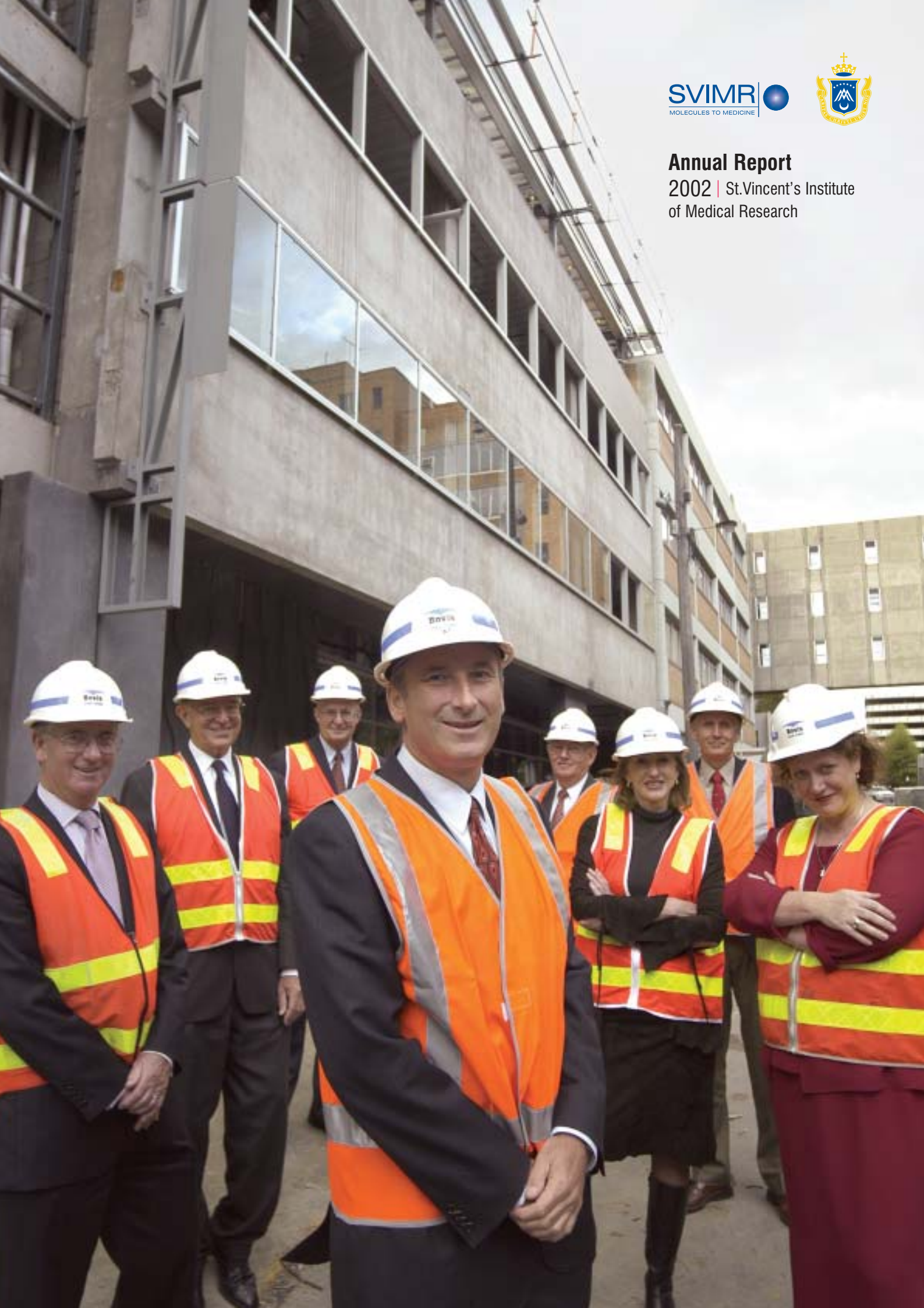


SVIMR
MOLECULES TO MEDICINE

Annual Report

2002 | St.Vincent's Institute of Medical Research

MISSION STATEMENT

St. Vincent's Institute of Medical Research is a centre of excellence in medical research. Its mission is to take molecules to medicine thereby promoting human well-being through the prevention and treatment of diseases. Its programs of basic and clinical research are applied to the study of certain diseases that are of great cost to the Australian community. These include osteoporosis, and other bone diseases, cancers (breast, lung and prostate) that spread to bone, diabetes, virology and also diseases of the heart and blood vessels.

The Institute is an independent one, founded in 1955 as an initiative of the Congregation of the Sisters of Charity and St. Vincent's Hospital. It is a member institution of Australia-wide health care facilities of the Sisters of Charity, and is sponsored and supported by the Congregation in many ways.

The contribution made by the research of the Institute to advancement of health care in Australia is an important one, and is conducted in close co-operation with a major teaching hospital, St. Vincent's Hospital Melbourne, and with The University of Melbourne. Through these links its research programs provide a valuable service to clinical medicine, graduate education and community welfare.

SVIMR
MOLECULES TO MEDICINE

Images: *(Front cover) Left to right at rear*: Institute Board Members - GEN Rogers, ID Reid, BJ Jackson, CA Griss, M Griffin, JD Best and NM Feely. *Foreground*: Director TW Kay. *(Back cover)* Construction workers - Bovis Lend Lease.

Editor/Project Coordinator: Dr. William McKinstry
Graphic Design: Origin Design
Photography: Ric Wallis
Print: Manark Printing Pty Ltd
Paper Stock: K.W. Doggett Fine Paper

Special thanks to Brenda Shanahan, Doug Wright, David Rees and Jock Campbell for their involvement.

Contents

Investing in Excellence

The purpose of the Institute's excellence in medical research is to serve the community, and we strive to achieve this in partnership with the community. To build this partnership we are working to increase community awareness of the Institute and its science through our Appeal for Life campaign.

Images: Pictured above architects (Design Inc.) rendering of the new Institute Building

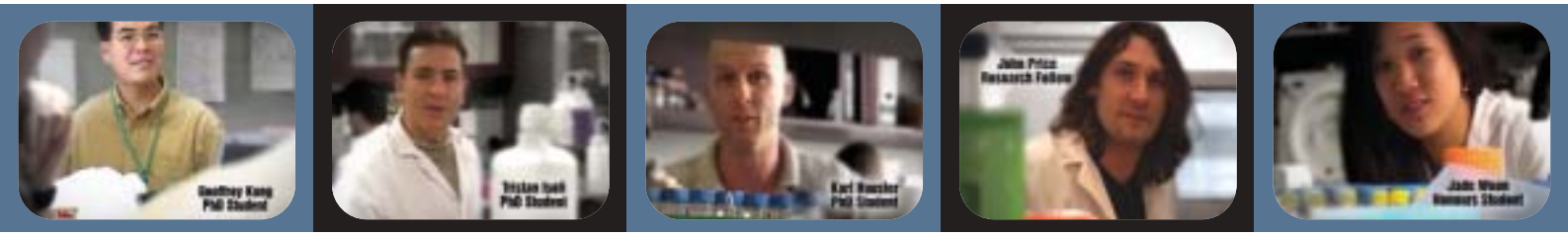
Creating Awareness

The success of the Institute is dependent on a strong partnership with the community, and we are striving to increase community awareness of the scientific success of the Institute and its implications for the health of the community.

We have had many successes in our Appeal for Life campaign. In the past year we doubled the number of new supporters of the Institute and the number of donors increased four-fold. We co-hosted the Mississippi Magic Ball with the St. Vincent's Hospital Foundation in May, 2002. More than 800 guests attended the gala event and helped to raise funds for cancer research and care conducted on the St. Vincent's campus. Our special thanks go to Crown, Marshall White Real Estate, and Toohey's for their very generous support.

To promote the Institute in the community we produced promotional material and we continued our Director's series of lunches and dinners. Our Appeal for Life Brochure, produced with generous sponsorship from Origin Design, Manark Printing Pty Ltd and K.W. Doggett Fine Paper won the Gold Medal at the National Print Awards and has been instrumental in attracting supporters. Our Director's series has featured guest speakers including Scobie Breasley, David Parkin, Stephen Gough, Patrick Ness, Stephanie Alexander, Vivienne James, John Eales, Simon O'Donnell, and James Halliday. These dinners and lunches have been sponsored by Crown, ANZ, and BT Australia. From these events have come many expressions of support for the Institute and lasting collaborations.

Gaining Community Support



Special thanks to Craig Williams, who sponsored our TV advertisement 'Hot on the track to beating bone disease' and to Channel 7 Melbourne who provided free airplay.

Following one dinner Craig Williams of Far East Consortium recommended that we promote the Institute on television. Craig sponsored the production of our first TV commercial in collaboration with Wilson Everard Advertising, who developed the concept and script, and Harry Benholtz of SMR Productions, who produced the advertisement. Our TV advertisement 'Hot on the track to beating bone disease' has been shown on Channel 7 over the past year, and also at the MCG during the AFL elimination and Semi Finals for 2002.

SVIMR Support Group

We have re-established the St. Vincent's Institute of Medical Research Support Group, led by Claire O'Callaghan and her committee. This group of volunteers raises funds for the Institute by hosting dinners, conducting raffles, and promoting the work of the Institute to the wider community.

The members of the Support Group committee are Annette Bongiorno, Judy Brady, Maureen Breheny, Joan Chappell, Cathy Clancy, Anne Courtney, Mary Duggan, Cathy Gilbert, Carole Hart, Maureen Hoare, Dawn Hill, Anna Kennedy, Barbara Lonergan, Margaret Lorkin, Diana Lowe, Gail McHale, Claire O'Callaghan, Judy Walsh, Christina Westmore-Payton, Natalie Woodley, Therese Whiting and Thecla Xipell. Through their efforts the Institute has gained increased recognition and donor support from the community.

1000 Club

Our fundraising was stimulated by the formation of the 1000 Club at a dinner hosted by BT Australia. We aim to have 1000 members, each donating $1000 for the funding of laboratories in the new Institute building currently under construction.

Our partnership continues with our friends at Yering Station, from the Yarra Valley. Yering Station provides the wines for our annual wine fundraising promotion. The funds raised from this promotion help fund one of our most difficult tasks, that of the maintenance and renewal of research equipment, essential to all facets of research within the Institute.

Image: Above (*left to right)*: SVIMR Support Group - Therese Whiting, Gayle McHale, Dawn Hill, Carol Hart, Claire O'Callaghan, Joan Chappell, Thecla Xipell and Anna Kennedy.

Science for the Community

Image: Stephanie Alexander

Special thanks to Stephanie and the staff of Richmond Hill Café and Larder for their ongoing support as hosts for our fund raising events.

SVIMR gratefully acknowledges the following organisations for their support

YERING Station

ANZ

STAGING CONNECTIONS

RED

WILSON EVERARD ADVERTISING

K.W.DOGGETT Fine Paper

the one to watch

ORIGIN

KORIMCO

SMR

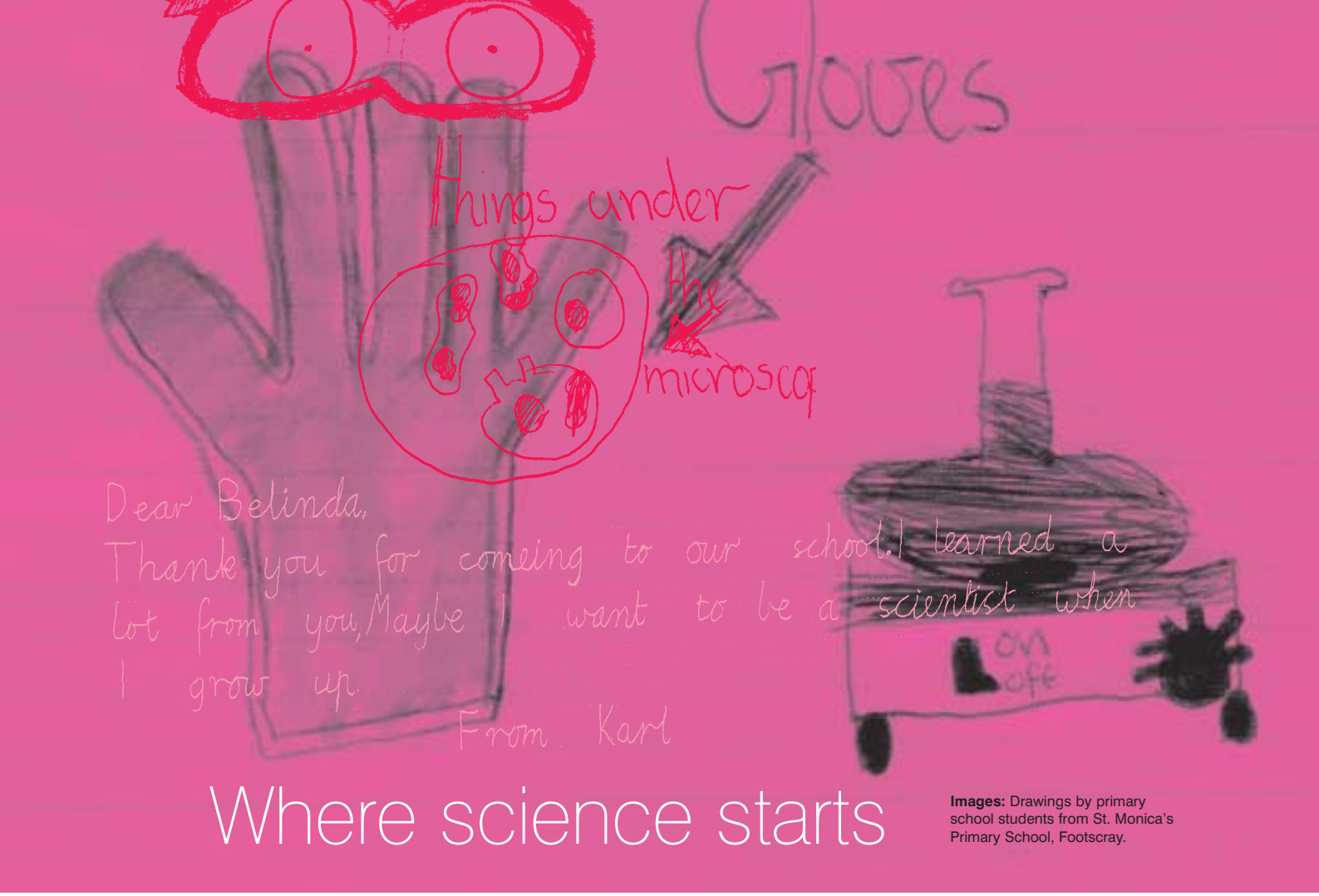
CROWN TOWERS

MANARK printing

Taking science to schools

As a result of our television commercial 'Hot on the track to beating bone disease' one of our medical research scientists, Belinda Rizzo, was contacted by her Aunt, Mrs Antiounette Stasi, a grade 4 teacher at St. Vincent's Primary School in Essendon. She invited Belinda to visit the school and tell her students what being a scientist is all about. Belinda gave a short presentation to the students about experiments and safety in the laboratory. Word soon spread about Belinda's presentation and she has since visited a number of other primary schools. Belinda believes that it is important that primary school children are exposed to the enjoyment of science, particularly as children become more aware of advances in medical research though the media and the internet.

The Institute has a limited number of work experience student placements each year where year 10 and 11 students spend a week working alongside research scientists. This brief exposure to the research environment has resulted in one such student, Jenny Leung, going on to undertake tertiary studies and returning to commence her PhD studies with Associate Professor Rik Thompson. Another student, Sarah Wongseelashote, went on to be Dux of her school and is now studying medicine.



Where science starts

Images: Drawings by primary school students from St. Monica's Primary School, Footscray.

SVIMR has a major commitment to the education of school students. By introducing students to the wonders of science we hope to foster an interest that will encourage them to consider science as a career path, thereby ensuring the community continues to reap the benefits of medical research into the future.

Strengthening our ties

How research supports medicine

The prevention and control of diabetes has been designated a Health Priority Area in Australia because of its rising incidence and its serious impact on health. The diabetes research team at SVIMR is working on type 1 diabetes, which is one of the most common chronic disorders beginning in children. It currently affects one in every 200 Australians and its incidence is increasing, particularly in children less than five years of age.

Links to the Hospital

Diabetes research is an area of strength and growth not only at SVIMR but also in other centres located on the St. Vincent's Hospital precinct. Together, these centres cover the entire spectrum of diabetes research from molecular biology to population studies. The quality and breadth of this research is unique in Australia. This strong research focus, combined with the highly regarded clinical diabetes services at St. Vincent's, has formed the basis of a new Diabetes Centre of Excellence that was launched in November 2002. Partnerships within this Centre will promote high-quality diabetes research, facilitate links between laboratory studies and clinical research and, in so doing, provide excellence in patient care.

Image: Pictured above (*left to right*): Prof. T d'Apice Director, Immunology Research Centre St. Vincent's Health; Prof. T Kay Director SVIMR; Prof. J Best Head of Dept. of Medicine, The University of Melbourne, St. Vincent's Health.

Director Tom Kay

In April 2002, the Board of St. Vincent's Institute of Medical Research appointed Professor Thomas Kay MBBS PhD FRACP FRCPA, a diabetes researcher and immunologist, as its fourth Director.

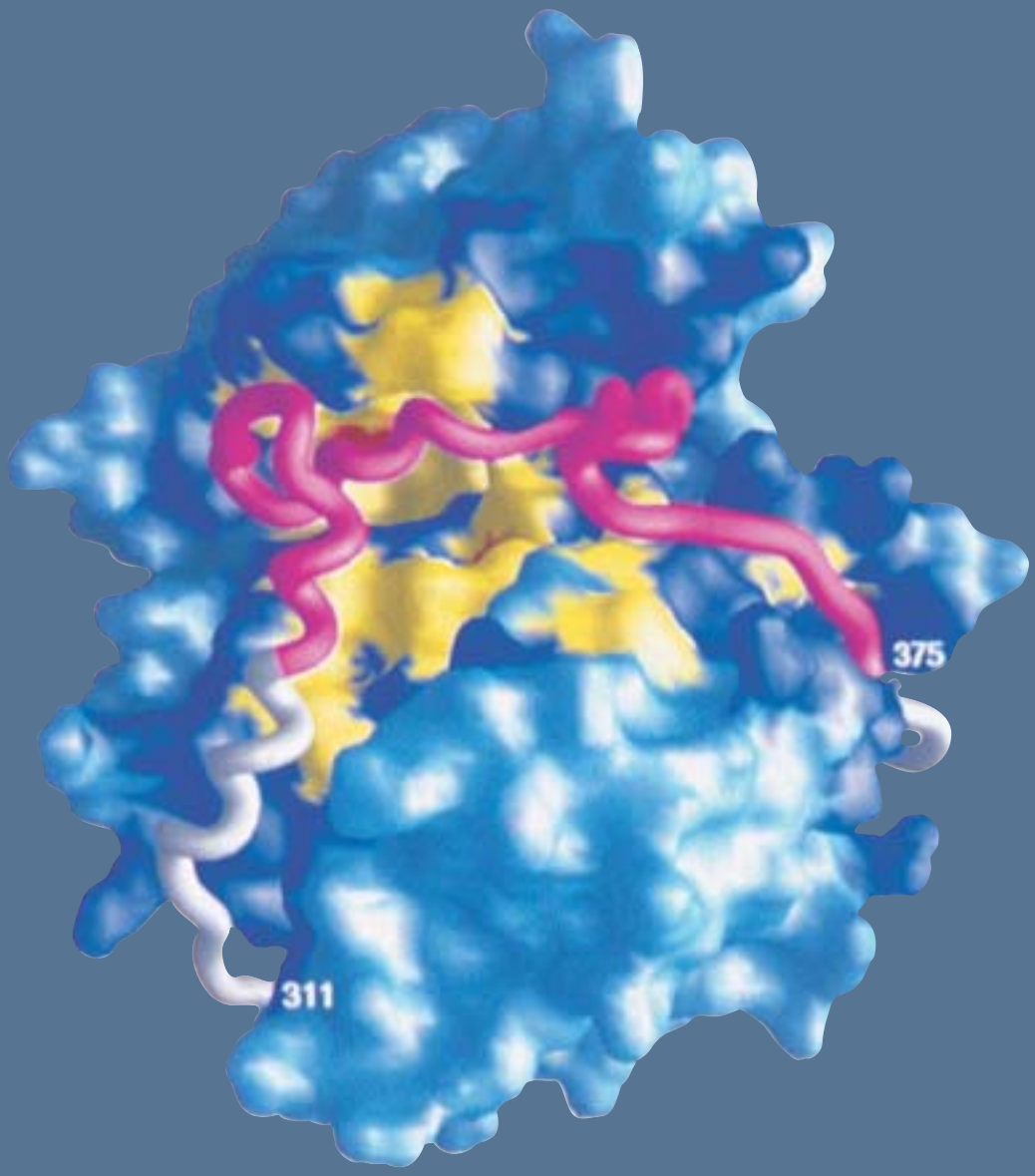
Tom first developed his taste for medical research during his medical training at the University of Melbourne, taking a year off from his medical course to study for a B.Med.Sci. degree under the supervision of Professor Gustav Nossal at the Walter and Eliza Hall Institute for Medical Research (WEHI). Upon completion of his training as a physician specialising in endocrinology at the Royal Melbourne Hospital Tom returned to WEHI to undertake a PhD with Len Harrison, working on the immunological basis of type 1 (juvenile) diabetes. This research set the stage for much of what he has achieved in more recent years.

After obtaining his PhD, Tom worked as a Clinical and Research Fellow at Harvard Medical School in Boston for 3 years. He then returned to WEHI and the Royal Melbourne Hospital as first assistant in the Burnett Clinical Research Unit. Here, Tom focused his research on understanding how the insulin-producing beta cells of the pancreas are destroyed, leading to type 1 diabetes. In addition to his research, Tom supervised the Diagnostic Serology Laboratory which he saw as an essential link for clinical immunology in the hospital and for the training of future clinical immunologists. He also treated patients in the Diabetes and General Medicine clinics at the Royal Melbourne Hospital. Tom's career training places him among a small group of clinician-scientists whose abilities enable them to operate seamlessly in the echelons of science as well as in the hospital ward.

Tom's major achievements are the discovery that cytotoxic T cells are the main white blood cells responsible for destroying beta cells, and the development of a range of genetic approaches to examine the role of different protein molecules potentially involved in beta cell destruction. Tom's research contributions have been recognised by many prestigious honours and awards, including a Career Development Award from the Juvenile Diabetes Research Foundation and a 5-year NIH/JDRF Program Grant. He has a prominent national and international profile, and is often invited to speak at International Scientific meetings. His expertise in the area of type 1 diabetes has resulted in many published review articles and book chapters, and in his appointment to the Editorial Boards of the journals Autoimmunity and the Journal of Molecular Endocrinology. Tom is married to Barbara Demediuk, a gastroenterologist at St. Vincent's Hospital, and they have 3 young children.

This article is a brief synopsis of Tom's history prepared with the assistance of Len Harrison and Anne Thorburn.

switched on proteins



switched on science

During the year Professor Bruce Kemp was made a Fellow of the Royal Society, UK, in recognition of his pivotal contributions towards understanding a basic control mechanism of the body's metabolism. This control mechanism is a switching process that attaches or removes phosphate from proteins. This has the effect of causing subtle changes to the shape of the protein that changes the way it behaves. Bruce's ideas were confirmed when a number of protein structures determined by his research group proved his concept of protein regulation. Illustrated here is the three-dimensional structure of one such protein, twitchin kinase, that Bruce studied to formulate his ideas.

Premier Protein Institute

St. Vincent's Institute of Medical Research (SVIMR) is Australia's foremost protein research institute and one of the leading protein research institutes in the world. The Institute is part of a global network of scientists collaborating to combat disease and improve health.

The study of proteins is today recognised as the foundation of medical research, with virtually all advances in knowledge of disease based on an improved understanding of proteins.

Our discoveries are producing new treatments to prevent cancers spreading to bone, and to prevent osteoporosis and arthritis. We are developing improved treatments for heart disease, obesity, and diabetes. And we are performing fundamental research into infection by the AIDS and hepatitis viruses, the causes of cancer, and brain diseases including Alzheimer's disease and epilepsy.

Major Awards

Professor Bruce Kemp
The Royal Society, UK, recognised Professor Bruce Kemp's important contribution to the field of protein kinases with his appointment as a Fellow of the Royal Society. The Royal Society, established in 1660, elects Fellows from all scientific disciplines within the UK and the Commonwealth as a mark of their distinguished contribution to understanding nature. Professor Kemp joins Professor Jack Martin and a host of other eminent scientists on the honour roll.

Professor Tom Kay
Associate Professor Tom Kay was appointed Professor within the Department of Medicine, The University of Melbourne, in recognition of his contribution to medical research, particularly in the fields of diabetes and autoimmune diseases, and his recent appointment as Director of St. Vincent's Institute of Medical Research.

Professor TJ Martin
TJ Martin was appointed Emeritus Professor of Medicine, The University of Melbourne, and received the Holt Fellowship from the Board of St. Vincent's Institute of Medical Research in recognition of his research in the field of bone biology and for services as Director of St. Vincent's Institute of Medical Research.

Professor Michael Parker
The Royal Society of New South Wales awarded Professor Michael Parker the Walter Burfitt Prize for work of the highest scientific merit performed in Australia or New Zealand over the last 6 years.

Chairman's 2002 Report

This year has been one of achievement and change for SVIMR. We have a new Director. Our new building is progressing and should be completed by September 2003. Our scientists continue to be recognised for their work and our fund-raising efforts are reaping rewards.

Professor Tom Kay, our new director and the fourth since the Institute was founded in 1957, took up his appointment in mid-2002. Tom is a highly regarded diabetes researcher. We welcome him and look forward to a long association.

The new building has progressed rapidly and is now at the stage of the exterior being complete and the interior fit-out is continuing. Good weather and other factors have meant that Bovis Lend Lease have the building a little ahead of schedule at this stage and should with luck be ready for us to move in around the start of September. Already parts of the new building are being occupied with the mass spectrometer room recently being handed over by the builders. The building will approximately double the size of the Institute and bring much needed laboratory space for our current scientists and enable us to recruit new staff. It will also provide much better seminar and conference facilities than have previously been available. An important part of the project has been the planning of new animal facilities, particularly for genetically modified mice that have become an essential tool of modern medical research. The Institute and the Hospital are partnering to build a new facility that will provide shared state-of-the-art housing for mice. We are very grateful for the support of the Hospital in this.

