
and

MISSION

■ To undertake research to study and improve healthcare delivery and outcomes, including epidemiological studies, particularly among the veteran and war widow community and children of veterans.

■ To optimise support from the wider community in order to facilitate our vision.

■ To promote and conduct health research in the areas of lifestyle and ageing that significantly impact on Australians and their families, and in doing so focus upon the needs of the community serviced by Concord Repatriation General Hospital.

■ To construct a state of the art Research Institute on the campus of Concord Repatriation General Hospital.

■ To provide leadership and excellence in research activities throughout Australasia.

■ To apply research to product development within Australia where possible.

■ To sponsor education and training in relevant health disciplines.
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## ANZAC RESEARCH INSTITUTE RESEARCH REPORTS

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## ANZAC HEALTH & MEDICAL RESEARCH FOUNDATION

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This report was submitted by Professor John Young before his death in February 2004. His vision and contribution have been instrumental in the formation and continued guidance of the ANZAC Health and Medical Foundation throughout its first decade. It is in recognition of his service that his report is retained as that of the chair for 2002-03 Annual Report. Vale.

As Chairman of ANZAC Health and Medical Research Foundation, it is with pleasure that I present the Annual Report for the year 2002-03.

This year the Foundation has been pleased to observe the ANZAC Research Institute's continuing success in its early years of development. Those include the securing several new NHMRC project grants, the graduation of its first PhD students and attracting international scientist to work there. All demonstrating that it is rapidly growing into a front rank biomedical research organisation on an international and national scale.

This year the Foundation has had to consider how it adapts to its changing role from being primarily a charitable fund-raising Foundation aiming to establish and support first class medical research on the Concord campus to operating as the Board overseeing a dynamic and growing medical research institute. To accommodate this, over the year the Constitution was revised and modernised. The new Constitution ratified by the Council in June 2003 has created a governing Board with 12 members reduced from the Council with 24 members but maintaining the key stakeholders.

On this occasion I extend thanks to the retiring councillors who shared the burdens of the Foundation's first decade of development. I thank them most sincerely for their time, energy and expertise given so readily to support the shared vision to establish a strong and effective medical research facility on the Concord Hospital campus. Their valued contributions have paid off in the conspicuous success of the ANZAC Research Institute. Its future success will always owe a debt to the vision and efforts of these pioneers.

In October, the Foundation held a very successful Annual Dinner aimed at raising the Institute's profile in the community as well as raising funds to support the Institute's operation. The 238 guests and supporters enjoyed a fine night of entertainment including the very topical speaker, Richard Butler, former Chief Weapons' Inspector in Iraq and soon to be Governor of Tasmania, who gave an enlightening speech on bioterrorism and modern weapons control.

The growth and development of the ANZAC Research Institute depends critically on the support both of private donors and corporate sponsors of the Foundation. We thank our loyal supporters for their generosity and urge everyone to keep the Foundation's work in mind when opportunities arise to foster the highest quality Australian science and medical progress particularly in the field of ageing.

Once again I am especially pleased to record on behalf of the Foundation thanks to Dr Diana Horvath, CEO of the CSAHS, for her ongoing support of the Institute's growth and work.

I am sure you will all join me in wishing the Director and his staff continued success and another productive year at the ANZAC Research Institute.

Professor J A Young AO, FAA
It is a pleasure to report on the ANZAC Research Institute’s 3rd year of operation. This year has been marked by further exceptional growth and development of the Institute, its company of scientists and its scientific reach.

During this year the ANZAC Research Institute had over 80 closely affiliated researchers on campus and provided a research home to over 40 scientists. In 2002, it attracted $2.2 million of peer-reviewed grant funds including $1.4 million from NHMRC. The Institute’s scientists also won 5 new NHMRC grants for a greater than 50% success rate which greatly exceeds the national average of ~25%. Among other achievements for the year, the Bone Biology laboratory attracted one of the four NSW Government’s first round of BioFirst biotechnology awards.

A major event this year was the renewal of the Institute’s NSW Health Research and Development Infrastructure Grant of $1.3 million over the next three years. This provides the crucial infrastructure to operate the Institute’s basic office and general facility functions. For this application, the Institute made a combined submission with the Centre for Education and Research on Ageing (CERA) to reflect a strategic alliance and convergent research interests of both organisations. While the grant was less than expected and has necessitated economies on desirable research services, we can continue operating and growing. This does however signal that there is a pressing need to diversify our sources of infrastructural support for the Institute’s basic operations.

We are proud that this year saw the graduation of the Institute’s first research degree candidates. These include 5 PhD’s completed by Peter Liu, Lam P Ly, Gloria Foxley, Tian Zhuang and Victoria Cogger together with the first Science Honours graduate, Alvaro Garcia. The University of Sydney has also awarded research academic titles to Institute senior scientists Drs Colin Dunstan, Charles Allan, Michael Muller, Matt Harris, Vadim Dedov and Marina Kennerson.

A key issue for the Institute is purchasing and maintaining new high-tech, specialist research equipment required by the Institute’s scientists. During the last year, with help from the University’s Equipment Grants, the Institute has now acquired a mass spectrometer and a flow cytometer. Further equipment high on our priority list is a Faxitron micro x-ray machine and scanning electron microscope.

This year saw the inception of the Institute’s Transgenic Unit headed by Dr Charles Allan as scientific director and Karen Brennan as manager. Here we aim to become a transgenic production facility creating complex genetic models for human physiology and disease research where these cannot be studied directly in humans. The Andrology laboratory has developed several unique constructed transgenic mouse models to evaluate hormone action in the testis, prostate, blood vessels and other androgen sensitive tissues.

The Institute’s laboratories and facilities are now working actively on many fronts in ageing-focused research. Professor Garth Nicholson’s Neurobiology laboratory continues to be an international leader in neurogenetic research. For his outstanding work, Professor Nicholson was awarded a Personal Chair by the University of Sydney becoming a Professor in Neurogenetics to recognise his sustained and outstanding achievements in this field. His laboratory continues to be highly successful in identifying the genetic causes, and the environmental triggers for, neurodegenerative diseases of the peripheral nervous system such as sensory and motor neuropathies, Parkinson’s disease, acoustic neuroma and Friedrich’s ataxia. During this year his group acquired new NHMRC grant and international funding from the US Muscular Dystrophy Association.

The Andrology laboratory studies male reproductive health, medicine and biology at all phases of life. This year it announced the completion of a world first, a proof of principle study in humans demonstrating a highly effective prototype of a hormonal male contraceptive. This news, highlighted in international and national media as one of the top international news stories of the week, gave prominence to the Institute as a place for world leading research. During this year also Peter Liu, the Andrology Fellow, was invited to participate in the highlight clinical trials presentations and press conference for the 2nd year in a row at the Clinical Trials Symposium of the US Endocrine Society Scientific Meeting. His paper on the trial in the top journal JCEM was featured by the US Endocrine Society Scientific Meeting. His paper on the trial in the top journal JCEM was featured by the US Endocrine Society Scientific Meeting.

Later Peter was awarded the highly competitive and prestigious NHMRC Neil Hamilton Fairley Fellowship to further his postdoctoral studies at the Mayo Clinic in the USA for 2 years and a further 3 years back in Australia.

The Biogerontology laboratory, headed by Professor David Le Couteur from the Centre for Education and Research on Ageing, have continued to research into their important new concept, published originally in the Lancet, linking functional ageing of the liver as a key step in accelerating cardiovascular disease. Using cell culture, animal and human models, this research provides important new leads and targets to...
prevent, treat and reverse heart disease, which remains the major cause of death in our community. During this year, the Biogerontology laboratory recruited back the NHMRC CJ Martin Fellow, Dr Mathew Harris, from America to continue his cutting edge research into the molecular genetics of liver function. Among the accolades this year, Dr Sarah Hilmer, a geriatrician undertaking her PhD studies, was awarded the Pfizer Royal Australasian College of Physicians Advanced Trainee Research Prize. Dr Victoria Cogger also won the Australian Association of Gerontology RM Gibson Scientific Award in recognition of her contribution.

The Bone Biology laboratory headed by Professor Markus Seibel, a top clinician and researcher recruited from Germany to the Institute, has established a strong osteoporosis and bone cancer research program. In securing the return from the USA of Dr Colin Dunstan, an expatriate Australian protein biochemist who was working on bone biology at Amgen, a leading international biotech company. To support Dr Dunstan’s recruitment, Professor Seibel and the Institute won one the inaugural NSW Health BioFirst biotechnology awards fulfilling its aim to bring in new biotechnology researchers to NSW by reversing the “brain drain” for expatriate scientists. During the year, this laboratory also attracted a Research Fellow, Dr Christian Meier, from the Swiss National Fund.

It is planned that within the next year the Institute will establish a Vascular Biology laboratory in conjunction with the Cardiology Department of Concord Hospital and will recruit the top young scientist Dr Paul Witting.

During this year the Institute welcomed a start-up biotechnology research and development company Avastra, which has leased space for its Tissue Welding laboratory within the Institute. In conjunction with burns and plastic surgeon, Dr Peter Maitz AM, this company is commercialising the development of a patented suture-free vascular anastomosis that could revolutionise microvascular surgery.

While the Institute’s scientists have been extraordinarily successful in obtaining competitive grants and commercial contracts, a key determinant of the Institute’s overall future success is its fundraising. The Institute also needs expensive, high technology research equipment as well as funding for scholarships and fellowships. These are crucial for the Institute to maintain its competitive edge both in training the new generation of scientists as well as attracting and retaining top class scientists. Tax-free scholarship stipends are a highly cost-effective way to support research for scientific training of the next generation of scientists. Post-doctoral fellowships help create the critical mass of diverse scientific skills that would recruit and retain valuable scientific expertise. Scientific trainees and scientists form the core of future medical research in this country and the Institute’s focus on ageing is an important way to ensure that vital research into healthy ageing in Australia is encouraged. Our fundraising efforts must try to match the successes of our scientists in grant funding and scientific discoveries.

It continues to be an enormous privilege to have had the continual support of our Chairman, Professor John Young AO. To him I am personally truly grateful. This young organisation could never have started so quickly and effectively without his wisdom and strategic guidance. The vital goodwill and practical support from Dr Diana Horvath, CEO of the Central Sydney Area Health Service and from Mathew Daley and Dr Margaret Sanger of Concord Hospital, continue to be most gratifying. Once again it is my pleasure to acknowledge the Institute’s scientists for their wonderful collegiality and dedication to doing great things for themselves, for their labs and the Institute. The Institute is more than the sum of their parts because of their productive collaborations. We are fortunate indeed to have such fine, accomplished and highly motivated scientific staff. My thanks also go to the Institute’s management team of the General Manager, Christine Harrison with Colleen Fitzgibbon, Marco Fabiani and Annet Doss. Mamdouh Khalil continues to run the Molecular Physiology Unit with exemplary skill and dedication so that we can maintain the highest quality animal facilities. The Institute is fortunate indeed to have such strong organisational support of its goals. Thanks are also due to the members of the Council over the Foundation’s first decade whose support has laid the solid groundwork for the Institute’s development. Finally, I thank our many friends and supporters whose continuing efforts will determine how well the ANZAC Research Institute succeeds in fulfilling its wonderful promise and potential.

The ANZAC Research Institute, now a recognised independent medical research institute by NHMRC and the NSW Government, is proud of its growing achievements in grants, publications and scientific training outlined in the following pages. I hope you will agree that this progress has been remarkably fast and extraordinarily successful.

Prof David Handelsman
Director, ANZAC Research Institute & Professor of Reproductive Endocrinology and Andrology

Institute-wide Grants
NSW Health: $1,332,000, 2000-03
NHMRC Equipment: $65,000, 2003
University of Sydney Equipment: $75,000, 2002; $75,000, 2003
Ramaciotti Foundation: $30,000, 2003

Andrology is the study of male reproductive health, medicine and biology. It covers several key dimensions notably fertility, sexuality and the effects of androgens, male hormones like testosterone, on health and disease. Relevant areas include testicular disorders, male infertility, male contraception, androgen deficiency, hormonal influences on prostate and cardiovascular diseases, and male ageing. Our Andrology group conducts a range of integrated clinical and basic research studies with a focus on male reproductive health across all ages. Comprising of the Andrology Laboratory at the ANZAC Research Institute and Department of Andrology at Concord Hospital, it features an integrated bench-to-bedside approach intended to facilitate successful translational research.

Our research focuses on improvement in these broad aspects of men’s health:
- Development of hormonal male contraception
- Causes, prevention and treatment of prostate disease
- Understanding testicular function, notably hormonal control of sperm production
- Androgen therapy to improve health and well-being
- Understanding the effect of male hormones in health

Development of hormonal male contraception
L Turner, M Jimenez, R McLachlan (Prince Henry’s Institute of Medical Research), DJ Handelsman

The development of a hormonal male contraceptive is a major practical application of improved knowledge about how hormones control sperm production. Men continue to have strong interest in effective family planning with over one third of all contraceptive-using couples relying on traditional male methods of contraception. Yet, not a single new male contraceptive method was introduced over the last century despite the development over the last 4 decades of numerous highly effective, reversible contraceptives for women that have produced profound changes in our society.

