DipEd (Qld), MSc (WVU), PhD (Monash)

SENIOR SCIENTIFIC
Mark Nelson MB BS, MFM, FRACGP, PhD

PROFESSIONAL & TECHNICAL
Melinda Rockell BSc Hons
Louise Shiel BSc, Grad Dip App Sci, Grad Dip Ed, Grad Dip Clin Epi

Anne Bruce SRN
Debbie Hilton BPhy, MPH (Queensland)
Sloane Birrell BA, Grad Dip Health Soc Sci, MMed Sc
Claudia Retigan BA
Carol Bear
Elizabeth Artingstall BA, MA
Ann Nadonza BSc
Jesselle Vinluan

Risk Reduction Clinic

HEAD
Janis Jennings Nurse SRN

NURSES
Virginia Cable
Elizabeth Jenkins
Marijke Tress
Di Wilson

ADMINISTRATION
Amanda Coats BA (Monash)

Alfred Baker Medical Unit

SENIOR SCIENTIFIC
James Cameron BEElecHons, MengSc, MBBS (Melbourne) CPEBiomed

Stephen Duffy MD, BSc Hons, PhD, FRACP, MRCP, DipRACOG
Jane Thompson MD, MBBS (Monash)
Larrence Schneider MBBS

PROFESSIONAL & TECHNICAL
Leslie Delcourt
Sally Kay Nurse SRN, BBM (Monash)

Alfred & Baker Gene Bank

SCIENTIFIC
Carolyn Williams

PROFESSIONAL & TECHNICAL
Kevin Burke BSc (RMIT)

STUDENTS
PhD

Aggarwal, Anne
Ahimastos, Anna
Ahlens, Belinda
Arabia, Anna-Maria
Ballinger, Mandy
Berry, Karen
Burns, Wendy
Byrne, Melissa
Calkin, Anna
Chellappah, Jessica
Connelly, Nathan
Davis, Belinda
Dawood, Tye
De Dios, Stephanie
Dunlop, Felicity
Eikelis, Nina
Gifford-Garner, Jennifer
Gould, Paul
Harrison, Sharon
Huggins, Catherine
Jastrzebski, Katarzyna
Krawczynszyn, Mark
Lee, David
Lien, Hong
McCrystal, Graham
Medley, Tanya
Miller, Francis
Nair, Raj
Ngoc, Ngan Huyah
Nigro, Julie
Norman, Ursula
Onan, Done
Parnell, Melinda
Rasaratnam, Brindi
Rizkalla, Bishoy
Sheeran, Freya
Smith, Nicola
Taylor, Andrew
Thomas, Merlin
Tochon-Danguy, Nathalie
Van den Brink, Oliver

Vasilevski, Oliver
White, Tony
MD

Guo, Xiao Ming
Lee, Fiona
Lyon, William
MSc

Douglas, Gabrielle
Garner, Sarah
Pham, Tam
B Med Sci

Tan, Tze
AMS
Kang, Seah, Kwee (Ray)
Yee, Louis Teo Loon

Honours
Anthony Hadj
Boak, Lauren
Brasacchio, Daniella

Chan, Hsui-Wen
Cychil, Martha
Desiree, Anthony
Georges, Suzan
Henstridge, Darren

Ivey, Melanie
Khalil, Nadine
Laskowski, Adrian
Lister, Kerrie
Lloyd, Robyn

Markman, Phuong
Mathers, Jessica
Ngo, Khao
Olchawa, Beata
Osborne, Joanne

Raj, Tina
Rancie, Helen
Rose, Honor
Ross, Richard
Rothschild, Lauren

Ruddell, Brent
Thearle, Daniel
Thiloshini, Herath
Witlox, Kristie

DIRECTORATE, OPERATIONAL, ADMINISTRATIVE & SUPPORT STAFF

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

CHIEF OPERATING OFFICER
Erica Hughes BA, ASIA

SENIOR PRINCIPAL RESEARCH FELLOWS
Prof. Colin I Johnston AO, MBBS, (Sydney), MD (Hon) (Melb), FRACP, FAHA
Prof Paul J Nestel AO MD (Sydney) FTSE FRACP FAHA

FINANCE AND ADMINISTRATION
Anita Furnell Director, B.Comm (Melb) ACA
Gary Loetsch Accountant, BEc (Acc), CPA, DipOD
Montse Becker Finance Officer
Nada Watson Finance Officer
Vicky Wootton Executive Assistant to the Director
Annetta Conlan Executive Manager
Angela Baker Administrative Assistant, BA (Melbourne)
Jackie Fiegert Receptionist

COMMERCIALISATION
Tina Rankovic Head of Commercialisation, AssDipDiagRad, GradDipMgmt (RMIT), AIMM, AMAMI
Louisa Garone Commercialisation Manager, BSc (Hons), PgradDipMgmt

HUMAN RESOURCES
Cathy LeMoignan Head of Human Resources, BA Grad Dip (I&ER)
Bryan Quinn Human Resources Manager MNIA, MAHRI

INFORMATION SERVICES GROUP
Simon Vergers Informations Services Manager, B.Eng MBA
Damian Lee Support Officer
Darek Smallek Support Officer

IMAGING APPLICATIONS
Brian Jones Imaging Applications Manager BSc RIT

BAKER RESEARCH FOUNDATION
Kristen Boschma Head Marketing & Communication
BBus, AMFIA, AIMM
Bobbie Renard Manager Community Relations MFIA
Elizabeth Veal Manager Fundraising MFIA
Myra De La Rue Donor Liaison & Planned Giving Officer MFIA
Ian Misson Donor Liaison & Planned Giving Officer
Geoffrey Tolson Donor Liaison & Planned Giving Officer
Kylie Nelson Database & Donor Liaison Officer

LABORATORY SERVICES
Tony Hendy Laboratory Manager/Safety Officer BAgSc (Hons) Melb
Meghan Street Media Prep / Washroom Services BSc (Hons, Microbiology)

BUILDING INFRASTRUCTURE MANAGEMENT
Steve Droste Building Infrastructure Manager BEng (Melbourne)

BIOMEDICAL ENGINEERING
Philip Carruthers-Bleasdale Electronic Engineer

SUPPLY & SUPPORT SERVICES
Beatrice Garces Technical Assistant

PRECINCT ANIMAL CENTRE
Debra Ramsey PAC Operations Manager AppSc (Animal Tech) BHIT
Susan Mooney PAC Client Service Manager & Experimental Supervisor AppSc (Animal Tech) BHIT
Kylie Aquilina Production Technical Assistant AppSc (Animal Tech) VUT

Corina Backhouse Production Supervisor AppSc (Animal Tech) BHIT
Josephine Balzan Support Technical Assistant Experimental AppSc (Animal Tech) BHIT

Samantha Fennell Support Technical Assistant Production
Rhianna Hoyle Experimental Services Technical Assistant (P/T)
Samantha Hulme Experimental Services Technical Assistant AppSc (Animal Tech) VUT

Fiona Keurentjes Technical Assistant AppSc (Animal Tech) FIT
Kirsty Lee Production Technical Assistant
Sandra Miljavec Technical Assistant AppSc (Animal Tech) BHIT
Susan Myers Experimental Services Technical Assistant (P/T) AppSci (Animal Tech) BHIT

Rajani Jasti Admin officer, MSC (Animal Sci)
Fiona Share Large animals, procedure and other services technical officer

Sandra Miljavec Large animals, procedure and other services technical officer
Kim Hauser Technical Assistant AppSc (Animal Tech) FIT
Wilfred Villareal Technical Assistant AppSc (Animal Tech) VUT

Shirley Godwin Technical Assistant BAppSc (RMIT)
Hayley Bristow Technical Assistant BAppSc (Animal Tech) VUT
Amanda Blaik Technical Assistant
Marija Mikasinovic Technical Assistant
Kelly Goldring Animal Tech AppSci, AppSci (Animal Tech) BHIT

VET
Lynda Bonning Veterinarian BVSc Hons

THE BAKER RESEARCH FOUNDATION

The Baker Research Foundation was established in 2001 to build upon the considerable community and corporate support enjoyed by the Baker Heart Research Institute. It acts as a welcome bridge between our scientists and our donors.

In addition to raising funds for Baker research through donor retention and acquisition and managing its bequest program, the Foundation works to raise the profile of the Baker through advertising, public relations and other initiatives.

A rewarding year

In the past 12 months, the Foundation achieved some key goals:

- developed and implemented a new corporate identity
- managed the official opening (in partnership with the Alfred Hospital PR team) of the new Commercial Road building
- successfully launched a scholarship program to provide funds for students to continue their studies at the Baker
- increased interest and commitment to the Baker deferred-giving donor program
- staged another of our popular and fund-raising Wine Lovers Dinners, and
- initiated an extensive planning and recruitment program to further build on our work.

We're also extremely pleased to welcome Alan Stockdale as our Foundation Chairman. His wealth of experience and energy will be a great asset in helping us going forward.

The year ahead

The Foundation has been a hive of activity with the planning of an exciting program for 2003. Its highlights are:

- working with pro-bono agencies Sugar Advertising and Publicis Mojo on mass media campaigns
- a series of seminars and events that range in topic from recent medical science discoveries to fine cooking and financial planning
- special fundraising events
- a new and refined donor club structure
- dynamic new media and public relations strategies to publicise the Baker's name and work, and
- new ways of deferred-giving for potential donors.

Once again, the Foundation team gratefully acknowledges the significant support of all our donors. Whether it is a major gift, a small donation, time as a volunteer, a pro bono agency gift, a gift in memory of a loved one or a bequest, your contributions are always immensely welcome. You can be assured they make a significant difference. With your help the vital work of the Baker could not continue.

We thank you from the bottom of our hearts, and look forward to sharing our exciting new program of events with you.

BAKER SUPPORTERS

The Institute is grateful for major contributions from:

Anti-Cancer Council
Australian Rotary Health Research Foundation
Baker Foundation
Juvenile Diabetes Research Foundation
National Heart Foundation
National Health & Medical Research Council
National Institute of Health
The Welcome Trust
Victorian Government

Baker Research Foundation (Founding Members)

GSA Group Pty Ltd
Gurry AO, Mr William P
Kodak (Australasia) Pty Ltd
O'Bryan, Mr Norman J
Ross, Mrs Margaret S

Major Donors

Mr G I Anderson
Mr L M Berkowitz
Mr William D Bowness
Mr Edward Cook
Mr Stephen J Cook
Dr Richard Cranswick
Mr Leigh Devine
Mrs L C Dickson
Mrs Joan E Donaldson
Mr & Mrs A & B Edwards
Ms P M Holmes
Mr A P Kelly
Mr A S Leslie
Mr Robert Lyng
Mr & Mrs R & P Metzke
Miller Foundation
Mr & Mrs Lynton E Morgan
Dame Elisabeth Murdoch AC DBE
Miss Loris N Peggie
Mr Simon Price
Mr & Mrs E Rabinowicz
Mr L Ian Roach AO
Mr & Mrs B B S & R Robertson
Mrs Margaret S Ross AM
Mrs Alison H Rowland
Mr Rob Stewart
Mr J Thompson
Mrs P Wellington

Trusts & Foundations

Bayside Health
Feilman Foundation
H & L Hecht Trust
Joe White Bequest
Marion & E H Flack Trust
Ramaciotti Foundations for Biomedical Research
William Buckland Foundation

Endowments

Baker Foundation
Bell Charitable Fund
E.E.E. Stewart Charitable Trust
Estate Kenneth W Hesse
Estate Lindsay J Baldy
George F Little Settlement
Grace & Herbert Foulkes Charitable Trust
Hazel & Pip Appel Fund
James & Elsie Borrowman Trust
M A & V L Perry Foundation
Thomas, Annie & Doris Burgess
William Buckland Research Fund

Scholarships

John T Reid Charitable Trusts – Post Doctoral Fellowship
Noel Dickson Scholarship Fund
Peter & Ilse Arnhold Memorial Scholarship
Ray Shrimpton Memorial Travel Award

Robbie Eisner Scholarships
Ruth Webster Scholarship
Sheila Duke Memorial Scholarship
The Bertalli Family Scholarship
The Cybec Foundation Scholarship

Baker Gold Club

Mr E V Carroll
Mr Stephen J Cook
Mr K Eisner
Mr E L Garner AM
Miss H D Glascodine
Mr Richard P Harbig
Mr Frank A Roberts
Mr & Mrs B B S & R Robertson
Mr & Mrs E & J Ross
Mrs Pauline Row
Mr John Shalit

Baker Silver Club

Mr Geoff Bade
Mr Martin P Bade
Mr L M Berkowitz
Miss Mavis Bowskill
Mrs Alison Bult
Mrs L C Dickson
Mrs J Ferrarin
Mr M Alex Garfield
Mrs J E Grimwade
Emerit Prof P I Korner AO
Mr & Mrs S & M Marks
Mr D I McCullough
Mr Ronald G Pitcher
Lady Reid
Mrs Denzil Smith
Mrs C Y Sullivan
Mr Peter Swindells OAM
Ms Jennifer Tatchell
Mr H E Vivian
Mr & Mrs A C Weber