The building is being funded by the Commonwealth and State Governments and the Sisters of Charity. There is an approximately $4m gap between funding from these sources and the budget for the building and members of the Board, particularly Brenda Shanahan, Terry Power and Douglas Wright, have been very active in raising money. The fund-raising brochure they produced, "Appeal for Life", designed by Elaine Hogarty of Origin Design, is an outstanding document that has won industry awards for printing. A large variety of fund-raising activities has been organised including the series of "Director's Dinners" and "Evenings with a Hero" and the establishment of the 1000 Club with the aim of getting 1000 individuals to donate $1000 each. We are very happy with the results of the TV commercial that was produced for the Institute by Wilson Everard. We are grateful for the support we have already received but are very conscious of the big job ahead. We continue to work closely with St Vincent's Hospital Foundation to partner events such as the Ball held in May 2002. In addition we are establishing our own Foundation to support fund-raising for the building and for future research.
A special thank you must go to our Development Officer, Diane Losa, who continues to work with great enthusiasm and commitment on all these projects.

The Constitution of the Institute has been reviewed and updated with the help of Mr Jim Baillie from Cutler Hughes. The main changes are to replace the existing membership structure with three members reflecting our principal stakeholders: St Vincent's Health Melbourne, the Trustees of the Sisters of Charity, and the Sisters of Charity Health Service. The present members have retired, but we are planning new ways to involve them and we continue to value their support and involvement over many years. The new Constitution preserves the close affiliation of the Institute with the University of Melbourne by providing for the University to nominate a director of the Board on the recommendation of the Dean of the Faculty of Medicine. A further part of our ongoing collaboration with the University is our taking up joining membership in the re-launched Bio21 project. Bio21 is a consortium of mainly University associated centres working together to improve facilities for medical research and biotechnology in Melbourne. We look forward to making a strong contribution to the future of Bio21.

There have been several Board changes in the last year. Mr Barry Jackson joined the Board mid-way through the year. Later in the year Mr Terry Power stepped down from the Board and Ms Kerrie Cross was replaced by the new CEO of

St Vincent's Health, Ms Nicole Feely. We were very saddened by the death of Sr Kathleen Higgs late in the year. Further changes have occurred with the introduction of the new Constitution.

We congratulate Prof Richard Larkins both on being made an Officer of the Order of Australia (AO) and on his new position as Vice-Chancellor of Monash University.

The Institute continues to be outstandingly successful in achieving success with peer-reviewed grants from the NHMRC and other organisations. The election of the Deputy Director, Professor Bruce Kemp, as a Fellow of the Royal Society was an outstanding achievement. Of particular note last year was the success of A/Prof DJ "Jock" Campbell in being awarded a grant from the United States National Heart Lung and Blood Institute, a Fellowship from the National Heart Foundation and a Project Grant from the NHMRC. Contracts with the Therapeutics Goods Administration for management of the NRL and the Victorian Breast Cancer Consortium were renewed, which will allow valuable parts of the Institute's activities to continue.

In conclusion, it has been another highly successful year for the Institute with the exciting prospect of the new building nearing completion. Our progress depends on so many people – the scientists of course who work so hard to sustain their success and to discover ways of improving medicine, but thanks must also go to the Administration and to the Members of the Board, as well as to the wider community for their support.

Ian Reid
Chairman

Members of the Institute

Current Institute Members

The following is a list of current Members:

The Memorandum and Articles of Association provides for the appointment of Members of the Institute. They comprise some members of the Hospital Senior Medical Staff, and others from business, the profession and academic life, who are interested in the Institute and wish to promote its activities. Members are kept informed of Institute activities, and are represented on the Institute Board.

Dr. F.P. Alford
Professor J.D. Best
Dr. K.J. Breen
Dr. D.H. Campbell
Mr. J.C. Chappell
Mr. W.J. Clancy
Dr. L.E. Clemens
Sr. Maryanne Confoy
Ms. K.L. Cross
Ms. N. Feely
Dr. J.J. Griffin
Ms. M. Griffin
Mr. C.A. Griss
Mr. J.F. Gurry
Mr. J. Gutman
Sr. Kathleen Higgs (deceased)
Dr. D.J. Hillis
Mr. B.J. Jackson
Ms. M.A. Jackson
Professor E.D. Janus
Professor B.E. Kemp
Professor R.G. Larkins
Mr. Justice A. McDonald
Dr. I.G. McDonald
Professor T.J. Martin
Mr. H.J. Nicholas
Professor D.G. Penington
Sr. Paulina Pilkington
Mr. T. Power
Mr. I.D. Reid
Mr. G.E.N. Rogers
Professor G.B. Ryan
Mr. A.F. Sallmann
Ms. B. Shanahan
Mr. C. Smith
Mr. P. Spry-Bailey
Mr. M.J. Walsh
Mr. D. Wright

Members of the Board

Professor James Best
MD, BS, FRACP, FRC Path, FRCP Edin.

Professor Best is Professor and Head of The University of Melbourne Department of Medicine, St. Vincent's Hospital, Melbourne. He is the Co-Head of St. Vincent's Hospital's Department of General Internal Medicine, and is a Member of the Board of Directors of St. Vincent's Health.

Dr Laurence Clemens
MBBS, FRACP.

Dr Clemens is Director of The Department of Rheumatology, St Vincent's Hospital, Melbourne and Chairman of the Division of Medicine at St Vincent's Hospital Melbourne.

Ms Kerrie Cross
BA, BSW, MHA.
Retired 15th July 2002

Ms Cross recently retired as the Chief Executive Officer of the Sisters of Charity Health Service Melbourne. She is a member of the Boards of Wesley Mission Melbourne and the Brotherhood of St. Laurence.

Ms Nicole Feely
BCom, LLB.
From 22nd July 2002

Ms Feely was recently appointed as Chief Executive Officer, St. Vincent's Health. She has a background in business law, politics and administration in both the private and public sectors.

Ms Marcia Griffin
BA, DipEd, BCom.

Ms Griffin is a Board Member of PMP Communications Ltd, National Pharmacies and is on the Advisory Board for the Carr Design Group.

Mr Barry J Jackson
B.Com (Hons), MAICD.
From 27th May 2002

Mr Jackson is a Director of Paperlinx Ltd, Alesco Corporation Ltd, and Equity Trustees Ltd. He was formerly Managing Director of Pacifica Group Ltd (1995-2001) and has over 30 years experience in manufacturing and industrial marketing.

Mr Charles Griss
FCPA, FCA, FAICD.

Mr Griss is a former Senior Executive of ANZ Banking Group Ltd and former Managing Director of Esanda Finance Corporation Ltd. He is a Director of the SCHS Melbourne Region Board, and Chairman of both the Quality of Audit Committee and Community Advisory Committee for the SCHS Melbourne Region Board.

Sr Kathleen Higgs
RSC, RN, BA, Grad. Dip.
Health Services Management.
Deceased 22nd November 2002

Sr Higgs was missioned to the role of Clinical Risk Manager, St. Vincent's Hospital Melbourne (SVHM). She had a broad background in healthcare having worked extensively in both clinical and administrative areas.

Mr Terrence Power
FCPA, FAICD.
Retired 25th October 2002

Mr Power was responsible for Business Development, Marketing and Client Services in the Funds Management Division of the BT Financial Group until January 2000. He was a former non-executive Director of BT Funds Management Ltd, is Chairman of the Investor Group (a listed public company) and is an Industry Member of the Financial Industry Complaints Services Ltd Panel. He is an Associate Fellow of the Australian Marketing Institute and Executive Member of the American Marketing Association.

Professor Richard G Larkins
MDBS, PhD, FRACP, FRCP.

Professor Larkins is Dean of the Faculty of Medicine, Dentistry and Health Sciences and Head of the School of Medicine at The University of Melbourne. Past positions have included: Chair of NHMRC, President of the Endocrine Society of Australia, Chair of the Accreditation Committee of the Australian Medical Council and President of the Royal Australasian College of Physicians.

Mr Ian D Reid
BE (Chem), ASA, FIEAust, MAICD.

Mr Reid comes from a manufacturing and industry background. He is a Director of Advanced Riverina Holdings and a Board Member of the Brotherhood of St. Laurence and the Melbourne Anglican Foundation.

Mr Graham Rogers
FIA, FIAA, ASA.

Mr Rogers is Chairman of SMF Funds Management and serves on the Boards of RACV Financial Services, the Private Health Insurance Administrative Council, and the Athaeneum Club. He is Senior Vice President of the Institute of Actuaries of Australia, Chairman of The University of Melbourne Actuarial Foundation and is Principal of the Offley House Group. His background includes more than 25 years as a chief executive in the financial services industry including Colonial Investment Management, Jacques Martin Group and Equitable Life.

Ms Brenda Shanahan
BEc, BCom.

Ms Shanahan has a research background in finance in Australian and overseas economies and sharemarkets. She is a Non-Executive Director of AWB Ltd and AWB International Ltd, a Director of St. Vincent's Health, and is a Board Member of Challenger International Ltd. and JM Financial Group Ltd. She is a former member of the Australian Stock Exchange and former Executive Director of a stockbroking firm, a fund management company and an actuarial company.

Mr Douglas Wright
FAICD.

Mr Wright is a Founder and Managing Director of Wrights, an Australian-owned creative communications consultancy. He is a public affairs strategist, and has worked in the media and business in Australia and Europe. He is Chairman of the Victorian Government's Small Business Advisory Council. Mr Wright is a Member of the Public Relations Institute of Australia, the Counsellors' Academy of the Public Relations Society of America, and an Associate Member of the Australian Marketing Institute and Institute of Public Relations (UK).

VALE - Sr Kathleen Higgs RSC. 1938 - 2002

We are sad to record the death of Sister Kathleen Higgs RSC, a greatly respected and loyal member of the Institute Board, who passed away after a long illness on November 22, 2002. In taking on Board membership some years ago to represent the Sisters of Charity, Sister Kathleen took part in the Institute activities with all the thoroughness and dedication that she applied in other areas throughout her life.

Born in Bathurst, NSW, Kathleen was one of four children born to Jack and Madge Higgs who predeceased her. Her family were with the Sisters of Charity and the St Vincent's community at the wonderful liturgy that celebrated her life. The liturgy was graced by the beautiful music that Kathleen herself chose for the occasion.

After spending her childhood in Bathurst and undertaking her nursing training at St. Vincent's Hospital, she worked in several hospitals in country NSW, living experiences that developed in her a tremendous compassion for all those who needed her care. Her close contact through those years with the Sisters led Kathleen to enter the Congregation of the Sisters of Charity in the early 1960's. Her ministries in health care within the congregation included intensive care nursing, nursing administration and hospital management. Kathleen had a deep commitment to the nursing profession and was instrumental in introducing many innovative practices into the profession. Many of these innovations continue today.

Her commitment to nursing was recognised by her admission to Fellowship of the Royal College of Nursing (Australia). She was overjoyed with this privilege.

One of the highlights of Kathleen's life was the 9 months she spent in Vietnam as a member of the medical team from St. Vincent's Hospital Sydney. This experience deeply influenced her concern for the poor and the sick in Third World countries, especially those torn by war.

In the midst of her busy nursing life she had the desire to study comparative religions and for this reason she completed a Bachelor of Arts Degree at Sydney University majoring in Celtic spirituality. Kathleen also attained post graduate qualifications in health administration. Her diverse interests included painting, music, lapidary, ceramics and photography.

When diagnosed with cancer last year Kathleen faced her illness with her usual courage and with the deep faith that sustained her during this time, and took her to the end of her illness with great dignity. In speaking of Sr Kathleen in the Eulogy, Sr Paulina Pilkington RSC expressed our belief that Kathleen is now enjoying the peace that we all seek. She concluded with the words of St. Thomas Moore,: "Pray for me, as I will for thee, that we may merrily meet in heaven."

Edited transcript of the Eulogy delivered by Sr. Paulina Pilkington RSC.

2002 in Review

The Institute has its focus on future changes in medicine and improvement of individuals' lives by discovering new knowledge.

Taking up my new position has made the last 12 months an incredibly exciting if daunting time. I would like to thank everybody at the Institute and in the Hospital who have been so welcoming and helpful. In accepting the Directorship I was very aware of the outstanding position that the Institute occupies in Australian and international scientific life. Most of you will be aware that the Institute is regularly ranked at the highest level of Australian medical research institutes for publications, patents and grants, particularly when its relatively small size is taken into account.

It receives more competitive funding from the NHMRC than many Universities. This reputation has been won through continued effort over the 45-year history of the Institute – from the historic early days of Pehr Edman and his automated protein sequenator through to the present. Jack Martin has left the Institute in great shape and we are lucky to continue to have his input and guidance as the John Holt Fellow of the Institute.

The Institute has its focus on future changes in medicine and improvement of individuals' lives by discovering new knowledge. We do this primarily by basic laboratory based research that usually takes years of testing before it can be applied in the clinic. The discovery of the hormone PTHrP by Jack Martin and his group in the late 1980's is only now being applied clinically to patients with breast cancer. In my own field of Type 1 (or juvenile) diabetes, research into transplantation of insulin-producing pancreatic islets begun about 20 years ago is now bearing fruit with successful transplants being carried out over the last 3 or 4 years. Medicine is always changing and in most fields becomes dramatically different over a 20-year period. It is inspiring to think about which current basic laboratory projects of the Institute will impact on the practice of medicine in the future – not all will, of course, but some certainly will.

The theme of the Institute continues to be a combination of disease-focused programs underpinned by outstanding "enabling" technologies. The Institute has major programs in diseases of bone, particularly metastasis of cancers to bone, but also osteoporosis and arthritis, and in cancer, diabetes, and cardiovascular diseases, as well as efforts directed towards neurological diseases (epilepsy and Alzheimer's disease) and virology. We have excellent facilities and resources for protein chemistry and crystallography, continuing themes that can be traced back to the Edman days. An example of this is the new mass spectrometer purchased with the support of several philanthropic trusts and in use for most of the past year.

With the development of the new building, the increased level of funding through the NHMRC, and increased infrastructure support for Medical Research Institutes announced in the last State budget, the Institute will naturally grow, with opportunities for young Australians working overseas to return to the Institute and opportunities to bring in some more senior researchers. The Institute will remain smallish however. This means that we will always rely on collaboration to achieve critical mass. Our key collaborators will include researchers on the Hospital campus and researchers nearby including The University of Melbourne and its affiliated centres. The Institute has become a joining member of Bio21, The University's Biotechnology Consortium. There are some advantages of small size including autonomy and lack of bureaucracy that should help us remain a dynamic and responsive research centre. An especially important collaboration to mention is in the area of diabetes. Diabetes groups on the St Vincent's Hospital campus have joined together to form a Centre of Excellence with the strong support of the Hospital.

Our new focus on diabetes in the Institute will contribute to this, as will the expertise in diabetes complications in The University of Melbourne Department of Medicine, expertise in transplantation in the Hospital's Department of Clinical Immunology, and the clinical expertise within the Department of Endocrinology and Diabetes in the Hospital. These collaborations that bring together activities from basic laboratory research and best clinical practice must be the way of the future in enabling research and innovation in medicine, to improve the health of the community.

2002 Achievements
of SVIMR scientists

Discovery of proteins that protect against diabetes
We found that the protein called "suppressor of cytokine signaling" (SOCS-1) protects the insulin-producing pancreatic islets from the damaging effects of pro-inflammatory cytokine molecules.

Discovery of proteins that control the strength of bone
We identified several proteins that stop bone breakdown. These may lead to new treatments for diseases where bone loss is a major problem such as osteoporosis, rheumatoid arthritis, and cancer-induced bone diseases. We determined that proteins contained within bone can stimulate the production of bone-destroying cells.

Discovery of a fish hormone will teach us more about human bones
We were the first to discover parathyroid hormone in fish.

Cancer metastasis model
We developed the first model system for human mammary epithelial-mesenchymal transition, a pathway to metastasis in cancer.

Matrix metalloproteinases in bone metastasis
We are among the first to show a major role for this class of enzymes in the spread of breast cancer to bone.

Development of animal models of prostate cancer
These animals develop prostate cancer very similar to that which occurs in patients, and will help us develop new treatments for this disease.

Gene array technology reveals secrets of cancer and diabetes
Our application of gene array technology has revealed those genes that behave differently in cancer and in diabetes, and will assist the development of new treatments.

Control of blood vessel relaxation
We identified chemical switches on the enzyme endothelial nitric oxide synthase that controls blood vessel relaxation.

Discovery how the enzyme AMP-activated protein kinase is regulated in cells
We developed a technique to search for proteins that "switch on" the stress-monitoring enzyme, AMP-activated kinase.

Understanding McArdle's disease
We discovered how patients with muscle weakness due to McArdle's disease, a glycogen storage disease, regulate their muscle metabolism during exercise.

Understanding the actions of blood pressure medications on the kidney
We showed that different medications for high blood pressure have different effects on the hormone angiotensin in kidney. These results help explain why some medications provide better protection of the kidney from disease than other medications.

Understanding how cells protect themselves from becoming cancers
We identified mRNA poly(A)-tail length control as a novel mechanism involved in the cellular response to DNA damage.

Molecular analysis of a beating heart
We demonstrated that the S100A1 calcium binding protein is required for the normal contractile response of the heart to increased haemodynamic demand.

Protein switches in the brain
We have built a molecular model of the GABA receptor, a protein in the brain that is the target for various anti-anxiety drugs, anti-convulsants, sedatives, depressants, anti-epilepsy drugs, alcohol, and anaesthetics. This model will assist the design of new and improved drugs for the treatment of many neurological disorders.

Understanding Alzheimer's disease
We determined the 3-D structure of a domain of the amyloid precursor protein, a key determinant in Alzheimer's disease. The structure reveals how copper binds to the protein and provides insights for the design of drugs to combat this disease.

Understanding liver inflammation in hepatitis
We determined the E2 binding site of the human CD81 molecule. This has important implications for understanding the inflammation that occurs when the liver is infected by the hepatitis C virus.

Diabetes

Approximately one in every 200 Australians has Type 1 diabetes.

Diabetes Unit

Thomas Kay
Nadine Dudek
Eugene Estella
Rochelle Fernandes
Amanda Handley
Emma Jamieson
Helen Thomas
Ann Thorburn

Overview of Research and Benefits to Society

People with Type 1 diabetes lack insulin, which is a hormone that regulates the metabolism of glucose. This occurs because their immune system mistakenly attacks and destroys the insulin-producing cells in their pancreas. These insulin-producing beta cells (ß-cells) are contained within small clumps of cells called islets in the pancreas. Type 1 diabetes is a major burden because of the lifelong need for several daily insulin injections and finger prick tests to control blood glucose levels, as well as the problems of long-term complications. Approximately one in every 200 Australians has Type 1 diabetes. The incidence of Type 1 diabetes is increasing, especially in children less than five years of age. Reduced insulin production is also frequently seen in the more common form of diabetes called Type 2 diabetes. Scientists in the Diabetes Unit at SVIMR are investigating how ß-cells are destroyed. Our discovery of several proteins that cause ß-cells to self-destruct provides new approaches to the treatment and prevention of Type 1 diabetes.

Fellowships and Prizes

Dr Helen Thomas
JDRF Advanced Postdoctoral Fellow

Grants

THW Kay and HE Thomas
T-cell mechanisms of ß-cell destruction.
NHMRC/JDRF Special Program Grant. (5-year grant support)

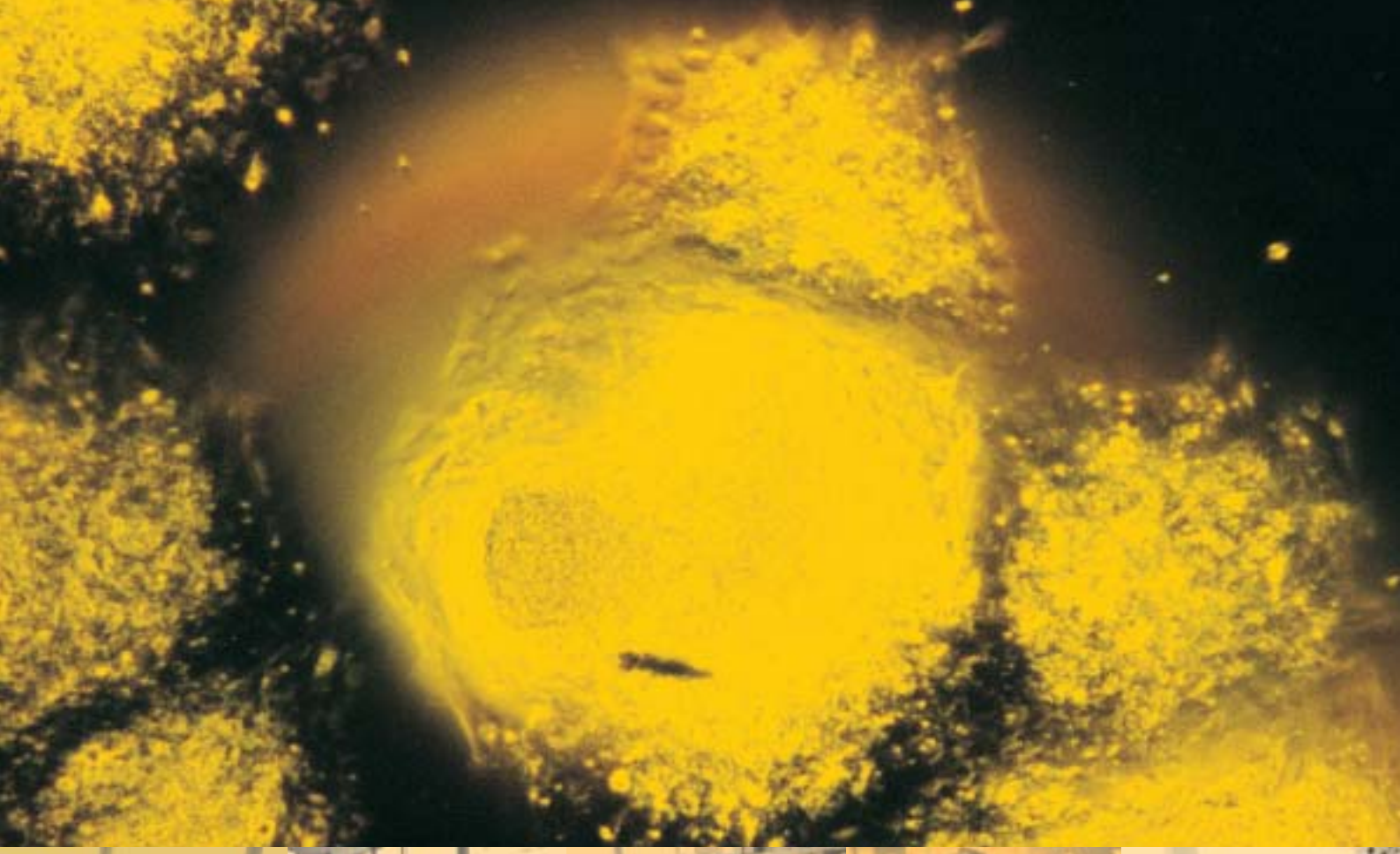
Identification of Multiple Pathways of ß-cell Death

Type 1 diabetes is a complex process and many different proteins and cells play a role in this disease. In addition, the disease process can be different in different individuals. To identify the proteins that play a role, we are using a range of different mouse models that develop Type 1 diabetes similar to the disease that occurs in patients. We are using genetic techniques to investigate how changes in certain proteins modify the disease process, with the aim to discover new treatments and possible ways to prevent Type 1 diabetes.

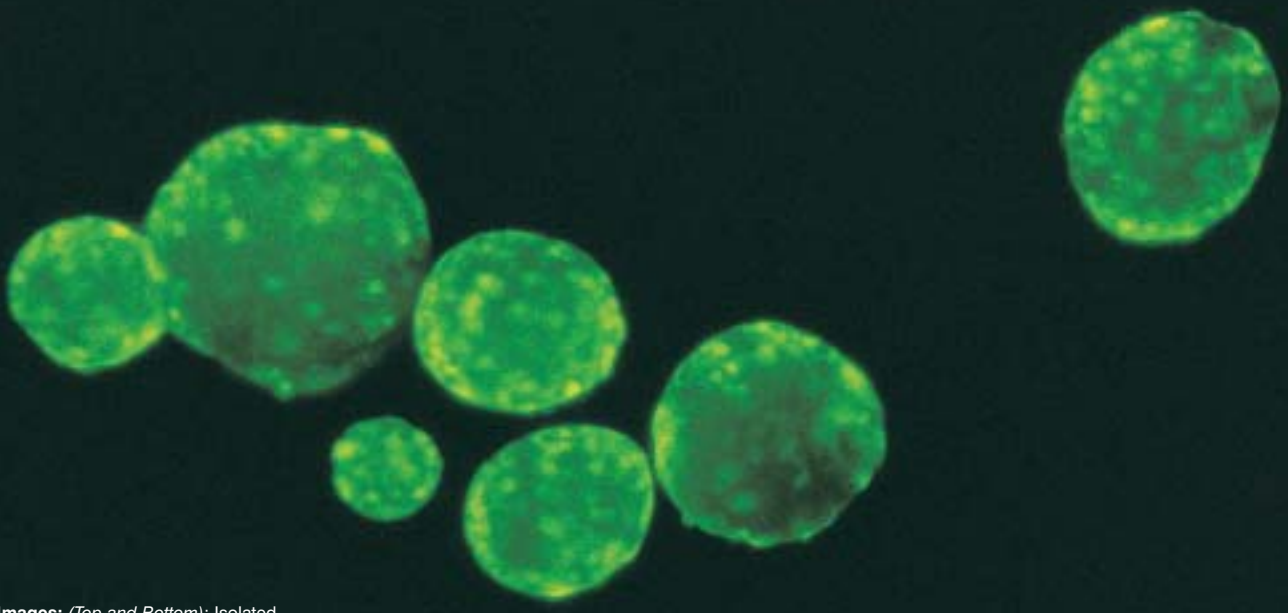
Several cytokines (hormones of the immune system that are involved in inflammation) and cell surface receptors that lead to cell death when activated such as Fas and TNFR1 have been implicated in ß-cell destruction, as well as perforin, a molecule packaged within cytotoxic or "killer" T-cells. We have blocked these pathways in diabetic mice by introducing additional genes that inhibit them or by removing genes required for their activity. These studies showed that ß-cell destruction in diabetic mice proceeds by several mechanisms. Just as different mechanisms operate in different strains of diabetic mice, it is likely that a similar spectrum of mechanisms also operates to a variable extent in different individuals who develop diabetes.