Throughout the past decade our group has been the largest centre participating in landmark WHO studies into production of effective male contraception. In the first ever study we have tested and defined the features necessary for a reliable reversible hormonal male contraceptive. Important for safety and reliability was the preference for a long lasting injectable, rather than an oral pill. This uses a combination of the hormones testosterone and progestin, the synthetic analogue of the pregnancy hormone that is used in female contraception.

In a series of detailed clinical studies we have used a depot form of testosterone, defined the lowest effective dose with sufficient suppression but without undesirable side effects and tested it with a progestin to identify the best combination. Using this information, we recently completed a proof-of-principle study showing very high contraceptive efficacy with acceptable short-term safety and acceptability for this prototype depot combination hormonal male contraceptive.

This outstandingly good result for a prototype hormonal regimen is a large step forward. The announcement of the very positive findings of this study, funded by CONRAD, an American public sector agency, made international headline news. These studies lead the world and make great progress in the development of a practical hormonal male contraceptive regimen. They have re-ignited the flagging interest of two multinational pharmaceutical companies in further practical product development based on these findings.

Clinical pharmacology of testosterone
S Kelleher, A Conway, L Turner, DJ Handelsman

The Andrology Department has continued its studies into the clinical pharmacology of testosterone and androgen replacement therapy. Having established the modern clinical pharmacology of subdermal testosterone pellet implants, this method has now become among the main forms of testosterone replacement therapy in Australia. Although very convenient for young men with androgen deficiency, implantation of pellets involves a minor surgical procedure where the major drawback is the occasional extrusion of one or more pellets. Several clinical studies have been employed to identify the cause of extrusion and to prevent or reduce its occurrence. These outcomes allowed us to offer differing sites for implementation and a newer less invasive procedure. Additionally, the data obtained has helped us to determine the effective therapeutic duration of testosterone pellets and at what threshold blood levels of testosterone equal the physical manifestations of insufficient circulating testosterone.

This year we completed a clinical research trial of a new Australian manufactured testosterone cream. Overseas, the development of transdermal patches and topical testosterone products has substantially enhanced the practical options available for men who require testosterone to use convenient products. Yet in Australia only one form of relatively irritating skin
patch and no topical products are available. In this context, we have recently completed a study of thirty androgen deficient men in three Australian centres. Each of them used the cream for three months, whilst they were monitored for hormone levels and quality of life outcomes to assess the effectiveness of this novel form of treatment. Compared with the other available safe options, a lotion or cream has the advantages of being pain-free, non-invasive, convenient and self-administered. Preliminary findings suggest that this cream is well suited to some androgen deficient men and that it provides a useful option among the range of testosterone products available for replacement therapy.

**Genetic models for androgen action in the prostate – androgen receptor (AR) excision models**

P Lim, CM Allan, J Spaliviero, J Singh, G Risbridger (Monash Institute of Reproduction and Development), DJ Handelsman

Using the opportunity provide by modern genetic engineering tools, this research is aiming to create a more specific genetic model to study the way hormones act within the prostate during development and disease. Testosterone has vital effects on the prostate and it is known that these are markedly amplified by conversion to the very potent androgen DHT (dihydrotestosterone), the purest natural androgen. Beyond the fact of this amplification effect, it remains virtually impossible to study the interplay of various hormones within the prostate in the intact animal. In this research project, new transgenic models will be developed to cut out the AR from the prostate in specific locations so as to identify the chain of events in how testosterone acts on the prostate of healthy intact animals. This may provide new targets for prevention and treatment for prostate diseases including prostate cancer.

**Characterisation of androgen-independent genes in the prostate**

J Singh, Q Dong (Sydney Cancer Centre, University of Sydney), DJ Handelsman

Like all prostate cells, prostate cancers start out being very responsive to testosterone. As a result even when prostate cancer is diagnosed at an advanced and incurable stage, removal of testosterone by surgery or drug treatment can reduce prostate cancer cell growth and prolong life. However, during the later stages of prostate cancer, the continued growth of these cancerous cells becomes more aggressive and independent of testosterone. The reasons for this later development during the terminal stages of prostate cancer are not really understood. Better knowledge of why this occurs will provide the chance for even more effective treatments to reduce the impact of spreading prostate cancers. Our work to understand the mechanism of androgen-independent cancer (AIC) is of great importance to reduce debility, and delay death, from prostate cancer.

Earlier studies have shown that androgen signalling pathway can remain active in AIC during androgen ablation therapy. As androgen action is mediated through target genes, identification of these androgen sensitive genes is an important step in gaining insight into the growth and progression of AIC. Using a new experimental approach where genes active in the prostate of mice are compared in different hormonal conditions, we have identified several candidate genes that are regulated by androgens. We have found one gene is over-expressed in prostate cancer. Once fully confirmed, these findings will provide a new marker not only for diagnosis but also for potential therapeutic interventions in prostate cancer.

**Prenatal factors in male reproductive health**

S Wishart, B Jin, J Fonda, J Rodgers, I Chan, A Conway, DJ Handelsman

In contrast to previous studies of male reproductive health, which have focused on genetic or environmental factors in adult life, this study takes a different approach by considering factors before and immediately after birth. This NHMRC funded project is creating a birth cohort of young men now in their early thirties. At this age, testis and prostate development are complete but it is before prostate disease begins. Our underlying concept is that prenatal hormonal environment may be an important, but previously unrecognised, determinant in the development and diseases of the human testis and prostate. We believe that the perinatal gonadotrophin and androgen surge is a critical event in creating hormonal imprinting upon many androgen sensitive tissues. It enables both the optimal development of the testis and prostate at and after puberty, but also sets the stage for late-life androgen-dependent organ diseases. It sets the limits on testicular functional reserve i.e. spermatogenesis, fertility, and androgen secretion, and so impacts subsequent pathological insults dependant on the underlying spermatogenic reserve. Similarly, early life hormonal imprinting may set in train later life prostate diseases, notably nodular prostatic hyperplasia, perhaps by permanently modifying hormonal sensitivity of the prostate stroma and epithelium.

The birth cohort is of men born in inner Sydney around 1970 who have been identified from hospital birth records. Unlike a birth cohort developed by our collaborators in Adelaide involving tracing and non-invasive studies of girls, the recruitment of boys in Sydney for more invasive study has proved more difficult and complex.
Prompted by the necessity to improve recruitment, we have streamlined the procedures as far as possible and have now embarked on a detour to develop a less invasive method of prostate volume measurement. With urological colleagues we have devised a novel, simpler, less invasive ultrasound technique that avoids the need for a transrectal probe. We are currently evaluating this improved strategy before resumption of recruitment of the original birth cohort.

**Role of DHT in androgen effects and prevention of prostate disease**

L Turner, S Kelleher, M Jimenez, A Tourians & L Gooren (Free University, Amsterdam), DJ Handelsman

One advance in understanding the development and diseases of the prostate was the recognition that the prostate has an inbuilt androgen amplification system. The prostate has high expression of type 2 $\alpha$–reductase, which avidly converts virtually all testosterone entering the prostate to DHT. The highly selective expression of type 2 $\alpha$–reductase in the prostate allows for the development of specific type 2 $\alpha$–reductase inhibitors such as finasteride.

In a collaborative study with the Department of Andrology and Endocrinology at the Free University, Amsterdam, we studied the contribution of the prostate to circulating blood DHT concentrations. All circulating DHT is converted from testosterone by the enzyme $\alpha$–reductase. It remains unclear whether DHT has any role as a circulating hormone or is merely a passive reflection by overflow of tissue generation of DHT from testosterone. Previous indirect studies had suggested that the type 2 $\alpha$–reductase contributes 50-80% of circulating DHT. To study the quantitative contribution of the prostate to circulating DHT, we studied patients who were being treated with a fixed dose of testosterone and compared those lacking a prostate i.e. females and male transgender, to androgen deficient men with a prostate. Unexpectedly, this study found that the prostate contributes only very small amounts of DHT to the circulation, indicating that most circulating DHT arises elsewhere.

A recent finding from a study has shown striking effects of DHT in reducing apparent prostate growth rate in otherwise healthy older men. This clue has led to the development of a major study to be conducted at the Andrology Department, Concord Hospital, to evaluate in detail how effective DHT is at preventing prostate growth in middle-aged men without known prostate disease. Sponsored by an overseas pharmaceutical company and in collaboration with the Departments of Endocrinology, Concord Hospital and Cardiology, Royal Prince Alfred Hospital, this study, will also monitor carefully whether the DHT treatment has any adverse effects on bone or the cardiovascular system.

**The role of FSH: genetic models to study FSH action**

CM Allan, J Spaliviero, M Jimenez, DJ Handelsman

The two pituitary-derived hormones, the gonadotrophins FSH and LH, regulate normal testis development, sperm production and male fertility. LH has long been known to be crucial to stimulate the production of testosterone from Leydig cells in the testis. This testosterone has critical function within the testis to stimulate sperm production and is also crucial for male sexual and bodily development. Yet, the role of FSH has remained difficult to study and it has not really been understood. Our current NHMRC-funded research has made unique contributions to understanding how FSH acts in the testis.

In men, FSH is produced by the pituitary and travels to the testis via the bloodstream. There it binds to and activates a specific receptor found exclusively on Sertoli cells, the nurse cells that support, coordinate and nourish sperm production, spermatogenesis. Since the absolute number of Sertoli cells determines the final capacity of sperm production in adult testes, our findings that FSH has its main role in controlling Sertoli proliferation reveals a new dimension to its importance, especially for its most important effects that occur on testicular development and male fertility during early life.

We established new genetic models to study the specific actions of FSH on Sertoli proliferation and spermatogenesis. Our unique transgenic approach selectively restored FSH activity in genetically infertile hypogonadal mice that lack endogenous FSH and LH secretion. We revealed that FSH alone, independently of LH activity, induces complete Sertoli cell proliferation and initiates early stages of sperm development. We also confirmed, these findings with Dr F Zhang of Finland’s complementary mouse model that isolated FSH activity due to targeted disruption of the LH receptor gene.

We have shown previously that testosterone alone was essential to complete all the steps to production of mature sperm. By contrast, our research shows that although not necessary for complete Sertoli cell proliferation, testosterone is required to stimulate full Sertoli cell function that is needed to complete the final stages of sperm development. In combination, FSH and testosterone have strong synergistic but independent effects on sperm production and maturation.

Our research has shown that not only can FSH establish the full Sertoli cell complement of the testis, but also that LH-mediated effects are also a critical determinant for Sertoli function and spermatogenesis. Future studies will focus on the biological pathways activated by FSH during early postnatal life, which ultimately dictate the amount of sperm production in adult males.
The role of testosterone: genetic models for androgen receptor function

CM Allan, P Lim, J Spaliviero, DJ Handelsman

Our previous research has shown that androgens, primarily testosterone, alone could initiate and maintain qualitatively normal spermatogenesis, and restore fertility, in mice that lack all pituitary gonadotrophins. Androgen actions are exerted via a specific androgen receptor (AR) through which expression of a wide variety of androgen-sensitive genes is controlled. Despite the fundamental role of testosterone in spermatogenesis, remarkably little is known about the identity or function of the specific genes or pathways by which testosterone does this.

We recently initiated new projects to identify the pathways and targets of the androgen response. One strategy uses microarray technology to simultaneously examine the expression of over 10,000 genes during testosterone acting for the first time on the testis. This analysis of global gene expression profiles will allow the initial identification of candidate pathways and key factors regulated by androgen, which may ultimately provide new targets for more effective treatments of male infertility, or new strategies for male contraception.

In related research, Patrick Lim commenced his PhD studies in 2003 and will focus on developing new transgenic models to modify AR expression in the testis. Maturing germ cells, which ultimately form spermatozoa, do not express the AR; so androgen-dependent germ cell development must be initiated via cells expressing AR, like Sertoli cells. In future studies new genetic mouse models will be used to evaluate the role and contribution of Sertoli cell AR to the overall steroid response in the testis.

Androgen exposure and its effects on cardiovascular disease in men

AK Death (Heart Research Institute), DS Celermajer (Royal Prince Alfred Hospital), DJ Handelsman

Funded by the NHMRC and National Heart Foundation of Australia, this research aims to study why heart disease affects men earlier and more frequently than women. The deposition of cholesterol in blood vessel walls, atherosclerosis, is the most important cardiovascular disease and is the leading cause of death in developed countries. Several factors including genetic, lifestyle or hormonal, have been recognised as playing a role in cardiovascular disease. ANZAC Research Institute links with the Heart Research Institute to focus on how androgens may be involved in early and late stages of the pathogenesis of heart disease.