Baker Bronze Club

Mr Alan K Abbey
Mr & Mrs B C Allison
Mr & Mrs S E & M L Barker
Miss Paula N Barry
Mr & Mrs D L Birch
Mr & Mrs I G Bird
Mrs L Bitterfeld
Mr James M Bland
Mrs Gwendoline Bowman
Miss J Bromley
Mr John W Brown
Mrs B L Butcher
Mr P F Canobio
Mrs D Carter
Mrs L G Cheary
Mr & Mrs J R Cheney
Miss Pamela R Christensen
Mr N S Cohen
Miss Verna A Cook
Dame Joyce Daws
Mr & Mrs E A & M P Dodd
Mr Raymond S Downey
Miss J D Duffield
Mrs Heather Eather
Mrs R A Edwards
Mrs J W Engelbert
Mrs M I Euhus
Mrs K L Fairweather
Mr Greg J Farmer
Mr & Mrs J & B Filgate
Mrs F M Findlay
Mr Keith A Forbes
Mr John R Franklin
Mrs Maya Friberg
Dr J M Gardiner
Mr Vincent M Gawne
Mrs J M Gibson
Mrs Joan P Gillespie
Mr & Mrs J & K Godfrey
Dr James S Guest AM

Mr A G Hammet
Mrs L K Hancock
Dr John K Harcourt OAM
Mr R J Harden
Mr & Mrs L & Y Harrison
Dr C S Haughton
Mr & Mrs F & S Hawkins
Mrs Ida L Hicks
Dr A David Hore
Mr Robert Hudson
Miss Nada Hunter
Mr K Johnston
Dr & Mrs F C Jones
Miss G Jones
Dr Victor Kalfif
Mr R D Kerr
Mr John W Leslie
Dr & Mrs J C Lill
Mr & Mrs M W Ling
Mrs Clarissa A Linton-Smith
Mrs Margery Little
Miss Joy A Macdonald
Mr Howard Macmillan
Mrs Phyllis L Maggs
Miss M Marriott
Mr C Leon Martin
Mr & Mrs W A & T A Matthews
Miss R O McIntyre
Mr Neil S McLaren
Miss Jean W McNaughton
Mrs G J McPhee
Mr Donald Michell
Mr Robert G Miller
Mr W M Miller
Mr D Bruce Moore
Mr Frederick Moore
Mr F R D Morgan CBE
Mrs Gweny Mueller
Mr & Mrs N & M Myers
Miss E A Nihill
Miss V H Notley
Mr E P Oldham
Mrs Carmel Opray
Mr G J Paruit
Ms Diana Peatt
Mr & Mrs R I Pender
Mr W J Pollock AM
Mr & Mrs A & R Proudlock
Mrs Joan Ray
Mr E G Reid
Mr & Mrs N & G Reid
Ms Bobbie Renard
Mrs J A Repper
Mrs Patricia Robertson
Mr William M Rooney
Mrs Valda Rowland
Mr Peter W Ryall
Mr & Mrs J B Ryan
Mr Keith J Scott
Miss Leila M Skewes
Mr & Mrs I H & B Y Smith
Dr W J Smith
Mr Warren R Smith
Mr & Mrs George Smorgon
Mr G C Snell
Mr & Mrs C J & E D Soutar
Mr Philip Spry-Bailey
Mr Harold F Stevens RFM
Mrs Edna J Stock
Mr Brian R Talbot
Mr & Mrs R & J Taylor
Miss J L Thompson
Mrs Stella Thomson
Mr & Mrs Ken & Sue Trezise
Miss J A Turnbull
Mr J L Vuillemain
Mrs J E Watkins
Dr W G Wicks
Mrs G E Williams
Mr Siew C Wong
Mr Kenneth Woolfe

Event Sponsors and Donors

Majella Wines
Plunkett's Winery
Michelton Wines
Casella Wines
R L Buller & Son
Cleveland Winery
Dominique Portet Wines
Milawa Cheese Company
Brian Chalmers-Leask
Lofty Connections P/L
Pink Lady Chocolates

Bequests

Estate Donald Stanley Blair
Estate E M Gaborit
Estate D A Galbraith
Estate Walter Henry Ironside
Estate of Violet M Lowe
Estate George Pearson McKaige
Estate Dorothy Stanley-Low
Estate Isobel Ivy Thomas
Estate John Donald Wilson

Volunteers

Robert & Jan Ashe
Denise Bailey
Paula N Barry
George & Betty Bird
Ida Bourke
Ken Bracher
Frances Brown
Patricia Brown
Robyn Brown
Elaine Callow
Bob Clemmens
Bev Cohen
Margot Dixon
David & Audrey Doig
Alex Eberbach
Laurie & Sandra Feldman
Alan & Flora Fellows
Gladys Fone
Joanne Fox
Shirley E Gilbert
Keith Gillespie
Edith Godbar
Ron Hancock
John K Harcourt OAM
Heather Heath
Lindsay & June Jenkins
Fred & Kathleen Kidd
Heather Lanyon
June Lawrence
Rosemary Lawton
Elsa Lindsay
Bill & Betty Ling
Jill Loudon
Marjorie Marris
David Maxwell
Dot McCoy
Margit Meier
Wanda Nelson
Lana Newton
Keith Nicholson
Kay Nugent
Margaret O'Brien
Patricia O'Shaughnessy
Alma Parker
Lorraine Ratcliffe
Ken Rattray
Joan Riseley
Heather Rolls
Patricia Singleton
Denzil Smith
Tess Van Staveren
Marge Watson
Dan Webb
Leonisa Wenden
Ian Wood



SUPPORTING THE BAKER



HOW YOU CAN SUPPORT

The Baker Heart Research Institute relies on non-government sources, including donations from members of the public, for a substantial part of its operating income. The Baker enjoys an international reputation for the high quality of its basic and applied research into the causes of cardiovascular disease (in particular hypertension and atherosclerosis). It is an established centre for training in medical research, providing post-graduate education, and on the job training in specialised techniques.

USE OF DONATED FUNDS

All donations are used to support the Baker's medical research program, and in particular to assist with the purchase of equipment and laboratory supplies. Donations are not directed towards administrative costs, nor are they used to support our fund raising activities.

- **ALL DONATIONS OVER \$2 ARE TAX DEDUCTIBLE**
- **WE ARE PLEASED TO DIRECT DONATIONS AS REQUESTED**
- **ALL DONATIONS ARE ACKNOWLEDGED BY LETTER**

THERE ARE MANY WAYS TO SUPPORT

Depending on the size and nature of your donation, it may be in your interest to obtain advice from your solicitor, accountant or financial adviser concerning taxation, probate and other financial matters.

- Donation (Pre tax payroll deductions available)
- Bequest & Endowments
- Gift of Assets or Property
- Trust or Named Fund
- Scholarships
- Volunteering

If you would like any further information on how you can support the Baker please contact us:

Telephone 1300 728 900

Mail Baker Heart Research Institute

PO Box 6492, St Kilda Road Central Melbourne 8008

www.baker.edu.au

BOARD MEMBERS' REPORT

FOR THE YEAR ENDED 31 DECEMBER 2002

The Board of Management presents its report together with the financial statements of the Institute for the year ended 31 December, 2002 and the audit 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 & Hon. Treasurer

Professor G L R Jennings

Professor D Alcorn

Dr P G Habersberger AM

Mr P Munz

Professor N Saunders

Professor R Smallwood AO

Mr R Stewart

Dr M Walsh

Mr W Gurry AO and Mr R E Barker were Board Members from the beginning of the financial year until their resignations on 6 March 2002 and 12 August 2002 respectively.

Mrs C Schwartz and Mr A Stockdale were appointed Board Members on 12 February 2003.

PRINCIPAL ACTIVITIES

The principal activities of the Institute are to conduct 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 \$1,907,292 (2001: deficit \$2,488,928). After allowing for the capital and specific purpose funds, the Institute's result for the year was a deficit of \$1,624,601 (2001: deficit of \$13,006,505).

REVIEW OF OPERATIONS

A review of the operations of the Institute during the year is contained in the President's and the Director's reports. The Institute's activities continued to be dedicated to medical research into the basic causes of cardiovascular disease. The Institute is a body corporate formed by 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 Environment Protection Act in respect of its operations.

INSURANCE OF OFFICERS

During the financial year, the Institute paid a premium of \$8,355 to insure the board members and certain officers 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

On 23 May 2002 the new Baker Building was officially opened by the Premier of Victoria. The new building has provided the Institute with the necessary facilities to support the achievement of its mission.

In the first half of 2003, we anticipate commencing the fit-out of our two floors in the Burnet Institute building and we expect it will be completed by the end of 2003.

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, other than salaries, 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 7th day of April 2003

Signed in accordance with a resolution of the Board of Management



Norman O'Bryan SC
President



Garry Jennings
Director

FINANCIAL REPORT

BAKER MEDICAL RESEARCH INSTITUTE

Statement of Financial Performance for the year ended 31 December 2002

	Note	2002 \$	2001 \$
Revenue from ordinary activities	3	16,721,266	28,447,013
Expenses for building works		(802,939)	(25,228,178)
Employee benefits expense		(10,940,461)	(11,488,939)
Laboratory consumables used		(2,371,329)	(1,673,071)
Depreciation and amortisation expenses	4	(1,129,122)	(902,548)
Borrowing costs expense	4	(41,067)	(27,969)
Laboratory support expenses		(1,408,001)	(1,331,447)
Other expenses from ordinary activities		(1,652,948)	(801,366)
Deficit from ordinary activities before income tax expense	4	(1,624,601)	(13,006,505)
Income tax expense	2(k)	—	—
Deficit from ordinary activities after income tax expense		(1,624,601)	(13,006,505)
Total changes in funds other than those resulting from transactions with owners as owners		(1,624,601)	(13,006,505)

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 2002

	Note	2002 \$	2001 \$
ASSETS			
Current assets			
Receivables	9	2,669,514	5,361,503
Inventories	2(h)	–	–
Prepayments		122,661	109,839
Total current assets		2,792,175	5,471,342
Non-current assets			
Investments	10	7,589,498	6,902,197
Plant & equipment	11	4,892,020	4,083,997
Total non-current assets		12,481,518	10,986,194
TOTAL ASSETS		15,273,693	16,457,536
LIABILITIES			
Current liabilities			
Interest bearing liabilities	12	718,386	1,307,833
Payables		2,561,444	1,838,234
Prepaid grants	13	34,189	91,509
Provisions	14	1,448,221	1,141,490
Total current liabilities		4,762,240	4,379,066
Non-current liabilities			
Interest bearing liabilities	15	126,879	96,720
Provisions	16	200,464	173,039
Total non-current liabilities		327,343	269,759
TOTAL LIABILITIES		5,089,583	4,648,825
NET ASSETS		10,184,110	11,808,711
FUNDS			
Accumulated funds			
Operating fund	5	(8,284,279)	(6,376,987)
Capital fund	6	18,075,377	17,887,164
Specific purpose fund	7	393,012	298,534
TOTAL FUNDS	8	10,184,110	11,808,711

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 2002

	Note	2002 \$	2001 \$
Cash flows from ordinary activities			
Receipts from granting bodies		7,718,807	6,380,274
Donations and bequests		8,154,583	6,586,615
Receipts for building works		2,357,044	10,516,036
Payments to suppliers & employees (inclusive of goods and services tax)		(15,332,528)	(39,273,196)
Dividends received		275,643	400,966
Interest received		23,269	320,311
General income		40,384	468,300
Net cash inflow from ordinary activities	19(b)	3,237,202	(14,600,694)
Cash flows from investing activities			
Payment for investment securities		(1,475,049)	(1,291,017)
Proceeds from sale of investment securities		834,592	4,159,974
Payment for property, plant & equipment		(1,965,892)	(2,080,598)
Proceeds from sale of property, plant & equipment		44,438	–
Net cash outflow from investing activities		(2,561,911)	788,359
Cash flows from financing activities			
Principal repayments under finance leases		(76,915)	(35,505)
Net cash outflow from financing activities		(76,915)	(35,505)
Net cash increase in cash held		598,376	(13,847,840)
Cash at beginning of the financial year		(1,267,762)	12,583,317
Effects of exchange rate changes on cash held in foreign currencies		(3,123)	(3,239)
Cash at the end of the financial year	19(a)	(672,509)	(1,267,762)

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

BAKER MEDICAL RESEARCH INSTITUTE

Notes to the Financial Statements 31 December 2002

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 Institute 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 Board and Urgent Issues Group Consensus Views.

Set out hereunder are the significant accounting policies adopted by the Institute in the preparation of its financial statements for the year ended 31 December 2002. These policies have been consistently applied unless otherwise indicated.

a) Basis of accounting

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.

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) 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 award.

ii) Interest revenue

Recognised when received.

iii) Investment revenue

Recognised on sale of investments.