Function of SOCS-1 in ß-cells and the Immune System

In addition to study of protein molecules that damage islets, we are studying molecules that protect islets from damage so that we may exploit these natural protective mechanisms to ensure survival of transplanted islets, and perhaps to prevent diabetes. One promising group of protective molecules is the suppressor of cytokine signalling (SOCS) family of proteins. We are investigating the role of SOCS proteins in the function of the immune system. The SOCS proteins are expressed in ß-cells at quite low levels. By increasing production of SOCS proteins, ß-cells are protected from the action of several cytokines and are protected in some, but not all, models of diabetes. We have also identified important roles for the SOCS-1 protein in metabolism. Mice deficient in the SOCS-1 protein have low blood glucose levels and we found that they have enhanced insulin sensitivity in the liver and also have ß-cells that secrete more insulin in response to glucose. These findings help us understand the role of SOCS proteins in disease and their potential as therapy.



Diabetes



Images: *(Top and Bottom)*: Isolated pancreatic islets.

Human Islet Transplantation

Transplantation of insulin-producing pancreatic islets is a successful treatment for Type I diabetes, but has the major drawback that large numbers of islets must be transplanted because many of the islets do not survive transplantation. The need to transplant large numbers of islets is a barrier to the wider application of this enormously promising therapy. We also need to find less harmful alternatives to the long-term use of immunosuppressive drugs to prevent rejection of transplanted islets. Toxic molecules produced by white blood cells play a key role in the death of islet cells that occurs during and after transplantation.

Protection of islets from these damaging molecules is thus likely to be critical to improving the success rate of islet transplantation, in addition to finding alternatives to immunosuppressive drugs. Over the next two years we will translate the work we have done in mouse models to a more clinical level by making human ß-cells more resistant to the toxic molecules that attack and destroy them after transplantation. With further development in this area and an adequate supply of ß-cells (potentially through stem cells or animal ß-cells), islet transplantation may become an effective treatment for the majority of patients with Type 1 diabetes.

Bone Biology

Osteoclasts are the cause of bone and joint damage in rheumatoid arthritis.

BONE BIOLOGY AND DISEASES

Our skeleton acts as the structural support for our body, and also provides a reservoir for growth factors and calcium that is important for many of our organs. In order for a healthy skeleton to be formed and maintained it needs to be continually built and remodelled during life. Attaining a peak bone mass at adulthood, we then lose bone as a normal consequence of ageing. Both the peak bone mass and rate of bone loss are influenced by our genes and environment, and our diet and exercise.

Our Aim

We have an integrated research program (basic to clinical) with the focus of understanding the processes that build and break down bone during normal growth and in conditions with accelerated bone loss, such as osteoporosis, arthritis and the spread of cancer to bone. We aim to identify those molecules responsible for building new bone, and/or stopping the breakdown of bone, so that we can use these molecules to design new treatments for these diseases.

Inhibitors of Bone Destruction

One of the main clinical approaches to maintaining the skeleton is to limit its destruction, using drugs that inhibit bone breakdown. Two examples of this type of drug that are commonly used in clinical practice are bisphosphonates and a class of drug known as SERMs. However, there is a pressing need to develop new treatments to prevent bone destruction. We have identified several new molecules that stop the formation or activity of cells called osteoclasts that have the specialised function to degrade bone. We demonstrated that these molecules prevent bone destruction in laboratory studies and we are now translating this work to appropriate animal models of bone disease, with the aim to ultimately develop new medicines. We have taken out patents to protect the intellectual property for each of the new molecules we discovered.

In identifying these new inhibitors of bone degradation, we discovered the role of a cell that is resident in bone, but which has been little investigated for its function in the control of bone structure. This cell is the T lymphocyte (T-cell) that is involved in the immune response against viral and bacterial infections. Our studies identified compounds that stimulate T-cells to produce a vast array of very potent protein molecules that can either inhibit or enhance bone destruction. These protein molecules made by T-cells are important in diseases where an immune response is evident, particularly in conditions such as rheumatoid arthritis and bone cancer. In addition to our previous discovery of the cytokine IL-18 as an inhibitor of osteoclast formation, we showed that IL-12 also inhibits osteoclast formation. We are investigating other inhibitors of osteoclast formation made by T-cells.

Towards Treatments for Rheumatoid Arthritis

Our laboratory work on osteoclast regulation is improving understanding of the bone loss associated with inflammatory bone diseases such as rheumatoid arthritis. Rheumatoid arthritis is an inflammation of joints that produces bone destruction. This bone loss causes painful joint deformities, progressive functional disability, an increased risk of bone fractures and increased mortality rates. Until recently, the bone destruction that occurs in rheumatoid arthritis was thought

Bone Biology and Diseases

TJ Martin, Head

Bone Physiology
Jane Moseley, Head
Hannelore Diefenbach-Jagger
Patricia Ho
Ginny Leopold
Pat Smith

Clinical Bone Endocrinology
Kong Wah Ng, Head
Jeanette Dickins
Christine Gange
Vicky Kartsogiannis
Ingrid Kriechbaum
Chi Ly
Natalie Sims
Hong Zhou

Comparative Endocrinology
Janine Danks, Head
Patricia Ho
Lisa McCarthy

Molecular Endocrinology
Matthew Gillespie, Head
Elizabeth Allan
Jan Elliott
Jane Fisher
Daphne Hards
Karl Häusler
Natasha Ilievska
Danijela Mirosavljevic
Julian Quinn
Evange Romas
Rachel Thomas

Fellowships and Prizes

Dr Natalie Sims
RD Wright Biomedical Career Development Award from the NHMRC to study novel mechanisms for the control of osteoclastogenesis in bone and joint diseases.

Dr Rachel Thomas
CJ Martin Post Doctoral Fellowship from the NHMRC to be undertaken with Prof. J-P Thiery at the Institut Curie, Paris, France.

Ms Natasha Ilievska
St. Vincent's Hospital Scholarship.

Ms Danijela Mirosavljevic
St. Vincent's Hospital Scholarship.

Grants

MT Gillespie and JL Fisher. Breast cancer growth in bone. Eli Lily Women's Health, USA. (1-year grant support)

MT Gillespie and JL Fisher. Role of osteoprotegerin in breast cancer growth in bone. National Breast Cancer Foundation Grant. (3-year grant support)

MT Gillespie and JL Fisher. Mechanisms of breast cancer metastasis. Department of Defense, US Army. (3-year grant support)

TJ Martin and MT Gillespie. Identification of new anabolics. Chugai Pharmaceuticals Co. Ltd. Japan. (1-year grant support)

E Romas and N Sims. Altering osteoclast formation and function in rheumatoid arthritis to prevent joint destruction. NHMRC Project Grant. (3-year grant support)

to be due to direct invasion of bone by the inflamed tissue. Our studies show that osteoclasts are the cause of bone and joint damage in rheumatoid arthritis, and we are investigating whether compounds that stop the activity of these cells may be used to treat the disease. Our initial studies showed that these treatments prevent joint damage, although they do not prevent inflammation or cartilage damage. In future studies we will investigate whether the combination of different treatments provides more effective control of this disease.

What Influences Breast Cancer Growth in Bone?

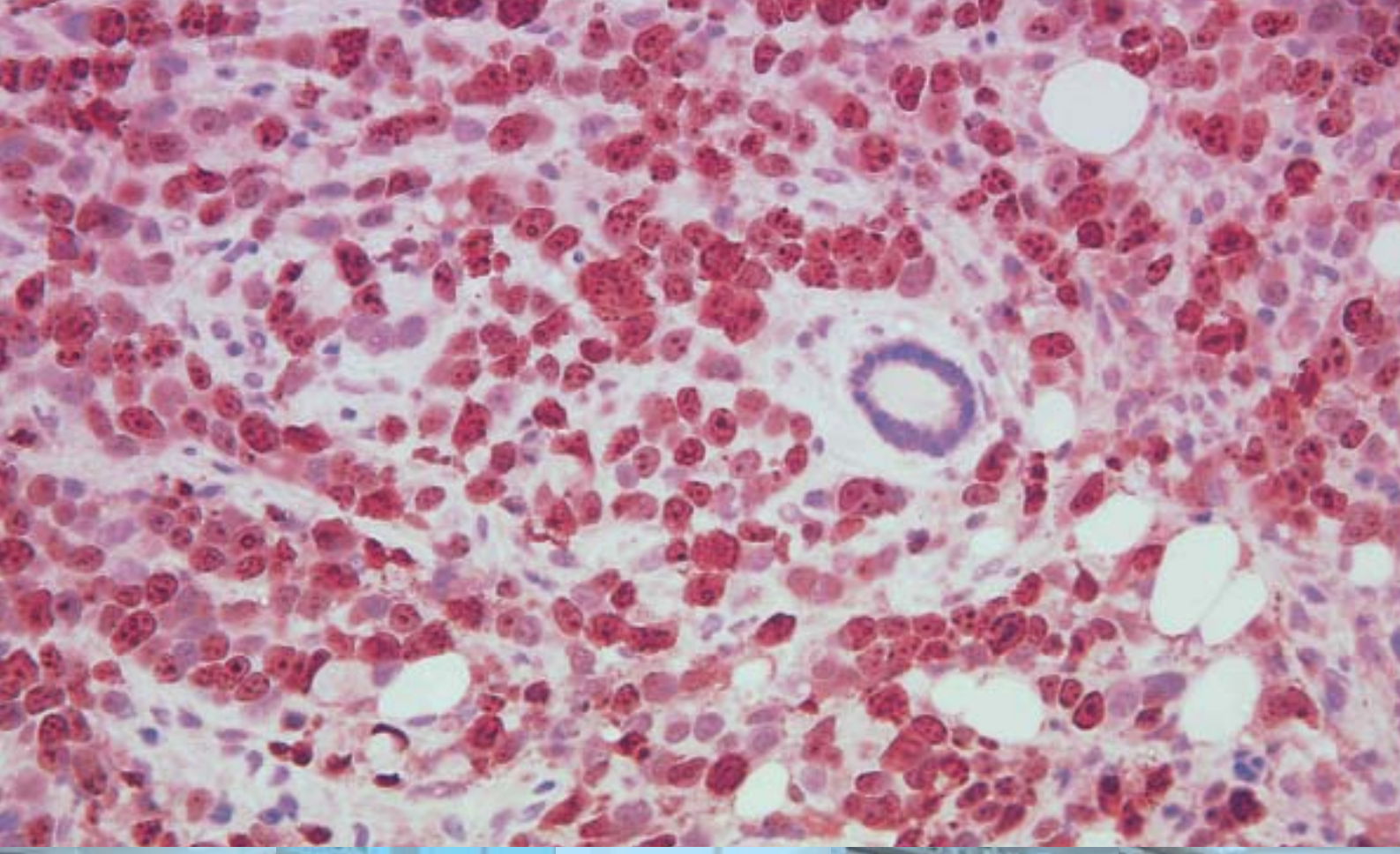
Several years ago we discovered a protein called parathyroid hormone-related protein (PTHrP) because of its close relationship to parathyroid hormone. We showed that PTHrP is produced by a number of different cancers, principally those of the head and neck, lung, breast and kidney. Our experimental studies showed that cancers that make increased amounts of PTHrP are able to grow in bone, causing bone destruction. These findings suggested that PTHrP production by breast cancers might contribute to the spread of these cancers to bone. However, in a large prospective clinical study of PTHrP expression in breast cancer, we found that PTHrP production by breast cancer is associated with improved survival and a lesser likelihood for the cancer to spread to other parts of the body, including bone.

Our experimental and clinical studies show that PTHrP can have different effects depending on where it is made. If a breast cancer makes PTHrP, it provides some protection against spread to other parts of the body. However, if a cancer has spread to bone, then PTHrP production can accelerate the growth of the cancer in bone.

In addition to its role in cancer, PTHrP plays an important role in the regulation of normal cells. We are investigating these functions of PTHrP and how it may regulate the function of genes in these cells.

Lessons from Fish

To better understand the role of PTHrP in normal cell function we are studying lower vertebrates, including fish. We have shown that PTHrP is expressed in a number of tissues in the fish, and we have for the first time identified parathyroid hormone in fish. These studies in fish will help us develop new treatments for bone disease or build new bone.



Bone Biology

Images: Histological tissue sections.(*Top)*: breast cancer cells stained for eostrogen receptor. *(Bottom)*: bone cells.

Cancer Research

The VBCRC group has focussed on matrix metalloproteinase-2, an enzyme that degrades proteins surrounding a cancer.

VBCRC Invasion and Metastasis Unit

Erik (Ric) Thompson, Head
Margaret Bills
Tony Blick
Masha Fridman
Nicolle Gibson
Marc Lafleur
Erin Ring
Elizabeth Williams

THE JOINT INVASION AND METASTASIS UNIT (JIMU)

The Joint Invasion and Metastasis Unit (JIMU) is a collaboration between a number of scientists whose research is focussed on the mechanisms by which cancer spreads to other parts of the body (a process called metastasis), and the development of new treatments to prevent metastasis. The JIMU includes the Victorian Breast Cancer Research Consortium (VBCRC) Invasion and Metastasis group and operates in close collaboration with The University of Melbourne Department of Surgery and the Bernard O'Brien Institute of Microsurgery (BOBIM) at St. Vincent's Hospital. A/Prof. Thompson leads an active research team performing research into invasion and metastasis of breast cancer. Other principals include Annet Hammacher (prostate cancer); John Price (SVIMR, cell migration and bone metastasis); Mark Waltham (SVIMR, pharmacogenomics) and Elizabeth Williams (BOBIM, prostate cancer progression and bone metastasis).





Grants

E Thompson, M Waltham, D Newgreen, L Ackland and M Henderson. Molecular markers for epithelio-meschymal transition in human breast cancer. Department of Defense, US Army. (3-year grant support)

MMP-2 Activation in Breast Cancer Progression

For many years, this VBCRC group has focussed on matrix metalloproteinase-2 (MMP-2), an enzyme that degrades proteins surrounding a cancer, thus allowing the cancer cells to escape from the primary site and to metastasise. This enzyme is produced by normal cells around the cancer and is activated on the cell surface. MMP-2 is also activated by individual cancer cells that have changed their properties (undergone the Epithelio-Mesenchymal Transition, EMT), and MMP-2 activation may help them spread from the primary cancer. Such activation on the cell surface is not automatic and we have shown that some tissue proteins can influence this process. Further knowledge of how these proteins enable the breast cancer cells, and normal host fibroblasts, to activate MMP-2 may assist the design of new treatments to block MMP-2 activation and to stop metastasis of cancer.

We showed that cancer cells that make MMP-2 grow faster and metastasise more frequently. These studies complement very well those of Dr Mark Waltham, who has shown that inhibition of all MMPs with a broad-spectrum inhibitor blocks both growth of the primary cancer and its metastasis to bone. We are also studying how MMP-2 is activated ("switched on") by other enzymes and proteins.

We have been recognised internationally for our contributions in this field. Dr Marc Lafleur was invited to present this work at the American Association for Cancer Research Special Conference on Proteases, the ECM and Cancer, Hilton Head Island, South Carolina, and A/Prof Thompson was invited to address this topic at the 25th San Antonio Breast Cancer Symposium in December 2002.

Molecular Basis of Breast-Bone Metastasis

The VBCRC group continues to work in collaboration with Dr John Price on the analysis of breast-bone metastasis with human cancer cell line model systems. We have performed post gene array validation of candidate genes and will continue to analyse clinical specimens, searching for genes that are turned on or off in metastasis. Further gene array analysis of the other bone metastasising variants of the cancer cell line MDA-MB-231 are in progress.

Prostate Cancer

Dr Elizabeth Williams has developed an animal model that develops metastasis to bone in a manner very similar to that which occurs in patients with prostate cancer. These studies will allow us to define better the mechanisms by which prostate cancer spreads to bone and enable us to develop drugs to treat this diseases.

Cancer Research

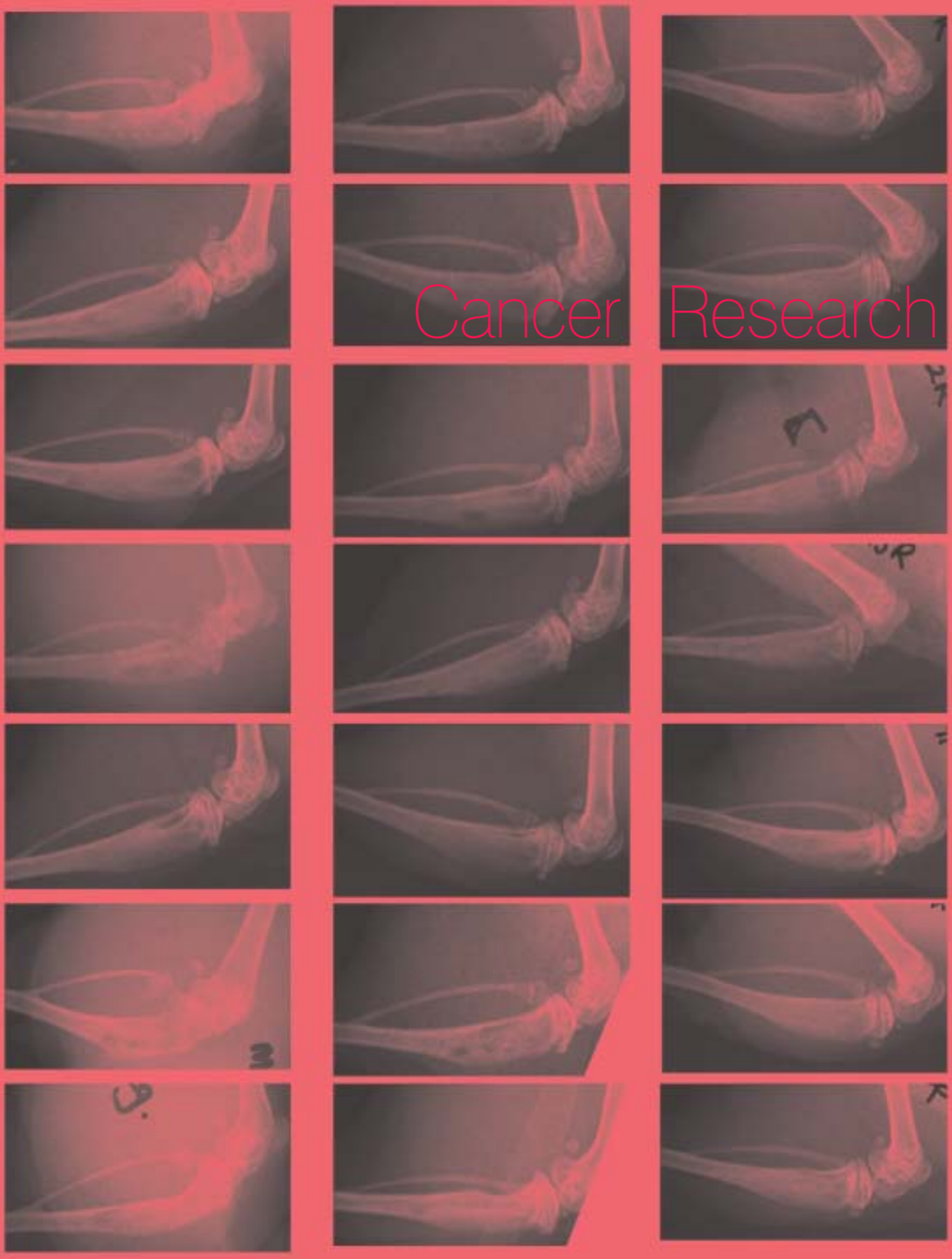


Image: Autoradiographs of osteolysis - tumour cells which dissolve the bone.

Cancer Research

An important part of cancer is the change of a normal cell to a cancer cell, one that keeps growing and dividing when it should not.

Bone Metastasis and Migration Laboratory

John Price, Head
Susan Docherty
Jessica Moore
Joe Pereira

Fellowships and Prizes

Dr John Price
St Vincent's Hospital Foundation Senior Investigator Oral Award, St Vincent's Hospital Research Week 2002.

BONE METASTASIS AND MIGRATION LABORATORY

An important part of cancer is the change of a normal cell to a cancer cell, one that keeps growing and dividing when it should not. This change from a normal cell to a cancer cell and then to a more aggressive cancer cell that spreads to other parts of the body is the major cause of poor clinical outcome in patients. Rather than being a totally random process, different cancers display preferential spread to particular organs. For example, breast cancer displays a high predilection to metastasise to bone. For those patients dying from carcinoma of the breast, approximately 85 percent have demonstrable metastasis to bone. Although metastasis is a highly complex and multi-step process, the ability of the cancer cells to actively migrate is fundamental to the process. Identification of molecules involved in the process of cell migration is important for the development of treatments that prevent metastasis.

One of our major objectives in the laboratory is to identify genes that are involved in the process of breast cancer cell migration and bone metastasis. To achieve this we identified a number of human breast cancer cells with increased potential to cause bone metastasis, and found that these cells also have an increased ability to migrate. Use of gene array technology has led us to identify a number of genes that may be involved in breast cancer cell migration and bone metastasis. We are currently focussing upon one of these molecules, HSP90, as drugs that inhibit its action currently exist. Through the use of these drugs we identified HSP90 as a molecule that is involved in breast and ovarian tumour cell migration and we are now investigating whether HSP90 also plays a role in metastasis of breast cancer to bone.

Our laboratory is using a high throughput migration screening strategy to identify new drugs that are able to directly inhibit cancer cell migration. These drugs may prevent metastasis and enable more effective treatment of cancer.

PHARMACOGENOMICS

The human genome project has provided an extremely valuable resource for the identification of genes that cause disease. As a parallel to this, the development of new drugs and the concept of tailoring patient-specific therapies are benefiting from this new information and the associated technological advances. The focus of the pharmacogenomics group is to apply these new technologies, principally gene expression profiling and proteomics, to define the mechanisms of cancer and to identify how drugs work.

One of the methods we use to identify genes that cause disease is called gene array or gene expression profiling. This method allows us to study thousands of different genes in a tissue to identify those genes that are expressed differently in disease and that may contribute to the disease.

One of our main focus areas is breast cancer where we are discovering genes that cause breast cancer cells to become more aggressive and to metastasise. Currently 15 genes and associated pathways have been identified and we are perusing these as potential diagnostics for clinical use and as novel therapeutic targets. This project is complemented by bioinformatic studies which utilise the ever growing tissue and disease expression databases that are available, and is performed in collaboration with local and international groups.

In another project area and using mouse models of breast cancer, we have identified two drug molecules that are capable of inhibiting onset of osteolytic bone damage associated with metastasis. While these drugs do not inhibit metastasis to other sites, this bone specific effect is potentially of considerable clinical significance. For this project we have also started gene expression profiling studies in order to determine the precise molecular action of these drugs and the reason for bone specificity.

We are also applying our expertise to the identification of genes associated with diabetic renal disease, in collaboration with R Gilbert, University of Melbourne Department of Medicine, St. Vincent's Hospital.

Pharmacogenomics

Mark Waltham, Head
Angela Arvanitis
Tony Blick
Maria Kamarinos
Emma Walker

Grants

M Waltham and R Gilbert. Microarray profiling micro-vasculature complications in diabetes. NHMRC/JDRF Special Program Grant. (5-year grant support)

Protein Chemistry and Regulation

PROTEIN CHEMISTRY AND REGULATION

Diet and exercise are key factors in maintaining health and extending life. We are interested in the biochemical events that occur in response to diet and exercise and how they determine well being. The opportunity to learn about this came from our identification of an enzyme called the AMP-activated protein kinase (AMPK) that is a major metabolic stress-sensing enzyme. AMPK has aroused tremendous interest by academic as well as pharmaceutical industry laboratories because of its potential importance in diabetes, obesity and other age onset diseases. AMPK functions in the body to control metabolism and gene function in response to energy demand (exercise) and supply (calories). During exercise AMPK is activated and it accelerates glucose uptake and fatty acid oxidation, the burning of fat, to produce energy. In tissues such heart, kidney, and placenta, it also accelerates glycolysis, the metabolism of glucose. In addition to enhancing metabolism to produce energy, AMPK switches off the synthesis of fatty acids, cholesterol and triglycerides in the liver and adipose tissue. AMPK does this by attaching a phosphate molecule (a process called phosphorylation) to key regulatory enzymes in these metabolic pathways, as well as to regulators of gene function. The importance of AMPK has been dramatically highlighted by the discovery that mutations in this enzyme cause heart disease. There is also evidence that AMPK may have a role in aging.

The AMPK enzyme is "switched on" by another enzyme called AMPK kinase (AMPKK). In collaboration with Dr Lee Witters at Dartmouth Medical College in New Hampshire, USA we developed a method for measuring AMPKK activity in muscle biopsies from exercising volunteers. Previously, we found that AMPK phosphorylates and activates the enzyme endothelial NO Synthase (eNOS) in ischaemic hearts. This enzyme is responsible for producing nitric oxide that is an important regulator in the cardiovascular system. We have now identified a number of enzymes that regulate eNOS activity that may contribute

Protein Chemistry and Regulation

Bruce Kemp, Head
Julian Adams
Zhiping Chen
Peter Hoffmann
Tristan Iseli
Ian Jennings
Frosa Katsis
Belinda Michell
Sid Murthy
Gregory Steinberg
Bryce van Denderen
Jade Woon

Visiting staff
Professor Erich A Nigg
Ms Elena Nigg

Image: *(Top of page)*: Purified AMP-Kinase on a gel.

The focus of our research is to understand how the AMPK enzyme performs its functions and how it is regulated. The AMPK is an enzyme that monitors the energy levels of the body.

to normal function of blood vessels, and also to disease of blood vessels.

We also undertook collaborative studies with Prof. Erick Richter's laboratory at the Copenhagen Muscle Research Centre, University of Copenhagen in Denmark. We studied patients with chronically high muscle glycogen stores and deficient glycogen breakdown (McArdle's disease) to assess the influence of high glycogen levels on AMPK activation.