The major male hormone is testosterone. Blood testosterone concentrations are at low levels in women and children and at much higher levels in young men after puberty and it remains high until late middle age when levels begin a gradual, slow decline. The timing of this decline coincides with peak incidence of cardiovascular events in men but whether this reflect a short-term effect of lower testosterone concentrations or a delayed effect of long-term exposure to higher testosterone concentrations has not yet been determined. Circumstantial evidence suggests that testosterone has both positive and negative effects on the development of atherosclerosis, the underlying cause of most heart disease. Our hypothesis is that in young males years of androgen conditioning may promote atherosclerosis and then, in later life, declining testosterone concentrations trigger vasoconstriction in already diseased arteries. Recently, it has been recognised that early life environmental exposures affect later life cardiovascular disease. An early life event in men is
the perinatal androgen surge where blood testosterone concentrations reach adult levels for months. We suggest that this androgen surge may trigger molecular and cellular events that lead to the earlier onset atherosclerosis in adult men.

Our studies are using an established and a newly created transgenic model of atherosclerosis. The established model of atherosclerosis is the ApoE-deficient mouse that develops hypercholesterolemia and subsequent plaques within twelve weeks of life. These mice are rendered androgen deficient by castration and subsequently are treated with testosterone or dihydrotestosterone (DHT) for a period of eight weeks. During this time, atherosclerosis lesions form in the sinus and aortic arches of the mice. We are now determining whether the lack of testosterone, restoration of normal testosterone levels, or DHT treatments affects the rate and extent of development of atherosclerosis in these mice.

In contrast, the novel model of atherosclerosis is an ApoE/hpg-deficient mouse. Which aims to combine the attractive features of both genetic expressions. The hpg mice are gonadotrophin deficient and produce no sex steroids, including testosterone. So they lack the perinatal androgen surge that causes hormonal imprinting. By crossing ApoE and hpg mouse lines, we are able to develop an androgen deficient mouse, which has no exposure to the perinatal androgen surge and yet will develop atherosclerosis. Through comparison we will be able to determine the effects of perinatal hormonal imprinting as well as later life androgen exposure have on late life cardiovascular disease.

The results will aid in the understanding of atherosclerosis and allow for the development of diagnostic indicators and possible new therapies for cardiovascular disease.

Grants

NHMRC: $770,000, 2001-03; $225,000, 2002-04; $360,000, 2003-05
University of Sydney Cancer Research Fund: $100,000, 2002-03
National Heart Foundation: $100,000, 2003-04
Andrology Australia: $27,250, 2002-03

Fellowships

Peter Liu
NHMRC Neil Hamilton Fairley Fellowship: $ 350,000, 2003-2006
The ageing liver and oxidative stress
VC Cogger, M Muller, D Le Couteur

Dr Victoria Cogger recently completed her PhD on the morphology of ageing and oxidative stress in the liver and is currently working as a post-doctoral scientist in the Biogerontology Laboratory.

Dr Cogger has been investigating how ageing and oxidative stress affects the ultrastructure of the liver's unique blood vessels, known as sinusoids, and what are the possible implications of these changes. The endothelium of the liver is normally very thin and perforated with pores called fenestrations that are grouped into clusters called sieve plates. The sinusoidal endothelium is strategically situated and structured to enable free exchange of proteins and other nutrients between the hepatocytes and blood. It also shields the hepatocytes from most blood cells, platelets, and the largest of colloids such as chylomicrons and large viruses. In ageing the fenestrations of the liver are reduced in number and become smaller in size and diffuse extracellular matrix deposition is seen in the areas under the endothelium. These changes have now been characterised in three species, rats, baboons and humans. When exposed to oxidative stress the fenestrations of the liver become disrupted resulting in the formation of large gaps in the endothelium. Under these conditions the endothelium offers no protection to the underlying hepatocytes. Changes seen in ageing and oxidative stress are thought to impact on the exchange of substances from the blood into the hepatocytes, reducing the liver's capacity for detoxification and metabolism. This may have implications for the pathogenesis of disease.

In the last year Victoria has presented her work at the International symposium on the cells of the hepatic sinusoid & their relation to other cells in Tucson, Arizona in September 2002.

The genetics of cholestatic liver disease and the ageing liver
M Harris

Dr Matthew Harris is a NHMRC CJ Martin Fellow who joined the Biogerontology Laboratory in December 2002 after two years at Tufts University School of Medicine, Boston USA. Working with the eminent liver researcher, Professor Irwin M. Arias, Dr Harris developed his expertise in liver secretion processes such as the clearance of drugs, cholesterol and bile. He is currently studying the genetic forms cholestasis. Cholestasis is a liver disease where normal secretion of bile is blocked and manifests as jaundice, itching and can progress to liver failure. A severe childhood liver disease, cholestasis has been found to be genetic in nature with the underlying mechanisms of disease remaining elusive. Unraveling the mechanism of this disease will determine the possibility of medication for its remedy.

Dr Harris has also initiated projects that aim to determine the possible genetic changes to the liver during the ageing process.
Oxidative stress and the endothelium

M Muller, D Le Couteur

Dr Michael Muller has established the use of endothelial cell tissue culture as a tool for investigating the effects of drugs and oxidative stress on endothelial morphology. Hepatic sinusoidal endothelial cells from a number of species have been successfully isolated and cultured under laboratory conditions. We are currently studying the relationship between oxidant-induced changes in intracellular redox status and the morphology of hepatic sinusoidal endothelial cells with particularly emphasis on liver sieve dynamics. To provide insight into this process, mobilisation of F-actin filaments, proteins controlling endothelial sieve morphology, is being visualised by confocal microscopy under conditions of oxidative stress. In conjunction with studies on oxidative stress and the hepatic endothelium, work has also been progressing on understanding the role of mitochondrial dysfunction, induced either by chemicals or age, and sinusoidal defenestration. This is an exciting project that may link the mitochondrial theory of ageing with our observations of age-related sinusoidal defenestration.

Chemical-induced oxidative stress and liver tumour formation

M Muller

In other liver related work, Dr Muller published an invited review paper on the mechanisms of mouse liver tumour formation in response to redox active chemicals and their relevance to human risk assessment. This work is of importance to international chemical regulatory agencies as it examines the mechanistic relationships between chemical exposure, hepatic metabolism, oxidative stress and the incidence of liver tumour formation.

Mechanisms of hepatic sinusoidal defenestration

VC Cogger, S Hilmer, M Muller, F Braet (Free University of Brussels, Belgium), D Le Couteur

Previous studies have shown that F-actin filaments are important in forming fenestrations and maintaining their morphology although the dynamics of this process are not well understood. The reorganisation of F-actin filaments is an energy dependent process requiring the energy rich molecule ATP. In collaboration with Dr Filip Braet, a visiting scientist to the Biogerontology Laboratory from the Free University of Brussels, Belgium, we explored the relationship between intracellular ATP levels and fenestration morphology. We subsequently showed that as ATP levels decreased so did the number of fenestrations, including loss of whole sieve plates. During this work a novel structure was identified that we have tentatively termed a defenestration centre. This work has been accepted for publication in the journal Hepatology.

As mitochondria are the subcellular structures responsible for ATP production we are following up our initial observations with other studies that examine the relationship between mitochondrial dysfunction, induced either by chemicals or age, and sinusoidal defenestration. We are also exploring the role of environmental influences on the sinuoids of the liver. In particular, we are interested in the role dietary oxidised fats may have on the hepatic sinusoids and their effect on endothelial fenestrations. Previously, we have shown that oxidants delivered directly into the liver via the portal vein induce morphological changes in the hepatic sinusoids, including loss of fenestrae. In preliminary experiments we have shown that dietary-derived oxidised fats reach the portal vein in concentrations high enough to induce oxidative stress. We are now investigating the effects of dietary oxidised fats on hepatic sinusoid morphology and function.

Cast of the liver sinusoidal system.
Grants
NHMRC: $360,000, 2003-05
NHMRC/DVA: $240,000, 2002-05

Fellowships
Matthew Harris
NHMRC CJ Martin Fellowship: $320,000, 2001-05

Scholarships
Sarah Hilmer
NHMRC Postgraduate Medical Scholarship: $85,296, 2002-04
Allesandra Warren
NHMRC Dora Lush Postgraduate Scholarship: $65,559, 2002-04

Prizes
Sarah Hilmer
Pfizer Royal Australasian College of Physicians
Advanced Trainee Research Prize: 2003
Victoria Cogger
Australian Association of Gerontology RM Gibson
Scientific Award: 2003
The Bone Research Program pursues research in basic bone biology, applied bone metabolism and clinical research into metabolic bone disease. Although the basic and applied streams of the Bone Research Program are still in their infancy, a number of exciting and competitive projects are already underway or nearing completion. Furthermore, the enthusiastic members of the research team were successful in attracting research funds both from within Australia and overseas.

Our future plan is to further develop a comprehensive research program that makes use of the many opportunities provided by a multidisciplinary institution such as the ANZAC Research Institute. Our goal is to intensify collaborations with basic and clinical researchers on the Concord campus, and to extend our research efforts to other areas relevant to bone biology and disease.

Preventing the spread of malignant tumours to bone
C Dunstan, MJ Seibel

Breast cancer and prostate cancer have a particular preference to form cancer metastases in bone. Breast cancer in bone is associated with bone destruction that frequently results in significant pain and disability. Prostate cancer cells in bone induce high rates of bone formation and bone resorption resulting in disorganisation of bone structure and severe pain. Appearance of bone metastases is an indication of poor prognosis in both of these cancers, and treatment frequently is only palliative. Bone provides a "fertile soil" for breast and prostate cancer cells that have moved from the original cancer site. Tumour cells grow in bone and induce normal cells of the bone marrow to destroy the surrounding hard bone. The destruction of bone releases factors that help cancer cells grow faster thus creating a vicious cycle that contributes to the serious consequences of bone metastases.

We are studying mice with transplanted breast cancer cells to understand what makes the bone marrow a receptive site for breast cancer metastasis. Using one of the body’s own bone protecting proteins, such as osteoprotegerin, to block bone destruction we will evaluate its ability to inhibit cancer cells to invade and grow in bone. In collaboration with Dr Julie Brown from Oncology Research Centre, Prince of Wales Hospital, we will conduct similar studies with prostate cancer. Treatment of breast and prostate cancer could be significantly improved if bone could be modified to either block the targeting of cancer to bone, or to inhibit its growth there.

We have also introduced methods for growing bone forming cells, osteoblasts, and bone resorbing cells, osteoclasts, in the laboratory. These will be used to investigate further potential interactions between bone cells and cancer cells in cell culture.

Hormonal and genetic determinants of age-related changes in bone turnover and predictors of fracture risk in elderly men
C Meier, K Brennan, J deWinter, MJ Seibel

Fractures resulting from osteoporosis are a major cause of hospital admission, loss of independence, and reduced life expectancy in the elderly. Osteoporosis affects not only women, but also men. It is estimated that approximately one third of osteoporotic fractures occur in men. Several independent risk factors for fracture have been identified, such as bone mineral density, bone turnover rate, family history, and presence of other diseases. However, not all men, and especially not all men with age-related hormone deficiency, exhibit a decrease of bone mass leading to osteoporosis. It is our hypothesis that variations in genes involved in hormone regulation could be a major determinant of osteoporosis in men.

In this study we aim to identify the forces behind age-related changes in bone metabolism and bone strength, and identify best predictors of fracture risk, and to evaluate the association between genetic variation with bone loss and fracture risk. In collaboration with Prof JA Eisman, Bone and Mineral Research Program, Garvan Institute of Medical Research, Sydney we have used for this study the large population of elderly men contained in the Dubbo Osteoporosis Epidemiology Study.

This project is expected to shed light on the presently poorly understood mechanisms causing osteoporosis in men. Results from this study are expected to provide opportunities for prevention and better treatment of osteoporosis.

Studies into the relative contributions of estrogen and testosterone on bone turnover in men
C Meier, P Liu, J de Winter, DJ Handelsman, MJ Seibel

Sex hormones have a major importance on accrual and the maintenance of bone mass. While the role of estrogen in regulating bone metabolism in women is well understood, the contribution of
estrogen and androgens in regulating bone turnover in men is unclear. Several investigations have demonstrated that estrogen, as estradiol is also important to maintain bone mass in men.

In collaboration with Dr P Liu and Prof DJ Handelsman we evaluated the effect of the most potent naturally occurring androgen, dihydrotestosterone (DHT), and recombinant human chorionic gonadotrophin (rhCG) compared to placebo on bone turnover in healthy, elderly men over 60 years. Our results confirm earlier studies that estradiol, secreted in rhCG treated men, has a prominent effect on bone in older men, although no direct effect of DHT on bone turnover was found.

Further studies to assess the impact of sex hormones on bone metabolism in men are planned. We are interested in finding the level of estradiol that is required by the skeleton to suppress bone turnover and maintain bone mass. This project attempts to find more effective treatment of osteoporosis in men.