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 agreements. The amount of grants received for specific purposes during the year but unspent at year end, is usually 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 (2-20 years)
- Furniture and fittings (5-10 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 statement of financial position. Finance leases effectively transfer from the lessor to the lessee substantially all the risks and benefits incidental to ownership of the leased property. Where assets are acquired 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 new building is written off against income during the year. Accordingly, the Institute's new building is not included as an asset in the accounts as the Institute does not own the property.

h) Inventories

During 2002, the Institute continued a shared services agreement with 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 statement of financial performance when receivable. Where the recoverable amount of securities is less than the carrying amount of the securities, the security is written down to its recoverable amount. The decrement in the carrying amount is recognised as an expense in net profit or loss in the reporting period in which the recoverable amount write-down occurs.

The recoverable amount of a security is the net amount expected to be recovered through cash inflows and outflows arising from its continued use and subsequent disposal.

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 grants and for the purchase of equipment and supplies. Foreign currency transactions are initially translated into Australian currency at the rate of exchange at the 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 when they are due for settlement. Collectibility of trade debtors is reviewed on an ongoing basis. Debts which are considered 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 ancillary costs incurred in connection with the arrangement of borrowings
- finance lease charges, and
- certain exchange differences arising from foreign currency borrowings.

q) Comparative figures

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

3 Revenue from ordinary activities	2002	2001
	\$	\$
Revenue from operating activities		
Government and Statutory Bodies	6,655,065	6,297,503
Baker Foundation	1,250,000	1,150,000
Revenue from outside the operating activities		
Fundraising, corporate & private support	7,288,424	5,847,571
Capital works campaign	333,000	12,540,080
Dividends	275,643	390,553
Interest	314,294	289,859
Proceeds from sale of non-current assets	93,290	1,457,935
General income	511,549	473,512
Total revenue	16,721,266	28,447,013
4 Operating 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	15,692	17,414
Investments	77,598	1,440,521
Foreign exchange gain	–	–
Expenses		
Borrowing costs		
Interest and finance charges paid/payable	114,859	60,235
Foreign exchange loss	3,123	3,239
	117,982	63,474
Less: Amount capitalised	(76,915)	(35,505)
Borrowing costs expensed	41,067	27,969
Depreciation - Plant and equipment	1,082,038	853,440
Amortisation - Motor vehicles under finance lease	47,084	49,108
Total depreciation and amortisation expenses	1,129,122	902,548
Write down of inventories to net realisable value	–	55,143
Employee entitlements	334,156	62,808
Rental expense relating to operating leases	207,988	254,780
5 Operating fund		
Balance at beginning of year	(6,376,987)	(3,888,059)
Deficit for year	(1,907,292)	(2,488,928)
Balance at end of year	(8,284,279)	(6,376,987)

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 2002, are carried forward.

The current fund balance is:

	2002	2001
	\$	\$
Balance at beginning of year	17,887,164	28,528,987
(Deficit) / Surplus for year	188,213	(10,641,823)
Balance at end of year	18,075,377	17,887,164

7 Specific purpose fund

The specific purpose fund comprises 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	298,534	174,288
Surplus for year	94,478	124,246
Balance at end of year	393,012	298,534

8 Fund balances

Balance at beginning of year	11,808,711	24,815,216
Surplus / (Deficit) for year		
Operating fund	(1,907,292)	(2,488,928)
Capital fund	188,213	(10,641,823)
Specific purpose fund	94,478	124,246
	(1,624,601)	(13,006,505)
Balance at end of year	10,184,110	11,808,711

9 Receivables

Trade debtors	2,669,514	5,361,503
Total receivables	2,669,514	5,361,503

10 Non-current assets – Investments

Shares and debentures (at cost)	7,733,132	6,902,197
Less: Provision for write down to recoverable amount	143,634	–
Total investments – at recoverable amount	7,589,498	6,902,197

The Institute's investments are shown at cost, with the exception of one unlisted security which has been written down to its recoverable amount. As at 31 December 2002, the market value of the Institute's non-current investments was \$8,678,036 (2001: \$8,865,007).

11 Non-current assets - Property, plant and equipment	2002	2001
	\$	\$
Plant and equipment (at cost or Board's valuation)	8,076,685	6,235,514
Less: Accumulated depreciation	(3,340,471)	(2,258,433)
	4,736,214	3,977,081
Motor vehicles under finance leases	254,489	200,262
Less: Accumulated amortisation	(98,683)	(93,346)
	155,806	106,916
Total plant and equipment	4,892,020	4,083,997

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 2002	6,235,514	200,262	6,435,776
Additions at cost	1,841,171	124,721	1,965,892
Disposals	–	(70,493)	(70,493)
Balance at 31 December 2002	8,076,685	254,490	8,331,175
Accumulated Depreciation			
Balance at 1 January 2002	(2,258,433)	(93,346)	(2,351,779)
Depreciation expense	(1,082,038)	(47,084)	(1,129,122)
Disposals	–	41,747	41,747
Balance at 31 December 2002	(3,340,471)	(98,683)	(3,439,154)
Net Book Value			
As at 1 January 2002	3,977,081	106,916	4,083,997
As at 31 December 2002	4,736,214	155,806	4,892,020

12 Current liabilities – Interest bearing liabilities	2002	2001
	\$	\$
Bank overdraft	672,509	1,267,762
Lease liability	45,877	40,071
Total current interest bearing liabilities	718,386	1,307,833

13 Current liabilities – Prepaid grants	2002	2001
Prepaid grants	34,189	91,509

14 Current liabilities – Provisions	2002	2001
Annual leave	519,333	542,584
Long service leave	928,888	598,906
Total current provisions	1,448,221	1,141,490

15 Non-current liabilities – Interest bearing liabilities	2002	2001
	\$	\$
Lease liability	126,879	96,720
Total non-current interest bearing liabilities	126,879	96,720

16 Non-current liabilities – Provisions	2002	2001
Long service leave	200,464	173,039
Total non-current provisions	200,464	173,039

17 Lease commitments

Finance lease commitments

Finance lease commitments are payable as follows:

Not later than 1 year	57,093	50,262
Later than 1 year and not later than 5 years	145,833	112,418
Minimum lease payments	202,926	162,680
Less: Future finance charges	(30,170)	(25,889)
Recognised as a liability	172,756	136,791

Representing lease liabilities:

Current	45,877	40,071
Non-current	126,879	96,720
	172,756	136,791

Operating lease commitments

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

Not later than 1 year	60,759	81,819
Later than 1 year and not later than 5 years	15,014	172,961
	75,773	254,780

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 2002 are:

Mr N O'Bryan SC

Dr G P Johnston

Mr R E Barker (resigned 12/8/02)

Professor G Jennings

Professor D Alcorn

Mr W P Gurry (resigned 6/3/02)

Dr P G Habersberger

Mr P Munz

Professor N Saunders

Professor R Smallwood AO

Mr R Stewart

Dr M Walsh

b) 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, other than salaries, 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 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. This facility has been extended for short periods of time throughout the year to meet short term funding requirements.

Cash at the beginning of the financial year as shown in the statement of cash flows is reconciled to the related items in the statement of financial position as follows:

	2002	2001
	\$	\$
Interest bearing liabilities	(672,509)	(1,267,762)
Cash and term deposits	–	–
Total	(672,509)	(1,267,762)
b) Reconciliation of operating deficit after income tax to net cash from ordinary activities		
Operating (deficit) / surplus from ordinary activities	(1,624,601)	(13,006,505)
Effects of exchange rate changes on cash held in foreign currencies	3,123	3,239
Depreciation and amortisation	1,129,122	902,548
Write-down of investments to recoverable amount	143,634	–
(Profit) on sale of non-current assets	(93,290)	(1,457,934)
Changes in net assets and liabilities		
(Increase) / decrease in debtors	2,691,989	(2,824,507)
Decrease in inventories	–	55,143
(Increase) / decrease in prepayments	(12,821)	167,183
Decrease in accrued interest	–	30,451
Increase in creditors	723,211	1,447,423
Increase / (decrease) in prepaid grants	(57,321)	91,509
(Decrease) / increase in provisions	334,156	(9,244)
Net cash inflow from ordinary activities	3,237,202	(14,600,694)

(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 \$254,489 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 2 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 statement of financial position, 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 10 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 2002	Variable Interest Rate	Less than 1 Year \$	1 to 5 Years \$	More than 5 Years \$	Non - Interest Bearing	Total
Financial assets						
Receivables	–	–	–	–	2,669,514	2,669,514
Investments	–	–	–	–	7,589,498	7,589,498
Total financial assets	–	–	–	–	10,259,012	10,259,012
Weighted average interest rate	–	–	–	–		
Financial liabilities						
Bank overdraft	672,509	–	–	–	–	672,509
Payables	–	–	–	–	2,514,135	2,514,135
Lease liabilities	–	45,877	126,879	–	–	172,756
Security deposits	–	–	–	–	47,309	47,309
Total financial liabilities	672,509	45,877	126,879	–	2,561,444	3,406,709
Weighted average interest rate	12.24%	19.65%	13.00%	–		
Net financial assets / (liabilities)	672,509	(45,877)	(126,879)	–	7,697,568	6,852,303
31 December 2001						
Financial assets						
Receivables	–	–	–	–	5,361,503	5,361,503
Investments	–	–	–	–	6,902,197	6,902,197
Total financial assets	–	–	–	–	12,263,700	12,263,700
Weighted average interest rate	–	–	–	–		
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
Security deposits	–	–	–	–	–	–
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)	(1,267,762)	(40,071)	(96,720)	–	10,425,466	9,020,913

21 Capital commitments

As at 31 December 2002, capital expenditure contracted for, in respect of completion of the building, at balance date but not provided for in the accounts of the Institute:

	2002	2001
	\$	\$
Not later than 1 year	–	209,834
Total capital commitments	–	209,834

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 Remuneration of auditors

Amounts received or due and receivable by the auditors of the Institute for:

– Audit of the financial report	20,000	17,700
---------------------------------	--------	--------

24 Segment information

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

25 Reconciliation of net financial assets / (liabilities) to net assets

	Note		
Net financial assets	20	6,852,303	9,020,913
Non-financial assets and liabilities:			
Other assets		122,661	109,839
Property, plant and equipment	11	4,892,020	4,083,997
Other liabilities	13	(34,189)	(91,509)
Provisions	14, 16	(1,648,685)	(1,314,529)
Net assets per statement of financial position		10,184,110	11,808,711

BAKER MEDICAL RESEARCH INSTITUTE

Board Members' Declaration

The Board Members declare that the financial statements and notes set out on pages 39 to 50:

- comply with Accounting Standards and other mandatory professional reporting requirements; and
- give a true and fair view of the Institute's financial position as at 31 December 2002 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:

- the financial statements and notes are in accordance with accounting standards and other mandatory professional reporting requirements; and
- 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
Melbourne, 7 April 2003



Garry Jennings
Director



INDEPENDENT AUDIT REPORT

to the Members of the Baker Medical Research Institute

QUALIFICATION

As stated in note 2(g) to the accounts, the Baker Medical Research Institute (The Institute) has written off to expense certain capital expenditures incurred on the new building, which is being partially occupied by The Institute and the remainder leased to other parties. This is a departure from Accounting Standard AAS4: '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 \$802,939 incurred in the current year (2001 – \$25,228,178) should have been recognised initially as capital works in progress and then transferred to property, plant and equipment on completion. Had this been done, non-current assets would be \$58,258,236 (2001 – \$55,959,973), total assets would be \$61,050,411 (2001 – \$61,431,315), deficit after income tax would be \$821,662 (2001 – surplus of \$12,221,673), and accumulated funds would be \$55,960,828 (2001 – 56,782,690). Furthermore, had the building been capitalised the depreciation charge would have commenced in June 2002 at a rate of 2% per annum resulting in depreciation charge for the year of approximately \$458,000 reducing non-current assets and increasing the deficit by \$458,000.

Had these changes been made, total non-current assets would have totalled \$57,800,236 and the deficit for the year would have been \$1,279,662. Given that the building was not complete until 2002, the depreciation adjustments would have had no impact in the prior year.

AUDIT OPINION

In our opinion, except for the effects on the financial report as set out in pages 39 to 50 of the matter referred to in the qualification paragraph, the financial report of the Institute:

- i) complies with Accounting Standards and other mandatory professional reporting requirements; and
- ii) gives a true and fair view of the Institute's financial position as at 31 December 2002 and of its performance for the year ended on that date.

SCOPE AND SUMMARY OF OUR ROLE

The financial statements – responsibility and content

The preparation of the financial report for the year ended 31 December 2002 is the responsibility of The Institute's Board Members. It includes the financial statements of The Institute and has been prepared for distribution to members of the Institute.

The auditor's role and work

We conducted an independent audit of the financial statements in order to express an opinion on it to members of The Institute. Our role was to conduct the audit in accordance with Australian Auditing Standards to provide reasonable assurance as to whether the financial report is free of material misstatement. Our audit did not involve an analysis of the prudence of business decisions made by the Board Members or management.

In conducting the audit, we carried out a number of procedures to assess whether in all material respects the financial statements presents fairly a view, in accordance with Accounting Standards and other mandatory professional reporting requirements in Australia, which is consistent with our understanding of The Institute's financial position, the results of its operations and its cash flows.