Another major milestone was the 2nd International Conference on AMP-activated protein kinase held in Dundee, Scotland, in September 2002 where Prof. Bruce Kemp gave the opening plenary lecture. Many of the Institute's staff working on AMPK attended the Dundee meeting and Bruce Kemp was elected to chair the 3rd International Conference on AMP-activated protein kinase to be held at Lorne, Victoria, in March 2004.

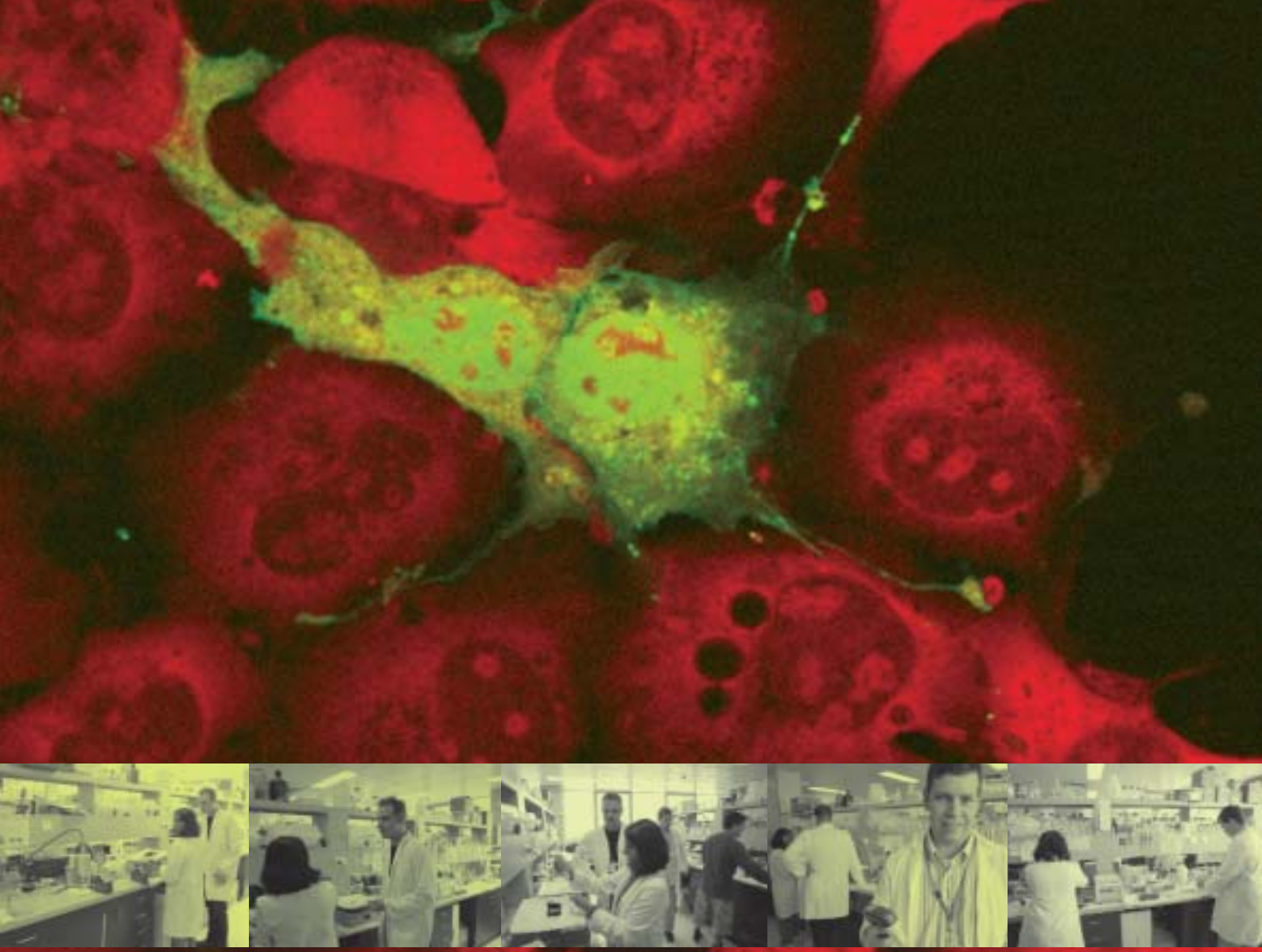
Functional Proteomics

David Stapleton, Head
Abhilasha Gupta
Mark Walter

FUNCTIONAL PROTEOMICS

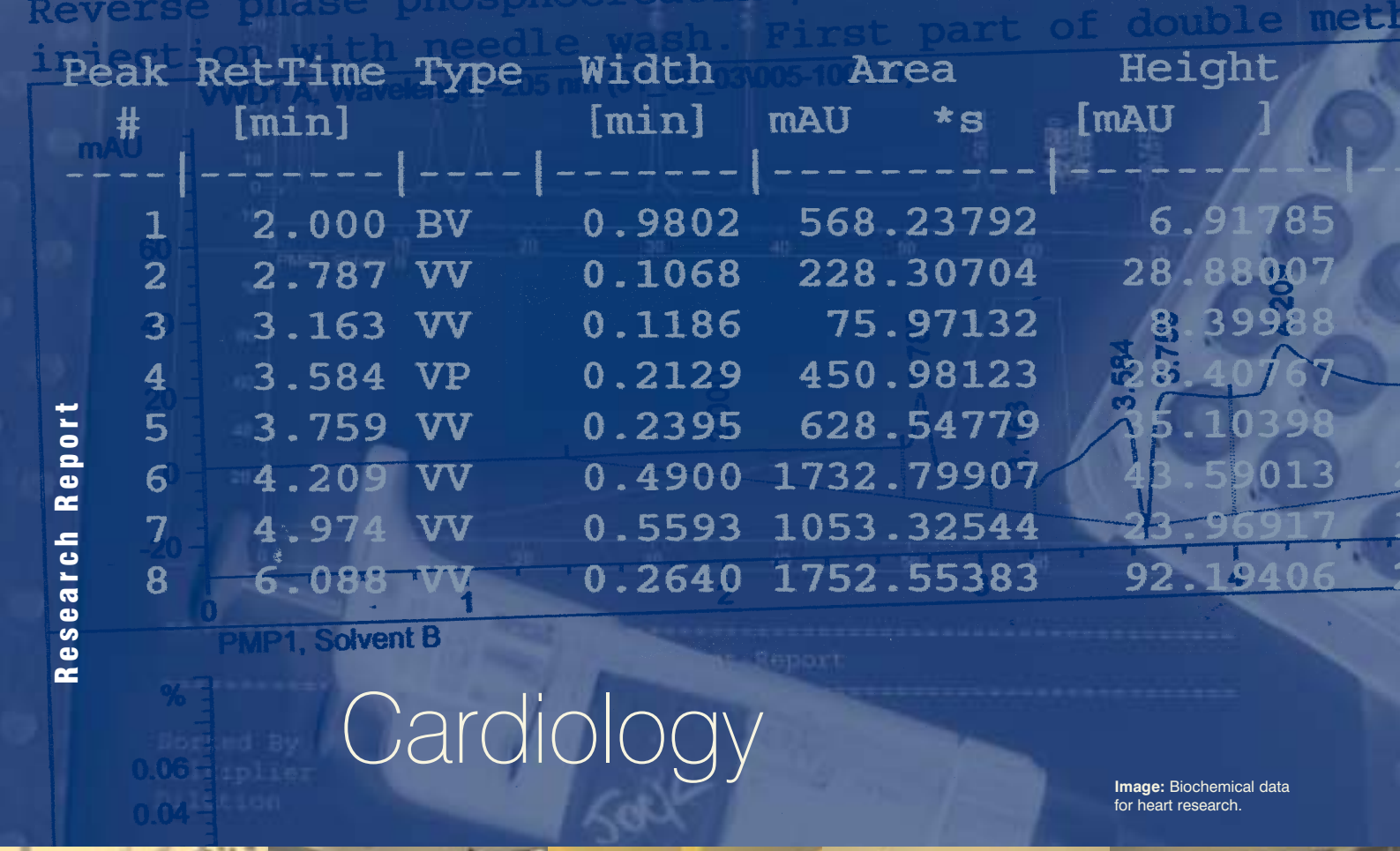
The AMPK enzyme is composed of three different proteins, called the α, β, and γ subunits, and each of these protein subunits has several regions (domains) with different functions. The focus of our research is to understand how the AMPK enzyme performs its functions and how it is regulated. The AMPK is an enzyme that monitors the energy levels of the body. When oxygen and nutrient levels decrease, the energy levels of a cell also decrease leading to activation of AMPK. This results in activation of energy-producing pathways and inhibition of energy-consuming pathways, allowing cells to match energy supply with demand to ensure their survival.

Using internet-based software and biochemical approaches we identified a domain within the AMPK β subunit that binds to glycogen, the cellular storage site for glucose. In collaboration with Prof. Michael Parker's laboratory we constructed a three-dimensional model of this glycogen-binding domain, based on similar domains found in glycogen branching enzymes. This enabled us to predict the amino acids important for glycogen binding. Mutation of these amino acids completely abolished binding to glycogen. Furthermore, we showed that AMPK, when bound to glycogen, retains full activity whilst activation by its upstream activator, AMPK kinase, was enhanced 2- to 3-fold by glycogen. These findings demonstrate that glycogen binding provides a link between AMPK and a major cellular energy store. Recently it has been shown that mutations in the γ subunit, that we have shown to fully activate AMPK, cause a novel glycogen storage disease in humans. The identification of a glycogen-binding domain may therefore help to explain this previously unknown molecular relationship between glycogen and AMPK.



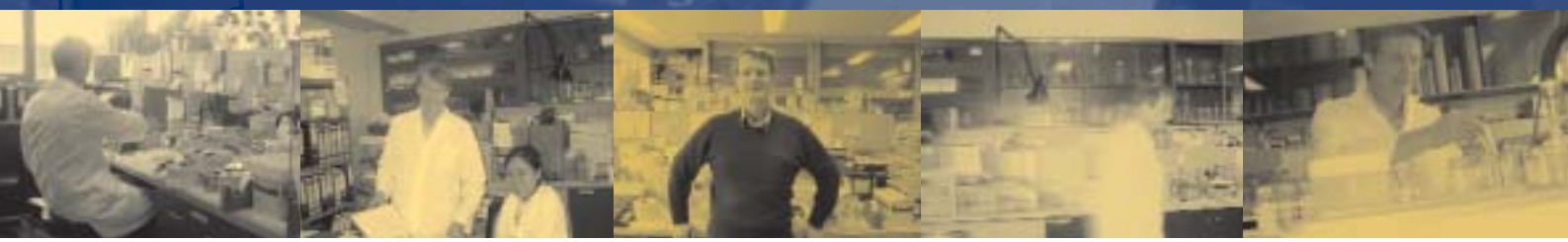
Functional Proteomics

Images: *(Top and Bottom)*: Fluorescently stained cells expressing AMP-Kinase.



Cardiology

Image: Biochemical data for heart research.



Molecular Cardiology

DJ Campbell, Head
Theodora Alexiou
Barry Dixon
Athena Kladis
Gareth Symons

Fellowships and Prizes

DJ Campbell was awarded a National Heart Foundation of Australia Career Development Fellowship to investigate the role of AMP-activated protein kinase in cardiovascular energetics and function.

Grants

DJ Campbell and BE Kemp. Function of AMP-activated protein kinase in the heart. NHMRC Project Grant. (3-year grant support)

DJ Campbell, BE Kemp, A Jenkins, BC Neal and M Woodward. Predictors of recurrent stroke in the PROGRESS study. National Institutes of Health, USA. (2-year grant support)

MOLECULAR CARDIOLOGY

Despite major advances in treatment and prevention, diseases of the heart and blood vessels remain the most common causes of death and illness in our community. These diseases include heart attack, stroke, and heart failure. Our research aims to improve understanding why these diseases occur, and how we can better prevent and treat them.

Stroke is one of the most devastating events anyone can experience. Once someone has had a stroke, even a very small stroke, they are at greatly increased risk of a second stroke. The PROGRESS study, which enrolled 6,105 patients who had had a stroke, showed that reduction of blood pressure with the drugs perindopril and indapamide dramatically reduced the risk of a second stroke. In collaboration with the Institute of International Health in Sydney, and The University of Melbourne Department of Medicine, St. Vincent's Hospital, Melbourne, we are using plasma samples collected from these patients at the beginning of the study to investigate those hormones and chemicals that predict whether a second stroke will occur. These studies will help us understand why strokes occur and how we can prevent them.

A second major research theme is the study of how the heart gets its energy. The heart is continuously pumping blood, and it has to work harder when we exercise. Diseases of the heart, such as heart attack and heart failure, markedly impair the ability of the heart to pump blood. This impaired ability to work is largely due to a problem with energy production in the heart. In collaboration with Bruce Kemp's laboratory, we are studying an enzyme in the heart called AMP-activated protein kinase (AMPK). This enzyme plays a major role in energy production in the heart. Our aim is to better understand how this enzyme contributes to energy production in the heart, so that we can correct the problems of energy production that occur in heart disease.

Molecular Genetics

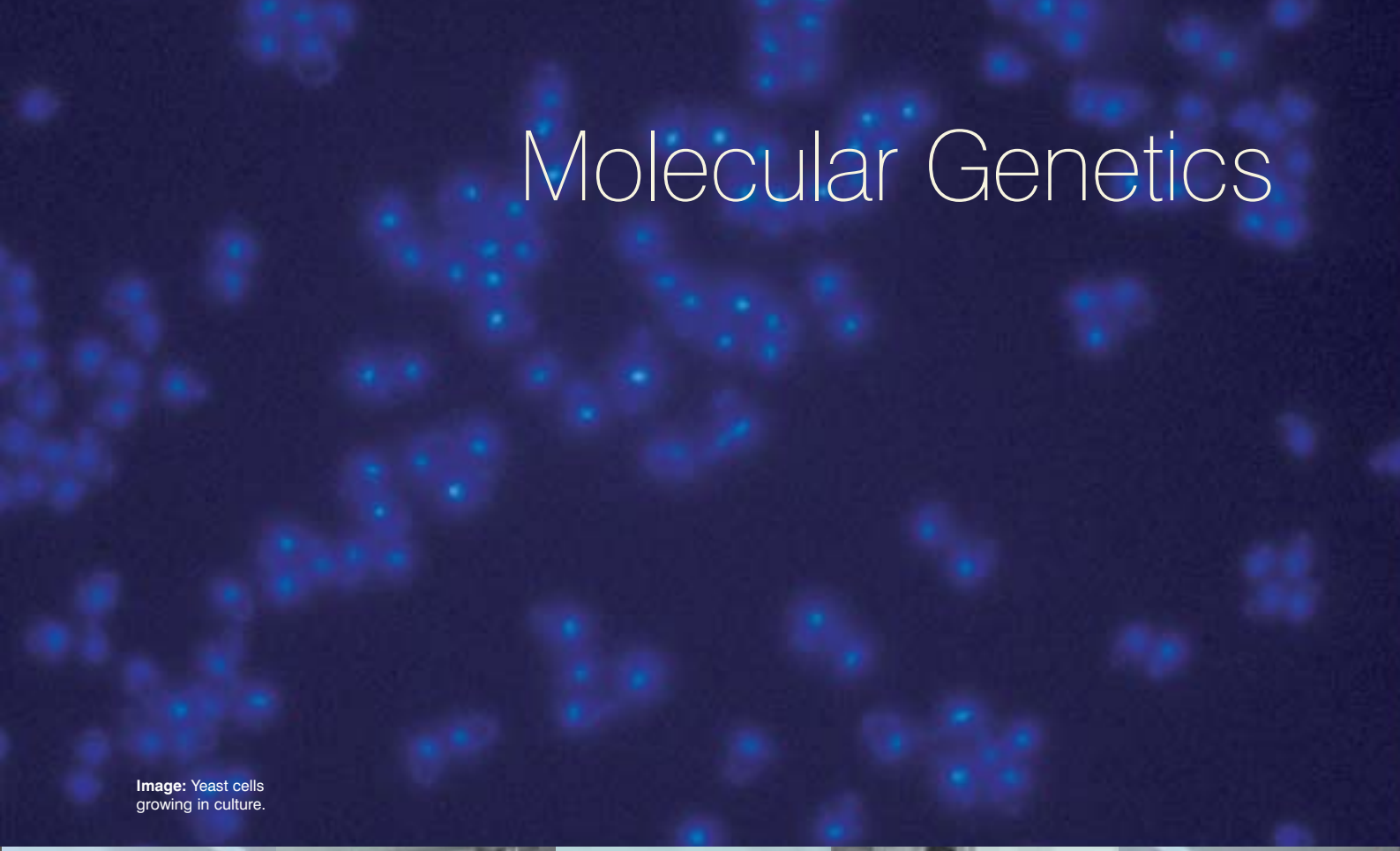


Image: Yeast cells growing in culture.





MOLECULAR GENETICS

As molecular geneticists we use gene manipulations to understanding the function of genes under normal and pathological conditions. Our main research interests are how cells repair DNA damage, and how this prevents the onset of cancer. In addition, we are studying the role of a small calcium binding protein, called S100A1, in the regulation of heart muscle contraction.

DNA damage is a major contributing factor to the onset of cancer. To prevent this all eukaryotic organisms, from yeasts to humans, contain remarkably conserved mechanisms (called checkpoint signalling pathways) that detect DNA damage and activate repair, and at the same time prevent cell division while damage persists. One mechanism by which checkpoints regulate DNA repair involves the activation of genes that code for certain repair proteins. We identified an additional mechanism by which checkpoint pathways regulate genes. Using yeast as a model system, we found that the checkpoint protein Dun1 interacts directly with an enzyme called poly(A)-nuclease (PAN) that regulates the stability of messenger RNA molecules that help in protein production by the cell. Combined genetic defects in Dun1 and PAN lead to a dramatically increased sensitivity of yeast to the DNA damaging compound hydroxyurea. Our studies show that Dun1 and PAN act together to regulate the cellular response to DNA damage.

In another project, we generated mice that lack the gene for the calcium binding protein S100A1 that is highly expressed in heart muscle cells. These mice appear to be entirely normal under basal conditions. However, in contrast to normal mice, hearts of S100A1 mutant mice are unable to pump blood with increased force in response to cardiovascular stress, for example, in response to exercise or chronic high blood pressure. These findings demonstrate that the S100A1 protein plays a crucial role in the response of the heart to stress.

Molecular Genetics

Jörg Heierhorst, Head
Lindus Conlan
Andrew Hammet
Carolyn McNees
Brietta Pike
Nora Tenis

Grants

J Heierhorst.
FHA domain-dependent functions of cell cycle checkpoint kinases.
NHMRC Project Grant.
(3-year grant support)

J Heierhorst.

A novel human DNA damage protein that interacts with the CHK2 and PML tumour suppressor genes.
Cancer Council of Victoria.
(3-year grant support)

Biota Structural Biology Laboratory

Crystallography structures provide an exciting foundation for the development of drugs that in the future might combat Alzheimer's disease.

Biota Structural Biology Laboratory

Michael Parker, Head
Julian Adams
Brett Cromer
Michelle Dunstone
Susanne Feil
Geoffrey Kong
William McKinstry
Craig Morton
Joanne Parsons
Galina Polekhina
Belinda Rizzo
Ian Walker
Mark Walter

Visiting Staff
Professor D Tsernoglou

Knowledge of Protein 3-D Structure Enables the Intelligent Design of New Drugs

Proteins are one of the body's most essential building blocks. In addition to contributing to the structure of the body, proteins are also the "molecules of life", in that they are the molecular engines that control all functions of the body. Knowledge of the structure of a protein is essential to understanding its function. Crystallography offers the means to determine the three-dimensional (3-D) structure of proteins at the atomic level. Knowledge of protein 3-D structure enables the intelligent design of new drugs for the treatment of disease. The major areas of protein crystallography research in the group involve proteins involved in mental disease, bacterial toxins that attack cell walls, and proteins that detoxify poisons.

Switches that Control the Electrical Activity of the Brain

Much of the working of the brain depends on electrical activity that is very precisely regulated by specialised "switches". These "switches" are large protein molecules. We have begun a major study of one of these switch proteins that receives chemical signals from a transmitting cell and converts them back to electrical signals in the receiving cell. This switch protein binds the chemical GABA (short for gamma-aminobutyric acid), and is called the GABA receptor; it functions by allowing chloride ions to enter the cell. The GABA receptor is particularly important from a medical point-of-view because it is the target for various anti-anxiety drugs (benzodiazepines such as valium or diazepam), anti-convulsants, sedatives, depressants (barbiturates such as pentobarbital), anti-epilepsy drugs, alcohol, and anaesthetics. A three-dimensional structure of the GABA receptor will allow the development of new drugs for the treatment of many brain conditions including epilepsy and anxiety.

This year we generated a model of the part of the receptor that binds the chemical GABA. The model has been extremely useful in understanding how GABA and some clinically available drugs bind to the protein. To obtain a much more complete and accurate picture of the GABA receptor we have embarked on a crystallography project to meet this aim.

Alzheimer's Disease – from Structure to Drug

Alzheimer's disease causes dementia, usually in the later decades of life, and is becoming an important health problem with the increased aging of society. After heart disease, cancer and stroke, Alzheimer's disease is the fourth leading cause of death in the developed world. The disease affects 1 in 20 people over 65 years of age increasing to 1 in 5 people over 80 years. The disease is characterised by the presence in the brain of plaques (tangled protein precipitates) composed principally of a protein called amyloid precursor protein (APP).

To understand the normal function of APP we need to determine its three-dimensional shape. We have determined the structures of components of APP by crystallography. The structures suggest APP may play important roles in the body such as the promotion of nerve cell growth and the binding of dangerous metals, particularly copper. The structures also provide an exciting foundation for the development of drugs that might combat Alzheimer's disease.

Our work on Alzheimer's disease is an international collaboration involving Professor Colin Masters, Drs Roberto Cappai and Kevin Barnham at The University of Melbourne Department of Pathology, and Professor Konrad Beyreuther and Dr Gerd Multhaup at the Centre for Molecular Biology, University of Heidelberg, Germany.

Fellowships and Prizes

Dr Galina Polekhina. RD Wright Biomedical Career Development Award from the NHMRC to study medically important protein complexes using X-ray crystallography.

Mr Geoffrey Kong. Dean's Roll of Excellence, Faculty of Health Sciences, The University of Melbourne.

Mr Geoffrey Kong. Amgen Australia Prize for best research project in biomedical subjects, Biochemistry Honours, The University of Melbourne.

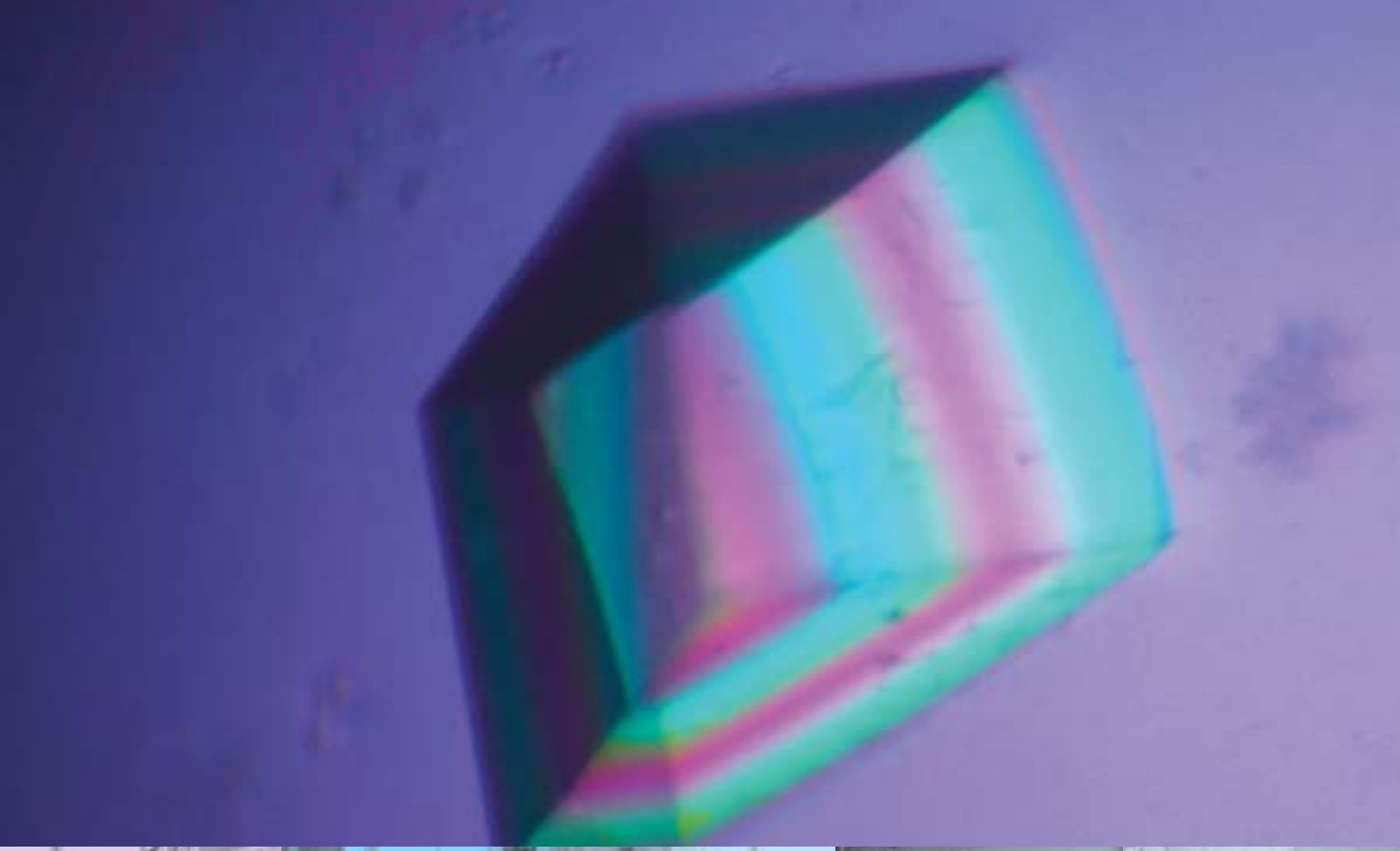
Ms Michelle Dunstone. Maslen 1987 Travelling Scholarship from the Society of Crystallographers of Australia and New Zealand.