Bone structure and turnover in hypogonadal mice

N Sims, K Brennan, J Spalivero, T Borovina, DJ Handelsman, MJ Seibel

In a collaborative effort between the Bone Biology and Andrology laboratories and Dr. Natalie Sims at St. Vincent’s Hospital, Melbourne, we are studying the effects of male sex hormones on the structure and turnover of bone. The effects of female sex hormones on bone density, specifically the withdrawal of estrogen after meno-pause, are already well known. Osteoporosis in elderly men is also a recognised problem, as are the effects of testosterone-deficiency on bone. We aim in this project to contribute to the understanding of the processes by which androgens influence bone strength in males.

Using the testosterone deficient hypogonadal (hpg) mouse to examine the bone structure and turnover, we have demonstrated significant differences in bone turnover in testosterone deficiency. We also demonstrated that hormone replacement was able to normalise bone turnover.

Glucocorticoid-induced changes in bone metabolism

MJ Seibel, C Dunstan, K Brennan

Our laboratory has a strong interest in transgenic models of bone disease. This project is looking at the mechanism governing normal and abnormal bone metabolism, in particular the effects of glucocorticoids (GC) on bone. New techniques have been developed and transgenic models are being used to elucidate the role of pre-receptor GC metabolism in the pathogenesis of GC-induced bone loss. In the long term, these studies hope to point the way to strategies for the reversal or even prevention the detrimental effects of cortisone on the skeleton.

Effects of Bisphosphonates on serum OPG and RANKL levels in patients with postmenopausal osteoporosis and Paget’s disease of bone

C Meier, C Dunstan, F. Lang, M Hooper, MJ Seibel

Osteoprotegerin (OPG) and RANK ligand (RANKL) are part of an important cytokine system regulating the differentiation and activity of bone resorbing cells, osteoclasts. Bisphosphonates have been used to successfully treat metabolic bone diseases characterised by bone resorption like in postmenopausal osteoporosis. Recently it has been shown in cell cultures that bisphosphonates modulate OPG production in human osteoblasts. There is however no data on the effects of bisphosphonates on OPG and soluble RANKL levels in patients with metabolic bone diseases.

In this study we will investigate the change in serum levels of OPG and RANKL and their correlation with different biochemical bone markers in patients on bisphosphonate treatment. The results of this trial will give us a better understanding by which mechanisms bisphosphonates reduce bone resorption.

Genetic determinants of susceptibility to Paget's disease of bone

C Meier, N Kormas, F Lang, MJ Seibel, M J Hooper

Paget’s disease of the bone (PDB) is the second most common form of metabolic bone disease after osteoporosis. The aetiology of Paget's disease of the bone is unknown, although it is often seen in...
multiple family members and seems exhibit autosomal dominant inheritance. We have collected DNA samples from both affected and unaffected family members in kindreds with familial PDB and from sporadic cases.

A previous study demonstrated a mutation in Exon 1 of the TNFRSF11A gene in a family with aggressive familial PDB. This gene is responsible for the production of RANK, the receptor that induces osteoclast differentiation and seems to be an appropriate candidate gene for mutations. In collaboration with Prof Garth Nicholson, Dr Marina Kennerson, ANZAC Research Institute, and Prof Stuart Ralston, Aberdeen, Scotland, we studied patients with familial PDB for mutations in the TNFRSF11A and SQSTM1 genes. We were unable to identify a mutation in either locus, suggesting that these mutations are rare in Australian families with PDB. A new polymorphism however was identified in 13 patients with PDB in our cohort but its clinical significance is yet unknown. Further studies are underway including a genome search for other candidate genes in familial PDB.

These bone markers have been developed recently and are still being refined and are already widely used as an assessment tool. We are developing an experimental and clinical validation method for novel or improved markers of bone turnover.

In association various collaborators, studies are focusing on evaluation of bone turnover in the very elderly, the effect of androgens on male bone health, the effect of growth hormones and androgens on bone metabolism in elite athletes, the use of bisphosphonates on the healing of prostheses used for total hip replacements, amongst other topics.

**Grants**

NSWHealth BioFirst Award: $300,000, 2002-04
National Institute of Health (USA): $35,000, 1999-2003
Swiss National Foundation with Roche Foundation: $310,000, 2002-04
International Osteoporosis Foundation: $40,000, 2002-03

**Industry Grants**

Amgen (USA): $83,400, 2003
Nova Nordisk (Switzerland): $40,000, 2003-04

**Studies on biochemical markers of bone metabolism**

J de Winter, D Gaston-Parry, C Meier, MJ Seibel

All metabolic bone diseases are characterised by changes in the two processes that keep bone alive, healthy and strong, bone formation and bone resorption. Measurement of specific bone markers in serum and urine determines their activity and these results can help to assess the severity and monitor therapy of bone diseases like osteoporosis.
The Neurobiology laboratory has an international reputation for finding new genes causing disease of peripheral nerves. The main interest is to determine how these gene mutations cause disease and in particular why they often lead to degeneration of nerves and brain tissues with ageing.

The focus of our work is the mechanism of adult onset neurodegenerations. As part of this work, we are developing new methods to study causes of cell death in disorders such as hereditary sensory neuropathy and motor neurone disease. New techniques have been developed to look at the accumulation of abnormal proteins in these disorders.

In the last year, the laboratory has mapped the chromosomal location for the mutation for two new diseases, a sensory neuropathy with gastro-oesophageal reflux and cough and a new form of Charcot-Marie-Tooth (CMT) neuropathy. The clinical features of this new form of CMT are described in a paper, which was recently published in the journal, Neurology.

Finding the gene for dominant intermediate Charcot-Marie-Tooth Neuropathy
ML Kennerson, D Zhu, G Walizada, GA Nicholson

Charcot-Marie-Tooth (CMT) neuropathy is one of the most common groups of human hereditary disorders. This syndrome is a disorder of peripheral nerve affecting both motor and sensory neurons. Our research focus has been to identify the gene causing a new form of CMT, known as dominant intermediate Charcot-Marie-Tooth neuropathy, which was earlier described by our group. We have localised the gene to chromosome 19 and have refined the region of DNA to search to 2.6 million base pairs. Database resources of the Human Genome Project and completion of sequencing the human genome has provided this project with a wealth of information about the genes in this chromosomal region. We are using this data to identify genes that are expressed in brain and nerve and to prioritise mutation screening.

Identification of genes causing CMT neuropathies will enhance our knowledge of the molecules involved in the maintenance of peripheral nerve function and provide insight into the cellular mechanisms of disease leading to neurone degeneration and the late onset degenerative disorders of nerve.

Finding gene for Hereditary Motor and Sensory Neuropathy with pyramidal signs
D Zhu, ML Kennerson, G Nicholson

Hereditary Motor and Sensory Neuropathy or Charcot-Marie-Tooth neuropathy (CMT) is the most common inherited peripheral neurological disease in humans. It is clinically characterised by weakness and atrophy of distal muscles, depressed or absent deep tendon reflexes and mild sensory loss. People with CMT usually show symptoms of clumsiness, weakness and difficulty in balance and movement early in life. This disease has a wide variety of genetic causes and maps, with about thirty other hereditary peripheral neuropathies, to several chromosomes.

The variant we are studying, CMT with pyramidal signs (CMT-P), is genetically distinct from other dominantly inherited axonal neuropathies. It presents with stiffness of the limbs unlike other types of CMT.

Our aim is to identify the chromosomal location and gene mutation causing CMT-P. We are studying a large family of several generations where eleven people are affected. Preliminary linkage analysis from genotyping data revealed two possible chromosomes associated with this disease. We are now looking at additional markers within these two regions to narrow down the linkage and find the gene eventually.

Charcot-Marie-Tooth Neuropathy with cough and gastroesophageal reflux
C Kok, ML Kennerson, GA Nicholson

We have linked the gene mutation causing a unique form of HSN type I, to a new locus on chromosome 3, in a single large Australian family. Patients affected with this syndrome have symptoms of a sensory neuropathy with a chronic cough and gastroesophageal reflux (GOR). A second family has been identified with similar clinical symptoms that have been linked to the same region and through genetic information from this family the locus region has been refined. Two of the three strong known candidate genes for pathogenic role in HSN with GOR and cough have been excluded. Currently a physical map of the region is been made to confirm predicted genes.

Finding the gene mutation causing this disorder will provide a diagnostic test for the family. It will also provide information about the pathogenic mechanism of this disorder. Our clinical studies indicate that GOR may be a frequently unrecognised effect of clinically neuropathies. Results of this study will be published in The American Journal of Human Genetics in the September 2004.
New genes causing familial motor neurone disease and hereditary motor neuropathies.

S Gopinath, ML Kennerson, GA Nicholson

Motor Neurone Disease (MND) is an adult onset neurodegenerative disorder characterised by death of motor neurons in the cortex, brainstem and spinal cord. In this condition muscle weakness and atrophy results in death usually within 3-5 years of symptom onset. Rapid advances being made in the study of MND suggest that it is caused by a combination of genetic and environmental factors.

We are studying families with MND, without mutations in the highly penetrant SOD1 gene, to locate more genes that can cause motor neurone disease. In collaboration with other overseas investigators, we are performing linkage studies for known familial MND loci using high throughput analyses to screen DNA. Identification of new genes causing MND will help to understand the biology of the motor neurons.

Our study extends to families with motor neuropathy, in whom we are screening known genes and loci causing hereditary motor neuropathy (HMN). Identification of HMN genes also contributes to our understanding of motor neuron pathology and possible therapeutic implications.

Understanding the molecular mechanisms of Hereditary Sensory Neuropathy type 1 (HSN1)

V Dedov, I Dedova, GA Nicholson

HSN1 is the most common hereditary disorder of peripheral sensory neurones. It is caused by autosomal dominant degeneration of dorsal root ganglia and motor neurones. HSN1 patients suffer from progressive sensory loss in the feet, distal muscle wasting and weakness, chronic skin ulcers and distal amputation. The pathogenesis of this disease is unknown.

However, our group found the gene and mutations responsible for this disease in the gene SPTLC1, encoding subunit 1 of serine palmitoyl transferase (SPT). This enzyme catalyses the first step in sphingolipid synthesis that is important for cell survival.

Now our aim is to uncover the molecular mechanisms causing the death of neurones in HSN1 and eventually find treatments to reverse or slow down the disease. To study HSN1 we are using cell cultures of patient’s lymphocytes, which carry the mutation. We are using advanced optical and biochemical techniques to study the biology, life span and characteristics of these cells in order to define a model that can be used to screen compounds to beat the disease.

HSN1 Lymphocytes in cell culture
Developing and characterising a neural degenerative transgenic mouse model as a first step towards finding a treatment for the human disease, hereditary sensory neuropathy type I (HSN1).

SJ Myers, GA Nicholson

This project is funded by USA-MDA and aims to address the precise mechanisms causing neural degeneration in HSN1. It plans to explore the function, trafficking and signaling of the causative mutant protein, serine palmitoyl transferase long chain subunit 1 (SPTLC1).

Very little is known about the pathogenesis of HSN1, we will use a transgenic mouse model to examine the impaired function of the mutant gene with respect to altered protein expression, activity and membrane localisation of SPTLC1. By reviewing impaired function in relation to the neuropathology of peripheral nerve and dorsal root ganglia, we will test compounds to prevent or slow the disease.

Grants
NHMRC: $615,000, 2001-03: $225,000, 2003-05
Muscular Dystrophy Association (USA): $241,320, 2002-05
Amyotrophic Lateral Sclerosis Association (USA): $87,500, 2002-05
NHMRC Equipment: $10,000, 2003
Ramociotti Foundation: $19,000, 2003

Scholarships
Sumana Gopinath
Motor Neurone Association Research Institute of Australia: $24,000, 2003

Prizes
Cindy Kok
Princes Beatrice Fonds (Netherlands): $18,000, 2003-04

Neurobiology Researchers
Back Row from left: Cindy Kok, Danqing Zhu, Garth Nicholson, Vadim Dedov, Simon Myers
Front Row: Gina Walizada, Marieke Tuuew, Sumana Gopinath, Marina Kennerson
The Effect of Rhinovirus infection on Asthmatics

BGG Oliver, J Black, N King, S Lim

The common cold virus or rhinovirus for most of us is relatively harmless and leads only to the development of cold symptoms in the upper respiratory tract. However for many people the same virus can result in hospitalisation due to worsening of an existing respiratory disease such as asthma or chronic obstructive pulmonary disease (COPD).

To understand better the inflammatory processes that cause such dramatic deterioration in certain people, we have been examining the role of rhinoviral infection in cells found in the airways. Following infection by rhinovirus the cells in the airways release small protein messengers collectively known as cytokines. These messenger molecules attract other cells from the immune system to the lungs where they accumulate and may be the trigger for an asthma attack or COPD exacerbation.