The procedures included:

- selecting and examining evidence, on a test basis, to support amounts and disclosures in the financial statements. This included testing, as required by auditing standards, certain internal controls, transactions and individual items. We did not examine every item of available evidence
- evaluating significant accounting estimates made by The institute in its preparation of the financial statements
- obtaining written confirmation regarding material representations made to us in connection with the audit.

Our audit opinion was formed on the basis of these procedures.

INDEPENDENCE

As auditor, we are required to be independent of the The Institute and free of interests which could be incompatible with integrity and objectivity. In respect of this engagement, we followed the independence requirements set out by The Institute of Chartered Accountants in Australia and the Auditing and Assurance Standards Board.

In addition to our audit work, we were engaged to undertake other services for The Institute. In our opinion the provision of these services has not impaired our independence.



PricewaterhouseCoopers



Paul Lewis

Partner

Melbourne, 10 April 2003

Liability is limited by the Accountant's Scheme under the Professional Standards Act 1994 (NSW)

BAKER HEART RESEARCH INSTITUTE WHISTLEBLOWERS PROTECTION POLICY

The Baker is partially government funded and therefore obliged to print this policy in accordance with the Whistleblowers Protection Act 2001.

1. Statement of support to whistleblowers

The Baker Heart Research Institute (BHRI) is committed to the aims and objectives of the Whistleblowers Protection Act 2001 (the Act). It does not tolerate improper conduct by its employees, officers or members, nor the taking of reprisals against those who come forward to disclose such conduct.

The BHRI recognises the value of transparency and accountability in its administrative and management practices, and supports the making of disclosures that reveal corrupt conduct, conduct involving a substantial mismanagement of public resources, or conduct involving a substantial risk to public health and safety or the environment.

The BHRI will take all reasonable steps to protect people who make such disclosures from any detrimental action in reprisal for making the disclosure. It will also afford natural justice to the person who is the subject of the disclosure.

2. Purpose of this policy

This policy establishes a system for reporting disclosures of improper conduct or detrimental action by BHRI or its employees. The system enables such disclosures to be made to the Protected Disclosure Coordinator (PDC) or to one of the nominated Protected Disclosure Officer (PDO). Disclosures may be made by employees or by members of the public.

This policy is designed to complement normal communication channels between supervisors and employees. Employees are encouraged to continue to raise appropriate matters at any time with their supervisors. As an alternative, employees may make a disclosure of improper conduct or detrimental action under the Act in accordance with this policy.

3. Objects of the Act

The Whistleblowers Protection Act 2001. The purpose of the Act is to encourage and facilitate the making of disclosures of improper conduct by public officers and public bodies. The Act provides protection to whistleblowers who make disclosures in accordance with the Act, and establishes a system for the matters disclosed to be investigated and rectifying action to be taken.

4. Definitions of key terms

Three key concepts in the reporting system are improper conduct, corrupt conduct and detrimental action. Definitions of these terms are set out below.

4.1 Improper conduct

A disclosure may be made about improper conduct by a public body or public official and must be serious enough to constitute, if proved, a criminal offence or reasonable grounds for dismissal. Improper conduct means:

- Conduct that is corrupt;
- A substantial mismanagement of public resources;
- Conduct involving substantial risk to public health or safety or to the environment.

4.2 Corrupt conduct

Corrupt conduct means:

- Conduct of any person (whether or not a public official) that adversely affects the honest performance of a public officer's or public body's functions;
- The performance of a public officer's functions dishonestly or with inappropriate partiality;
- Conduct of a public officer, former public officer or a public body that amounts to a breach of public trust;
- Conduct by a public officer, former public officer or a public body that amounts to the misuse of information or material acquired in the course of the performance of their official functions; or
- A conspiracy or attempt to engage in the above conduct.

4.3 Detrimental action

The Act makes it an offence for a person to take detrimental action against a person in reprisal for a protected disclosure.

Detrimental action includes:

- Action causing injury, loss or damage;
- Intimidation or harassment; and
- Discrimination, disadvantage or adverse treatment in relation to a person's employment, career, profession, trade or business, including the taking of disciplinary action.

5. The reporting system

5.1 Contact persons within the BHRI

Disclosures of improper conduct or detrimental action by BHRI or its employees, may be made to the following officers:

- The PDC – Bryan Quinn, Human Resources Manager
- The PDO – Cathy LeMoignan, Head Human Resources.

All correspondence, phone calls and emails will be referred to the PDC.

Where a person is contemplating making a disclosure and is concerned about approaching the PDC or a PDO in the workplace, he or she can request a meeting in a discreet location away from the workplace.

5.2 Alternative contact persons

A disclosure about improper conduct or detrimental action by BHRI or its employees, may also be made directly to the Ombudsman:

The Ombudsman Victoria

Level 22, 459 Collins Street

Melbourne Victoria 3000 (DX 210174)

Internet: www.ombudsman.vic.gov.au Email: ombudvic@ombudsman.vic.gov.au

Tel: 9613 6222 Toll Free: 1800 806 314

Ombudsman: Dr Barry Perry Tel: (03) 9613 6202

6. Roles and responsibilities

6.1 Employees

Employees are encouraged to report known or suspected incidences of improper conduct or detrimental action in accordance with these procedures.

All employees of the BHRI must support those who have made a legitimate disclosure and must refrain from any activity that is, or could be perceived to be, victimisation or harassment of a person who makes a disclosure and protect and maintain the confidentiality of a person they know or suspect to have made a disclosure.

6.2 Protected Disclosure Officer

PDO will:

- Be a contact point for general advice about the operation of the Act for any person wishing to make a disclosure about improper conduct or detrimental action;
- Make arrangements for a disclosure to be made privately and discreetly and, if necessary, away from the workplace;
- Receive any disclosure made orally or in writing;
- Commit to writing any disclosure made orally;
- Impartially assess the allegation and determine whether it is a disclosure made in accordance with Part 2 of the Act;
- Take all necessary steps to ensure the identity of the whistleblower and the person who is the subject of the disclosure are kept confidential; and
- Forward all disclosures and supporting evidence to the PDC.

6.3 PDC

The PDC has a central 'clearinghouse' role in the internal reporting system. He or she will:

- Receive all disclosures forwarded from the PDO;
- Receive all phone calls, emails and letters from members of the public or employees seeking to make a disclosure;
- Impartially assess each disclosure to determine whether it is a public interest disclosure;
- Refer all public interest disclosures to the Ombudsman;
- Be responsible for carrying out, or appointing an investigator to carry out, an investigation referred to the public body by the Ombudsman;
- Be responsible for overseeing and coordinating an investigation where an investigator has been appointed;
- Appoint a welfare manager to support the whistleblower and to protect him or her from any reprisals. Generally the welfare manager will be the Human Resources Manager or the Human Resources Officer.
- Advise the whistleblower of the progress of an investigation into the disclosed matter;
- Establish and manage a confidential filing system;
- Collate and publish statistics on disclosures made;
- Take all necessary steps to ensure the identity of the whistleblower and the identity of the person who is the subject of the disclosure are kept confidential; and
- Liaise with the chief executive officer of the public body.

6.4 Investigator

The investigator will be responsible for carrying out an internal investigation into a disclosure where the Ombudsman has referred a matter to the public body. An investigator may be a person from within an organisation or a consultant engaged for that purpose.

6.5 Welfare manager

The welfare manager is responsible for looking after the general welfare of the whistleblower. The welfare manager will:

- Examine the immediate welfare and protection needs of a whistleblower and seek to foster a supportive work environment;

- Advise the whistleblower of the legislative and administrative protections available
- Listen and respond to any concerns of harassment, intimidation or victimisation in reprisal for making disclosure;
- Ensure the expectations of the whistleblower are realistic.

7. Confidentiality

The BHRI will take all reasonable steps to protect the identity of the whistleblower, maintain confidentiality and ensure reprisals are not made against a whistleblower.

8. Collating and publishing statistics

A secure register to record the information required to be published in the annual report, and to generally keep account of the status of whistleblower disclosures. The register will be confidential and will not record any information that may identify the whistleblower.

9. Receiving and assessing disclosures

9.1 The PDO will assess the disclosure based on the following:

- Has the disclosure been made in accordance with Part 2 of the Act?
- Has the disclosure been made to the appropriate person?
- Does the disclosure contain the essential elements of a protected disclosure?
- Is the disclosure a public interest disclosure?

Where the PDC concludes that the disclosure amounts to a public interest disclosure, he or she will:

1. Notify the person who made the disclosure of that conclusion; and
2. Refer the disclosure to the Ombudsman for formal determination as to whether it is indeed a public interest disclosure.

Where the PDC concludes that the disclosure is not a public interest disclosure, he or she will:

1. Notify the person who made the disclosure of that conclusion; and
2. Advise that person that he or she may request the public body to refer the disclosure to the Ombudsman for a formal determination as to whether the disclosure is a public interest disclosure, and that this request must be made within 28 days of the notification.

In either case, the PDC will make the notification and the referral within 14 days of the conclusion being reached by the public body. Notification to the whistleblower is not necessary where the disclosure has been made anonymously.

10. Investigations

10.1 Introduction

Where the Ombudsman refers a protected disclosure to the BHRI for investigation, the PDC will appoint an investigator to carry out the investigation, this person will generally, be the COO.

10.2 Natural justice

The principles of natural justice will be followed in any investigation of a public interest disclosure. The principles of natural justice concern procedural fairness and ensure a fair decision is reached by an objective decision maker. Maintaining procedural fairness protects the rights of individuals and enhances public confidence in the process.

10.3 Reporting requirements

The PDC will ensure the whistleblower is kept regularly informed concerning the handling of a protected disclosure and an investigation.

The PDC will report to the Ombudsman about the progress of an investigation.

Where the Ombudsman or the whistleblower requests information about the progress of an investigation, that information will be provided within 28 days of the date of the request.

11. Action taken after an investigation

11.1 Investigator's final report

At the conclusion of the investigation, the investigator will submit a written report of his or her findings to the PDC.

11.2 Action to be taken

If the PDC is satisfied that the investigation has found that the disclosed conduct has occurred, he or she will recommend to the Director the action that must be taken to prevent the conduct from continuing or occurring in the future. The PDC may also recommend that action be taken to remedy any harm or loss arising from the conduct.

Where the investigation concludes that the disclosed conduct did not occur, the PDC will report these findings to the Ombudsman and to the whistleblower.

11. Managing the welfare of the whistleblower

12.1 Commitment to protecting whistleblowers

- The BHRI is committed to the protection of genuine whistleblowers against detrimental action taken in reprisal for the making of protected disclosures.

12.2 Keeping the whistleblower informed

The PDC will ensure the whistleblower is kept informed of action taken in relation to his or her disclosure, and the time frames that apply.

12.3 Occurrence of detrimental action

If a whistleblower reports an incident of harassment, discrimination or adverse treatment that would amount to detrimental action taken in reprisal for the making of the disclosure, the welfare manager will:

- Record details of the incident;
- Advise the whistleblower of his or her rights under the Act; and
- Advise the PDC or chief executive officer of the detrimental action.

The taking of detrimental action in reprisal for the making of a disclosure can be an offence against the Act as well as grounds for making a further disclosure. Where such detrimental action is reported, the PDC will assess the report as a new disclosure under the Act. Where the PDC is satisfied that the disclosure is a public interest disclosure, he or she will refer it to the Ombudsman. If the Ombudsman subsequently determines the matter to be a public interest disclosure, the Ombudsman may investigate the matter or refer it to another body for investigation as outlined in the Act.

12.4 Whistleblowers implicated in improper conduct

Where a person who makes a disclosure is implicated in misconduct, the BHRI will handle the disclosure and protect the whistleblower from reprisals in accordance with the Act, the Ombudsman's guidelines and these procedures. The BHRI acknowledges that the act of whistleblowing should not shield whistleblowers from the reasonable consequences flowing from any involvement in improper conduct.

13. Management of the person against whom a disclosure has been made

The BHRI recognises that employees against whom disclosures are made must also be supported during the handling and investigation of disclosures. The BHRI will take all reasonable steps to ensure the confidentiality of the person who is the subject of the disclosure during the assessment and investigation process. Where investigations do not substantiate disclosures, the fact that the investigation has been carried out, the results of the investigation, and the identity of the person who is the subject of the disclosure will remain confidential.

The BHRI will give its full support to a person who is the subject of a disclosure where the allegations contained in a disclosure are clearly wrong or unsubstantiated. If the matter has been publicly disclosed, the Director of the BHRI will consider any request by that person to issue a statement of support setting out that the allegations were clearly wrong or unsubstantiated.