Grants

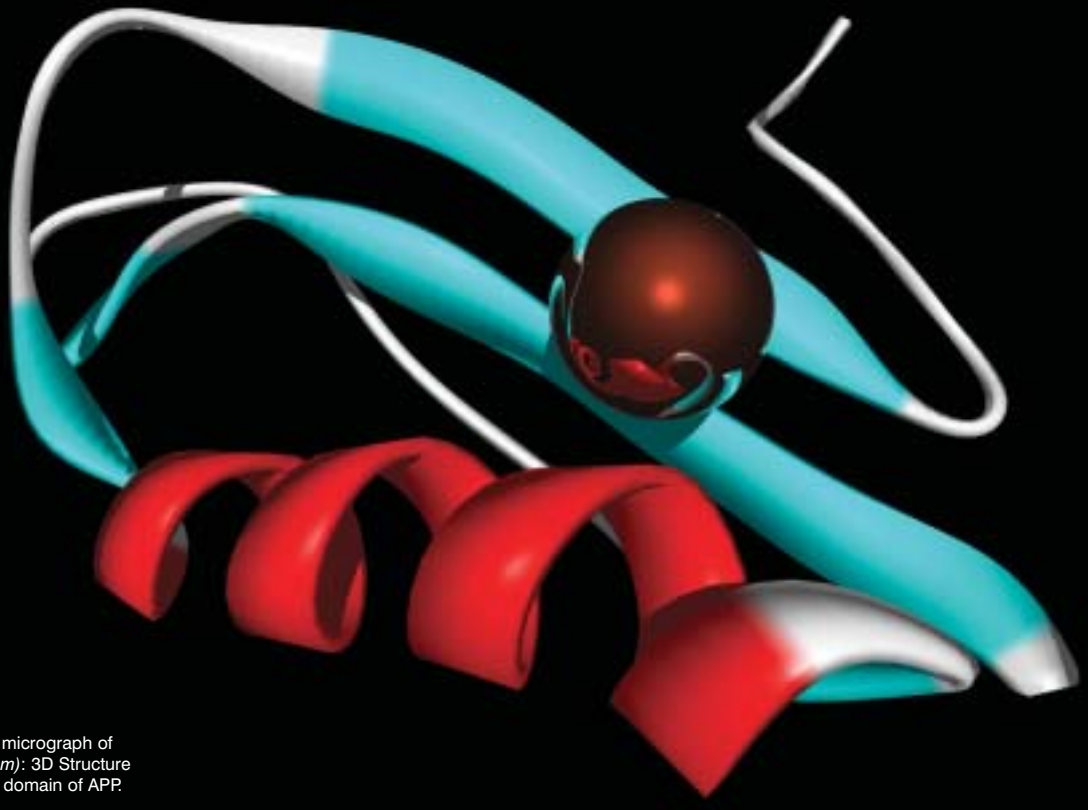
MW Parker. Structure of bacterial pore-forming protein toxins. NHMRC Project Grant. (3-year grant support)

MW Parker and B Cromer. Structural studies of the GABA receptor. NHMRC Project Grant. (5-year grant support)

MW Parker, G Polekhina, D Bowtell and CM House. Structural determinants of Siah ubiquitin ligase complexes NHMRC Project Grant. (3-year grant support)



Crystallography



Images: *(Top)*: Photo micrograph of protein crystals *(Bottom)*: 3D Structure of the copper binding domain of APP.

Virology

The focus of the Virology Unit is to understand the mechanisms these viruses use to enter and infect cells.

Virology

Andy Poumbourios, Head
Heidi Drummer
Chan-Sien Lay
Anne Maerz
Chris Vassos
Kirilee Wilson

Human immunodeficiency virus (HIV), human T-cell leukaemia virus (HTLV) and hepatitis C virus (HCV) have all had a devastating impact on the human population, infecting approximately 250 million individuals. HIV-1 and HTLV-1 are human retroviruses that predominantly infect T-cells that are essential for immune system function. HIV-1-infected individuals eventually succumb to opportunistic infections, dementia and cancer. HTLV-1 causes adult T-cell leukaemia and is associated with a neurological disorder called tropical spastic paraperesis. HCV is a member of the Flaviviridae family of viruses and is therefore more closely related to Dengue and Yellow Fever viruses than to the retroviruses HIV and HTLV.

HCV infects approximately 200 million humans world-wide with almost 200,000 infections in Australia alone. HCV infects liver cells and approximately one fifth of chronic HCV carriers develop liver cirrhosis and 1-5% develop liver cancer. HCV is now the leading single indicator for liver transplantation in developed countries.

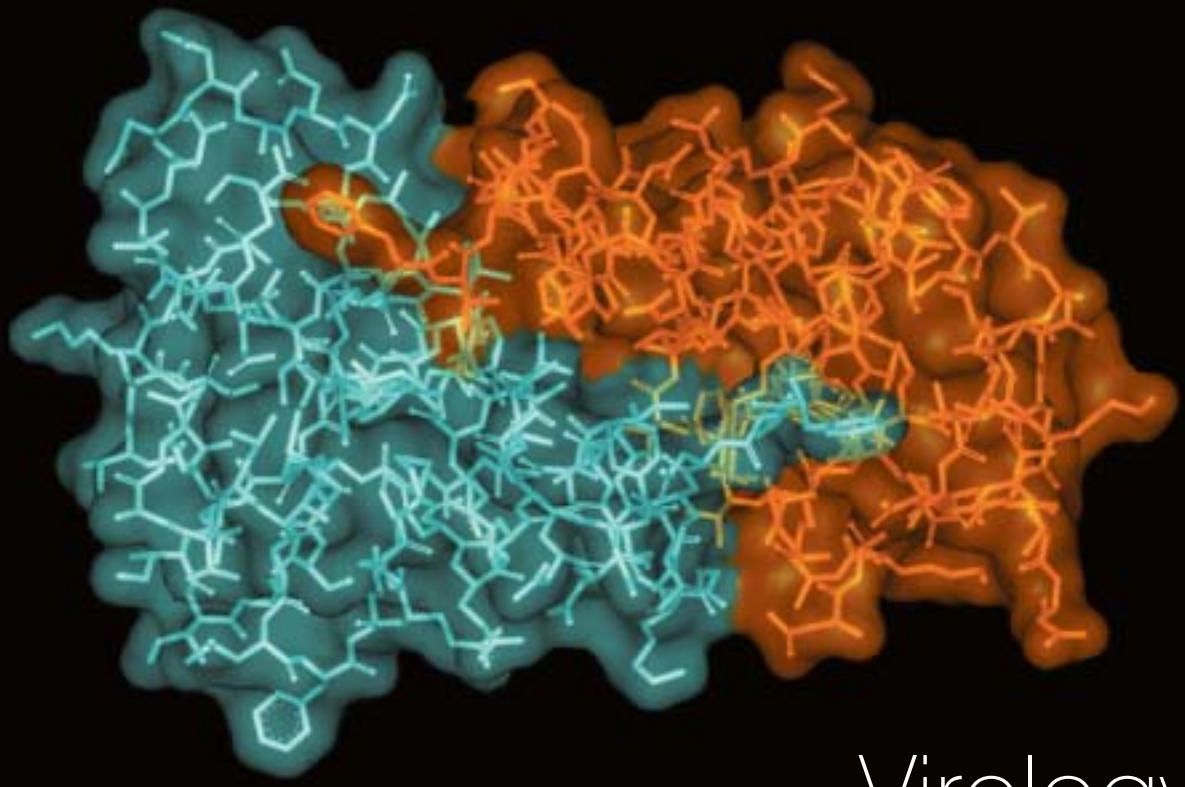
The focus of the Virology Unit is to understand the mechanisms these viruses use to enter and infect cells. HIV, HTLV and HCV are enveloped in a lipid membrane in which protein molecules are embedded. The viruses use these envelope proteins to interact with receptor molecules on the cell they infect. This binding event alters the molecular structure of the viral proteins such that the membrane of the virus fuses with the membrane on the cell, a process called fusion. This leads to internalisation of the genetic material of the virus and initiates viral replication. Understanding how HIV-1, HTLV and HCV achieve fusion at the molecular level will help us to identify new ways to prevent viral infection.

In 1999 we determined the structure of the HTLV-1 fusion protein. We have used this information to determine which regions of the fusion protein are essential for viral fusion to occur which has enabled us to better understand how fusion is mediated.

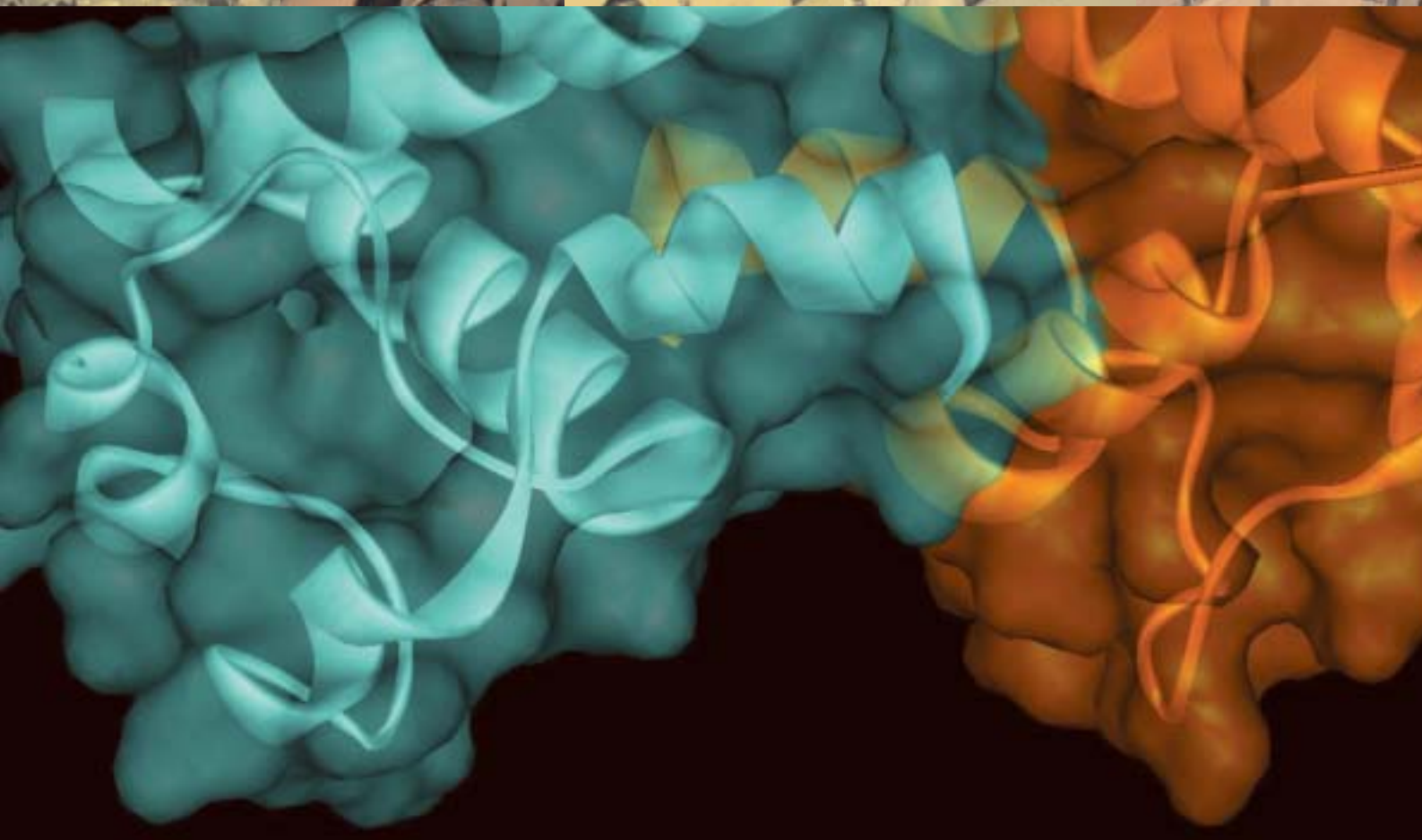
We also made major advances in our understanding of how the envelope protein of HIV-1 mediates fusion. Binding of the envelope protein to a target cell transmits a signal through a conserved region of the fusion protein to initiate fusion.

The envelope proteins of HCV also bind to cells and mediate viral fusion and infection. In addition one of the viral proteins, E2, also interacts with a cellular protein, CD81, that is present on nearly all nucleated cells including T cells. CD81 plays a key role in stimulating T cells as part of the normal immune response. However, when viral E2 protein binds CD81, T-cells become abnormally activated potentially damaging liver cells. We have identified the region of the CD81 molecule that interacts with viral E2 and this information will now allow us to design drugs that prevent this process.

Images: *(Opposite page top and bottom)*: 3D Structure of the cellular protein, CD81.



Virology



National Serology Reference Laboratory Australia™

Hepatitis C is a serious chronic disease and a significant public health burden. It has been estimated that over 200,000 people are currently infected in Australia.

National Serology Reference Laboratory

Elizabeth M Dax, Director
Susan Best, Senior Scientist
Elizabeth Johnson, Research Co-ordinator

Rod Chappel
Hayley Croom
Wayne Dimech
Larissa Doughty
Stacey-Lee Edwards
Darren Jardine
Sally Land
Kate McGavin
Kim Richards
Anita Sands
Kim Wilson

Research at the National Serology Reference Laboratory, Australia™(NRL), focuses on the development of new and improved diagnostic tests for infectious diseases. Commercial imperatives drive development of tests by diagnostics manufacturing companies and this leaves diagnostic issues, important to individuals and/or public health authorities, unaddressed. Our research programme tackles such problems.

The foundation of our research was laid in 2001 with the awarding of a major grant from the Centers for Disease Control and Prevention, USA (CDC) to establish a laboratory test to identify individuals infected with HIV within the past year. Such a test is important to health authorities for monitoring the spread of the disease, particularly in communities where a vaccine may be under trial. It is also important to individuals, as someone recently infected may choose to notify or trace contacts, and clinical management may be different compared to someone with long-standing infection. We used complementary techniques of specific antibody assays and detailed analysis of the interactions between antibodies and individual HIV proteins to characterise the maturation of the antibody-based immune response to HIV infection in fine detail. We discovered a factor that is present in the early stages of infection and disappears after about 4 months, as the immune response matures. This factor is a suitable candidate for a new diagnostic test to identify recent HIV infection and a provisional patent has been submitted for the invention. This project has allowed us to establish methodology that we are now applying to investigate human immune responses to other infectious diseases. By this means we will understand in far greater detail the way the body responds to infection and we hope to discover new specialised diagnostic tests for other infectious diseases.

Hepatitis C is a serious chronic disease and a significant public health burden. It has been estimated that over 200,000 people are currently infected in Australia. While diagnostic tests are extremely good at identifying infected individuals, a significant number of people, who are probably not infected, return a positive test when screened and their infection status is given as "indeterminate". This raises unnecessary doubts and anxiety for affected persons and their doctors. We identified two potential sources of false positive results in commonly used tests and we are now working to refine testing procedures so that this problem can be avoided in future.

In other work, we continue to collaborate with the developers of HIV vaccines in Australia. Our role is to investigate how the body makes antibodies in response to these vaccines. A first trial was completed in human subjects and further preclinical development is ongoing in macaque monkeys.

National Serology Reference Laboratory

Staff Members

PATRON
Gustav J.V. Nossal, *AC CBE MBBS BSc(Med) Syd PhD Melb HonLLD Mon HonLLD Melb HonMD Mainz HonMD Ncl HonMD Leeds HonMD UWA HonDSc Syd HonDSc Qld HonDSc ANU HonDSc UNSW HonDSc LaT HonDSc McMaster HonDSc Oxon FRCP FRACP FRCPA FRACOG(Hon) FRCPath FRACGP FRSE FTSE FAA FRS.*

ST. VINCENT'S INSTITUTE OF MEDICAL RESEARCH

RESEARCH

DIRECTORS
Thomas W. Kay, *BMedSci MB BS PhD Melb FRACP FRCPA; Professor (Medicine), The University of Melbourne. (from April 2002)*
T. John Martin, *AO MD DSc Melb Hon MD Sheffield FRACP FRCPA FAA FRS; Professor (Medicine), The University of Melbourne. (to April 2002)*

DEPUTY DIRECTOR
Bruce E. Kemp, *BAgSci Hons Adel PhD Flinders FAA FRS; Professor (Medicine), The University of Melbourne.*

ASSOCIATE DIRECTORS
Matthew T. Gillespie, *BSc Hons PhD Mon; Associate Professor (Medicine), The University of Melbourne.*
Michael W. Parker, *BSc Hons ANU DPhil Oxon; Head of Biota Structural Biology Laboratory, Professor and Professorial Fellow (Biochemistry and Molecular Biology), The University of Melbourne.*

HOLT FELLOW
T. John Martin, *AO MD DSc Melb Hon MD Sheffield FRACP FRCPA FAA FRS; Professor (Medicine), The University of Melbourne.*

PRINCIPAL RESEARCH FELLOWS
Elizabeth M. Dax, *AM MBBS MD Melb PhD Mon; Associate Professor (Medicine), The University of Melbourne.*
Erik Thompson, *BSc Hons PhD Griffith, Head of VBCRC Invasion and Metastasis Unit, Associate Professor (Surgery), The University of Melbourne.*

SENIOR RESEARCH FELLOWS
Duncan Campbell, *BMedSci MBBS PhD Melb FRACP Grad Dip Epid Biostat; Associate Professor (Medicine), The University of Melbourne.*
Janine Danks, *BSc LaTrobe MSc Melb PhD Mon; Research Fellow, NHMRC, Fellow (Zoology), The University of Melbourne.*
Jörg Heierhorst, *MD Hamburg; Senior Research Fellow, NHMRC.*
Elizabeth Johnson, *BSc Hons PhD Melb.*
Mark Waltham, *BSc Hons PhD Qld;*

RESEARCH FELLOWS
Zhiping Chen, *BSc Shanghai PhD ULP France.*
Heidi Drummer, *BSc Hons PhD Melb.*
William McKinstry, *BSc Hons Tas PhD Melb; NHMRC Industry Fellow.*
Pantelis Poumbourios, *BSc Hons PhD Melb.*
John Price, *BSc Hons PhD Aberdeen.*
Julian Quinn, *BSc Hons MSc DPhil Oxon.*
David Stapleton, *BSc Hons La Trobe PhD Melb; R.D. Wright Fellow, NHMRC.*
Helen Thomas *BSc Hons UWA PhD Melb.*
Hong Zhou, *MBBS Ningxia PhD Melb.*

VISITING SCIENTISTS
Gregory Steinberg, *BSc PhD Uni Guelph; NSERC Postdoctoral Fellow.*
Professor Demetrius Tsernoglou, *University of Rome "La Sapienza", Italy.*
Professor Erich A. Nigg and Ms Elena Nigg, *Max-Planck-Institute for Biochemistry in Martinsried, Germany.*

RESEARCH SCIENTISTS
Julian Adams, *BSc MSc Cantab PhD Massey.*
Elizabeth Allan, *BSc Otago, PhD Melb.*
Lindus Conlan, *BSc Hons PhD Melb.*
Brett Cromer, *BSc Hons PhD ANU.*
Nadine Dudek, *BSc Hons ANU PhD Melb.*
Jane Fisher, *BSc Hons PhD Mon.*
Masha Fridman, *BSc Hons MSc PhD Mon.*
Andrew Hammet, *BSc Hons PhD Melb.*
Peter Hoffmann, *PhD Saarbrücken.*
Ian Jennings, BSc PhD Melb.
Vicky Kartsogiannis, *BSc Hons PhD Melb.*
Maria Kamarinos *BSc Hons PhD Melb.*
Frosa Katsis, *BAppSc IIC PIT.*
Marc Lafleur, *BSc Hons Ottawa PhD East Anglia.*
Belinda Michell, *BSc Hons MBA Mon PhD Melb.*
Rachel Mudge, *BSc Hons PhD Melb.*
Galina Polekhina, *MSc Hons Moscow State PhD Aarhus.*
Bryce van Denderen, *BSc Hons PhD Melb.*
Ian Walker, *BSc Hons Curtin PhD ANU.*
Elizabeth Williams, *BSc Hons PhD Melb.*
Kirilee Wilson, *BSc Hons PhD Melb.*

RESEARCH ASSISTANTS
Margaret Bills, *BSc Macquarie.*
Tony Blick, *BSc Hons Mon.*
Susan Docherty, *BSc Hons LaTrobe.*
Susanne Feil, *BSc MSc Stockholm.*
Rochelle Fernandes, *BSc Melb.*
Christine Gange, *BSc Hons Melb.*
Amanda Handley, *BBSc Hons Latrobe MPH Melb*
Karl Häusler, *BAppSc PIT MAppSc RMIT.*
Emma Jamieson, *BSc Hons Curtin*
Athena Kladis, *BSc Melb.*
Mark Lam, *BSc Hons PhD Melb.*
Chi Ly, *BSc Hons Melb.*
Anne Maerz, *BAppSc RMIT.*
Joanne Parsons, *BSc Hons Swinburne.*
Belinda Rizzo, *BSc Hons Melb.*
Nora Tenis, *BSc Mon, Grad Dip Med Lab Sc Uni of SA.*
Chris Vassos, *BSc Hons Melb.*
Emma Walker, *BSc Hons Deakin.*

CHIEF TECHNICAL OFFICERS
Hannelore Diefenbach-Jagger, *Chem TechCert Augsburg.*
Daphne Hards, *BAppSc RMIT.*
Virginia Leopold, BSc Hons LaTrobe.

SENIOR TECHNICAL OFFICERS
Patricia Wai Ming Ho, *BSc Mon.*
Patricia Smith, *Dip Med Lab Tech RMIT.*

TECHNICAL OFFICERS
Nicolle Gibson, *Assoc Dip Animal Tech Box Hill.*
Jan Elliott.
Marija Mikasinovic, *Cert App Sci Animal Tech Dip App Sci Animal Tech VUT*

TECHNICAL ASSISTANT
Ingrid Kriechbaum, *Dip Health (Med Lab) RMIT.*

LABORATORY ASSISTANT
Shadie Emini.
James Katsis.
Erin Ring.

SENIOR PRINCIPAL RESEARCH ASSOCIATES
Peter Choong, *MBBS MD Melb FRACS FAORTHA; Professor of Orthopaedics, St. Vincent's Hospital and The University of Melbourne.*
Robin Marks, *AM MBBS MPH Mon FRACP FACD; Professor of Dermatology, St. Vincent's Hospital and The University of Melbourne.*
Jane Moseley, *BSc PhD Lond; Associate Professor (Medicine), The University of Melbourne.*
Kong Wah Ng, *MBBS Hons Mon MD Melb FRACP FRCP Edin; Associate Professor (Medicine), The University of Melbourne.*

PRINCIPAL RESEARCH ASSOCIATES
Michael Henderson, *MBBS FRACS; Associate Professor of Surgery , St. Vincent's Hospital and The University of Melbourne.*
John Slavin, *MBBS FRACPA; Department of Pathology, St. Vincent's Hospital, Melbourne.*

SENIOR ASSOCIATES
Vivian Grill, *MBBS Syd FRACP; Senior Lecturer (Medicine), The University of Melbourne.*
Damien Myers, *BAppSc MSc RMIT PhD Melb; Department of Clinical and Biomedical Sciences, Barwon Health, The University of Melbourne.*
Evange Romas, *MBBS PhD Melb FRACP; Senior Lecturer (Medicine), The University of Melbourne, Department of Rheumatology, St. Vincent's Hospital, Melbourne.*
Mary Wlodek, *BSc MSc PhD Mon; Department of Physiology, The University of Melbourne.*

ASSOCIATES
Craig Morton, *BSc Hons PhD Melb; Biota Structural Biology Laboratory, Melbourne.*
Sue Rogers, *BSc Hons PhD Lond; Department of Medicine, The University of Melbourne.*
Julie Sharp, *BAppSc RMIT PhD Melb, Thomaïy Fellow.*
Natalie Sims, *BSc Hons PhD Adel; R.J. Gleghorn Fellow, The University of Melbourne.*
David Thomas, *MBBS PhD Melb FRACP; Neil Hamilton Fairley Fellow, NHMRC.*

ADMINISTRATION

BUSINESS MANAGER
David Rees, *Bach Bus CPA.*

LABORATORY AND TECHNICAL SERVICES MANAGER
David Murfitt, *HNC AppBiol Cambridge CAT.*

DEVELOPMENT MANAGER
Diane Losa.

DIABETES PROGRAM MANAGER
Anne Thorburn, *BSc Hons PhD Syd*

ADMINISTRATIVE OFFICERS
Gayle McMurray.
Claire Tanswell, *GCertBusAdmin Swinburne.*

ASSISTANT ACCOUNTANT
Lisnawati Wirawan-Liauw, *SE Atmajaya Katolik Uni.*

ADMINISTRATIVE ASSISTANTS
Beth Castles.
Dimitra Samaras.

NATIONAL SEROLOGY REFERENCE LABORATORY

RESEARCH

DIRECTOR
Elizabeth Dax, *AM MBBS Melb PhD Mon; Associate Professor (Medicine), The University of Melbourne.*

SENIOR SCIENTIST
Susan Best, *MAppSc RMIT.*

RESEARCH CO-ORDINATOR
Elizabeth Johnson, *BSc Hons PhD Melb.*

QUALITY MANAGER
Roderick Chappel, *BAgrSc PhD Melb.*

PROJECT MANAGER
Wayne Dimech, *BAppSc RMIT FAIMS GDipMgt Deakin.*

SCIENTISTS
Thein Thein Aye, *MBBS Yangoon PhD Japan.*
Hayley Croom.
Larissa Doughty, *BSc Hons Mon.*
Stacey-Lee Edwards, *BSc Hons Melb.*
Barbara Francis, *BSc Melb GDipAppSc (Health Stats) PhD Swinburne.*
Fernando Garcia, *BAppSc RMIT.*
Rosina Gribben, *BSc Syd.*
Callum Haig, *BSc UWA.*
Chris Hamilton, *BSc Hons Otago PhD Mon.*
Darren Jardine, *BSc Hons PhD LaTrobe.*
Marina Karakaltsas, *BSc LaTrobe.*
Sally Land, *BSc Hons Dip Ed Melb.*
Kate McGavin, *BSc AmusA Melb.*
Leah O,Mahony, *BSc Hons Ireland.*
Lena Panagiotopoulos, *BSc LaTrobe.*
Scott Read, *BSc Hons Lond.*
Kim Richards, *BSc Hons VUT.*
Anita Sands, *BSc Hons Melb.*
Joanne Schlegel, *BAppSc RMIT.*
Kathy Smeh, *BSc Hons BEd Studies MEd Melb.*
Sandy Walker, *BSc Hons LaTrobe.*
Kim Wilson, *BAppSc QIT PhD Melb.*

DATA MANAGEMENT & WEBSITE OFFICER
Rosanna Fahmy.