We are examining the ability of macrophages to limit a rhinoviral infection. Macrophages are cells found within the lungs that limit infections by both bacteria and viruses. We have found that rhinoviruses are able to infect these cells, but the macrophage is capable of limiting viral infection. The macrophage can be thought of as a sponge that is able to mop up viral particles and stop them from infecting other cells.

We are also examining the interactions that occur between rhinovirus and the smooth muscle cells of the airway. When the virus infects these cells, it replicates and spreads to other cells. Infection of these cells causes the cells to die and to release various cytokines. We think that the combination of these effects will cause the smooth muscle cells to contact reducing the airway diameter thus making it harder for people to breath.

We are examining these mechanisms in the hope that it will lead to the development of a treatment for the common cold virus.

The Dose-Dependent Molecular Mechanisms of Corticosteroids in Asthmatic Inflammation.

M Baraket, J Black, N King, S Lim

Corticosteroids are the most effective medication for the prevention and treatment of asthma. Although unfavourable side effects are more numerous and more likely to occur in response to orally or intravenously administered corticosteroids, inhaled corticosteroids may be associated with adverse effects, particularly at high doses and when used for prolonged periods.

This study is examining the effects of asthma medication on airway inflammation in patients with mild to moderate asthma who are prescribed different doses of the same inhaled corticosteroid for six weeks. The expression of various relevant cytokines or inflammatory proteins and cytokine genes are examined by analysing lung cells obtained via bronchoscopy, before and after treatment. The aim is to explore the question of whether there is a different effect on the way corticosteroids work after low versus high dose inhaled corticosteroids.

The Regulation of Inflammation in Chronic Obstructive Pulmonary Disease (COPD)

LJ Seeto, M Roth, J Black, P Johnson, S Lim

This project aims to find more effective therapies for COPD sufferers through examination of their inflammatory processes. To further this research, this year has been spent continuing collaborative research at the National Heart and Lung Institute, Royal Brompton Hospital, London, which is one of the key research institutes in respiratory medicine in the world. New experimental techniques have been acquired to study differences between lung cells and airway tissues from smokers and never smokers in an attempt to further determine the mechanisms underlying COPD.

Two papers were presented at the national and international Thoracic Society conferences: one detailing enzyme interactions for the development of smoking-related disease and the other discussing the anti-inflammatory effects of newer steroid and bronchodilator therapy on lung macrophages in people with COPD.

Grants
NHMRC: $390,000, 2002-04

Scholarships
Melissa Baraket
Australian Postgraduate Award, PhD Scholarship: $58,977, 2002-04
STAFF & STUDENTS 2002-2003

Director
Prof David Handelsman MB BS, PhD, FRACP

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Prof David Le Couteur MB BS, PhD, FRACP
Dr Sam Lim MB BS, PhD, FRACP
Prof Garth Nicholson MB BS PhD
Prof Markus Seibel MD, PhD, FRACP

Clinical Research Associates
Dr Ann Conway MB BS, FRACP

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Dr Christian Meier MD (Switzerland)

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Dr Colin Dunstan PhD
Dr Vadim Dedov MD (Russia), PhD
Dr Jillian Kril PhD
Dr Marina Kennerson PhD
Dr Michael Muller MA, PhD
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Dr Jaskarit Singh PhD
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Dr Lam Ly MD (Vietnam) PhD

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Ben Marshan BSc (Hons)
Gina Walizada BSc (Hons)

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Gloria Foxley BSc (Hons)
Dr Sumana Gopinath MB BS, FRACP
Dr Sarah Hilmer BSc (Hons), MB BS, FRACP
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Drs Cindy Kok DRS
Brian Oliver MSc
Dr Linda Seeto MB BS
Dr Soji Swaraj MB BS, FRACP, MBA
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Sabina Horky
Ljubica Vrga BSc
Matilda Webby

Administration
General Manager
Christine Harrison BSc (Hons), FIBMS, Grad Dip Bus, AFACHSE, AIMM

Molecular Physiology and Animal Services
Manager
Mamdouh Khalil BAgSci

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S Craig Rudd BCom, Grad Dip Acc, CA
Annet Doss Dip Acc, Dip Comp Prog

Computer Support
Marco Fabian BMath, Dip Comp Prog

Office Managers
Lydia Andreas BNS
Dianne Quinn JP

Receptionist
Colleen Fitzgibbons Dip Sec
# Growth at ANZAC Research Institute

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*Full Time Equivalent
ANZAC Health and Medical Research Foundation

**Scientific papers**

**Andrology**


Bianco JJ, Handelsman DJ, Pederson JS, Rijgersberg BR. Direct response of the murine prostate gland and seminal vesicles to estradiol. Endocrinology 143:493-8, 2002


Haywood M, Spaliviero J, Jimenez M, King N, Handelsman DJ, Allan CM. Sertoli and germ cell development in hypogonadotropic hypogonadism: expression of transgenic follicle-stimulating hormone alone or in combination with androgens. Endocrinology 144:509-17, 2002


Spaliviero JA, Jimenez M, Allan CM, Handelsman DJ. LH receptor mediated effects on initiation of spermasthenia in gonadotrophin deficient (hpg) mice are independent of luteinizing hormone (LH) receptor mediated proliferation induced by follicle-stimulating hormone. Journal of the American College of Cardiology 91:1399, 2003

Harris MI and Arias IM. F1C, a Type 1 P-selectin ligand: cholinergic effect on intravascular leukocyte, homologues (ATPβR2 and ATPβR3) expressed throughout the body. Biochemistry & Biophysics Acta-Molecular and Cell Biology of lipids, 1633:127-131, 2002


Muller M. Pycocin induces oxidative stress in human endothelial cells and modulates the glutathione redox cycle. Free Radical Biology and Medicine 33:1527-1533, 2002


Hofbauer LC, Kluger S, Kuhne CA, Dunstan CR, Burchert A, Schoppet M, Zielek A, Heufelder AE. Detection and characterization of RANK ligand and


Neuroendocrinology


Koschel M, Huber P, Staub JJ, Müller B. Prediction of disease progression in women with subclinical hypothyroidism: Beneficial effect of L-Thyroxine replacement therapy. Thyroid 2003; in press.

Neurobiology


Koschel M, Huber P, Staub JJ, Müller B. Prediction of disease progression in women with subclinical hypothyroidism: Beneficial effect of L-Thyroxine replacement therapy. Thyroid 2003; in press.

Neuroendocrinology


Koschel M, Huber P, Staub JJ, Müller B. Prediction of disease progression in women with subclinical hypothyroidism: Beneficial effect of L-Thyroxine replacement therapy. Thyroid 2003; in press.

Neurobiology


Koschel M, Huber P, Staub JJ, Müller B. Prediction of disease progression in women with subclinical hypothyroidism: Beneficial effect of L-Thyroxine replacement therapy. Thyroid 2003; in press.
THE FOUNDATION

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ANZAC Health and Medical Research Foundation

▲ GOVERNANCE

Emeritus Professor John Young AO FAA (Vale)
Professor Young served Chairman of the ANZAC Health and Medical Research Foundation. He was the Pro Vice-Chancellor (Health Sciences) at the University of Sydney. He also served as a Director of the Royal Alexandra Hospital for Children, Deputy Chair of the Central Sydney Area Health Service Board, and Vice President and Secretary (Biological) of the Australian Academy of Science, President, Federation of Asian and Oceanian Physiological Societies, and Member of the National Health and Medical Research Council and the Medical Board of NSW.

Mr Godfrey (Rusty) Priest AM
Mr Rusty Priest served as Deputy Chairman of the ANZAC Health & Medical Research Foundation. He is Immediate Past President of the Returned and Services League of Australia (NSW Branch), Having held office between 1993 and 2002. Rusty enlisted in the 2nd AIF in June 1945, serving in Japan with the British Commonwealth Occupation Forces from April 1946 to December 1948, the Australian Regular Army from 1946 to 1967 and the Emergency Reserve until 1975. Then he took a management position at the University of Sydney, retiring in 1990. He is extensively involved in all matters affecting the welfare of veterans and their dependants. He is the current Chairman, Board of Directors of the Kokoda Track Memorial Walkway Ltd.

Mrs Marie Beach
Mrs Beach joined the War Widows’ Guild of Australia NSW Ltd in July 1995. Mrs Beach was Hon Secretary of Coff’s Harbour War Widows’ Guild Club for four years, a Director of the Guild’s Board for five years and State President for one year. She developed strong working relationships with the ex-service community both in Coff’s Harbour and in Sydney and made an active contribution to the welfare of war widows.

The Hon Ms Kerry Chikarovski MP
Graduating from the University of Sydney with degrees in Economics and Law, majoring in Industrial Relations, Ms Chikarovski was elected to the NSW Parliament in May 1991, as the Member for Lane Cove. Before entering politics, she worked as a solicitor in private practice, specialising in property and commercial law, and lectured at the College of Law. She held the positions of Shadow Minister for Corrective Services and Emergency Services, and the Environment and Women and Public Private Partnerships. Whilst Leader of the Opposition in the NSW 1998-2002 she held portfolio responsibilities of the Arts, Ethnic Affairs, Infrastructure and Major Projects.

Mr Gary J Collins
Mr Collins has a career in public service since 1970 in the Department of Veterans’ Affairs. Where he has worked in all areas, Compensation, Health and Corporate Services and in many Australian locations, Brisbane, Canberra, Adelaide, Perth and Sydney. Gary was Chief Executive Officer of Repatriation General Hospitals in Perth and Brisbane, has been Deputy Commissioner in Perth, Brisbane and Sydney and worked briefly as Division Head Compensation in Canberra. Born in Lismore he grew up in Queensland and Malaysia as the son of a RAAF Air-frame fitter who saw service in both Army and Navy during the Second World War.
Mr Matthew Daly

For the last twenty years Mr Daly has been involved in management in health including teaching hospitals, district hospitals, private hospitals, and community health services. He is Executive Director of two University of Sydney teaching hospitals, Concord Repatriation General Hospital and Canterbury Hospital. At present he is seconded to South Western Area Health Service as Deputy CEO. He holds tertiary qualifications in health administration and business and has been surveyor for the Australian Council of Healthcare Standards and NSW State Branch Councillor with Australian College of Health Service Executives.

Mr Alan Davidson AM MBE

Former test cricketer Mr Davidson has served President NSW Cricket Association and Chairman of the Fresh Food Australia Holdings Pty Ltd. Mr Davidson is a former Australian Test cricketer and member of NSW Sports Advisory Council. He has been Trustee of the Bradman Museum Trust and Director of the Bradman Foundation.

The Hon Mr Tim Fischer

Born at Lockhart in southern NSW and educated at Boree Creek and Melbourne, Tim Fischer is a former Army Officer serving both in Australia and Vietnam. First elected to NSW Parliament in 1970 to represent the Country Party, in 1984 he was elected to Federal Parliament as representative for Farrer for the National Party. He served on many Committees and held several Shadow portfolio positions in Opposition, and was elected Leader of the National Party after the 1990 election. He served as Deputy Prime Minister and Minister for Trade from 1996 until his retirement from politics July 1999.

Professor David Handelsman

Professor Handelsman, has been Director of the ANZAC Research Institute since 1998, He is an international expert in Andrology in which whilst studying for his PhD he established the first Andrology centre in Australia. He has served as adviser to the WHO Human Reproduction Programme and Secretary of the International Society of Andrology. After receiving the Susman Prize from the Royal Australasian College of Physicians in 1994, he took up a visiting Professorship at University of Munster, Germany. On his return a Personal Chair was created for him at the University of Sydney and he became the first Professor in Andrology in Australia.

Dr Diana Horvath AO

Dr Horvath was appointed Chief Executive Officer of the Central Sydney Area Health Service in 1992. She has chaired the National Health and Medical Research Council, been President of the Australian Hospital Association, and served a five-year term as a Commissioner with the Health Insurance Commission. She is an active member of the Trade Policy Advisory Commission. She was recognised for her work in Australian public health when she was made an Officer of Australia in 1995.
Dr Edward Kremer OAM

Dr Kremer is a general practitioner in Bondi with a particular interest in fund raising. He is a member of the Royal Australian Army Medical Corps and consultant to the Director of Health Services NSW. Dr Kremer has been the AMA representative to the Medical Advisory Panel, Department of Veterans’ Affairs, Canberra. He has been a member of the Faculty Board, Royal Australian College of General Practitioners, NSW, and the representative on the NSW Advisory Panel to the Department of Veterans’ Affairs.

Mrs Ethel Lane AM MBE

Mrs Lane joined Concord Hospital in 1942 as an Australian Army nursing sister. She has been actively involved in services for ex-servicemen and women for many years and in 1994 was honoured by the naming of the EM Lane Chair of Surgical Nursing, Faculty of Nursing, University of Sydney.