14. Criminal offences

The BHRI will ensure officers appointed to handle protected disclosures and all other employees are aware of the following offences created by the Act:

1. It is an offence for a person to take detrimental action against a person in reprisal for a protected disclosure being made. The Act provides a maximum penalty of a fine of 240 penalty units (\$24,000) or two years imprisonment or both.
2. It is an offence for a person to divulge information obtained as a result of the handling or investigation of a protected disclosure without legislative authority. The Act provides a maximum penalty of 60 penalty units (\$6,000) or six months imprisonment or both.
3. It is an offence for a person to obstruct the Ombudsman in performing his responsibilities under the Act. The Act provides a maximum penalty of 240 penalty units (\$24,000) or two years imprisonment or both.
4. It is an offence for a person to knowingly provide false information under the Act with the intention that it be acted on as a disclosed matter. The Act provides a maximum penalty of 240 penalty units (\$24,000) or two years imprisonment or both.

15. Review

These procedures will be reviewed annually to ensure they meet the objectives of the Act and accord with the Ombudsman's guidelines.

16. Conclusion

Procedures regarding the conduct of investigations are available for perusal or discussion with the Human Resources Department.

PUBLICATIONS 2002

- 1 **Aggarwal A, Esler MD, Lambert GW, Hastings J, Johnston L, Kaye DM.** Norepinephrine turnover is increased in suprabulbar subcortical brain regions and is related to whole-body sympathetic activity in human heart failure. *Circulation* 2002 Mar 5; 105(9): 1031-3.
- 2 **Aggarwal A, Kaye DM.** Neurohormonal assessment of cardiac function. *Coron Artery Dis* 2002 Dec; 13(8): 415-9.
- 3 **Andrews RK, Suzuki-Inoue K, Shen Y, Tulasne D, Watson SP, Berndt MC.** Interaction of calmodulin with the cytoplasmic domain of platelet glycoprotein VI. *Blood* 2002 Jun 1; 99(11): 4219-21.
- 4 **Antioch KM, Jennings G, Botti M, Chapman R, Wulfsohn V.** Integrating cost-effectiveness evidence into clinical practice guidelines in Australia for acute myocardial infarction. *Eur J Health Economics* 2002; 3(1): 26-39.
- 5 **Boner G, Cao Z, Cooper M.** Combination antihypertensive therapy in the treatment of diabetic nephropathy. *Diabetes Technol Ther* 2002; 4(3): 313-21.
- 6 **Bonnet F, Candido R, Carey RM, Casley D, Russo LM, Osicka TM, Cooper ME, Cao Z.** Renal expression of angiotensin receptors in long term diabetes and the effects of angiotensin type 1 receptor blockade. *J Hypertens* 2002 Aug; 20(8): 1615-24.
- 7 **Bonnet F, Tikellis C, Kawachi H, Burns WC, Wookey PJ, Cao Z, Cooper ME.** Nephron expression in the post-natal developing kidney in normotensive and hypertensive rats. *Clin Exp Hypertens* 2002 Jul; 24(5): 371-81.
- 8 **Brunner-La Rocca HP, Woods RL, Kaye DM, Hastings J, Thomas CJ, Lambert E, Esler MD.** Divergent effects of ANP and BNP in acute heart failure: evidence for a putative BNP-selective receptor? *J Hypertens* 2002 Jun; 20(6): 1195-201.
- 9 **Byrne MJ, Raman JS, Alferness CA, Esler MD, Kaye DM, Power JM.** An ovine model of tachycardia-induced degenerative dilated cardiomyopathy and heart failure with prolonged onset. *J Card Fail* 2002 Apr; 8(2): 108-15.
- 10 **Calkin AC, Sudhir K, Honisett S, Williams MR, Dawood T, Komesaroff PA.** Rapid potentiation of endothelium-dependent vasodilation by estradiol in postmenopausal women is mediated via cyclooxygenase 2. *J Clin Endocrinol Metab* 2002 Nov; 87(11): 5072-5.
- 11 **Cameron JD, Gatzka CD, Kingwell BA.** Assessment of large artery function. *Coron Artery Dis* 2002 Dec; 13(8): 405-13.
- 12 **Candido R, Allen TJ.** Haemodynamics in microvascular complications in type 1 diabetes. *Diabetes Metab Res Rev* 2002 Jul-Aug; 18(4): 286-304.
- 13 **Candido R, Fabris B, Cooper ME.** Treatment for diabetic nephropathy: a review of the recent patent literature. *IDrugs: Investigational Drugs Journal* 2002; 5: 237-65.
- 14 **Candido R, Jandeleit-Dahm KA, Cao Z, Nesteroff SP, Burns WC, Twigg SM, Dilley RJ, Cooper ME, Allen TJ.** Prevention of accelerated atherosclerosis by angiotensin-converting enzyme inhibition in diabetic apolipoprotein E-deficient mice. *Circulation* 2002 July 9; 106(2): 246-53.
- 15 **Cao Y, Li H, Deb S, Liu JP.** TERT regulates cell survival independent of telomerase enzymatic activity. *Oncogene* 2002 May 9; 21(20): 3130-8.
- 16 **Cao Y, Li H, Mu FT, Ebisui O, Funder JW, Liu JP.** Telomerase activation causes vascular smooth muscle cell proliferation in genetic hypertension. *FASEB J* 2002 Jan; 16(1): 96-8.
- 17 **Cao Z, Bonnet F, Candido R, Nesteroff SP, Burns WC, Kawachi H, Shimizu F, Carey RM, de Gasparo M, Cooper ME.** Angiotensin type 2 receptor antagonism confers renal protection in a rat model of progressive renal injury. *J Am Soc Nephrol* 2002 July; 13(7):1773-87.
- 18 **Cao Z, Cox A, Bonnet F.** Increased osteopontin expression following renal ablation is attenuated by angiotensin type 1 receptor antagonism. *Exp Nephrol* 2002; 10(1): 19-25.
- 19 **Cooper ME, Mundel P, Boner G.** Role of nephron in renal disease including diabetic nephropathy. *Semin Nephrol* 2002 Sep; 22(5): 393-8.
- 20 **Dart AM, Du XJ, Kingwell BA.** Gender, sex hormones and autonomic nervous control of the cardiovascular system. *Cardiovasc Res* 2002 Feb 15; 53(3): 678-87.
- 21 **Dart AM, Martin JL, Kay S.** Association between past infection with Chlamydia pneumoniae and body mass index, low-density lipoprotein particle size and fasting insulin. *Int J Obes Relat Metab Disord* 2002 Apr; 26(4): 464-8.
- 22 **Delles C, Jacobi J, Schlaich MP, John S, Schmieder RE.** Assessment of endothelial function of the renal vasculature in human subjects. *Am J Hypertens* 2002 Jan; 15(1 Pt 1): 3-9.
- 23 **Devi R, Stewart K, Branson K, Harte M, Lew R, Perlmutter P, Smith AI, Aguilar MI.** Inhibitors of aminopeptidase P incorporating beta-amino acids. In: Peptides 2002, Proceedings of the 27th European Peptide Symposium, 2002.
- 24 **Du XJ.** Clues to understanding the role of estrogen receptors in mediating cardiovascular protection. *Cardiovasc Res* 2002 Oct; 56(1): 4-7.
- 25 **Du XJ.** Changes in adrenergic signal transduction in the failing myocardium. In: Zhang ZB, Cheng TO, Zhang CY eds. *Congestive heart failure*. 2nd ed. Scientific and Technical Documents Publishing House, Beijing, 2002: 126-32.
- 26 **Du XJ.** Molecular biology of the failing myocardium. In: Zhang ZB, Cheng TO, Zhang CY eds. *Congestive heart failure*. 2nd ed. Scientific and Technical Documents Publishing House, Beijing, 2002: 109-26.
- 27 **Du XJ, Cole TJ, Tennis N, Gao XM, Kontgen F, Kemp BE, Heierhorst J.** Impaired cardiac contractility response to hemodynamic stress in S100A1-deficient mice. *Mol Cell Biol* 2002 Apr; 22(8): 2821-9.
- 28 **Duffy SJ, Meredith IT.** Is it just the money? *Catheter Cardiovasc Interv* 2002; 55: 272-3.
- 29 **Duffy SJ, Meredith IT.** Prostanoids to not affect endothelium-dependent vasodilation in healthy humans. *Am J Cardiol* 2002 Sept 15; 90(6): 686.
- 30 **Epstein M, Cooper ME.** Diabetic nephropathy: focus on ACE inhibition and calcium channel blockade. In: *Calcium antagonists in clinical medicine*. Epstein M. ed. 3rd Ed. Philadelphia, Hanley & Belfus 2002: 383-410.
- 31 **Esler M.** Differentiation in the effects of the angiotensin II receptor blocker class on autonomic function. *J Hypertens* 2002 Jun; 20 Suppl 5: S13-9.
- 32 **Esler M, Hastings J, Lambert G, Kaye D, Jennings G, Seals DR.** The influence of aging on the human sympathetic nervous system and brain norepinephrine turnover. *Am J Physiol Regul Integr Comp Physiol* 2002 Mar; 282(3): R909-16.
- 33 **Esler M, Lambert G, Kaye D, Rumantir M, Hastings J, Seals DR.** Influence of ageing on the sympathetic nervous system and adrenal medulla at rest and during stress. *Biogerontology* 2002; 3(1-2): 45-9.
- 34 **Esmore DS, Rosenfeldt FL, Mack JA, Richardson M, Bergin P, Griffiths A.** Heterotopic heart transplantation: an attractive option in 2002. *J Heart Lung Transplant* 2002 Jan; 21(1): 93-4.
- 35 **Esmore DS, Rosenfeldt FL, Mack JA, Waters KN, Bergin P.** Long ischaemic time allografts (>6hr) further expand the transplant donor pool. *J Heart Lung Transplant* 2002 Jan; 21(1): 161-2.
- 36 **Feng S, Resendiz JC, Christodoulides N, Lu X, Arboleda D, Berndt MC, Kroll MH.** Pathological shear stress stimulates the tyrosine phosphorylation of alpha-actinin associated with the glycoprotein Ib-IX complex. *Biochemistry* 2002 Jan 29; 41(4): 1100-8.
- 37 **Ferrier KE, Muhlmann MH, Baguet JP, Cameron JD, Jennings GL, Dart AM, Kingwell BA.** Intensive cholesterol reduction lowers blood pressure and large artery stiffness in isolated systolic hypertension. *J Am Coll Cardiol* 2002 Mar 20; 39(6): 1020-5.
- 38 **Forbes JM.** Diabetes drugs of the future could help us all look and feel younger. *New Scientist*, 2002 Oct 5: 13.
- 39 **Forbes JM, Bonnet F, Russo LM, Burns WC, Cao Z, Candido R, Kawachi H, Allen TJ, Cooper ME, Jerums G, Osicka TM.** Modulation of nephron in the diabetic kidney: association with systemic hypertension and increasing albuminuria. *J Hypertens* 2002 May; 20(5): 985-92.
- 40 **Forbes JM, Cooper ME, Thallas V, Burns WC, Thomas MC, Brammar GC, Lee F, Grant SL, Burrell LA, Jerums G, Osicka TM.** Reduction of the accumulation of advanced glycation end products by ACE inhibition in experimental diabetic nephropathy. *Diabetes* 2002 Nov; 51(11): 3274-82.
- 41 **Gao GD, Du XJ.** Actions of cytokines and free radical in the failing heart. In: Zhang ZB, Cheng TO, Zhang CY eds. *Congestive heart failure*. 2nd ed. Scientific and Technical Documents Publishing House, Beijing, 2002: 132-43.
- 42 **Gao S, Johansson M, Rundqvist B, Lambert G, Jensen G, Friberg P.** Reduced spontaneous baroreceptor sensitivity in patients with renovascular hypertension. *J Hypertens* 2002 Jan; 20(1): 111-16.
- 43 **Gao XM, Dilley RJ, Samuel CS, Percy E, Fullerton MJ, Dart AM, Du XJ.** Lower risk of postinfarct rupture in mouse heart overexpressing beta2-adrenergic receptors: importance of collagen content. *J Cardiovasc Pharmacol* 2002 Oct; 40(4): 632-40.
- 44 **Goldstein DS, Robertson D, Esler M, Straus SE, Eisenhofer G.** Dysautonomias: clinical disorders of the autonomic nervous system. *Ann Intern Med* 2002 Nov 5; 137(9): 753-63.
- 45 **Goldstein DS, Holmes C, Frank SM, Dendi R, Cannon RO, Sharabi Y, Esler MD, Eisenhofer G.** Cardiac sympathetic dysautonomia in chronic orthostatic intolerance syndromes. *Circulation* 2002 Oct 29; 106(18): 2358-65.
- 46 **Gould PA, Kaye DM.** Clinical treatment regimens for chronic heart failure: a review. *Expert Opin Pharmacother* 2002 Nov; 3(11): 1569-76.
- 47 **Grassi G, Esler M.** The sympathetic nervous system in renovascular hypertension: lead actor or 'bit' player? *J Hypertens* 2002 Jun; 20(6): 1071-3.
- 48 **Hantzis V, Albiston A, Matsacos D, Wintour EM, Peers A, Koukoulas I, Myles K, Moritz K, Dodic M.** Effect of early