LABORATORY ASSISTANT
Frank Torzillo.

ADMINISTRATION

EXECUTIVE ASSISTANT
Debra Irvine.

COMMUNICATIONS OFFICER
Romy Johnston.

COMPUTER SYSTEMS MANAGER
John Tomasov, *BSc Hons PhD LaTrobe Grad Dip Comp Sci Mon.*

OFFICE MANAGER
Louie Opasinov, *BSc Dip Ed Melb.*

Staff Members

Graduates

"Working alongside internationally renowned scientists with varied research backgrounds has provided me with a well-rounded grasp of biological sciences necessary to enter the world stage"

Dr Lindus Conlan
Postdoctoral Fellow
Molecular Genetics Laboratory

Lindus obtained her PhD in 2000 for her studies on PTHrP within the Bone Biology Group. She then joined the Molecular Genetics Laboratory, investigating a new protein involved in the cellular response to DNA damage.

GRADUATES

THE FOLLOWING GRADUATED DOCTOR OF PHILOSOPHY:

Lindus Conlan *'Transcriptional properties of PTHrP'*

Andrew Hammet *'Regulation of DNA damage repair mechanisms by protein phosphorylation'*

Nirada Dhanesuan *'SPARC/Osteonectin regulation of MMP-2 activation at the cell surface'*

Rachel Thomas *'The involvement of PTHrP in the metastasis of breast cancer to bone'*

THE FOLLOWING GRADUATED BACHELOR OF SCIENCE HONOURS:

Stacey-Lee Edwards *'Resolution of the nature of HCV indeterminate serological results'*

STUDENTS

POSTGRADUATE SCHOLARS - DOCTOR OF PHILOSOPHY

Theodora Alexiou, *BSc Grad Dip Mon.*
'Generation and function of brain angiotensin'

Angela Arvanitis, *BSc Hons Melb.*
'Gene expression profiling in breast cancer cells'

Barry Dixon, *MBBS Syd FRACP.*
'Characterisation of systemic inflammation following cardiopulmonary bypass'

Michelle Dunstone, *BSc Hons Mon.*
'Structural studies of human complement pathway proteins'

Eugene Estella, *MBBS Qld FRACP.*
'Mechanism of β-cell destruction'

Susanne Feil, *MSc Stockholm.*
'Structural studies of medically important proteins'

Abilasha Gupta, *BSc Hons Melb.*
'The nuclear localisation of AMP-activated protein kinase'

Karl Häusler, *BAppSc Phillip MAppSc RMIT.*
'Osteoblastic and lymphocytic factors affecting osteoclastogenesis'

Natasha Ilievska, *BSc Hons VUT.*
'Role of PTHrP in DNA repair'

Tristan Iseli, *BSc Hons Melb.*
'Structure and function of the glycogen binding AMP-activated protein kinase β-subunit'

Geoffrey Kong, *BSc Hons Melb.*
'Structural studies of Alzheimer's disease amyloid precursor protein'

Chan-Sien Lay, *BSc Hons RMIT*
'Structural and functional features of retroviral envelope glycoproteins'

Lisa McCarthy, *BSc Hons Deakin.*
'Investigation of cancer cell inhibition by a novel extract of shark cartilage'

Sid Murthy, *BSc Hons Melb.*
'Regulation of AMP-activated protein kinase kinase'

Danijela Mirosavljevic, *BSc Hons LaTrobe.*
'Lymphocyte-derived factors affecting osteoclastogenesis'

Joseph Pereira, *BSc Hons LaTrobe.*
'An investigation into the role of the integrin αvβ3 and the matrix-metalloproteinase-2 in cancer'

Brietta Pike, *BSc Hons Melb.*
'FHA domains in the regulation of cell cycle check point protein kinases'

Mark Walter, *BSc Hons LaTrobe BEc Adel.*
'Structure and function of the γ-subunit of AMP-activated protein kinases'

POSTGRADUATE SCHOLARS - DOCTOR OF MEDICINE

P. Scott Mackie, *MBBS Melb.*
'The role of bisphosphonates as an adjunct treatment for osteosarcoma'

UNDERGRADUATE SCHOLAR - BACHELOR OF SCIENCE (HONOURS)

Stacey-Lee Edwards, *BSc Melb.*
'Resolution of the nature of HCV indeterminate serological results'

Jade Woon, *BSc Melb.*
'Post-translational modifications of haemoglobin in type I diabetes mellitus'

SEMINAR PROGRAM

Professor William Hamlett
Department of Anatomy and Cell Biology, Indiana University School of Medicine, Notre Dame, Indiana, USA.
'From egg to placenta: diversity of reproductive models in Chondricthyan fishes'

Dr Rachel Thomas
St. Vincent's Institute of Medical Research.
'The involvement of PTHrP in the metastasis of breast cancer to bone'

Professor Erich Nigg
Department of Cell Biology, Max-Planck Institute for Biochemistry, Martinsried, Germany.
'The regulation of cell division: focus on the centrosome cycle, the spindle checkpoint and chromosomal instability'

Dr Patrick Sexton
Howard Florey Institute of Experimental Physiology and Medicine.
'Recent developments in our understanding of RAMP function'

Dr Janine Danks
St. Vincent's Institute of Medical Research.
'Sharks and PTHrP'

Dr Philip Robinson
Cell Signalling Unit, Children's Medical Research Institute, Sydney.
'Dynamin and molecular mechanisms of endocytosis in neurones'

and Students

"SVIMR has provided me with great opportunities to work both independently and within a team environment in an exciting area of virology"

Chan-Sien Lay
Postgraduate Scholar
Virology Unit

Chan is investigating the structural and functional features of retroviral envelope glycoproteins - molecules on the surface of viruses that help them infect cells.

Dr Silviu Itescu
St. Vincent's Hospital, Melbourne and Columbia University, New York, USA.
'Characterisation and use of human bone marrow-derived angioblasts for cardiovascular disease'

Ms Maria Macheda
Department of Medicine, St Vincent's Hospital and The University of Melbourne.
'Characterisation of GLUT12, a novel glucose transporter'

Dr Rebecca Ritchie
Howard Florey Institute of Experimental Physiology and Medicine.
'New targets for the prevention of cardiac hypertrophy and ischaemia'

Dr Rick Pearson
Peter MacCallum Cancer Institute.
'The Akt protein kinase, novel signalling pathways and potential role in ovarian cancer'

Dr Margaret Hibbs
Ludwig Institute for Cancer Research.
'Exploring the role of the Lyn tyrosine kinase using mouse mutants'

Mr Colin House
Peter MacCallum Cancer Institute.
'Siah later - directing proteins to the proteasome'

Dr Lorraine Robb
The Walter & Eliza Hall Institute of Medical Research.
'Using gene targeting to uncover redundant and non-redundant roles for interleukin II in the mouse'

Dr Rebecca Heyes
Victorian Forensic Science Centre.
'Modern forensic investigations; from the crime scene to the court room'

Dr Andy Poumbourios
St. Vincent's Institute of Medical Research.
'HIV and HTLV helical hairpin structure: implications for membrane fusion function'

Dr Steve Petrou
Department of Physiology, The University of Melbourne.
'GABA receptor mutations in human epilepsy'

Dr Darren Kelly
Department of Medicine, St Vincent's Hospital and The University of Melbourne.
'Diabetic complications and the transgenic (mRen-2)27 rat'

Dr Philippe Bouillet
The Walter & Eliza Hall Institute of Medical Research.
'Role of the pro-apoptotic Bcl-2 relative Bim in auto-immune and degenerative diseases'

Dr Ian Campbell
Reid Rheumatology Laboratory, The Walter & Eliza Hall Institute of Medical Research.
'Severe Inflammatory Arthritis and Lymphadenopathy in the absence of TNF'

Associate Professor Tom Kay
St. Vincent's Institute of Medical Research.
'Immune mechanisms that cause type 1 diabetes'

Mr Karl Häusler
St. Vincent's Institute of Medical Research.
'TNF receptor family members and secreted frizzled related proteins in osteoclastogenesis'

Dr Margaret Jones
Prince Henry's Institute of Medical Research.
'Life without estrogen: the aromatase knockout mouse'

Dr Bill McKinstry
St. Vincent's Institute of Medical Research.
'Structure of parathyroid hormone related protein complexed to a monoclonal antibody: insights into hormone receptor interactions'

Dr Tony Tiganis
Department of Biochemistry & Molecular Biology Monash University.
'Protein tyrosine phosphatase TCPTP: A nuclear phosphatase regulating cytoplasmic signalling'

Dr Louise Purton
Stem Cell Biology Laboratory, Peter MacCallum Cancer Institute.
'Distinct roles of retinoic acid receptors in haemopoiesis'

Dr Mark Smyth
Cancer Immunology, Peter MacCallum Cancer Institute.
'Natures TRAIL - on a path to new tumour immunotherapies'

Dr Douglas Hilton
The Walter & Eliza Hall Institute of Medical Research.
'A foray into mouse genetics'

Dr Dennis Carney
Department of Medicine, St Vincent's Hospital and The University of Melbourne.
'Arsenic revisited'

Dr Julian Adams
St. Vincent's Institute of Medical Research.
'Studies in Protein Structure: The structures of Beta-lactoglobulin in two new crystal forms. The structure and properties of the iron superoxide dismutase from Methanobacterium thermoautotrophicum'

Mr Steve Christov
Department of Medicine, St Vincent's Hospital and The University of Melbourne.
'Lp(a) and its role in diabetes complications'

Dr Joan Heath
Ludwig Institute for Cancer Research.
'Fishing for insights into gut development and colon cancer'

Dr Grant McArthur
Peter MacCallum Cancer Institute.
'Regulation of cell size, division and differentiation of granulocytes by the MYC-antagonist MAD1'

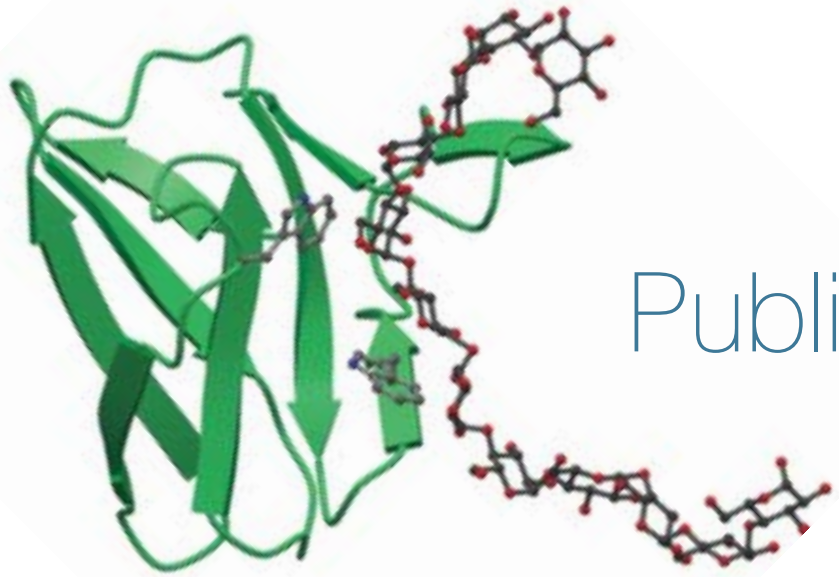
Dr Mandy Fosang
Murdoch Children's Research Institute.
'New Angles on Aggrecan Degradation'

Ms Susanne Feil
St. Vincent's Institute of Medical Research.
'Crystallographic studies of pore-forming protein toxins'

Ms Michelle Dunstone
St. Vincent's Institute of Medical Research.
'Structural studies of human complement pathway proteins'

Ms Brietta Pike
St. Vincent's Institute of Medical Research.
'A novel checkpoint protein important for cell cycle progression and DNA damage tolerance in S. cerevisiae'

Dr Philip Stork
Vollum Institute, Oregon, USA
'New insights into the mechanism of action of the small GTPases Ras and Rap1.'



Publications

Image: 3D structure of the glycogen-binding domain of AMP- Kinase.

REFERRED ARTICLES

Allocati, N., Masulli, M., Casalone, E., Santucci, S., Favaloro, B., Parker, M.W. & Di Ilio, C. Glutamic acid-65 is an essential residue for catalysis in Proteus mirabilis glutathione S-transferase B1-1. *Biochem. J.*, 363: 189-193, 2002.

Bonifacio, E., Atkinson, M., Eisenbarth, G., Serreze, D., Kay, T.W., Lee-Chan, E. & Singh, B. International Workshop on Lessons from Animal Models for Human Type 1 Diabetes: analyzing target autoantigens of humoral immunity in nonobese diabetic mice. *Ann. N. Y. Acad. Sci.*, 958: 1-2, 2002.

Boo, Y.C., Hwang, J., Sykes, M., Michell, B.J., Kemp, B.E., Lum, H. & Jo, H. Shear stress stimulates phosphorylation of eNOS at Ser(635) by a protein kinase A-dependent mechanism. *Am. J. Physiol. Heart Circ. Physiol.*, 283: H1819-H1828, 2002.

Bowser, D.N., Wagner, D.A., Czajkowski, C., Cromer, B.A., Parker, M.W., Wallace, R.H., Harkin, L.A., Mulley, J.C., Marini, C., Berkovic, S.F., Williams, D.A., Jones, M.V. & Petrou, S. Altered kinetics and benzodiazepine sensitivity of a GABAA receptor subunit mutation [gamma 2(R43Q)] found in human epilepsy. *Proc. Natl. Acad. Sci. USA.*, 99: 15170-15175, 2002.

Caccuri, A.M., Antonini, G., Allocati, N., Di Ilio, C., Innocenti, F., De Maria, F., Parker, M.W., Masulli, M., Polizio, F., Federici, G. & Ricci, G. Properties and utility of the peculiar mixed disulfide in the bacterial glutathione transferase B1-1. *Biochemistry*, 41: 4686-4693, 2002.

Caccuri, A.M., Antonini, G., Allocati, N., Di Ilio, C., De Maria, F., Innocenti, F., Parker, M.W., Masulli, M., Lo Bello, M., Turella, P., Federici, G. & Ricci, G. GSTB1-1 from Proteus mirabilis: a snapshot of an enzyme in the evolutionary pathway from a redox enzyme to a conjugating enzyme. *J. Biol. Chem.*, 277: 18777-18784, 2002.

Carpinelli, M.R., Wicks, I.P., Sims, N.A., O'Donnell, K., Hanzinikolas, K., Burt, R., Foote, S.J., Bahlo, M., Alexander, W.S. & Hilton, D.J. An ethyl-nitrosourea-induced point mutation in phex causes exon skipping, x-linked hypophosphatemia, and rickets. *Am. J. Pathol.*, 161: 1925-1933, 2002.

Chen, Z., Dudek, N., Wijburg, O., Strugnell, R., Brown, L., Deliyannis, G., Jackson, D., Koentgen, F., Gordon, T. & McCluskey, J. A 320-kilobase artificial chromosome encoding the human HLA DR3-DQ2 MHC haplotype confers HLA restriction in transgenic mice. *J. Immunol.*, 168: 3050-3035, 2002.

Chong, M.M.W., Thomas, H.E. & Kay, T.W. Suppressor of cytokine signaling-1 regulates the sensitivity of pancreatic β cells to tumor necrosis factor. *J. Biol. Chem.*, 277: 27945-27952, 2002.

Ciconte, A., Mills, A., Shipley, A. & Marks, R. Subacute cutaneous lupus erythematosus in a six year old child: A report of a case and a review of the literature. *Austral. J. Dermatol.*, 43: 62-64, 2002.

Cingolani, G., Bednenko, J., Gillespie, M.T. & Gerace, L. Molecular basis for the recognition of a non-classical nuclear localization signal by importin β. *Mol. Cell*, 10: 1345-1353, 2002.

Coggan, M., Flanagan, J.U., Parker, M.W., Vichai, V., Pearson, W.R. & Board, P.G. Identification and characterization of GSTT3, a third murine Theta class glutathione transferase. *Biochem. J.*, 366: 323-332, 2002.

Conlan, L.A., Martin, T.J. & Gillespie, M.T. The COOH-terminus of parathyroid hormone-related protein (PTHrP) interacts with beta-arrestin 1B. *FEBS. Lett.*, 527: 71-75, 2002.

Cromer, B.A., Morton, C.J. & Parker, M.W. Anxiety over GABA(A) receptor structure relieved by AChBP. *Trends Biochem. Sci.*, 27: 280-287, 2002.

Cromer, B.A., Morton, C.J., Board, P.G. & Parker, M.W. From glutathione transferase to pore in a CLIC. *Eur. Biophys. J.*, 31: 356-364, 2002.

Deng, X., Williams, E.D., Thompson, E.W., Gan, X. & Gu, M. Second-harmonic generation from biological tissues: effect of excitation wavelength. *Scanning*, 24: 175-178, 2002.

Dhanesuan, N., Sharp, J.A., Blick, T., Price, J.T. & Thompson, E.W. Doxycyclin-inducible expression of SPARC/ Osteonectin/ BM40 in MDA-MB-231 human breast cancer cells results in growth inhibition. *Breast Cancer Res. Treat.*, 75:73-85, 2002.

Drummer, H.E., Wilson, K.A. & Poumbourios, P. Identification of the hepatitis C virus E2 glycoprotein binding site on the large extracellular loop of CD81. *J. Virol.*, 76: 11143-11147, 2002.

Du, X.J., Cole, T.J., Tenis, N., Gao, X.M., Kontgen, F., Kemp, B.E. & Heierhorst, J. Impaired cardiac contractility response to hemodynamic stress in S100A1-deficient mice. *Mol. Cell. Biol.*, 22: 2821-2829, 2002.

Einoder, P.A. & Choong, P.F. Tumours of the head of the fibula: good function after resection without ligament reconstruction in 6 patients. *Acta. Orthop. Scand.*, 73: 663-666, 2002.

Ernst, M., Inglese, M., Scholz, G.M., Harder, K.W., Clay, F.J., Bozinoyski, S., Waring, P., Darwiche, R., Kay, T., Sly, P., Collins, R., Turner, D., Hibbs, M.L., Anderson, G.P. & Dunn, A.R. Constitutive activation of the SRC family kinase Hck results in spontaneous pulmonary inflammation and an enhanced innate immune response. *J. Exp. Med.*, 196: 589-604, 2002.

Farrow, K.A., Lyras, D., Polekhina, G., Koutsis, K., Parker, M.W. & Rood, J.I. Identification of essential residues in the Erm(B) rRNA methyltransferase of Clostridium perfringens. *Antimicrob. Agents Chemother.*, 46: 1253-1261, 2002.

Fisher, J.L., Schmitt, J.F., Howard, M.L., Mackie, P.S., Choong, P.F. & Risbridger, G.P. An in vivo model of prostate carcinoma growth and invasion in bone. *Cell. Tissue Res.*, 307: 337-345, 2002.

Fulton, D., Fontana, J., Sowa, G., Gratton, J.P., Lin, M., Li, K.X., Michell, B., Kemp, B.E., Rodman, D. & Sessa, W.C. Localization of endothelial nitric-oxide synthase phosphorylated on serine 1179 and nitric oxide in Golgi and plasma membrane defines the existence of two pools of active enzyme. *J. Biol. Chem.*, 277: 4277-4284, 2002.

Gillett, A.M., Wallace, M.J., Gillespie, M.T. & Hooper, S.B. Lung expansion increases calmodulin mRNA levels in the fetal lung. *Am. J. Physiol. - Lung Cell. Mol. Physiol.*, 282: L440-L447, 2002.

Grovender, S., Csimma, C., Genant, H.K., Valentin-Opran, A., Amit, Y., Arbel, R., Aro, H., Atar, D., Bishay, M., Borner, M.G., Chiron, P., Choong, P., Cinats, J., Coutenay, B., Feibel, R., Geulette, B., Gravel, C., Haas, N., Raschke, M., Hammacher, E., van der Velde, D., Hardy, P., Holt, M., Josten, C., Ketterl, R.L., Lindeque, B., Lob, G., Mathevon, H., McCoy, G., Marsh, D., Miller, R., Munting, E., Oeyre, S., Nordsletten, L., Patel, A., Pohl, A., Rennie, W., Reyndens, P., Rommens, P.M., Rondia, J., Rossouw, W.C., Daneel, P.J., Ruff, S., Ruter, A., Santavirta, S., Schidhauer, T.A., Gekle, C., Schnettler, R., Segal, D., Seiler, H., Snowdowne, R.B., Stapert, J., Taglang, G., Verdonk, R., Vogels, L., Weckbach, A., Wentzensen, A. & Wisniewski, T. Recombinant human bone morphogenetic protein-2 for treatment of open tibial fractures: a prospective, controlled, randomized study of four hundred and fifty patients. *J. Bone Joint Surg. Am.*, 84A: 2123-2134, 2002

Guise, T.A., Yin, J.J., Thomas, R.J., Dallas, M., Cui, Y. & Gillespie, M.T. Parathyroid hormone-related protein (PTHrP)-(1-139) isoform is efficiently secreted in vitro and enhances breast cancer metastasis to bone in vivo. *Bone*, 30: 670-676, 2002.

Hamilton, S.R., O'Donnell, J.B.Jr., Hammet, A., Stapleton, D., Habinowski, S.A., Means, A.R., Kemp, B.E. & Witters, L.A. AMP-activated protein kinase kinase: detection with recombinant AMPK alpha1 subunit. *Biochem. Biophys. Res. Commun.*, 293: 892-898, 2002.

Hammet, A., Pike, B.L. & Heierhorst, J. Posttranscriptional regulation of the RAD5 DNA repair gene by the Dun1 kinase and the Pan2-Pan3 poly(A)-nuclease complex contributes to survival of replication blocks. *J. Biol. Chem.*, 277: 22469-224774, 2002.

Irawaty, W., Kay, T.W.H. & Thomas, H.E. Transmembrane TNF and IFNγ induce caspase-independent death of primary mouse pancreatic beta cells. *Autoimmunity*, 35: 369-375, 2002.

Jenner, N., Campbell, J., Plunkett, A. & Marks, R. Cost of psoriasis: A study on the morbidity and financial effects of having psoriasis in *Australia. Australas. J. Dermatol.*, 43: 255-261, 2002.

Kiely, P., Eliades, L., Kebede, M., Stephenson, M. & Jardine, D. Anti-HCV conformity testing of voluntary blood donors: comparisons of the sensitivity of two immunoblot assays. *Transfusion*, 42: 1053-1058, 2002.

Kooptiwut, S., Zraika, S., Thorburn, A.W., Dunlop, M.E., Darwiche, R., Kay, T.W., Proietto, J. & Andrikopoulos, S. Comparison of insulin secretory function in two mouse models with different susceptibility to β-cell failure. *Endocrinology*, 143: 2085-2092, 2002.

Kunisada, T., Moseley, J.M., Slavin, J.L., Martin, T.J. & Choong, P.F. Co-expression of parathyroid hormone-related protein (PTHrP) and PTH/PTHrP receptor in cartilaginous tumours: a marker for malignancy? *Pathology*, 34: 133-137, 2002.

Kunisada, T., Ngan, S.Y., Powell, G. & Choong, P.F.M. Wound complications following pre-operative radiotherapy for soft tissue sarcoma. *Eur. J. Surg. Oncol.*, 28: 75-79, 2002.

Kveiborg, M., Chiusaroli, R., Sims, N.A., Wu, M., Sabatakos, G., Horne, W.C. & Baron, R. The increased bone mass in deltaFosB transgenic mice is independent of circulating leptin levels. *Endocrinology*, 43: 4304-4309, 2002.

Lafleur, M.A., Handsley, M.M., Knauper, V., Murphy, G. & Edwards, D.R. Endothelial tubulogenesis within fibrin gels specifically requires the activity of membrane-type-matrix metalloproteinases (MT-MMPs). *J. Cell Sci.*, 115: 3427-3438, 2002.

Lam, M.H., Thomas, R.J., Loveland, K.L., Schilders, S., Gu, M., Martin, T.J., Gillespie, M.T. & Jans, D.A. Nuclear transport of parathyroid hormone (PTH)-related protein is dependent on microtubules. *Mol. Endocrinol.*, 6: 390-401, 2002.

Lam, Y.H., Morton, C.J. & Separovic F. Solid-state NMR conformational studies of a melittin-inhibitor complex. *Eur. Biophys. J.*, 31: 383-388, 2002.

Land, S., Tabrizi, S., Gust, A., Johnson, E., Garland, S. & Dax, E.M. External quality assessment program for Chlamydia trachomatis diagnostic testing by nucleic acid amplification assays. *J. Clin. Microbiol.*, 40: 2893-2896, 2002.

Image: Molecular model of the GABA Receptor.

Lelie, P.N., van Drimmelen, H.A., Cuypers, H.T., Best, S.J., Strammer, S.L., Hyland, C., Allain, J.P., Moncharmont, P., Defer, C., Nubling, M., Glauser, A., da Silva Cardoso, M., Viret, J.F., Lankinen, M.H., Grillner, L., Wirthmuller, U., Coste, J., Schottstedt, V., Masecar, B. & Dax, E.M. Sensitivity of HCV RNA and HIV RNA blood screening assays. *Transfusion*, 42: 527-536, 2002.

Mackie, F.E., Meyer, T.W. & Campbell, D.J. Effects of antihypertensive therapy on intrarenal angiotensin and bradykinin levels in experimental renal insufficiency. *Kidney Int.*, 61: 555-563, 2002.