Dr Charles Pawsey

Dr Pawsey graduated from the University of Adelaide in 1967. He spent three years at Queen Elizabeth Hospital in Adelaide and then at Greenslopes in Brisbane as a National Heart Foundation Research Assistant undertaking research into the Renin-Angiotension system and hypertension. He undertook his physician traineeship at Sydney Hospital in 1972-73 and his Cardiology training at Royal Prince Alfred Hospital in 1974-75 and at Johns Hopkins Hospital in 1976. Since 1977 he has been a Staff Cardiologist at Concord Repatriation General Hospital.

Mr Graham Richardson

Mr Richardson is a former Senator and member of both Hawke and Keating Ministries, where he held several portfolios including Minister for Health. Mr Richardson resigned from federal parliament in 1992 and currently is a political commentator with Channel Nine. He was a member of the Sydney Organising Committee for the Olympic games and Mayor of the Olympic Village.

Ms Kerry Russell

Ms Russell is the Director of Nursing, Concord Repatriation General Hospital. She has an extensive health background and has been employed by CSAHS for a period of many years. From 1996-98 Kerry was seconded to the NSW Health Department, as Associate Director, Nursing Branch. She has an interest in strategic planning, financial management, resource allocation, and recruitment and retention issues. Holding a Bachelor of Administration from the University of New England, she is a member of the NSW College of Nursing and a surveyor with ACHS.
Ms Ann Sanders

Ann Sanders is the senior news anchor for Channel Seven News. For many years she has presented major news events from around the world to Australian viewers. Her presenting roles have included Australia's Most Wanted, 11am, local and federal elections and the Sydney Olympics. Ann is married with two sons and is an active supporter of many charitable organisations.

Dr Margaret Sanger

Dr Margaret Sanger is Director of Medical Services at Concord Repatriation General Hospital. Graduating with honours in Medicine from the University of Sydney she spent five years practising clinical medicine at Concord and Royal Prince Alfred Hospitals. She holds a Master of Health Administration and Fellowship of Royal Australian College of Medical Administration.

Rear Admiral Peter Sinclair AC

Rear Admiral Sinclair born in Sydney 1934 and educated at North Sydney Boys High School and the Royal Australian Naval College. His service in the Royal Australian Navy 1948-1989 included command of HMAS Duchess, HMAS Hobart, HMAS Penguin, Maritime Commander Australia and Commandant Australian Defence Force Academy. He was coordinator Flood Relief Operation Bogan Shire 1990 and Governor NSW 1990-1996. He was Chairman of the Council of the Order of Australia and is Deputy Chair of the Newcastle Permanent Building Society. Holding Honorary Doctorate from the University of Sydney and Southern Cross University and life membership of the RSL, he is Patron of Hunter Medical Research Foundation and Australian Surf Live Saving Foundation. He operates 'Flagship' Poll Hereford Stud near Tea Gardens NSW.

Sir Bruce Williams KBE

Sir Bruce has been a former Vice Chancellor of the University of Sydney, member of the Reserve Bank Board, member of the Finance Committee, University of Sydney, and Chair of the Sydney International Piano Competition of Australia. Sir Bruce was Chair of the Council's Building Subcommittee, overseeing the capital works project for the ANZAC Research Institute.
FINANCIAL PERFORMANCE

Synopsis of Financial performance

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<td>Peer reviewed funding</td>
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<td>Fundraising</td>
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<td><strong>Total Income:</strong></td>
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<tr>
<td>Salary costs</td>
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<td>Depreciation</td>
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<td><strong>Total Expenditure:</strong></td>
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<td><strong>$1,621,240</strong></td>
<td><strong>$1,938,741</strong></td>
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<tr>
<td><strong>Net Profit:</strong></td>
<td><strong>$236,634</strong></td>
<td><strong>$479,366</strong></td>
<td><strong>$1,075,677</strong></td>
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The ANZAC Research Institute continues to attract scientists of renown who are recognised by their peers in the awarding of research grants and invitation to perform clinical trials. The donations income was augmented in 2002-03 by the release of the funds associated with the Northcott Bequest.

Expenditure has risen to reflect the continuing growth in the Institute’s research efforts and the completion of refurbishment of the laboratories with equipment.

Detailed, audited financial statements are available in the pocket in the back cover of this report.
The Foundation's 8th Annual Gala Dinner and Auction was once again held at the Four Season's Hotel in Pitt Street, Sydney. The guest speaker was former Australian weapons' inspector, and now Governor of Tasmania, Richard Butler AM, who generously donated his time. He shared with the audience his first hand knowledge the Iraqi weapons situation and offered many personal insights on the topic. His address was a great success and gave us all much food for thought.

The other speaker for the evening was PhD student Victoria Cogger who spoke about her work in the ageing liver and about what it meant to work at the ANZAC Research Institute.

We are also most grateful for Helen Daly from Channel Nine who was our gracious Master of Ceremonies, whilst John Gatfield from Sky News acted in the role of auctioneer. Other entertainment was provided by a Quartet from St Andrew's Choir School, Four of a Kind, whose rendition of the national anthem was superb. Lily Dior and her jazz ensemble provide excellent musical accompaniment throughout the evening.

The evening was a complete success, attended by 235 guests and raising in excess of $48,000 for the Foundation. We are grateful for the unstinting support of our major sponsor Baxter Healthcare and generous gift of wine from Tintilla Estate and Vineyard. Special thanks are given to Tessy Bananis for her efforts in gaining the support of so many generous sponsors.

Friends of the ANZAC Research Institute (FOTARI) continue to be a constant source of support, as do the many RSL Sub branches and Veterans groups. These generous supporters together with NSW Health grants allow the ANZAC Research Institute the ability to offer excellent facilities to the many researchers who choose to work here.
DONOR HONOUR ROLL

The Council of the ANZAC Health and Medical Research Foundation wishes to express their sincere thanks to the following supporters for their generosity and contribution.

Bequests
Northcott Bequest for Research into Neural Disease
$564,204

Veterans and Community Organisations
$10,000 and over
Council of ExService Women’s Association of NSW
Centre for Vascular Research
Military Health Symposium

$1,000 and over
Second AIF Council
Chesterhill-Carramar Sub Branch
C M T Association of NSW

$500 and over
Ashfield RSL Sub-Branch

$100 and over
Enfield and Croyden RSL Sub Branch
Clovelly RSL Sub Branch
Concord RSL Women’s Bowling Club
North Sydney RSL Sub Branch
Pittwater RSL Sub Branch
Returned Sisters RSL Sub Branch

FOTARI
$1,000 and over
Paul Collett
Michael Hayes
Neville Jeffress
John Linsley
Charles Pawsey
Margaret Sanger
GM Smith
Paul Waizer

$500 and over
Ross Bradbury
Ramon Bullock
Eileen Collins
Gregory Falk
Steven Kalowski
Gary Pearce
Majorie J Pink

$100 and over
Eric Appleton
AXA Australia
JR Belcher
Dr David Breiger
Gary Browne
Francis Burns
John Chalmers
Alan Davidson
Reg Elliott
Christine Evans,
Robert Evans,
LI Ford
Prof Ben Freedman
Peter Frolich

$100 and over continued
Christine Harrison
Edmund Hasche
Margaret Haylen
G Hertzberg & Co Pty Ltd
Edna Hill
Mark Jimenez
Mrs Ethel Lane
Dr Peter Liu
Wallace & Margaret Lowden
Mary Manning
John Murphy MP
Betty Pillans
ML Roberts
PL Souza
SL Tivey
Ronald Walls
WB & MJ White
D Williams
Prof John Young

$50 and over
R Bateup
Rebecca Britt
William E Carter
Danny Chalis
H R Clarke
DJ Dyson
KJ Farrugia
Richard & Lisa Glass
Bryn Jones
Ross L Jones
Desmond Maguire
Brigadier RW Morris
Ron O’Connor
John Rasko
Denis Ryan
KJB Selig
Robert & Susan Sonnenschein
CR Waud
John M White

Corporate Sponsors
$60,000 and over
Baxter Healthcare Pty Ltd

$5,000 and over
Commonwealth Bank of Australia

$1,000 and over
Johnson & Johnson Medical
Becton Dickinson

$500 and over
John Morris Scientific Pty Ltd
Hofagu Pty Limited

$100 and over
DNA Consultants
Spizvac Marketing
Hewlett Packard
## EVENT SPONSORS

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<tr>
<th>Sponsorship for Annual Dinner and Auction</th>
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<td><strong>Major Sponsors</strong></td>
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<td>Tintilla Estate Vineyard and Olive Grove</td>
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<td><strong>Table Sponsors</strong></td>
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<td>Becton Dickinson</td>
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<td>Veritage Press</td>
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<td>Astral Scientific</td>
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<td>Auspep</td>
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<td>Australian Open</td>
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<td>Balloons Aloft</td>
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<td>Becton Dickinson Australia &amp; New Zealand</td>
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<td>Bulmer Australia</td>
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<tr>
<td>Captain Cook Cruises</td>
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<td>Catherine Freeman Enterprises</td>
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<td>Climb Services</td>
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<td>Concord Beauty Spot</td>
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<td>Mr Alan Davidson</td>
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<tr>
<td>Dolci D’oro</td>
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<td>Elegance Limousines</td>
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<td>Faros Seafood</td>
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<td>Fisher Biotec</td>
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<tr>
<td>Flower Trade</td>
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<td>Freight on Board</td>
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<td>Integrated Sciences</td>
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<td>Interpath Services</td>
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<td>Inthanon Thai Restaurant</td>
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<td>Invitrogen</td>
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<td>J &amp; M Promotion Consultants</td>
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<td>Jan Neil Studio</td>
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<td>Johnson &amp; Johnson Medical Pty Ltd</td>
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<tr>
<td>Kennedy &amp; Wilson Chocolates</td>
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<tr>
<td>Kosta Tszyu</td>
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<tr>
<td>Majors Bay Café</td>
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<tr>
<td>Matilda Cruises</td>
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<tr>
<td>Merrylands Floor Covering</td>
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<tr>
<td>Murdoch Magazines</td>
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<td>Nicole Anderson Photography</td>
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<td>O-Sea Travel</td>
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<td>Parramatta Eels</td>
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<tr>
<td>Patrice Newell and Phillip Adams</td>
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<tr>
<td>Phil Farero The Good Guys</td>
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<td>Qantas</td>
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<td>Robert Timms</td>
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</table>

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**Dinner Sponsors continued**

Roses Only
Rothsay Accounting
Saxton Speakers’ Bureau
Shering
Simon’s Framing and Printing
Spanish Terrazas
Sydney Aquatic Centre
Sydney Seafood School
Sydney Symphony
The Provan Group
The Windsor, Melbourne
Total Entity Pty Ltd
Concord Hospital Volunteers
V-Travel
Wadin Valley Estate
Western Suburbs Soccer Sports & Community Club Ltd
Zaffran Restaurant

**Acknowledgements**

Prof Bob Lusby
Robert Lavigne
John Rohannan
Tessy Bananis
Christine Harrison
Staff and Students of ANZAC Research Institute
**BEQUEST - AN OPPORTUNITY**

A bequest to ANZAC Health & Medical Research Foundation is a way of helping future generations to enjoy longer and more fulfilling lives.

**Estate Planning**

More and more supporters of ANZAC Health & Medical Research Foundation are ensuring that our vital research continues by including a bequest in their Will. This can also be done by codicil to an existing Will. There are usually four types of bequests to consider:

**A Specific Bequest**

This specifies the type of gift clearly, whether it is a gift of money, shares, property or life assurance policy.

**A Percentage Bequest**

This is the most flexible method of giving; the gift is automatically determined by the size of the estate and takes inflation into account.

**A Residual Bequest**

This is the amount that remains after the provisions for family and relatives have been made.

**Your Whole Estate**

This usually occurs when there are no living relatives and the Benefactor wishes to achieve something significant with their Estate.

The suggested wording for a bequest to ANZAC Health and Medical Research Foundation is:

"I GIVE to ANZAC Health & Medical Research Foundation, ABN 48 066 780 005, (the whole), or (a specific sum or piece of property), or (a percentage), or (the residue) of my Estate free of all duties and a receipt from the Treasurer or other authorised person shall be a complete and sufficient discharge for the Executor."

**OTHER GIVING OPPORTUNITIES**

- Yes. I would like to help ANZAC Health & Medical Research Foundation.
- I would like to meet a representative from the Foundation to discuss making a bequest.
- I have already remembered ANZAC Health & Medical Research Foundation in my Will.
- Please send me more information about the Foundation’s Bequest Program, including the recognition of benefactors.
- A gift to continue vital scientific research.
- Please send me more information on the FOTARI.