- glucocorticoid treatment on MR and GR in late gestation ovine kidney. *Kidney Int* 2002 Feb; 61(2): 405-13.
- 49 **Hastings JA, Wiesner G, Lambert G, Morris MJ, Head G, Esler M.** Influence of leptin on neurotransmitter overflow from the rat brain in vitro. *Regul Pept* 2002 Feb 15; 103(2-3): 67-74.
- 50 **Head GA.** Spontaneous baroreflex sensitivity: towards an ideal index of cardiovascular risk. *J Hypertens* 2002 May; 20(5): 829-31.
- 51 **Head GA, Saigusa T, Mayorov DN.** Angiotensin and baroreflex control of the circulation. *Braz J Med Biol Res* 2002 Sep; 35(9): 1047-59.
- 52 **Hilton DJ, O'Rourke PK, Welborn TA, Reid CM.** Diabetes detection in Australian general practice: a comparison of diagnostic criteria. *Med J Aust* 2002 Feb 4; 176(3): 104-7.
- 53 **Hilton DJ, Welborn TA, O'Rourke PK, Reid CM.** Forgot to fast? The importance on plasma glucose values. *Diabetes Care* 2002 Nov; 25(11): 2112.
- 54 **Holloway AC, Qian H, Pipolo L, Ziogas J, Miura S, Karnik S, Southwell BR, Lew MJ, Thomas WG.** Side-chain substitutions within angiotensin II reveal different requirements for signaling, internalization, and phosphorylation of type 1A angiotensin receptors. *Mol Pharmacol* 2002 Apr; 61(4): 768-77.
- 55 **Houlihan CA, Akdeniz A, Tsalamandris C, Cooper ME, Jerums G, Gilbert RE.** Urinary transforming growth factor-beta excretion in patients with hypertension, type 2 diabetes, and elevated albumin excretion rate: effects of angiotensin receptor blockade and sodium restriction. *Diabetes Care* 2002 Jun; 25(6): 1072-7.
- 56 **Houlihan CA, Allen TJ, Baxter AL, Panagiotopoulos S, Casley DJ, Cooper ME, Jerums G.** A low sodium diet potentiates the effects of losartan in Type 2 diabetes. *Diabetes Care* 2002 Apr; 25(4): 663-71.
- 57 **Jandeleit-Dahm K, Cooper ME.** Hypertension and diabetes. *Curr Opin Nephrol Hypertens* 2002 Mar; 11(2): 221-8.
- 58 **Janssen BJ, Lukoshkova EV, Head GA.** Sympathetic modulation of renal blood flow by rilmenidine and captopril: central vs. peripheral effects. *Am J Physiol Renal Physiol* 2002 Jan; 282(1): F113-23.
- 59 **Jennings G.** The patient with multiple risk factors. In: Manual of Hypertension. Mancia G, et al eds. Philadelphia, Churchill Livingstone Harcourt, 2002: 555-64.
- 60 **Jennings GL, Dilley RJ.** Left ventricular remodelling impacts on coronary flow reserve in hypertensive patients: is there a vascular mechanism? *J Hypertens* 2002 Jul; 20(7): 1291-3.
- 61 **Kahn DF, Duffy SJ, Tomasina D, Holbrook M, Rescorl L, Russell J, Gokce N, Loscalzo J, Vita JA.** Effects of black race on forearm resistance function. *Hypertension* 2002 Aug; 40(2): 195-201.
- 62 **Kalinina N, Agrotis A, Tararak E, Kanellakis P, Antropova Y, Ilyinskaya O, Quinn MT, Smirnov V, Bobik A.** Cytochrome b558-dependent NAD(P)H oxidase-phox units in smooth muscle and macrophages of atherosclerotic lesions. *Arterioscler Thromb Vasc Biol* 2002 Dec 1; 22(12): 2037-43.
- 63 **Kanellis J, Paizis K, Cox AJ, Stacker SA, Gilbert RE, Cooper ME, Power DA.** Renal ischemia-reperfusion increases endothelial VEGFR-2 without increasing VEGF or VEGFR-1 expression. *Kidney Int* 2002 May; 61(5): 1696-706.
- 64 **Kataoka S, Kudo A, Hirano H, Kawakami H, Kawano T, Higashihara E, Tanaka H, Delarue F, Sraer JD, Mune T, Krozowski ZS, Yan K.** 11beta-hydroxysteroid dehydrogenase type 2 is expressed in the human kidney glomerulus. *J Clin Endocrinol Metab* 2002 Feb; 87(2): 877-82.
- 65 **Kaye DM.** Alterations in oxygen consumption and sympathetic nervous activity in heart failure: independent or associated mechanisms? *Eur Heart J* 2002 May; 23(10): 764-6.
- 66 **Kaye DM, Parnell MM, Ahlers BA.** Reduced myocardial and systemic L-arginine uptake in heart failure. *Circ Res* 2002 Dec 13; 91(12): 1198-203.
- 67 **Kelly DJ, Cox AJ, Tolcos M, Cooper ME, Wilkinson-Berka JL, Gilbert RE.** Attenuation of tubular apoptosis by blockade of the renin-angiotensin system in diabetic Ren-2 rats. *Kidney Int* 2002 Jan; 61(1): 31-9.
- 68 **Kimura M, Jefferis AM, Watanabe H, Chin-Dusting J.** Insulin inhibits acetylcholine responses in rat isolated mesenteric arteries via a non-nitric oxide nonprostanoid pathway. *Hypertension* 2002 Jan; 39(1): 35-40.
- 69 **Kingwell BA.** Large artery stiffness: implications for exercise capacity and cardiovascular risk. *Clin Exp Pharmacol Physiol* 2002 Mar; 29(3): 214-7.
- 70 **Kingwell BA, Cameron JD, Dart AM.** Large artery stiffness and baroreflex function. *Circulation* 2002 Feb 26; 105(8): e56.
- 71 **Kingwell BA, Formosa M, Muhlmann M, Bradley SJ, McConell GK.** Nitric oxide synthase inhibition reduces glucose uptake during exercise in individuals with type 2 diabetes more than in control subjects. *Diabetes* 2002 Aug; 51(8): 2572-80.
- 72 **Kingwell BA, Gatzka CD.** Arterial stiffness and prediction of cardiovascular risk. *J Hypertens* 2002 Dec; 20(12): 2337-40.
- 73 **Kingwell BA, Waddell TK, Medley TL, Cameron JD, Dart AM.** Large artery stiffness predicts ischemic threshold in patients with coronary artery disease. *J Am Coll Cardiol* 2002 Aug 21; 40(4): 773-9.
- 74 **Kiriazis H, Sato Y, Kadambi VJ, Schmidt AG, Gerst MJ, Hoyt BD, Kranias EG.** Hypertrophy and functional alterations in hyperdynamic phospholamban-knockout mouse hearts under chronic aortic stenosis. *Cardiovasc Res* 2002 Feb 1; 53(2): 372-81.
- 75 **Komesaroff P.** Ethics culture and psychiatry: international perspectives. *Aust NZ J Psychiat* 2002 Feb; 36(2): 148-9.
- 76 **Komesaroff PA, Fullerton M, Esler MD, Jennings G, Sudhir K.** Oestrogen supplementation attenuates responses to psychological stress in elderly men rendered hypogonadal after treatment for prostate cancer. *Clin Endocrinol* 2002 Jun; 56(6):745-53.
- 77 **Komesaroff PA, Kafanelis B, Black C, Cable V, Sudhir K, Daly J.** Experiences at menopause of women in a non-English-speaking community: a qualitative study. *Climacteric* 2002 Mar; 5(1): 78-86.
- 78 **Komesaroff PA, Kerridge IH.** Ethical issues concerning the relationships between medical practitioners and the pharmaceutical industry. *Med J Aust* 2002 Feb 4; 176(3): 118-21.
- 79 **Lambert E, Du XJ, Percy E, Lambert G.** Cardiac response to norepinephrine and sympathetic nerve stimulation following experimental subarachnoid hemorrhage. *J Neurol Sci* 2002 Jun 15; 198(1-2): 43-50.
- 80 **Lambert EA, Thompson J, Schlaich M, Laude D, Elghozi J-L, Esler MD, Lambert GW.** Sympathetic and cardiac baroreflex function in panic disorder. *J Hypertens* 2002 Dec; 20(12): 2445-51.
- 81 **Lambert G.** Depression, the bête noire of cardiology? *Mol Psychiatry* 2002; 7(1): 17.
- 82 **Lambert G, Naredi S, Edén E, Rydenhag B, Friberg P.** Sympathetic nervous activation following subarachnoid hemorrhage: influence of intravenous clonidine. *Acta Anaesthesiol Scand* 2002 Feb; 46(2): 160-5.
- 83 **Lambert G, Naredi S, Eden E, Rydenhag B, Friberg P.** Monoamine metabolism and sympathetic nervous activation following subarachnoid haemorrhage: influence of gender and hydrocephalus. *Brain Res Bull* 2002 May; 58(1): 77-82.
- 84 **Lambert GW, Reid C, Kaye DM, Jennings GL, Esler MD.** Effect of sunlight and season on serotonin turnover in the brain. *Lancet* 2002 Dec 7; 360(9348): 1840-2.
- 85 **Laurent S, Kingwell BA, Bank A, Weber M, Struijker-Boudier H.** Clinical applications of arterial stiffness: therapeutics and pharmacology. *Am J Hypertens* 2002 May; 15(5): 453-8.
- 86 **Lew RA, Chai SY, Mustafa T, McDowell SG, Albiston AL.** Identification of the angiotensin IV receptor as insulin-regulated aminopeptidase: enzyme inhibition by AT4 ligands. In: Peptides 2002, Proceedings of the 27th European Peptide Symposium, 2002.
- 87 **Li H, Liu JP.** Signaling on telomerase: a master switch in cell aging and immortalization. *Biogerontology* 2002; 3(1-2): 107-16.
- 88 **Ling S, Dai A, Williams MR, Myles K, Dilley RJ, Komesaroff PA, Sudhir K.** Testosterone (T) enhances apoptosis-related damage in human vascular endothelial cells. *Endocrinology* 2002 Mar; 143(3): 1119-25.
- 89 **Ling S, Little PJ, Williams MR, Dai A, Hashimura K, Liu JP, Komesaroff PA, Sudhir K.** High glucose abolishes the antiproliferative effect of 17beta-estradiol in human vascular smooth muscle cells. *Am J Physiol Endocrinol Metab* 2002 Apr; 282(4): E746-51.
- 90 **Little PJ, Tannock L, Olin KL, Chait A, Wight TN.** Proteoglycans synthesized by arterial smooth muscle cells in the presence of transforming growth factor-beta1 exhibit increased binding to LDLs. *Arterioscler Thromb Vasc Biol* 2002 Jan; 22(1): 55-60.
- 91 **Lu X, Du XJ.** Compensation and decompensation of heart failure. In: Zhang ZB, Cheng TO, Zhang CY eds. Congestive heart failure. 2nd ed. Scientific and Technical Documents Publishing House, Beijing, 2002: 86-109.
- 92 **Mayorov DN, Head GA.** Ionotropic glutamate receptors in the rostral ventrolateral medulla mediate sympathetic responses to acute stress in conscious rabbits. *Auton Neurosci* 2002 Jun 28; 98(1-2): 20-3.
- 93 **McLennan SV, Kelly DJ, Cox AJ, Cao Z, Lyons JG, Yue DK, Gilbert RE.** Decreased matrix degradation in diabetic nephropathy: effects of ACE inhibition on the expression and activities of matrix metalloproteinases. *Diabetologia* 2002 Feb; 45(2): 268-75.
- 94 **Medley TL, Cole TJ, Gatzka CD, Wang WY, Dart AM, Kingwell BA.** Fibrillin-1 genotype is associated with aortic stiffness and disease severity in patients with coronary artery disease. *Circulation* 2002 Feb 19; 105(7): 810-5.
- 95 **Menshikov M, Elizarova E, Plakida K, Timofeeva A, Khaspekov G, Beabealashvili R, Bobik A, Tkachuk V.** Urokinase upregulates matrix metalloproteinase-9 expression in THP-1 monocytes via gene transcription and protein synthesis. *Biochem J* 2002 Nov 1; 367(3): 833-9.
- 96 **Mifsud SA, Skinner SL, Cooper ME, Kelly DJ, Wilkinson-Berka JL.** Effects of low-dose and early versus late perindopril treatment on the progression of severe diabetic nephropathy in (mREN-2)27 rats. *J Am Soc Nephrol* 2002 Mar; 13(3): 684-92.
- 97 **Morton JB, Byrne MJ, Power JM, Raman J, Kalman JM.** Electrical remodeling of the atrium in an anatomic model of atrial flutter: relationship between substrate and triggers for conversion to atrial fibrillation. *Circulation* 2002 Jan 15; 105(2): 258-64.
- 98 **Navakatikyan MA, Barrett CJ, Head GA, Ricketts JH, Malpas SC.** A real-time algorithm for the quantification of blood pressure waveforms. *IEEE Trans Biomed Eng* 2002 Jul; 49(7): 662-70.
- 99 **Nelson MR, Reid CM, Krum H, Muir T, Ryan P, McNeil JJ.** Predictors of normotension on withdrawal of antihypertensive drugs in elderly patients: prospective study in second Australian national blood pressure study cohort. *BMJ* 2002 Oct 12; 325(7368): 815.