Matthias, L.J., Yam, P.T., Jiang, X.M., Vandegraaff, N., Li, P., Poumbourios, P., Donoghue, N. & Hogg, P.J. Disulfide exchange in domain 2 of CD4 is required for entry of HIV-1. *Nat Immunol.*, 3: 727-732, 2002.

Michell, B.J., Harris, M.B., Chen, Z.P., Ju, H., Venema, V.J., Blackstone, M.A., Huang, W., Venema, R.C. & Kemp, B.E. Identification of regulatory sites of phosphorylation of the bovine endothelial nitric-oxide synthase at serine 617 and serine 635. *J. Biol. Chem.*, 277: 42344-42351, 2002.

Mozsolits, H., Unabia, S., Ahmad, A., Morton, C.J., Thomas, W.G. & Aguilar, M.I. Electrostatic and hydrophobic forces tether the proximal region of the angiotensin II receptor (AT1A) carboxyl terminus to anionic lipids. *Biochemistry*, 41: 7830-7840, 2002.

Nielsen, J.N., Wojtaszewski, J.F., Haller, R.G., Hardie, D.G., Kemp, B.E., Richter, E.A. & Vissing, J. Role of 5'AMP-activated protein kinase in glycogen synthase activity and glucose utilization: insights from patients with McArdle's disease. *J. Physiol.*, 541: 979-989, 2002.

Pasquini, G.M.F., Davey, R.A.M., Ho, P.W.M., Michelangeli, V.P., Grill, V., Kaczmarczyk, S.J. & Zajac, J.D. Local secretion of parathyroid hormone-related protein by an osteoblastic osteosarcoma (UMR 106-01) cell line results in growth inhibition. *Bone*, 31: 598-605, 2002.

Polekhina, G., House, C.M., Traficante, N., Mackay, J.P., Relaix, F., Sassoon, D.A., Parker, M.W. & Bowtell, D.D. Siah ubiquitin ligase is structurally related to TRAF and modulates TNF-alpha signaling. *Nat. Struct. Biol.*, 9: 68-75, 2002.

Quinn, J.M., Whitty, G.A., Byrne, R.J., Gillespie, M.T. & Hamilton, J.A. The generation of highly enriched osteoclast-lineage cell populations. *Bone*, 30: 164-170, 2002.

Romas, E., Sims, N.A., Hards, D.K., Lindsay, M., Quinn, J.W., Ryan, P.F., Dunstan, C.R., Martin, T.J. & Gillespie, M.T. Osteoprotegerin reduces osteoclast numbers and prevents bone erosion in collagen-induced arthritis. *Am. J. Pathol.*, 161: 1419-1427, 2002.

Ruangpanit, N., Price, J.T., Holmbeck, K., Birkedal-Hansen, H., Guenzler, V., Huang, X., Chan, D., Bateman, J.F. & Thompson, E.W. MT1-MMP-dependent and -independent regulation of gelatinase A activation in long-term, ascorbate-treated fibroblast cultures: regulation by fibrillar collagen. *Exp. Cell. Res.*, 272: 109-118, 2002.

Schuurman, R., Brambilla, D., de Groot, T., Huand, D., Land, S., Bremer, J., Benders, I. & Boucher, C.A. Underestimation of HIV type 1 drug resistance mutations: results from the ENVA-2 genotyping proficiency program. *AIDS Res. Hum. Retrovir.*, 18: 243-248, 2002.

Sims, N.A., Dupont, S., Krust, A., Clement-Lacroix, P., Minet, D., Resche-Rigon, M., Gaillard-Kelly, M. & Baron, R. Deletion of estrogen receptors reveals a regulatory role for estrogen receptors-beta in bone remodeling in females but not in males. *Bone*, 30: 18-25, 2002.

Sims, N.A., Sabatakos, G., Chen, J.S., Kelz, M.B., Nestler, E.J. & Baron, R. Regulating DeltaFosB expression in adult Tet-Off-DeltaFosB transgenic mice alters bone formation and bone mass. *Bone*, 30: 32-39, 2002.

Stephens, T.J., Chen, Z.P., Canny, B.J., Michell, B.J., Kemp, B.E., McConell, G.K. Progressive increase in human skeletal muscle AMPKalpha2 activity and ACC phosphorylation during exercise. *Am. J. Physiol. Endocrinol. Metab.*, 282: E688-E694, 2002.

Tester, A.M., Sharp, J.A., Dhanesuan, N., Waltham, M. & Thompson, E.W. Correlation between extent of osteolytic damage and metastatic burden of human breast cancer metastasis in nude mice: real-time PCR quantitation. *Clin. Exp. Metastasis*, 19: 377-383, 2002.

Thomas, H.E., Darwiche, R., Corbett, J.A. & Kay, T.W. Interleukin-1 plus γ-interferon-induced pancreatic β-cell dysfunction is mediated by beta-cell nitric oxide production. *Diabetes*, 51: 311-316, 2002.

Trivett, M.K., Walker, T.I., Macmillan, D.L., Clement, J.G., Martin, T.J. & Danks, J.A. Parathyroid hormone-related protein (PTHrP) production sites in elasmobranchs. *J. Anat.*, 201: 41-52, 2002.

Tsitrin, Y., Morton, C.J., el-Bez, C., Paumard, P., Velluz, M.C., Adrian, M., Dubochet, J., Parker, M.W., Lanzavecchia, S. & van der Goot, F.G. Conversion of a transmembrane to a water-soluble protein complex by a single point mutation. *Nat. Struct. Biol.*, 9: 729-733, 2002.

White, A.R., Multhaup, G., Galatis, D., McKinstry, W.J., Parker, M.W., Pipkorn, R., Beyreuther, K., Masters, C.L. & Cappai, R. Contrasting, species-dependent modulation of copper-mediated neurotoxicity by the Alzheimer's disease amyloid precursor protein. *J. Neurosci.*, 22: 365-376, 2002.

Wilkinson-Berka, J.L., Kelly, D.J., Rong, P., Campbell, D.J. & Skinner, S.L. Characterisation of a thymic renin-angiotensin system in the transgenic m(Ren-2)27 rat. *Mol. Cell. Endocrinol.*, 194: 201-209, 2002.

Wojtaszewski, J.F., MacDonald, J.N., Nielsen, Y., Hellsten, G.D., Hardie, G.D., Kemp, B.E., Kiens, B. & Richter, E.A. Regulation of 5'AMP-activated protein kinase activity and substrate utilization in exercising human skeletal muscle. *Am. J. Physiol. Endocrinol. Metab.*, 17:17, 2002.

Yang, X., Halladay, D., Onyia, J.E., Martin, T.J. & Chandrasekhar, S. Protein Kinase C is a mediator of the synthesis and secretion of osteoprotegerin in osteoblast-like cells. *Biochem. Biophys. Res. Commun.*, 290: 42-46, 2002.

Zhou, H., Kartsogiannis, V., Quinn, J.M., Ly, C., Gange, C., Elliott, J., Ng, K.W. & Gillespie, M.T. Osteoclast inhibitory lectin, a family of new osteoclast inhibitors. *J. Biol. Chem.*, 277: 48808-48815, 2002.

REVIEWS and BOOK CHAPTERS

Campbell, D.J. Renin-angiotensin system inhibition: how much is too much of a good thing? *Internal Med. J.*, 32: 616-620, 2002.

Campbell, D.J. Valsartan in chronic heart failure (Letter to Editor). *N. Engl. J. Med.*, 346: 1173-1174, 2002.

Choong, P.F. Orthopaedics. *Med. J. Aust.*, 176: 30, 2002.

Choong, P.F. Bone and soft tissue tumours. *ANZ. J. Surg.*, 72: 317, 2002.

Choong, P. Malignant periphseral nerve sheath tumours. In: "Oxford Textbook of Orthopedics & Traumatology". Eds. Blustrode, C., Buckwalter, J., Carr, A., Marsh, L., Fairbank J., Wilson-MacDonald, J. et.al., Oxford University Press, p. 315-318, 2002.

Choong, P. Synovial sarcoma. In: "Oxford Textbook of Orthopedics & Traumatology". Eds. Blustrode, C., Buckwalter, J., Carr, A., Marsh, L., Fairbank J., Wilson-MacDonald, J. et.al., Oxford University Press, p. 312-315, 2002.

Choong, P. Leiomyosarcoma of soft tissue. In: "Oxford Textbook of Orthopedics & Traumatology". Eds. Blustrode, C., Buckwalter, J., Carr, A., Marsh, L., Fairbank J., Wilson-MacDonald, J. et.al., Oxford University Press, p. 309-312, 2002.

Choong, P. Fibrosarcoma of soft tissue. In: "Oxford Textbook of Orthopedics & Traumatology". Eds. Blustrode, C., Buckwalter, J., Carr, A., Marsh, L., Fairbank J., Wilson-MacDonald, J. et.al., Oxford University Press, p. 307-309, 2002.

Choong, P. Malignant fibrous histiocytoma. In: "Oxford Textbook of Orthopedics & Traumatology". Eds. Blustrode, C., Buckwalter, J., Carr, A., Marsh, L., Fairbank J., Wilson-MacDonald, J. et.al., Oxford University Press, p. 304-307, 2002.

Choong, P. Liposarcoma. In: "Oxford Textbook of Orthopedics & Traumatology". Eds. Blustrode, C., Buckwalter, J., Carr, A., Marsh, L., Fairbank J., Wilson-MacDonald, J. et.al., Oxford University Press, p. 300-304, 2002.

Choong, P. Staging and surgical margins in musculoskeletal tumours. In: "Oxford Textbook of Orthopedics & Traumatology". Eds. Blustrode, C., Buckwalter, J., Carr, A., Marsh, L., Fairbank J., Wilson-MacDonald, J. et.al., Oxford University Press, p. 110-112, 2002.

Choong, P. Malignant vascular tumours of soft tissue. In: "Oxford Textbook of Orthopedics & Traumatology". Eds. Blustrode, C., Buckwalter, J., Carr, A., Marsh, L., Fairbank J., Wilson-MacDonald, J. et.al., Oxford University Press, p. 318-323, 2002.

Choong, P. Management of the patient with carcinoma of unknown origin metastatic to bone. In: "Oxford Textbook of Orthopedics & Traumatology". Eds. Blustrode, C., Buckwalter, J., Carr, A., Marsh, L., Fairbank J., Wilson-MacDonald, J. et.al., Oxford University Press, p. 323-329, 2002.

Hammet, A., Pike, B.L., McNees, C.J. & Heierhorst, J. FHA domains as phosphothreonine binding modules. *Austral. Biochem.*, 33: 9-12, 2002.

Marks, R. The changing incidence and mortality of melanoma in Australia. Recent *Results in Can. Res.*, 160: 113-121, 2002.

Marks, R., Foley, P.A. & Sinclair, R.D. Dermatology. *Med. J. Aust.*, 176: 10, 2002.

Martin, T.J. Manipulating the environment of cancer cells in bone: a novel therapeutic approach. *J. Clin. Invest.*, 110: 1399-401, 2002.

Martin, T.J. Some light shines on the resorption cavity. IBMS BoneKey, 2002.

Martin, T.J. RANKL limits its own osteoclastogenic effect. IBMS BoneKey, 2002.

Martin, T.J. & Cornish, J. Other factors controlling bone growth and development - calcitonin, CGRP, osteostatin, amylin and adrenomedullin. In: "Paediatric bone: biology and diseases". Eds. Glorieux, F.H., Pettifor, J.M. & Jueppner, H. Academic Press, San Diego, 2002.

Parker, M.W. Synchrotron light sources: essential tools for revealing protein structures and for drug design. *ATSE Focus* (Magazine of the Australian Academy of Technological Sciences and Engineering), 121: 9-12, 2002.

Romas, E., Gillespie, M.T. & Martin, T.J. Involvement of receptor activator of NFkappaB ligand and tumor necrosis factor-alpha in bone destruction in rheumatoid arthritis. Bone, 30: 340-346.

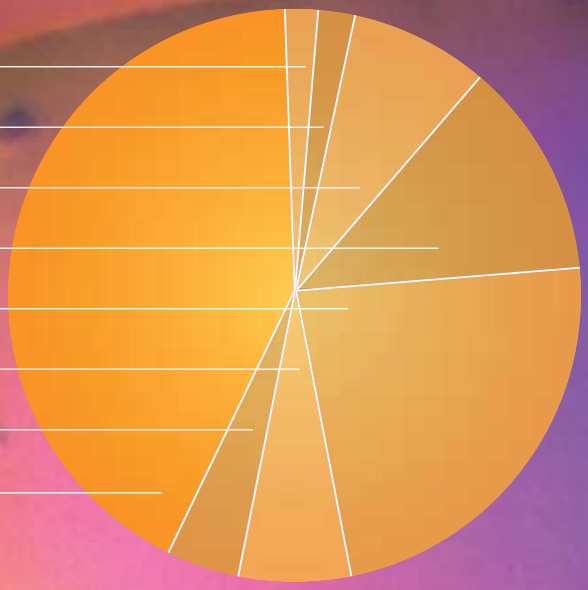
Sims, N. & Baron, R. Bone: Structure, Function, Growth and Remodeling. In: Orthopaedics. Eds: Fitzgerald, R.H., Kaufer, H., Malkani, A. & Mosby, M.O. p147-159, 2002.

Sinclair, R & Marks, R. A guide to the performance of diagnostic procedures used in the management of common skin diseases. Department of Dermatology, St. Vincent's Hospital Melbourne, 2002.

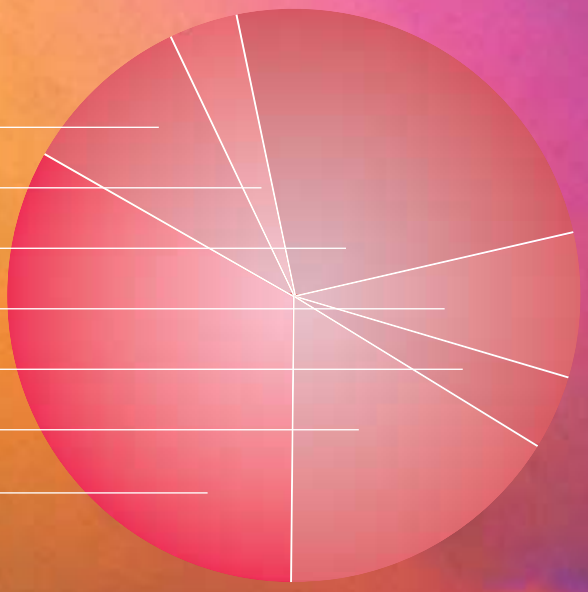
Thompson, E.W. & Price, J.T. Mechanisms of tumour invasion and metastasis: emerging targets for therapy. Expert Opin. Ther. Targets, 6: 217-233, 2002.

Financial Snapshot

INCOME	$ 000's
Interest and Dividends	176
Other	142
Victorian Govt Infrastructure	882
Legacies and Donations	1,205
Commonwealth & State Building Grants	2,353
Industry	583
Commonwealth Govt Service Contract	321
Competitive Research Grants	4,526
TOTAL	**7,783**



EXPENDITURE	$ 000's
Infrastructure - Admin &Tech Support Salaries	909
Infrastructure - General	341
Leasehold improvements	2,306
Depreciation	877
Transfers to Collaborators	272
Research Consumables	1,565
Research Salaries	3,097
TOTAL	**4,433**



DIRECTORS' FINANCIAL REPORT

Your Directors present their report on the company for the financial year ended 31 December 2002.

1. Board of management

The names of Directors in office at any time during or since the end of the year are:

Mr James D Best
Ms Marcia Griffin
Mr Richard G Larkins
Mr Graham EN Rogers
Mr Douglas A Wright
Mr Laurence Clemens
Mr Charles A Griss
Mr Ian D Reid
Ms Brenda M Shanahan

Mr Barry J Jackson (from 27 May 2002)
Ms Nicole M Feely (from 22 July 2002)

Ms Kerrie L Cross (retired 15 July 2002)
Mr Terrence R Power (retired 25 October 2002)

Sr Kathleen Higgs (deceased 22 November 2002)

Directors have been in office since the start of the financial year to the date of this report unless stated otherwise.

2. Principal activity

The principal activity of the company during the financial year was medical research. There was no significant change in the nature of the company's principal activity during the financial year.

3. Operating results

The operating surplus of the company amounted to a surplus of $820,847.

4. Dividends

In accordance with the company's constitution no funds are distributed either to members of the Board or members of the company.

5. Review of operations

During the financial year the company's revenue increased by $2,634,457 on last year. The major reason for this was the major capital works grant income of $2,353,181 for the building extensions currently in progress. The company, in terms of operating research activities, showed a more balanced picture with revenue and expenses being more closely aligned.
The company has undertaken a $10.5 million building extension and refurbishment program, due for completion in September 2003.

6. Significant changes in state of affairs

No significant changes in the company's state of affairs occurred during the financial year.

7. After balance date events

No matters or circumstances have arisen since the end of the financial year which significantly affected or may significantly affect the operations of the company, the results of those operations, or the state of affairs of the company in future financial years.

8. Future developments

The expansion of the existing building will overcome space limitations and enhance the company's research activities.

9. Environmental issues

The company operates predominantly within the medical research sector and is committed to conducting its business activities with respect for the environment while continuing to meet expectations of members, employees, customers and suppliers. During the period from 1 January 2002 to the date of this report, this company has complied with the requirements of the *Environmental Protection Act.*

DIRECTORS' FINANCIAL REPORT

10. Meetings of directors

During the financial year, 29 meetings of directors (including committees) were held. Attendees were:

	Directors' Meeting		Committee Meetings					
			Appeal		Finance		Building	
	Number eligible to attend	Number Attended	Number eligible to attend	Number Attended	Number eligible to attend	Number Attended	Number eligible to attend	Number Attended
JD Best	6	6	-	-	-	-	-	-
L Clemens	6	3	-	-	-	-	-	-
KL Cross	3	3	-	-	-	-	-	-
NM Feely	3	2	-	-	-	-	-	-
M Griffin	6	5	-	-	-	-	-	-
CA Griss	6	5	-	-	7	7	6	6
Sr K Higgs	5	3	-	-	-	-	-	-
BJ Jackson	5	5	-	-	-	-	-	-
RG Larkins	6	4	-	-	-	-	-	-
TR Power	4	4	6	6	-	-	-	-
ID Reid	6	6	-	-	7	6	6	4
GEN Rogers	6	4	-	-	7	6	-	-
BM Shanahan	6	5	10	10	7	7	6	4
DA Wright	6	5	10	10	-	-	-	-

11. Directors' and auditors' indemnification

The company has not, during or since the financial year, in respect of any person who is or has been an officer or auditor of the company or a related body corporate:

- indemnified or made any relevant agreement for indemnifying against a liability incurred as an officer, including costs and expenses in successfully defending legal proceedings;
- paid or agreed to pay a premium in respect of a contract insuring against a liability incurred as an officer for the costs or expenses to defend legal proceedings; with the exception of the following matters.

During or since the financial year the company has paid premiums to insure each of the following directors against liabilities for costs and expenses incurred by them in defending any legal proceedings arising out of their conduct while acting in the capacity of director of the company, other than conduct involving a wilful breach of duty in relation to the company: JD Best, L Clemens, KL Cross, NM Feely, M Griffin, CA Griss, Sr K Higgs, BJ Jackson, RG Larkins, TR Power, ID Reid, GEN Rogers, BM Shanahan, DA Wright.

12. Proceedings on behalf of company

No person has applied for leave of Court to bring proceedings on behalf of the company or intervene in any proceedings to which the company is a party for the purpose of taking responsibility on behalf of the company for all or any part of those proceedings. The company was not a party to any such proceedings during the year.

Signed in accordance with a resolution of the Board of Directors.

Director
ID Reid

Director
CA Griss

Dated this 14th day of April 2003, Melbourne, Australia

STATEMENT OF FINANCIAL PERFORMANCE FOR THE YEAR ENDED 31 DECEMBER 2002

	Note	2002 ($)	2001 ($)
Revenues from ordinary activities	2	10,188,119	7,553,662
Consumables used	3	(1,201,208)	(1,313,329)
Employee benefits expense	3	(3,983,487)	(4,310,726)
Depreciation and amortisation expenses		(877,304)	(484,003)
Other expenses from ordinary activities	3	(3,305,273)	(669,954)
Net surplus from ordinary activities	14	820,847	775,650
Total changes in equity		**820,847**	**775,650**

The accompanying notes form part of these financial statements.

STATEMENT OF FINANCIAL POSITION FOR THE YEAR ENDED 31 DECEMBER 2002

	Note	2002 ($)	2001 ($)
CURRENT ASSETS			
Cash assets	8	5,683,110	2,845,601
Receivables	7	694,541	617,355
TOTAL CURRENT ASSETS		6,377,651	3,462,956
NON-CURRENT ASSETS			
Receivables	7	250,000	250,000
Other financial assets	9	366,708	67,588
Property, plant & equipment	10	1,981,861	2,646,763
TOTAL NON-CURRENT ASSETS		2,598,569	2,964,351
TOTAL ASSETS		8,976,220	6,427,307
CURRENT LIABILITIES			
Payables	11	804,121	167,099
Funds held in trust for NSRL accrued leave		138,280	138,280
Provisions	12	511,688	729,381
Other – grants in advance	13	1,824,690	597,982
TOTAL CURRENT LIABILITIES		3,278,779	1,632,742
NON-CURRENT LIABILITIES			
Provisions	12	167,092	85,063
TOTAL NON-CURRENT LIABILITIES		167,092	85,063
TOTAL LIABILITIES		3,445,871	1,717,805
NET ASSETS		**5,530,349**	**4,709,502**
EQUITY			
Retained surplus	14	5,530,349	4,709,502
TOTAL EQUITY	16	**5,530,349**	**4,709,502**

The accompanying notes form part of these financial statements.

STATEMENT OF CASH FLOWS FOR THE YEAR ENDED 31 DECEMBER 2002

	Note	2002 ($) Inflows (Outflows)	2001 ($) Inflows (Outflows)
CASH FLOW FROM OPERATING ACTIVITIES			
Grants received		9,006,598	6,167,430
Payments to suppliers and employees		(6,107,564)	(6,315,880)
Donations, legacies and bequests		1,108,448	611,377
Other revenue		1,195,768	245,853
Interest received		153,133	169,632
Dividends		8,390	2,641
Leasehold improvements		(2,305,973)	-
Net cash used in operating activities	20	**3,058,800**	**881,053**
CASH FLOW FROM INVESTING ACTIVITIES			
Purchase of plant and equipment		(219,680)	(1,114,992)
Payments for investments		(1,611)	(21,157)
Cash transfer (reclassification of investments as cash)		-	842,920
Net cash used in investing activities		**(221,291)**	**(293,229)**
Net Increase/(decrease) in cash held		2,837,509	587,824
Cash at the beginning of the year		2,845,601	2,257,777
Cash at the end of the year	20	5,683,110	2,845,601

The accompanying notes form part of these financial statements.

NOTES TO THE FINANCIAL STATEMENTS FOR THE YEAR ENDED 31 DECEMBER 2002

Note 1: Statement of Significant Accounting Policies

The financial report is a general purpose financial report that has been prepared in accordance with Accounting Standards, Urgent Issue Group Consensus Views, other authoritative pronouncements of the Australian Accounting Board and the *Corporations Act 2001*. The financial report covers St. Vincent's Institute of Medical Research, a company limited by guarantee, incorporated and domiciled in Australia. The financial report has been prepared on an accrual basis and is based on historical costs and does not take into account changing money values or, except where stated, current valuations of non-current assets. Cost is based on the fair values of the consideration given in exchange for assets.

The following is a summary of the material accounting policies adopted by the company in the preparation of the financial report. The accounting policies have been consistently applied, unless otherwise stated.

NOTES TO THE FINANCIAL STATEMENTS FOR THE YEAR ENDED 31 DECEMBER 2002

Note 1: Statement of Significant Accounting Policies continued

(a) Income Tax
The company is granted exemption from income tax under Subdivision 50-B of the Income Tax Assessment Act 1997 because of the charitable nature of the business within which it operates.

(b) Property, Plant and Equipment
Plant and equipment are carried at cost, less, where applicable, any accumulated depreciation or amortisation. The company does not own property. The carrying amount of plant and equipment is reviewed annually by directors to ensure it is not in excess of the recoverable amount from these assets. The recoverable amount is assessed on the basis of the expected net cash flows which will be received from the assets' employment and subsequent disposal. The expected net cash flows have not been discounted to their present values in determining recoverable amounts. Directors resolved to write off $264,374 of obsolete assets, including computers and research equipment, for the 2002 financial year.

(c) Depreciation
Depreciable assets with a cost in excess of $2,000 are capitalised and depreciation has been provided over their estimated useful lives using the diminishing value method for pre 1 January 1998 and straight line method for assets purchased after this date. The depreciation rates used for Plant and Equipment range from 10% to 33%.

(d) Foreign Currency Transactions and Balances
Foreign currency transactions during the year are converted to Australian currency at the rates of exchange applicable at the dates of the transactions. Amounts receivable and payable in foreign currencies at balance date are converted at the rates of exchange ruling at that date. The gains and losses from conversion of short-term assets and liabilities, whether realised or unrealised, are included in the surplus from ordinary activities as they arise.

(e) Employee Entitlements
Provision is made for the company's liability for employee entitlements arising from services rendered by employees to balance date. Employee entitlements expected to be settled within one year together with entitlements arising from wages and salaries and annual leave, which will be settled after one year, have been measured at their nominal amount. Other employee entitlements payable later than one year have been measured at the present value of the estimated future cash outflows to be made for those entitlements. Contributions are made by the company to employee superannuation funds and are charged as expenses when incurred. The company's long service leave liability of $254,535 represents a gross liability of $506,264 offset by net present value contractual obligations of $251,729 from National Health and Medical Research Council (NHMRC), applicable up to 31 December 2001. This payment will be receivable upon payment of long service leave by the company on behalf of eligible employees. NHMRC reimburse long service leave payments on a pro-rata basis for the period of their grant support.

(f) Cash
For the purpose of the statement of cash flows, cash includes cash on hand and at call deposits with banks or financial institutions, investments in money market instruments maturing within less than two months and net of bank overdrafts.