I / We wish to make a donation of $___________ to ANZAC Health and Medical Research Foundation

Name:_________________________________________________________________________

Address:_______________________________________________________________________

Postcode:_____________________________________________________________________

Telephone: H: _________________ W: ___________________

Mobile _____________________________________________

Email: ________________________________________________

Payment is by:

☐ Cheque  ☐ Money Order

☐ Visa  ☐ Bankcard  ☐ Mastercard

___ ___ ___ ___   ___ ___ ___ ___   ___ ___ ___ ___   ___ ___ ___ ___

Expiry:  ____ / ____

Signature: _____________________________________________

Date: __________

For further information contact:

ANZAC Health & Medical Research Foundation

ANZAC Research Institute

Hospital Road CONCORD NSW 2139

Telephone: (02) 9767 9100 or Facsimile: (02) 9767 9101

www.anzac.edu.au

Email: anzac@anzac.edu.au

All gifts over $2.00 are tax deductible
DIRECTORS REPORT TO THE MEMBERS

The directors hereby present their report for the year ended 30 June 2003

Directors

The names and details of directors in office at the date of this report are:

Emeritus Professor John A Young AO BSc(Qld) MB BS(Qld) MD(Qld) DSc(Qld) FAA FRACP MMedSci
Chair
Emeritus Professor and Honorary Professor of Physiology, University of Sydney
Deputy Chair, Central Sydney Area Health Service
Director, Royal Alexandra Hospital for Children
Director, Arthur T George Foundation
Trustee, Sydney Foundation of Medical Research
Director, CRC Asthma
Member, National Health and Medical Research Council
Member, Medical Board of NSW
President, Federation of Asian and Oceanian Physiological Societies

Mrs Felicity Barr BA(LaTrobe) FAICD FAAG
Deputy Chair
Chairman, War Widows’ Guild of Australia (NSW) Ltd
Honorary Governor, Ageing and Alzheimer’s Research Foundation
Vice President, Australian Association of Gerontology (NSW)
Member, National Institute for Governance

Mr Godfrey E Priest AM
Director
Chairman, Kokoda Track Memorial Walkway
Chairman, Lady Davidson Private Hospital Advisory Committee
Executive Member, Australian Red Cross (NSW Division)
Community Member, Graduate Medical Program, University of Sydney
Member, NSW Health Participation Council

Mr Brian M Lee
Director
Area Managing Director (Australia & New Zealand), Baxter Healthcare Pty Ltd
National President, Leukaemia Foundation of Australia
National President, Medical Industries Association of Australia
Member, Parramatta Development Board

Mr Matthew Daly ADipHA BBus AFACHSE
Director
Executive Director, Concord Repatriation General Hospital and Canterbury Hospital

Professor Andrew J S Coates MA DM(Oxon) MB BChir(Cantab) FRACP FRCP FESC FAACC FAHA MBA(London)
Dean, Faculty of Medicine, University of Sydney

Professor David Cook MB BS(Syd) MSc MD(Syd) FRACP
Director
Professor, Department of Physiology, University of Sydney

Professor David J Handelsman MB BS(Melb) FRACP PhD(Syd)
Director
Director, ANZAC Health & Medical Research Foundation
Director, ANZAC Research Institute
Sub-Dean Research (Concord), University of Sydney
Director, Department of Andrology, Concord Repatriation General Hospital
Dr Diana G Horvath  AO MB BS(Syd) MHP(NSW) FRACMA  FAFPHM  FCHSE  
Director  
Chief Executive Officer, Central Sydney Area Health Service  
Board Member, Institute of Clinical Excellence  
Board Member, Centenary Institute for Cancer Medicine and Cell Biology  
Board Member, Institute for International Health  
Board Member, Sydney Cancer Institute  

Mr Paul McClintock  BA LLB(Syd)  
Director  
CEO McClintock Associates Pty Ltd  
Director, MacQuaire Infrastructure Investment Management Ltd  
Director, Thales Group  
Director, Australian Strategic Policy Institute  
Member, Advisory Board of Profile Ray and Berndtson  

Dr Charles G Pawsey  MB BS(Adel) FRACP DDU  
Director  
Chair, Concord Repatriation General Hospital Medical Staff Council  
Medical Practitioner  

At this date no director has any interest in the equity of the company.  

Principle Activities  
The principal activities of the ANZAC Health and Medical Research Foundation during the year were that of acting as trustee for the ANZAC Health and Medical Research Foundation Trust Fund. There was no significant change in the nature of that activity during the year.  

Operating Results  
The company did not trade in its own right, and made neither a profit nor a loss.  

Significant Changes In State Of Affairs  
There were no significant changes in the state of affairs of the ANZAC Health and Medical Research Foundation during the year.  

Matters Subsequent To The End Of The Financial Period  
No matter or circumstances have arisen since the end of the financial period which significantly affected or may significantly affect the operations of the ANZAC Health & Medical Research Foundation, the results of those operations, or the state of affairs of the ANZAC Health & Medical Research Foundation in subsequent years.  

Likely Developments  
The company will continue to act as trustee of the ANZAC Health and Medical Research Foundation Trust Fund.  

Review Of Operations  
The combined effect of all fund raising campaigns conducted by the trust throughout the financial year has raised $177,045 in cash ($222,643 for 2001/2002) and $1,000 in pledges ($42,500 for 2001/2002).
Meetings Of Directors
During the financial period, meetings of directors, including committees, were held. Attendances are tabulated below:

### Council Meetings

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<th>Number Attended</th>
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<td>Mr. G. Priest AM (Deputy Chair)</td>
<td>2</td>
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<td>Mrs. K. Chikarovski MP</td>
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<td>Mr. G. Collins</td>
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<td>0</td>
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<tr>
<td>Mr. M. Daly</td>
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<tr>
<td>Mr. A. Davidson AM MBE</td>
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<td>The Hon. T. Fischer MP</td>
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<td>Professor D. Handelsman</td>
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<td>Mrs. E. Lane AM MBE</td>
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<td>Mrs. M. Beach</td>
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<td>0</td>
</tr>
<tr>
<td>Dr. C. Pawsey</td>
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<td>2</td>
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<tr>
<td>Mr. G. Richardson</td>
<td>2</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Ms. K. Russell</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Ms. A. Sanders</td>
<td>2</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Dr. M. Sanger</td>
<td>2</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Rear Admiral Peter Sinclair AC</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Sir Bruce Williams KBE</td>
<td>2</td>
<td>2</td>
<td>0</td>
</tr>
</tbody>
</table>

### Executive Board Meetings

<table>
<thead>
<tr>
<th></th>
<th>Eligible to Attend</th>
<th>Number Attended</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emeritus Professor J. Young AO (Chair)</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Mr G. Priest AM (Deputy Chair)</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Mr G. Collins</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Mr M. Daly</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Mr A. Davidson AM MBE</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Mr G Hartigan</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Professor D. Handelsman</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Dr D. Horvath AO</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Mrs E. Lane AM MBE</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Dr C. Pawsey</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Ms K. Russell</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Dr M. Sanger</td>
<td>3</td>
<td>3</td>
</tr>
</tbody>
</table>
Directors’ Indemnification
The ANZAC Health and Medical Research Foundation during or since the financial year, in respect of any person who is or has been an officer, has not been
- indemnified or made any relevant agreement for indemnifying against a liability incurred as an officer, including costs or expenses in successfully defending legal proceedings; or
- 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.

The NSW Treasury Managed Fund extends appropriate insurance to all directors appointed by the Central Sydney Area Health Service in accordance with Article of Association 29 [a][b][c]. The NSW Treasury Managed Fund insures all 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.

Directors’ Benefits
Neither since the financial year nor during the financial year has a director received or become entitled to receive a benefit by reason of a contract made by the company, an entity controlled by the Company, or a body corporate that was related to the company when the contract was made or when the director received or became entitled to receive the benefit with the director or with a firm of which the director is a member, or an entity in which the director has a substantial financial interest.

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

Felicity Barr
Deputy Chair
Diana Horvath
Director

Dated this tenth day of December 2003 at Sydney.
INDEPENDENT AUDIT REPORT

ANZAC Health and Medical Research Foundation

To Members of the New South Wales Parliament and Members of the ANZAC Health and Medical Research Foundation

Audit Opinion

In my opinion, the financial report of ANZAC Health and Medical Research Foundation is in accordance with:

(a) the Corporations Act 2001, including:
   (i) giving a true and fair view of the ANZAC Health and Medical Research Foundation’s financial position at 30 June 2003 and financial performance for the year ended on that date, and
   (ii) complying with Accounting Standards in Australia, and the Corporations Regulations 2001,

(b) other mandatory financial reporting requirements in Australia, and

(c) section 418 of the Public Finance and Audit Act 1983.

My opinion should be read in conjunction with the rest of this report.

The Directors’ Role

The financial report is the responsibility of the company’s directors. It consists of the statement of financial position, the statement of financial performance, the statement of cash flows and the accompanying notes, and directors’ declaration.

The Auditor’s Role and the Audit Scope

As required by the Public Finance and Audit Act 1983 and the Corporations Act 2001, I carried out an independent audit to enable me to express an opinion on the financial report. My audit provides reasonable assurance to Members of the New South Wales Parliament and the members of the ANZAC Health and Medical Research Foundation that the financial report is free of material misstatement.

My audit accorded with Australian Auditing and Assurance Standards and statutory requirements, and I:

• evaluated the accounting policies and significant accounting estimates used by the directors in preparing the financial report, and
• examined a sample of the evidence that supports the amounts and other disclosures in the financial report.

An audit does not guarantee that every amount and disclosure in the financial report is error free. The terms ‘reasonable assurance’ and ‘material’ recognise that an audit does not examine all evidence and transactions. However, the audit procedures used should identify errors or omissions significant enough to adversely affect decisions made by users of the financial report or indicate that the company’s directors had not fulfilled their reporting obligations.
My opinion does not provide assurance:

- about the future viability of the Foundation,
- that it has carried out its activities effectively, efficiently and economically, or
- about the effectiveness of its internal controls.

Audit Independence

The Audit Office complies with all applicable independence requirements of Australian professional ethical pronouncements and the Corporations Act 2001. The Public Finance and Audit Act 1983 further promotes independence by:

- providing that only Parliament, and not the executive government, can remove an Auditor-General, and
- mandating the Auditor-General as auditor of public sector agencies but precluding the provision of non-audit services, thus ensuring the Auditor-General and the Audit Office are not compromised in their role by the possibility of losing clients or income.

R J Sendt
Auditor-General

SYDNEY
18 December 2003
### STATEMENT OF FINANCIAL PERFORMANCE
**FOR THE YEAR ENDED 30 JUNE 2003**

<table>
<thead>
<tr>
<th>Note</th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$</td>
<td>$</td>
</tr>
<tr>
<td>Expenses from ordinary activities</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Revenue from ordinary activities</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Profit from ordinary activities before income tax expense</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Income tax expense relating to ordinary activities</td>
<td>1(C)</td>
<td>0</td>
</tr>
<tr>
<td>Profit from ordinary activities after related Income tax expense</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total changes in equity other than those resulting from transactions with owners as owners</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

The accompanying notes form an integral part of this statement of financial performance.

### STATEMENT OF FINANCIAL POSITION
**AT 30 JUNE 2003**

<table>
<thead>
<tr>
<th>Note</th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$</td>
<td>$</td>
</tr>
<tr>
<td>Total Assets</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total Liabilities</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Net Assets</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Equity</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

The accompanying notes form an integral part of this statement of financial position.

### STATEMENT OF CASH FLOWS
**FOR THE YEAR ENDED 30 JUNE 2003**

<table>
<thead>
<tr>
<th>Note</th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$</td>
<td>$</td>
</tr>
<tr>
<td>CASH FLOWS FROM OPERATING ACTIVITIES</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Net operating cash flows</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>CASH FLOWS FROM INVESTING ACTIVITIES</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Net investing cash flows</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>CASH FLOWS FROM FINANCING ACTIVITIES</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Net financing cash flows</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Net increase (decrease) in cash held</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Cash at the beginning of financial year</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Cash at the end of the financial year</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

The accompanying notes form an integral part of this statement of cash flows.
NOTES TO THE FINANCIAL STATEMENTS

NOTE 1: Statement of Significant Accounting Policies

(A) Basis of Accounting
The financial statements have been prepared as a general purpose financial report that complies with the requirements of the Corporations Act, Australian Accounting Standards, Urgent Issues Group Consensus Views and with the requirements of the Public Finance and Audit Act 1983 and the Health Services Act 1999 and its regulations. The accounting policies used are consistent with those adopted in the previous year. The financial statements have also been prepared in accordance with the historical cost convention and do not take account of changes in the general purchasing power of the dollar.

(B) Accounting Records
As required by Section 45C of the Public Finance and Audit Act 1983, the Foundation has kept proper accounts and records in relation to all its activities.

(C) Income Tax
The Foundation is exempt from income tax under Section 23(j) of the Income Tax Assessment Act.

(D) Trustee
The company acts as trustee for the ANZAC Health and Medical Research Foundation Trust Fund. The accounting policies adopted by the company in the preparation of the financial statements for the year ended 30 June 2003 reflect the fiduciary nature of the company's responsibility for the assets and liabilities of the ANZAC Health and Medical Research Foundation Trust Fund which are set out in note 4.