- 100 **Nestel P.** Biomarkers, surrogates for nutritional evidence. *Curr Opin Lipidol* 2002 Feb; 13(1): 1-2.
- 101 **Nestel PJ.** Adulthood - treatment: Cholesterol-lowering with plant sterols. *Med J Aust* 2002 Jun 3; 176(11 Suppl): S122.
- 102 **Nestel PJ.** Adulthood - prevention: Cardiovascular disease. *Med J Aust* 2002 Jun 3; 176(11 Suppl): S118-9.
- 103 **Nestel PJ.** Husband A. Effects of free (aglycone) phytoestrogens and metabolites on cardiovascular functions and cancer. In: Phytoestrogens and Health. Am Oil Chemist Soc 2002; 318-30.
- 104 **Nestel P, Shige H, Pomeroy S, Cehun M, Abbey M, Raederstorff D.** The n-3 fatty acids eicosapentaenoic acid and docosahexaenoic acid increase systemic arterial compliance in humans. *Am J Clin Nutr* 2002 Aug; 76(2): 326-30.
- 105 **Nigro J, Dilley RJ, Little PJ.** Differential effects of gemfibrozil on migration, proliferation and proteoglycan production in human vascular smooth muscle cells. *Atherosclerosis* 2002 May; 162(1): 119-29.
- 106 **Nystrom HC, Jia J, Johansson M, Lambert G, Bergström G.** Neurohormonal influences on maintenance and reversal of two-kidney one-clip renal hypertension. *Acta Physiol Scand* 2002 Jul; 175(3): 245-51.
107. O'Dwyer DT, Smith AI, Matthew ML, Andronicos NM, Ranson M, Robinson PJ, Crock PA. Identification of the 49-kDa autoantigen associated with lymphocytic hypophysitis as alpha-enolase. *J Clin Endocrinol Metab* 2002 Feb; 87(2): 752-7.
- 108 **Oldfield MD, Cooper ME.** Growth factors in diabetic microangiopathy. *Mod Diabetes Management* 2002; 3(1): 10-14.
- 109 **Oldfield MD, Cooper ME.** How is diabetic microvascular disease best prevented and treated? In: Diabetes Annual 2002. Barnett AH, ed. London, Martin Dunitz, 2002: 61-86.
- 110 **Oldfield MD, Cooper ME.** The biochemistry and pathophysiology of renal lesions in type 2 diabetes. In: Diabetic nephropathy in type 2 diabetes. Mogensen CE, ed. London, Science Press, 2002: 41-56.
- 111 **Orsida BE, Krozowski ZS, Walters EH.** Clinical relevance of airway 11beta-hydroxysteroid dehydrogenase type II enzyme in asthma. *Am J Respir Crit Care Med* 2002 Apr 1; 165(7): 1010-14.
- 112 **Paizis G, Cooper ME, Schembri JM, Tikellis C, Burrell LM, Angus PW.** Up-regulation of components of the renin-angiotensin system in the bile duct-ligated rat liver. *Gastroenterology* 2002 Nov; 123(5): 1667-76.
- 113 **Parnell MM, Holst DP, Kaye DM.** Exercise training increases arterial compliance in patients with congestive heart failure. *Clin Sci (Lond)* 2002 Jan; 102(1): 1-7.
- 114 **Pepe S, McLennan PL.** Cardiac membrane fatty acid composition modulates myocardial oxygen consumption and posts ischemic recovery of contractile function. *Circulation* 2002 May 14; 105(19): 2303-8.
- 115 **Pepe S, Rosenfeldt FL, Lakatta EG, Sollott SJ.** Mitochondrial membrane alterations by aging and diet: impact on recovery of myocardial function after stress. *Adapt Biol Med* 2002; 3: 84-93.
- 116 **Petersson MJ, Rundqvist B, Johansson M, Eisenhofer G, Lambert G, Herlitz H, Jensen G, Friberg P.** Increased cardiac sympathetic drive in renovascular hypertension. *J Hypertens* 2002 Jun; 20(6): 1181-7.
- 117 **Raman JS, Ishikawa S, Power JM.** Epicardial radiofrequency ablation of both atria in the treatment of atrial fibrillation: experience in patients. *Ann Thorac Surg* 2002 Nov; 74(5): 1506-9.
- 118 **Romanov YA, Soboleva EL, Smirnov VN, Bobik A.** Human atherosclerosis: new cell participants? In: Proceedings of the World Heart Congress: Atherosclerosis and Hypertension, Winnipeg, 2001; Pierce GN et al eds. Boston, Kluwer Academic, 2002.
- 119 **Rosenfeldt FL, Esmore DS.** Preventing radial artery spasm. *Ann Thorac Surg* 2002 May; 73(5): 1695-6.
- 120 **Rosenfeldt FL, Pepe S, Linnane A, Nagley P, Rowland M, Ou R, Marasco S, Lyon W.** The effects of ageing on the response to cardiac surgery: protective strategies for the ageing myocardium. *Biogerontology* 2002; 3(1-2): 37-40.
- 121 **Rosenfeldt FL, Pepe S, Linnane A, Nagley P, Rowland M, Ou R, Marasco S, Lyon W, Esmore D.** Coenzyme Q10 protects the aging heart against stress: studies in rats, human tissues, and patients. *Ann NY Acad Sci* 2002 Apr; 959: 355-9.
- 122 **Shaw JA, Kingwell BA, Walton AS, Cameron JD, Pillay P, Gatzka CD, Dart AM.** Determinants of coronary artery compliance in subjects with and without angiographic coronary artery disease. *J Am Coll Cardiol* 2002 May 15; 39(10): 1637-43.
- 123 **Shen Y, Dong JF, Romo GM, Arceneaux W, Aprico A, Gardiner EE, Lopez JA, Berndt MC, Andrews RK.** Functional analysis of the C-terminal flanking sequence of platelet glycoprotein Ib alpha using canine-human chimeras. *Blood* 2002 Jan 1; 99(1): 145-50.
- 124 **Sheppard KE.** Nuclear receptors. II. Intestinal corticosteroid receptors. *Am J Physiol Gastrointest Liver Physiol* 2002 May; 282(5): G742-6.
- 125 **Sheppard KE, Autelitano DJ.** 11Beta-hydroxysteroid dehydrogenase 1 transforms 11-dehydrocorticosterone into transcriptionally active glucocorticoid in neonatal rat heart. *Endocrinology* 2002 Jan; 143(1): 198-204.
- 126 **Shrimpton CN, Smith AI, Lew RA.** Soluble metalloendopeptidases and neuroendocrine signaling. *Endocr Rev* 2002 Oct; 23(5): 647-64.
- 127 **Steer DL, Lew RA, Perlmutter P, Smith AI, Aguilar MI.** Beta-amino acids: versatile peptidomimetics. *Curr Med Chem* 2002 Apr; 9(8): 811-22.
- 128 **Steer DL, Lew RA, Perlmutter P, Smith AI, Aguilar MI.** Inhibitors of metalloendopeptidase EC 3.4.24.15 and EC 3.4.24.16 stabilized against proteolysis by the incorporation of beta-amino acids. *Biochemistry* 2002 Sep 3; 41(35): 10819-26.
- 129 **Stitt AW, Jenkins AJ, Cooper ME.** Advanced glycation end products and diabetic complications. *Expert Opin Investig Drugs* 2002 Sep; 11(9):1205-23.
- 130 **Suzuki-Inoue K, Tulasne D, Shen Y, Bori-Sanz T, Inoue O, Jung SM, Moroi M, Andrews RK, Berndt MC, Watson SP.** Association of Fyn and Lyn with the proline rich domain of GPVI regulates intracellular signalling. *J Biol Chem* 2002 Jun 14; 277(24): 21561-6.
- 131 **Sviridov D, Hoang A, Huang W, Sasaki J.** Structure-function studies of apoA-I variants: site-directed mutagenesis and natural mutations. *J Lipid Res* 2002 Aug; 43(8): 1283-92.
- 132 **Sviridov D, Miyazaki O, Theodore K, Hoang A, Fukamachi I, Nestel P.** Delineation of the role of pre-beta 1-HDL in cholesterol efflux using isolated pre-beta 1-HDL. *Arterioscler Thromb Vasc Biol* 2002 Sep 1; 22(9): 1482-8.
- 133 **Sviridov D, Nestel P.** Dynamics of reverse cholesterol transport: protection against atherosclerosis. *Atherosclerosis* 2002 Apr; 161(2): 245-54.
- 134 **Tannock LR, Little PJ, Wight TN, Chait A.** Arterial smooth muscle cell proteoglycans synthesized in the presence of glucosamine demonstrate reduced binding to LDL. *J Lipid Res* 2002 Jan; 43(1): 149-57.
- 135 **Taylor AJ, Bobik A, Berndt MC, Ramsay D, Jennings G.** Experimental rupture of atherosclerotic lesions increases distal vascular resistance: a limiting factor to the success of infarct angioplasty. *Arterioscler Thromb Vasc Biol* 2002 Jan; 22(1): 153-60.
- 136 **Thomas MC, Cooper ME.** Turning up the heat: heat shock proteins, hypertension and cardiovascular risk. *J Hypertens* 2002 Sep; 20(9): 1713-4.
- 137 **Thomas WG, Brandenburger Y, Autelitano DJ, Pham T, Qian H, Hannan RD.** Adenoviral-directed expression of the type 1A angiotensin receptor promotes cardiomyocyte hypertrophy via transactivation of the epidermal growth factor receptor. *Circ Res* 2002 Feb 8; 90(2): 135-42.
- 138 **Tikkanen I, Tikkanen T, Cao Z, Allen TJ, Davis BJ, Lassila M, Casley D, Johnston CI, Burrell LM, Cooper ME.** Combined inhibition of neutral endopeptidase with angiotensin converting enzyme or endothelin converting enzyme in experimental diabetes. *J Hypertens* 2002 Apr; 20(4): 707-14.
- 139 **Tsunoda N, Pomeroy S, Nestel P.** Absorption in humans of isoflavones from soy and red clover is similar. *J Nutr* 2002 Aug; 132(8): 2199-201.
- 140 **Turner AI, Rivalland ET, Clarke IJ, Lambert GW, Morris MJ, Tilbrook AJ.** Noradrenaline, but not neuropeptide Y, is elevated in cerebrospinal fluid from the third cerebral ventricle following audiovisual stress in gonadectomised rams and ewes. *Neuroendocrinology* 2002 Dec; 76(6): 373-80.
- 141 **Twigg SM, Cao Z, McLennan SV, Burns WC, Brammar G, Forbes JM, Cooper ME.** Renal connective tissue growth factor induction in experimental diabetes is prevented by aminoguanidine. *Endocrinology* 2002 Dec; 143(12): 4907-15.
- 142 **Van den Buuse M, Van Acker SA, Fluttert MF, De Kloet ER.** Involvement of corticosterone in cardiovascular responses to an open-field novelty stressor in freely moving rats. *Physiol Behav* 2002 Feb 1-15; 75(1-2): 207-15.
- 143 **Ward MR, Agrotis A, Kanellakis P, Hall J, Jennings G, Bobik A.** Tranilast prevents activation of transforming growth factor-beta system, leukocyte accumulation, and neointimal growth in porcine coronary arteries after stenting. *Arterioscler Thromb Vasc Biol* 2002 Jun 1; 22(6): 940-8.
- 144 **Whisstock JC, Shen Y, Lopez JA, Andrews RK, Berndt MC.** Molecular modeling of the seven tandem leucine-rich repeats within the ligand-binding region of platelet glycoprotein Ib alpha. *Thromb Haemost* 2002 Feb; 87(2): 329-33.
- 145 **Wilkinson-Berka JL, Kelly DJ, Koerner SM, Jaworski K, Davis B, Thallas V, Cooper ME.** ALT-946 and aminoguanidine, inhibitors of advanced glycation, improve severe nephropathy in the diabetic transgenic (mREN-2)27 rat. *Diabetes* 2002 Nov; 51(11): 3283-9.
- 146 **Williams MR, Ling S, Dawood T, Hashimura K, Dai A, Li H, Liu JP, Funder JW, Sudhir K, Komesaroff PA.** Dehydroepiandrosterone inhibits human vascular smooth muscle cell proliferation independent of ARs and ERs. *J Clin Endocrinol Metab* 2002 Jan; 87(1): 176-81.
- 147 **Wing LMH, Brown MA, Beilin L, Ryan P, Reid CM.** "Reverse white-coat hypertension" in older hypertensives. *J Hypertens* 2002 Apr; 20(4): 639-44.
- 148 **Woodcock EA, Wang BH, Arthur JF, Lennard A, Matkovich SJ, Du XJ, Brown JH, Hannan RD.** Inositol polyphosphate 1-phosphatase is a novel anti-hypertrophic factor. *J Biol Chem* 2002 Jun 21; 277(25): 22734-42.
- 149 **Zammit DJ, Berzins SP, Gill JW, Barnett L, Koentgen F, Lambert GW, Harvey RP, Boyd RL, Classon BJ.** An essential role for the lymphostromal plasma membrane Ly-6 superfamily molecule TSA-1 in development of the embryonic adrenal gland. *Mol Cell Biol* 2002 Feb; 22(3): 946-52.
- 150 **Zolk O, Frohne M, Maurer A, Kluxen FW, Hentsch B, Zubakov D, Hoheisel JD, Zucker IH, Pepe S, Eschenhagen T.** Cardiac ankyrin repeat protein, a negative regulator of cardiac gene expression, is augmented in human heart failure. *Biochem Biophys Res Commun* 2002 May 24; 293(5): 1377-82.