(g) Revenue
Grant income is recognised upon performing the research associated with the specific grant. Donation income is recognised upon receipt or when spent, if funds were received for a specific purpose. Interest income is recognised as it accrues.
Dividend revenue is recognised when the dividend is received.
All revenue is stated net of the amount of good and services tax (GST).

(h) Equipment Purchases
The company's revenue generated from ordinary activities includes funds raised for the purchase of assets. In the financial year ending 31 December 2002, revenue raised for asset purchases was $212,402.

(i) National Serology Reference Laboratory
The company is the host company for the National Serology Reference Laboratory (NRL). In this role the company provides administration services to the 31 employees. The NRL financial reporting is separate from the company and reported on a 30 June financial year basis to the Commonwealth Government.

(j) Goods and Services Tax (GST)
Revenues, expenses and assets are recognised net of the amount of GST, except where the amount of GST incurred is not recoverable from the Australian Tax Office. In these circumstances the GST is recognised as part of the cost of acquisition of the asset or part of an item of the expense. Receivables and payables in the statement of financial position are shown inclusive of GST.

Note 2: Revenue

	Note	2002 ($)	2001 ($)
Operating activities			
- grants	4-6	7,462,536	5,625,382
- infrastructure support (Victorian State Government)		882,225	716,677
- contract services		320,618	242,642
- legacies and bequests		846,967	415,440
- donations		357,725	195,937
- dividends	(a)	8,390	2,641
- interest	(b)	167,374	169,632
- royalty		-	82,794
- conference		19,716	43,660
- other		122,568	58,857
Total revenue		**10,188,119**	**7,553,662**
(a) Dividends from:			
- other corporations		8,390	2,641
		8,390	2,641
(b) Interest from:			
- other corporations		167,373	169,632
		167,373	169,632

Note 3: Surplus from Ordinary Activities

	2002 ($)	2001 ($)
Surplus from research activity has been determined after:		
(a) Expenses		
Direct cost of research activities:		
Direct research expenses		
- consumables	1,169,719	1,035,524
- salaries and on costs	3,096,550	3,368,816
- other	395,453	450,132
	4,661,722	**4,854,472**
Transfer of funds to external joint collaborators	**272,231**	**87,059**
Infrastructure cost of research activities:		
- administration	295,836	366,361
- salaries and on costs (includes laboratory technical support)	908,883	919,070
- other	45,323	67,047
	1,250,042	**1,352,478**
Depreciation and amortisation of non-current assets	**877,304**	**484,003**
(b) Significant Revenue and Expenses:		
The following revenue and expense items are relevant in explaining the financial performance.		
- Leasehold improvements	2,305,973	-
- Revenue raised for asset purchases	212,402	1,114,992
- Revenue raised for building refurbishment and extension	2,353,181	-

Note 4: Grants – Commonwealth Government

	2002 ($)	2001 ($)
National Health and Medical Research Council	3,040,857	2,615,828
Australian Research Council	194,920	184,445
Department of Health and Aging	1,535,000	-
	4,770,777	**2,800,273**

Note 5: Grants – Victorian State Government

	2002 ($)	2001 ($)
Department of Innovation, Industry & Regional Development	818,181	-
	818,181	**-**

NOTES TO THE FINANCIAL STATEMENTS FOR THE YEAR ENDED 31 DECEMBER 2002

Note 6: Grants – Other

	2002 ($)	2001 ($)
Agouron Pharmaceuticals Inc.	-	83,467
Assoc. International Cancer Research	22,417	86,418
AXA Asia Pacific Holdings Ltd.	-	150,000
Biota Holdings Ltd.	60,985	103,830
Chugai Pharmaceuticals Co.	288,285	225,000
Gastroenterological Society of Australia	27,258	27,234
Juvenile Diabetes Research Fdn.	147,118	-
Max Planck Research Award	100,352	10,444
Mercury Therapeutics Inc.	704	51,231
National Heart Foundation of Australia	86,880	82,780
National Institutes of Health	88,914	-
Novartis Pharma AG	36,251	69,994
Pfizer Pty. Ltd.	104,006	326,832
Servier Laboratories (Aust) Pty. Ltd.	-	25,000
Servier Laboratories International	11,260	-
Solvay Pharmaceuticals Co.	-	47,557
St. Vincent's Hospital, Melbourne	13,399	21,379
Susan G. Komen Cancer Fdn.	164,305	-
The Cancer Council of Victoria	152,115	173,050
Thomaïy Breast Cancer Fund	-	48,000
University of Melbourne	10,760	131,789
US Army Medical Research Command	-	127,433
Victorian Breast Cancer Research Consortium	458,587	503,877
Wellcome Trust	17,198	529,794
Victoria Department of Education	25,000	-
Transfer from other Institutes	57,784	-
	1,873,578	**2,825,109**

Note 7: Receivables

	2002 ($)	2001 ($)
CURRENT		
Grants and reimbursements	694,541	617,355
NON-CURRENT		
St. Vincent's Hospital - Imprest Advance	250,000	250,000
	944,541	**867,355**

Note 8: Cash Assets

	2002 ($)	2001 ($)
Cash at bank and on hand	3,671,789	918,106
Debentures – At cost		
- ANZ Bank Term Deposit	923,532	888,601
Deposits at call		
- Perpetual Trustees	926,807	885,206
- Macquarie Treasury Fund	160,982	153,688
	5,683,110	**2,845,601**

Note 9: Other Financial Assets

	2002 ($)	2001 ($)
Shares in listed Corporations – At cost	366,708	67,588
Market value of listed Corporations	386,568	62,687

Note 10: Property, Plant & Equipment

	2002 ($)	2001 ($)
Plant and equipment at:		
- Directors' valuation 1/1/90	-	841,359
Less accumulated depreciation	-	678,795
Written down value	**-**	**162,564**
- Post 1/1/90 assets at cost	4,692,745	6,348,332
Less accumulated depreciation	2,710,884	3,864,133
Written down value	1,981,861	2,484,199
Total plant and equipment	**1,981,861**	**2,646,763**

Movements in Carrying Amounts:
Movement in the carrying amounts for each class of property, plant and equipment between the beginning and end of the current financial year.

Balance at the beginning of the year	2,646,763	2,015,774
Additions	212,402	1,114,992
Disposals/write off	(264,374)	-
Depreciation expense	(612,930)	(484,003)
Carrying amount at the year end	**1,981,861**	**2,646,763**

Note 11: Payables

	2002 ($)	2001 ($)
CURRENT		
Trade creditors	682,314	165,823
Sundry creditors	121,807	1,276
	804,121	**167,099**

Note 12: Provisions

	Note	2002 ($)	2001 ($)
CURRENT			
Employee entitlements	(a)	511,688	729,381
		511,688	**729,381**
NON-CURRENT			
Employee entitlements	(b)	167,092	85,063
		167,092	**85,063**
(a) Aggregate employee entitlement liability		678,780	814,444
(b) Number of employees at year end		80	78

Note 13: Grants in Advance

	2002 ($)	2001 ($)
Australian Research Council	17,532	-
Carson, Mr G	-	25,000
Chugai Pharmaceutical Co.	361,360	95,000
Commonwealth Dept of Health and Aging	465,000	-
Eli Lilly Pty. Ltd.	26,348	-
John Holt Estate	-	87,000
Juvenile Diabetes Research Fdn.	221,181	-
Max Planck Award	-	44,721
National Health & Medical Research Council	263,950	64,485
National Institutes of Health	166,373	-
Novartis Pharma AG	38,744	-
Pfizer Pty. Ltd.	-	163,416
Servier Laboratories International	78,740	-
Shanahan, Ms B	-	45,000
Susan G Komen Fdn.	73,229	-
The Cancer Council of Victoria	16,543	-
Victorian Dept. of Human Services	-	56,860
Victorian Dept. of Education, Training and Development	-	16,500
Other	95,690	-
	1,824,690	**597,982**

Note 14: Retained Surplus

	2002 ($)	2001 ($)
Retained surplus at the beginning of the financial year	4,709,502	3,933,852
Net surplus/(deficit) attributed to the company	820,847	775,650
Retained surplus at the end of the financial year	**5,530,349**	**4,709,502**

Note 15: Capital Commitments

	2002 ($)	2001 ($)
Capital expenditure commitments contracted for:		
- capital expenditure projects	8,180,776	-
	8,180,776	-
Payable:		
- not later than 1 year	8,180,776	-
	8,180,776	-

The commitment will be financed from internal funds, grants, fund raising and credit standby facility.

Note 16: Members' Guarantee Funds

The company is limited by guarantee. Every member of the company undertakes to contribute to the assets of the company in the event of its being wound up while it or he/she is a member or within one year afterwards for payment of the debts and liabilities of the company contracted before the time at which he/she ceases to be a member and the costs, charges and expenses of winding up and for an adjustment of the rights of contributories among themselves such amount as may be required not exceeding twenty dollars. The number of members at 31 December 2002 is 38 (2001 : 38).

Note 17: Segment Reporting

The company operates in the medical research sector where it undertakes basic and clinical research in Australia.

Note 18: Related Party Transactions

Transactions between related parties are on normal commercial terms and conditions no more favourable than those available to other parties unless otherwise stated.

Transactions with related parties:

Ms B Shanahan, a Director, is a Director of a company which provided investment advice during the year under normal commercial terms and conditions.

NOTES TO THE FINANCIAL STATEMENTS FOR THE YEAR ENDED 31 DECEMBER 2002

Note 19: Funds held in Perpetuity

The accumulated funds at the end of the financial year of $5,530,349 include funds held in perpetuity of $400,418. The income from these funds is directed to the company's medical research program.

Note 20: Cash Flow Information

	2002 ($)	2001 ($)
(a) Reconciliation of cash:		
Cash at the end of the financial year as shown in the statement of cashflows is reconciled to items in the statement of financial position as follows:		
Cash on hand and cash advances	3,671,789	918,106
Deposits at call	2,011,321	1,927,495
	5,683,110	**2,845,601**
(b) Reconciliation of cash flows from operations with surplus from ordinary activities:		
Surplus from ordinary activities	820,847	775,650
Non-cash flows in surplus from ordinary activities:		
Depreciation - plant and equipment	877,304	484,003
Changes in assets and liabilities:		
(Increase)/Decrease in debtors & accrued revenue	(69,909)	(170,865)
(Increase)/Decrease in non-current receivables	-	(250,000)
Increase/(Decrease) in creditors	637,022	56,874
Increase/(Decrease) in grants & donations in advance	1,226,708	(176,888)
Increase/(Decrease) in provision for employee entitlements	(135,664)	162,279
Increase in non-cash/share donations	(297,508)	-
Cash flows from operations	**3,058,800**	**881,053**

(c) Credit Standby arrangement and loan facility:
On the 6th December 2002 the company established a $3.5m overdraft facility with the Catholic Development Fund as a standby arrangement for funding the building extension. The facility is renegotiable on 30 June 2005 and the overdraft interest rate is variable.

	3,500,000	-
	3,500,000	**-**

Note 21: Auditor's Remuneration

	2002 ($)	2001 ($)
- audit or reviewing the financial report	10,728	7,750
- other services	-	-
	10,728	**7,750**

Note 22: Remuneration and Retirement Benefits

	2002 ($)	2001 ($)
(a) Directors' Remuneration		
Income paid or payable to all the directors of the company, directly or indirectly, by the company or any related party.	-	-
(b) Retirement and Superannuation Payments		
Amounts of a prescribed benefit given during the year by the parent entity or a related party to a director or prescribed superannuation fund in connection with the retirement from a prescribed office.	-	-

The names of the company's directors, who held office during the financial year are:

ID Reid	BM Shanahan
JD Best	DA Wright
L Clemens	B Jackson (from 27 May 2002)
M Griffin	NM Feely (from 22 July 2002)
CA Griss	KL Cross (retired 15 July 2002)
RD Larkins	T Power (retired 25 October 2002)
GEN Rogers	Sr K Higgs (deceased 22 November 2002)

Note 23: Financial Instruments

(a) Interest Rate Risk
The company's exposure to interest rate risk, which is the risk that a financial instrument's value will fluctuate as a result of changes in market interest rates and the effective weighted average interest rates those financial assets and financial liabilities, is as follows:

	Weighted Average Effective Interest Rate 2002(%)	Weighted Average Effective Interest Rate 2001(%)	2002($)	2001($)
Financial Assets				
Cash at bank and on hand	0.3	0.25	3,671,789	918,106
Debentures	4.7	5.1	923,532	888,601
Deposits at call	5.6	5.4	1,087,789	1,038,894
Total Financial Assets			**5,683,110**	**2,845,601**
Financial Liabilities				
Funds held in trust			138,280	138,280
Total Financial Liabilities			**138,280**	**138,280**

(b) Credit Risk

The maximum exposure to credit risk, excluding the value of any collateral or other security, at balance date to recognised financial assets is the carrying amount of these assets, net of any provisions for doubtful debts, as disclosed in the statement of financial position and notes to the financial statements.

The company does not have any material credit risk exposure to any single debtor or group of debtors under financial instruments entered into by the company.

(c) Net Fair Values

The net fair values of assets and liabilities approximates their carrying value.

No financial assets are readily traded on organised markets in standardised form other than listed investments.

The aggregate net fair values and carrying amounts of financial assets and financial liabilities are disclosed in the statement of financial position and in the notes to the financial statements.

Note 24: Superannuation Commitments

The company contributes to employee superannuation funds managed by external fund managers. Members of the funds are entitled to benefits on retirement, disability or death. Employees contribute to the funds at 7% of their gross salaries and the company contributes 14% of employees' gross salaries. Contributions to the Tertiary Education Superannuation Scheme (TESS) are to meet the company's Superannuation Guarantee and Award obligations to all its employees and currently amount to 9% of employees' gross salaries for employees who are not members of the employee contribution schemes and 3% for employees who are members of the employee contribution schemes.

The company is under no legal obligation to make up any shortfall in the fund's assets of the superannuation schemes to meet payments due to employees. 93% of the company's superannuation contributions are made to Unisuper Ltd, which manages the Superannuation Scheme for Australian Universities and TESS. The last actuarial assessment of the Superannuation Scheme for Australian Universities defined benefits superannuation fund was completed by Mr. Grant Harslett FIA, FIAA of Towers Perrin on 30 June 2002.

As at 30 June 2002 (being the latest available information):

	2002 ($)
Fund assets at net market value	1,833,072
Accrued benefits	1,970,698
Shortfall of fund assets over accrued benefits	(137,626)
Vested benefits	1,970,698
Employer contributions to the various funds by the company for the 12 month period ending 31 December 2002	466,269

The accrued benefits for each member of the Superannuation Scheme for Australian Universities (SSAU) have been calculated as the greater of:

(a) the present value of future payments of benefits to the member which arise from membership of SSAU up to the reporting date, determined using the actuary's current expectations of earnings on SSAU's assets, future inflation and salary levels and other relevant assumptions, and

(b) the vested benefits.

Vested benefits are benefits which are not conditional upon the continued membership of the fund or any factor, other than resignation from the fund.

Note 25: Company Details

The registered office and principal place of business of the company is:

St. Vincent's Institute of Medical Research
9 Princes Street
Fitzroy, Vic 3065

DIRECTORS' DECLARATION

The directors of the company declare that:
1. The financial statements and notes, as set out on pages 45 to 54 are in accordance with the *Corporations Act 2001*:
a) comply with Accounting Standards and the Corporations Regulations 2001: and
b) give a true and fair view of the financial position as at 31 December 2002 and of the performance for the year ended on that date of the company:
2. In the directors' opinion there are reasonable grounds to believe that the company 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 Directors.

Director
ID Reid

Director
CA Griss

Dated this 14th day of April 2003, Melbourne, Australia

INDEPENDENT AUDIT REPORT TO THE MEMBERS OF ST. VINCENT'S INSTITUTE OF MEDICAL RESEARCH

Scope

We have audited the financial report of St. Vincent's Institute of Medical Research for the financial year ended 31 December 2002, comprising the Statement of Financial Performance, Statement of Financial Position, Statement of Cash Flows, Notes to the Financial Statements and Directors' Declaration. The company's directors are responsible for the financial report. We have conducted an independent audit of this financial report in order to express an opinion on it to the members of the company.
Our audit has been conducted in accordance with Australian Auditing Standards to provide reasonable assurance whether the financial report is free of material misstatement. Our procedures included examination, on a test basis, of evidence supporting the amounts and other disclosures in the financial report, and the evaluation of accounting policies and significant accounting estimates. These procedures have been undertaken to form an opinion whether, in all material respects, the financial report is presented fairly in accordance with Accounting Standards and other mandatory professional reporting requirements and statutory requirements so as to present a view which is consistent with our understanding of the company's financial position and performance as represented by the results of its operations and its cash flows.
The audit opinion expressed in this report has been formed on the above basis.

Audit Opinion

In our opinion, the financial report of St. Vincent's Institute of Medical Research is in accordance with:
a) the *Corporations Act 2001* including:
  i) giving a true and fair view of the company's financial position as at 31 December 2002 and of its performance for the year ended on that date, and
  ii) complying with Accounting Standards and the Corporations Regulations 2001; and
b) other mandatory professional reporting requirements.

WEBB CALLAWAY PATON
Chartered Accountants

AP MARKS

Dated this 14th day of April 2003, Melbourne, Australia

DONATIONS

Bequests and Donations from Estates and Charitable Trusts

Merna Dorothea Sheahan Estate	297,508
John Holt Medical Research Endowment Fund	232,229
Helen Macpherson Smith Trust	100,000
Marguerite Mary Holdern Estate	57,009
The Marian & EH Flack Trust	48,500
MJ Polinelli Foundation	29,889
K & A Bongiorno Medical Research Endowment Fund	28,832
Perpetual Trustees (Clive & Vera Ramaciotti Foundation)	27,000
George Castan Family Charitable Trust	15,000
Bell Charitable Fund	9,000
William Angliss (Vic) Charitable Fund	2,000
	846,967

LIST OF DONORS

$50,000 plus

Shanahan, B

$20,000 - $29,999

Carson, G

Anonymous

$10,000 - $19,999

ABN AMRO

Trust Company of Australia Ltd

$5,000 - $9,999

Anonymous

BT Funds Management

Jackson, B

O'Shannassy, M

Vermont Cancer Research Fund

$1,000 - $4,999

Barker, R

Power, T

Reid, I

Smith, S

Spry-Bailey, P

Wantirna Hill Club Patrons

Wark, R

$500 - $999

AMP Life Ltd

Campbell, A

Chappell, JC

Kelly, M

Mayo Consulting Pty Ltd

Rizzo, P

Sagitta Wealth Management Ltd

Standard & Poor's

White, K

Under $500

Adams, K

Adams, R

Andrews, D

Andrews, J

Anonymous

Appleton, C

Appleton, F

Attard, C

Baker-Smith, R

Bakker, P

Ball, L

Bell, B

Beroukas, D

Block, P

Bloomfield, N

Breheny, M

Brennan, J

Brown, A

Bourke, T

Cator, H

Chappell, J

Corrigan, H

Coutney, A

Cox, K

Croxton, S

D'Arcy, J

Dawes-Robinson, J

Diggins, L

Duggan, M

Eaton, L

Eaton Pty Ltd

Ellingham, J

Emerson, S

Fellowes, J

Foote, I

Fox, P

Frost, D

Garvey, A

Gee, L

Green, R

Griffin, M

Hall, T

Hansford, AE

Haworth, P

Hart, Mr & Mrs L

Haydon, M

Henderson, K

Henderson, J

Hickey, K

Hickey, P

Hill, D

Hogg, T

Hopper, T

Howe, W

Ives, C & N

Jackson, B

Kay, C

Keam, M

Klemke, M

Kong, R

Kritikos, P

Kruger, A

Lane, M

Larner, J & C

Lee, S

Leishman, J

Lemke, P

Liow, K

Lonergan, B

Mather, P

McArthur, Z

McHale, G

McVilly, D

Methodist Ladies' College

Middleton, D

Middleton, P

Monotti, J

Moseley, J

Newton, K

Nicoll, G

O'Bryan, NM

O'Callaghan, B

O'Connor, V

Parker, N

Paull, D

Pinford, TD & WM

Plymin, M

Polk, L

Porter, J

Rechner, RJ

Rees, R

Reeve, F

Renard, R

Richmond Hill Café & Larder

Ritchies Stores Pty Ltd

Roche, K

Rockman, S

Rosalion, A

Rose, A

Rossi, B & K

Santamaria, P

Scott, K

DONATIONS

Under $500 continued

Sinclair, K	Veiling, B
Sissiam, F	Victorian Vending
Spiteri, B	Wagner, C
Tan, R	Wagstaff, J
Templeton, M	Walter, D
ter Hedde, D	White, K
Thorburn, L	Whiting, T
Toohey, M	Williams, S
Townsend, T	Williamson, A
Trehy, M	Wood, P
UBS Warburg	Woodley, N
Vagg, B	Wurm, J

Memorial Gifts

Gifts in remembrance have been made in honour of the following:

Bert McMurray
Emma Smith

1000 Club Members

Ralston, M & family
Shanahan, B & family
Xipell, T & Dr J
Alberti AM, S
Barro, R
Best, J
Campbell Tuckfield, P
Carson, G
Chappell, J
Colman, J
Curlewis, D
Griss, C
Grogan, M
Gutman, J
Halliday, J
Kay, C
Kay, T
Kemp, B
Lowe, D
Martin, TJ
McHale, G
Scott, P
Slattery, P
Thomas, C & C
Turner, R

261,481

Permanent Invested Funds

The following permanent funds are included in the company's pool of invested funds with income being directed to the Institute's medical research program.

The Mary Potter Research Grant	90,797
Diane B Jones Endowment	970
Lorna M Miller Endowment	208,651
Albert H Maggs Endowment	100,000
	400,418

ACKNOWLEDGEMENTS

Acknowledgements

We are also grateful for the generous support provided by:

ANZ Bank
Austpaper Pty Ltd
Baby Touch
Beacon Imports
Beryl Shaw
BT Financial
Channel Seven Melbourne
Christine Schang
Crown Limited
Daylesford Getaways
Early Settler Aromatherapy Emu Oil
Flowers by Andrea Louise
Foeden Photography
Gourmet Lovers, Heidelberg
Guiney Mt Martha Tennis Ranch
Hair du Cutt
Hair Odyssey
Hayman Island Great Barrier Reef
K W Doggetts
Klass Kutters
Korimco
la Prairie Switzland
Linea Casa Pty Ltd
Manark Printing Pty Ltd
Marc Pascal
Mt Atkinson Olive Grove Café
Neverfail
New International Imports
Nola Häusler
Origin Design
Richmond Hill Café and Larder
Robert Gordon Australia
Robert Gordon Gallery
Sandhurst Ridge
Sandy Cowling
Scally & Trombone
Sheraton Hotel & Towers Brisbane
SMR Productions Pty Ltd
Solution Red
Staging Connections
Surprise Box
The Brunswick Street Bookstore
The Travel Company
Toohey's
Turnley's Haircare
Village Cinemas Gold Class
Western Bulldogs Football Club
Wilson Everard
Wright's Creative Communications
Yering Station

Your donation will accelerate the pace of our discovery of new treatments for illnesses.

DONATIONS AND BEQUESTS

DONATIONS

The field of research in which the Institute is engaged touch the lives of many Australians. The scientific research of the Institute aimed at the treatment and cure of illness has depended heavily on the support of the community. Your financial support will have a direct effect on the Institute's research.

There are many ways in which you can help. These include making annual or more frequent gifts, making bequests via a Will or making a donation in memory of a loved one or esteemed person. Donations to St. Vincent's Institute of Medical Research are tax deductible. In claiming a tax deduction you may be required to quote the Institute's ABN 52 004 705 640.

Enquiries will be welcomed by the Director of the Institute on (03) 9288 2480.

Contributions are used directly in research, not on administrative costs.

BEQUESTS

The Institute will be pleased to accept the directions of the donor and use capital and income arising from a bequest according to the donor's wishes. However, it is not necessary to specify a particular purpose as all available Institute funds are used solely for medical research. It is advisable that legal assistance be obtained in making such a provision.

Suggested wording for bequests:

"I ....., bequest unto St. Vincent's Institute of Medical Research, 9 Princes Street, Fitzroy, 3065 in the State of Victoria for its general purposes (indicate the amount and/or item and/or address of property) free of all succession, estate and other death duties and declare that the receipt of the Director or other proper officer of the Institute shall be sufficient discharge to my Executors in respect thereof."

DONATION FORM

Please detach form below and forward to
St. Vincent's Institute of Medical Research
41 Victoria Parade Fitzroy Vic 3065
Phone: 03 9288 2480 Fax: 03 9416 2676

My contribution to the Appeal for Life Fund

Name: _____

Address: _____

_____

Suburb: _____ Post Code: _____

Phone: _____ Fax: _____

I wish to donate the amount of: $ _____

I wish to pay by:

Single payment ☐ Annual payment ☐ Other

My payment is by: ☐ Cheque or money order is enclosed
(made payable to St.Vincent's Institute of Medical Research)

☐ Credit Card ☐ Amex ☐ Visa

☐ Bankcard ☐ Mastercard ☐ Diners

Expiry Date: ___ / ___ Signature: _____

☐ *Please do not send any further promotional material*

Thank you for your support

SVIMR Australian Business Number: 52 004 705 640
All amounts of $2.00 and over are fully tax deductible.

Appeal for Life Gift

SVIMR is seeking financial support from the community to build on our achievements by expanding our research program. Your donation will accelerate the pace of our discovery of new treatments for illnesses that many of us and members of our families may suffer.

For further information about the Institute and our Appeal for Life connect to our website or contact us on (03) 9288 2480 during business hours.

www.svimr.unimelb.edu.au

St. Vincent's Institute of Medical Research
41 Victoria Parade Fitzroy Vic 3065
Phone: 03 9288 2480 Fax: 03 9416 2676
Email: svimr@medstv.unimelb.edu.au

Appeal for Life | St.Vincent's Institute of Medical Research

Building for the future of medical research

St. Vincent's Institute
of Medical Research
ACN 004 705 640

Postal
41 Victoria Parade
Fitzroy Victoria 3065

Located at
9 Princes Street
Fitzroy Victoria 3065

Telephone: 03 9288 2480
Facsimile: 03 9416 2676
Email: svimr@medstv.unimelb.edu.au
Web: www.svimr.unimelb.edu.au

SVIMR
MOLECULES TO MEDICINE