IN PRESS

- 151 **Aggarwal A, Esler MD, Morris M, Lambert G, Kaye DM.** The effects of low dose clonidine on cardiac, renal and brain sympathetic activity in human heart failure. *Hypertension* (in press).
- 152 **Bui BV, Armitage JA, Tolcos M, Cooper ME, Vingrys AJ.** ACE inhibition salvages the visual loss caused by diabetes. *Diabetologia* (in press).
- 153 **Candido R, Forbes JM, Thomas MC, Thallas V, Dean RG, Burns WC, Tikellis C, Ritchie RH, Twigg SM, Cooper ME, Burrell LM.** Breaker of advanced glycation end products attenuates diabetes-induced myocardial structural changes. *Circ Res* (in press).
- 154 **Chai Z, Brereton P, Suzuki T, Sasano H, Obeyesekere V, Saffery R, Fuller P, Enriquez C, Escher G, Kaye D, Krozowski Z.** 17 β -hydroxysteroid dehydrogenase XI is a novel human dehydrogenase expressed in human steroidogenic cells. *Endocrinology* (in press).
- 155 **Clavant SP, Forbes JM, Thallas V, Jerums G, Comper WD.** Isolated kidney perfusion with excess levels of angiotensin II induces increases in the fractional clearance of albumin and disruption of the tubular epithelial cell cytoskeleton. *Nephron* (in press).
- 156 **Cooper ME.** The prevalence of hypertension in patients with diabetes. In: *Hypertension and diabetes*. Vol 2. Mogensen CE ed. London, Lippincott Williams and Wilkins (in press).
- 157 **Cooper ME, Thomas MC.** Interactions between growth factors in the kidney: implications for progressive renal injury. *Kidney Int* (in press).
- 158 **Davis B, Cao Z, de Gasparo M, Kawachi H, Cooper ME, Allen TJ.** Disparate effects of angiotensin II and calcium channel blockers on albuminuria in experimental diabetes and hypertension: potential role of nephrin. *J Hypertens* (in press).
- 159 **Davis BJ, Johnston CI, Burrell LM, Burns WC, Kubota E, Cao Z, Cooper ME, Allen TJ.** Renoprotective effects of dual ACE/NEP inhibition in an experimental model of diabetic nephropathy. *Diabetologia* (in press).
- 160 **Du XJ, Samuel CS, Gao XM, Zhao L, Parry LJ, Tregear GW.** Increased myocardial collagen and ventricular diastolic dysfunction in relaxin deficient mice: a gender-specific phenotype. *Cardiovasc Res* (in press).
- 161 **Duffy SJ, Vita JA.** Effects of phenolics on vascular endothelial function. *Curr Opin Lipidol* (in press).
- 162 **Escher G, Krozowski Z, Croft KD, Sviridov D.** Expression of sterol 27-hydroxylase (CYP27A1) enhances cholesterol efflux. *J Biol Chem* (in press).
- 163 **Esler M, Lambert G, Brunner-LaRocca H, Vaddadi G, Kaye D.** Sympathetic nerve activity and neurotransmitter release in humans: translation from pathophysiology into clinical practice. *Acta Physiol Scand* (in press).
- 164 **Febbraio MA, Steensberg A, Strakie RL, McConell GK, Kingwell BA.** Skeletal muscle IL-6 and TNF- β release in healthy subjects and patients with type 2 diabetes at rest and during exercise. *Metabolism* (in press).
- 165 **Gao SA, Ambring A, Lambert G, Karlsson AK.** Autonomic control of the heart and renal vascular bed during autonomic dysreflexia in high spinal cord injury. *J Autonom Nerv Sys* (in press).
- 166 **Gorge B, Percy E, Gao XM, Dart AM, Richardt G, Du XJ.** Altered calcium transient and development of hypertrophy in beta2-adrenoceptor overexpressing mice with and without pressure overload. *Eur J Heart Fail* (in press).
- 167 **Hannan RD, Jenkins A, Jenkins AK, Brandenburger Y.** Cardiac hypertrophy: a matter of translation. *Clin Exp Pharmacol Physiol* (in press).
- 168 **Hoang A, Huang, W., Sasaki, J., Sviridov, D.** Natural mutations of apolipoprotein A-I impairing activation of lecithin:cholesterol acyltransferase. *Biochim Biophys Acta* (in press).
- 169 **Kingwell BA, Formosa M, Muhlmann M, Bradley SJ, McConell GK.** Type 2 diabetics have impaired leg blood flow responses to exercise; role of endothelium dependent vasodilation. *Diabetes Care* (in press).
- 170 **Lambert G, Reid C, Kaye D, Jennings G, Esler M.** Increased suicide rate in the middle aged and its association with hours of sunlight. *Am J Psychiat* (in press).
- 171 **Lassila M, Davis B, Allen TJ, Burrell LM, Cooper ME, Cao Z.** Cardiovascular hypertrophy in diabetic spontaneously hypertensive rats: optimising blockade of the renin angiotensin system. *Clin Sci* (in press).
- 172 **Lee J, Mustafa T, McDowell SG, Mendelsohn FAO, Brennan M, Lew RA, Albiston AL, Chai SY.** Structure-activity study of LVV-hemorphin-7: ligand and inhibitor of insulin-regulated aminopeptidase (IRAP). *J Pharmacol Exp Ther* (in press).
- 173 **Lew RA.** The use of HPLC in the study of peptide metabolism. In: *HPLC of Peptides & Proteins: Methods & Protocols*, ed. Aguilar, M.-I., *Methods in Molecular Biology* (in press).
- 174 **Lew RA, Reeve SB, Little PJ, Dive V, Smith AI.** Role of endothelial cell endopeptidases EC 3.4.24.15 and EC 3.4.24.16 in bradykinin metabolism. *Am J Physiol* (in press).
- 175 **Little PJ, Allen TJ, Hashimura K, Nigro J, Farrelly CA, Dilley RJ.** High glucose potentiates mitogenic responses of cultured ovine coronary smooth muscle cells to platelet derived growth factor and transforming growth factor-B1. *Diabetes Res Clin Pract* (in press).
- 176 **Mansfield D, Kaye DM, Brunner la Rocca H, Solin P, Esler MD, Naughton MT.** Raised sympathetic nerve activity in heart failure and central sleep apnea is due to heart failure severity. *Circulation* (in press).
- 177 **Mayorov DN, Head GA.** Glutamate receptors in the RVLM modulate the sympathetic baroreflex in conscious rabbits. *Am J Physiol Regul Integr Comp Physiol* (in press).
- 178 **McCrystal GD, Pepe S, MacDonald P, Esmore DS, Rosenfeldt FL.** The challenge of improving donor heart preservation. *Heart Lung Circ* (in press).
- 179 **Moravski C, Skinner SL, Stubbs AJ, Sarlos S, Kelly DJ, Cooper ME, Gilbert RE, Wilkinson-Berka JL.** The renin-angiotensin system influences ocular endothelial cell proliferation in diabetes: transgenic and interventional studies. *Am J Pathol* (in press).
- 180 **Nestel PJ.** Isoflavones: their effects on cardiovascular risk and functions. *Curr Rev Lipidol* (in press).
- 181 **Nestel P, Teede H and McGrath B.** A biochanin-enriched isoflavone extract from red clover lowers LDL cholesterol in men. *Am J Clin Nutr* (in press).
- 182 **Norman MU, Lew RA, Smith AI, Hickey MJ.** Metalloendopeptidases EC 3.4.24.15/16 regulate bradykinin activity in the cerebral microvasculature. *Am J Physiol Heart Circ Physiol* (in press).
- 183 **Norman MU, Reeve SB, Dive V, Smith AI, Lew RA.** Endopeptidases EC 3.4.24.15 and 24.16 in endothelial cells: potential role in vasoactive peptide metabolism. *Am J Physiol* (in press).
- 184 **Parnell MM, Chin-Dusting J, Kaye DM.** Low dose atorvastatin therapy does not augment endothelial function in active, hypercholesterolaemic males. *Br J Clin Pharmacol* (in press).
- 185 **Pepe S.** Aging and dietary lipids modulate Ca²⁺-dependent mitochondrial function in the post-ischemic heart. In: *Nutrition and Cardiovascular Disease*. Dhalla, Angel, Pierce, eds. Boston, Kluwer (in press).
- 186 **Rice E, Tesch G, Cao Z, Cooper M, Metz C, Atkins R, Nikolic-Paterson D.** Induction of MIF synthesis and secretion by tubular epithelial cells – a novel action of angiotensin II. *Kidney Int* (in press).
- 187 **Rosenfeldt FL, McCrystal GD, Pepe S, Esmore DS.** Myocyte or heart preservation: what is the optimal strategy for donor heart preservation during prolonged ischaemia? *J Heart Lung Transplant* (in press).
- 188 **Sato Y, Schmidt AG, Kiriazis H, Hoit BD, Kranias EG.** Compensated hypertrophy of cardiac ventricles in aged transgenic FVB/N mice overexpressing casequestrin. *Mol Cell Biochem* (in press).
- 189 **Solin P, Kaye DM, Little PJ, Bergin P, Richardson M, Naughton MT.** Impact of sleep apnea on sympathetic nervous activity in heart failure. *Chest* (in press).
- 190 **Suzuki S, Koyama K, Darnel A, Ishibashi H, Kobayashi S, Kubo H, Suzuki T, Sasano H, Krozowski ZS.** Dexamethasone up-regulates 11 β -hydroxysteroid dehydrogenase type 2 in BEAS-2B cells. *Am J Respir Crit Care Med* (in press).
- 191 **Sviridov D, Hoeg, JM, Eggerman T, Demosky SJ, Safonova IG, Brewer HB.** Low density lipoprotein receptor and apolipoproteins A-I and B expression in human enterocytes: comparison with CaCo-2 and HepG2 cells. *Digestion* (in press).
- 192 **Sviridov D, Kingwell BA, Hoang A, Dart AM, Nestel P.** Single session exercise stimulates formation of pre-beta1-HDL in leg muscle. *J Lipid Res* (in press).
- 193 **Thomas MC, Cooper ME, Shahinfar S, Brenner BM.** Dialysis delayed is death prevented: a clinical perspective on the RENAAL study. *Kidney Int* (in press).
- 194 **Thomas MC, Tikellis C, Burns WC, Thallas V, Forbes JM, Cao Z, Osicka TM, Russo LM, Jerums G, Ghabrial H, Cooper ME, Kantharides P.** Reduced tubular cation transport in diabetes: prevention by ACE inhibition. *Kidney Int* (in press).
- 195 **Tikellis C, Johnston CI, Forbes JM, Burns WC, Burrell L, Risvanis J, Cooper ME.** Characterization of renal ACE2 in diabetic nephropathy. *Hypertension* (in press).
- 196 **Tikellis C, Xuereb L, Casley D, Brasier G, Cooper ME, Wookey PJ.** Calcitonin receptor isoforms expressed in the developing rat kidney. *Kidney Int* (in press).
- 197 **Tolcos M, Tikellis C, Rees S, Cooper M, Wookey P.** Ontogeny of calcitonin receptor mRNA and protein in the developing central nervous system of the rat. *J Comp Neurol* (in press).
- 198 **Van den Brink OWV, Rosenfeldt FL, Penny DJ, Esmore DS, Lloyd R, Rothschild L, Quick D, Pepe S.** Endogenous cardiac opioids: enkephalins in adaptation and protection of the heart. *Heart Lung Circ* (in press).
- 199 **Vita JA, Gokce N, Duffy SJ, Kahn DF, Tomasian D, Palmisano J, Thomas S, Holbrook M, Keaney JF Jr.** Effect of atorvastatin on endothelium-dependent vasodilation in patients with coronary artery disease. *Am J Cardiol* (in press).
- 200 **Wing LMH, Reid CM, Ryan P, Beilin LJ, Brown MA, Jennings GLR et al for the Second Australian National Blood Pressure Study Group.** A comparison of outcomes with angiotensin-converting enzyme inhibitors and diuretics for hypertension in the elderly. *N Engl J Med* (in press).
- 201 **Wookey PJ, Cooper ME.** The roles of amylin in the periphery: a review. *Scientific World* (in press).
- 202 **Young MJ, Moussa L, Dilley R, Funder JW.** Early inflammatory responses in experimental cardiac hypertrophy and fibrosis: effects of 11 β -hydroxysteroid dehydrogenase inactivation. *Endocrinology* (in press).