NOTE 2: Profit From Ordinary Activities
The company did not trade during the past financial year.

NOTE 3: Reconciliation Of Net Cash Used In Operating Activities To Operation Result

<table>
<thead>
<tr>
<th></th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td>Net profit</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Net cash</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>
## NOTE 4: Assets And Liabilities Of The Foundation

The company acts as trustee for the ANZAC Health and Medical Research Foundation Trust Fund. The assets and liabilities of the ANZAC Health and Medical Research Foundation Trust Fund as disclosed in the financial statements of the Trust are as follows:

<table>
<thead>
<tr>
<th></th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td>Current Assets</td>
<td>4,106,858</td>
<td>3,163,723</td>
</tr>
<tr>
<td>Non-Current Assets</td>
<td>5,899,985</td>
<td>5,622,854</td>
</tr>
<tr>
<td>TOTAL ASSETS</td>
<td>10,006,843</td>
<td>8,786,577</td>
</tr>
<tr>
<td>Current liabilities</td>
<td>128,194</td>
<td>232,637</td>
</tr>
<tr>
<td>Non-Current liabilities</td>
<td>4,392</td>
<td>0</td>
</tr>
<tr>
<td>TOTAL LIABILITIES</td>
<td>132,586</td>
<td>232,637</td>
</tr>
<tr>
<td>NET ASSETS</td>
<td>9,874,257</td>
<td>8,553,940</td>
</tr>
<tr>
<td>Represented by</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TRUST FUNDS</td>
<td>9,874,257</td>
<td>8,553,940</td>
</tr>
</tbody>
</table>

## NOTE 5: Related Party Disclosures And Remuneration And Retirement Benefits

No directors received, directly or indirectly income paid, payable, or otherwise made available from ANZAC Health and Medical Research Foundation during this financial year.

No amount was paid, on behalf of a director, to a superannuation plan.

The names of Foundation’s directors who have held office during the financial year are:

- Mrs M. Beach [Resigned Aug 2003]
- Ms K. A Chikarovski MP [Resigned Oct 2003]
- Mr Gary Collins [Resigned Oct 2003]
- Mr M. Daly [Resigned Oct 2003]
- Mr A. K Davidson AM MB [Resigned Oct 2003]
- Mr T. A. Fischer [Resigned Oct 2003]
- Mr G. J. Hartigan [Resigned Oct 2002]
- Prof D. J. Handelsman [Resigned Oct 2003]
- Dr D. G. Horvath AO [Resigned Oct 2003]
- Dr E. P. Kremer OAM [Resigned Oct 2003]
- Mrs E. M. Lane AM MBE [Resigned Oct 2003]
- R/Adm Peter Sinclair AC [Resigned Oct 2003]
- Dr C. G. Pawsey [Resigned May 2003]
- Mr G. E. Priest AM [Resigned Oct 2003]
- Mr G. Richardson [Resigned Oct 2003]
- Ms K. J. Russell [Resigned Oct 2003]
- Ms A. E. Sanders [Resigned Oct 2003]
- Dr M. M. Sanger [Resigned Oct 2003]
- Emeritus Prof J. A. Young AO [Resigned Oct 2003]
- Sir Bruce Williams KBE [Resigned May 2003]
NOTE 6: Guarantee
The Foundation is limited by guarantee. In the event that the Foundation is wound up, the Memorandum of
Association states that each director is required to contribute $20.00 each towards meeting outstanding obligations
of the Foundation.

NOTE 7: Segment Level Reporting
As the trustee for the ANZAC Health and Medical Research Foundation Trust Fund, the principal activity of the company
during the financial period was to promote and facilitate healthcare delivery and research on illness and disease associated
with lifestyle and ageing. The financial results of the company fully reflect the conduct of this activity.
The geographical location of the company is Australia.

Director's Declaration
The Directors declare that:
1) the financial statements and associated notes comply with the accounting standards and Urgent Issues Group
Consensus Views;

2) the financial statements and notes give a true and fair view of the financial position as at 30 June 2003 and
performance of the company for the year then ended;

3) in the directors’ opinion;
   i) there are reasonable grounds to believe that the company will be able to pay its debts as when they
become due and payable
   ii) the financial statements and notes are in accordance with the Corporations Act, including sections
296 and 297.

Made in accordance with a resolution of the directors.

Felicity Barr
Deputy Chair

Diana Horvath
Director

Dated this tenth day of December 2003 at Sydney.
TRUST FUND

FINANCIAL STATEMENTS AS AT 30 JUNE 2003
INDEPENDENT AUDIT REPORT

ANZAC Health and Medical Research Foundation Trust Fund

To Members of the New South Wales Parliament

Audit Opinion Pursuant to the Public Finance and Audit Act 1983

In my opinion, the financial report of the ANZAC Health and Medical Research Foundation Trust Fund:

(a) presents fairly the ANZAC Health and Medical Research Foundation Trust Fund’s financial position as at 30 June 2003 and its financial performance and cash flows for the year ended on that date, in accordance with applicable Accounting Standards and other mandatory professional reporting requirements in Australia, and

(b) complies with section 41B of the Public Finance and Audit Act 1983 (the PF&A Act).

Audit Opinion Pursuant to the Charitable Fundraising Act 1991

In my opinion:

(a) the accounts of the ANZAC Health and Medical Research Foundation Trust Fund show a true and fair view of the financial result of fundraising appeals for the year ended 30 June 2003

(b) the accounts and associated records of the ANZAC Health and Medical Research Foundation Trust Fund have been properly kept during the year in accordance with the Charitable Fundraising Act 1991 (the CF Act) and the Charitable Fundraising Regulation 1998 (the CF Regulation)

(c) money received as a result of fundraising appeals conducted during the year has been properly accounted for and applied in accordance with the CF Act and the CF Regulation, and

(d) there are reasonable grounds to believe that the ANZAC Health and Medical Research Foundation Trust Fund will be able to pay its debts as and when they fall due.

My opinions should be read in conjunction with the rest of this report.

The Board’s Role

The financial report is the responsibility of the Members of the Board of the ANZAC Health and Medical Research Foundation Trust Fund. It consists of the statement of financial position, the statement of financial performance, the statement of cash flows and the accompanying notes.

The Auditor’s Role and the Audit Scope

As required by the PF&A Act and the CF Act, I carried out an independent audit to enable me to express an opinion on the financial report. My audit provides reasonable assurance to Members of the New South Wales Parliament that the financial report is free of material misstatement.

My audit accorded with Australian Auditing and Assurance Standards and statutory requirements, and I:

- evaluated the accounting policies and significant accounting estimates used by the Board in preparing the financial report,
- examined a sample of the evidence that supports:
(i) the amounts and other disclosures in the financial report,
(ii) compliance with accounting and associated record keeping requirements pursuant to the CF Act, and

- obtained an understanding of the internal control structure for fundraising appeal activities.

An audit does not guarantee that every amount and disclosure in the financial report is error free. The terms 'reasonable assurance' and 'material' recognise that an audit does not examine all evidence and transactions. However, the audit procedures used should identify errors or omissions significant enough to adversely affect decisions made by users of the financial report or indicate that Board had not fulfilled their reporting obligations.

My opinions do not provide assurance:
- about the future viability of the ANZAC Health and Medical Research Foundation Trust Fund,
- that it has carried out its activities effectively, efficiently and economically, or
- about the effectiveness of its internal controls.

Audit Independence

The Audit Office complies with all applicable independence requirements of Australian professional ethical pronouncements. The PF&A Act further promotes independence by:

- providing that only Parliament, and not the executive government, can remove an Auditor-General, and
- mandating the Auditor-General as auditor of public sector agencies but precluding the provision of non-audit services, thus ensuring the Auditor-General and the Audit Office are not compromised in their role by the possibility of losing clients or income.

R J Sendt
Auditor-General

SYDNEY
18 December 2003
### STATEMENT OF FINANCIAL PERFORMANCE

**FOR THE YEAR ENDED 30 JUNE 2003**

<table>
<thead>
<tr>
<th>Note</th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$</td>
<td>$</td>
</tr>
<tr>
<td>Revenue from ordinary activities</td>
<td>2(b)</td>
<td>3,013,892</td>
</tr>
<tr>
<td>Expenses from ordinary activities</td>
<td>2(a)</td>
<td>1,938,741</td>
</tr>
<tr>
<td>Profit from ordinary activities before income tax expense</td>
<td>2</td>
<td>1,075,151</td>
</tr>
<tr>
<td>Income tax expense relating to ordinary activities</td>
<td>1(D)</td>
<td>0</td>
</tr>
<tr>
<td>Profit from ordinary activities after related income tax expense</td>
<td></td>
<td>1,075,151</td>
</tr>
<tr>
<td>Total changes in equity attributable to beneficiaries of the trust</td>
<td></td>
<td>1,075,151</td>
</tr>
</tbody>
</table>

The accompanying notes form an integral part of this statement of financial performance.

### STATEMENT OF FINANCIAL POSITION

**AT 30 JUNE 2003**

<table>
<thead>
<tr>
<th>Note</th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$</td>
<td>$</td>
</tr>
<tr>
<td><strong>CURRENT ASSETS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cash assets</td>
<td>4</td>
<td>271,362</td>
</tr>
<tr>
<td>Other financial assets</td>
<td>5</td>
<td>3,706,762</td>
</tr>
<tr>
<td>Receivables</td>
<td>6</td>
<td>89,123</td>
</tr>
<tr>
<td>Other</td>
<td></td>
<td>39,611</td>
</tr>
<tr>
<td><strong>TOTAL CURRENT ASSETS</strong></td>
<td></td>
<td>4,106,858</td>
</tr>
<tr>
<td><strong>NON-CURRENT ASSETS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Property, plant and equipment</td>
<td>7</td>
<td>5,899,985</td>
</tr>
<tr>
<td><strong>TOTAL NON-CURRENT ASSETS</strong></td>
<td></td>
<td>5,899,985</td>
</tr>
<tr>
<td><strong>TOTAL ASSETS</strong></td>
<td></td>
<td>10,006,843</td>
</tr>
<tr>
<td><strong>CURRENT LIABILITIES</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Payables</td>
<td>8</td>
<td>72,861</td>
</tr>
<tr>
<td>Provisions</td>
<td>9</td>
<td>55,333</td>
</tr>
<tr>
<td><strong>TOTAL CURRENT LIABILITIES</strong></td>
<td></td>
<td>128,194</td>
</tr>
<tr>
<td><strong>NON-CURRENT LIABILITIES</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Provisions</td>
<td>9</td>
<td>4,392</td>
</tr>
<tr>
<td><strong>TOTAL NON-CURRENT LIABILITIES</strong></td>
<td></td>
<td>4,392</td>
</tr>
<tr>
<td><strong>TOTAL LIABILITIES</strong></td>
<td></td>
<td>132,586</td>
</tr>
<tr>
<td><strong>NET ASSETS</strong></td>
<td></td>
<td>9,874,257</td>
</tr>
<tr>
<td><strong>TRUST FUNDS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Settlement Capital</td>
<td>12</td>
<td>100</td>
</tr>
<tr>
<td>Retained Profits</td>
<td>11</td>
<td>9,628,991</td>
</tr>
<tr>
<td>Reserves</td>
<td>13</td>
<td>245,166</td>
</tr>
<tr>
<td><strong>TOTAL TRUST FUNDS</strong></td>
<td></td>
<td>9,874,257</td>
</tr>
</tbody>
</table>

The accompanying notes form an integral part of this statement of financial position.
**STATEMENT OF CASH FLOWS**  
FOR THE YEAR ENDED 30 JUNE 2003

<table>
<thead>
<tr>
<th>Note</th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$</td>
<td>$</td>
</tr>
<tr>
<td><strong>CASH FLOWS FROM INVESTING ACTIVITIES</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Receipts from grants - State Government</td>
<td>492,400</td>
<td>442,400</td>
</tr>
<tr>
<td>Receipts from grants - Commonwealth</td>
<td>45,000</td>
<td>80,000</td>
</tr>
<tr>
<td>Receipts from donations</td>
<td>795,828</td>
<td>205,083</td>
</tr>
<tr>
<td>Receipts from peer Reviewed Funding</td>
<td>1,558,589</td>
<td>644,960</td>
</tr>
<tr>
<td>Payments to suppliers and employees</td>
<td>(1,771,374)</td>
<td>(1,285,222)</td>
</tr>
<tr>
<td>Interest received</td>
<td>164,179</td>
<td>97,136</td>
</tr>
<tr>
<td>Other receipts</td>
<td>123,789</td>
<td>427,335</td>
</tr>
<tr>
<td>Net cash provided by operating activities</td>
<td>3</td>
<td>1,408,411</td>
</tr>
</tbody>
</table>

| **CASH FLOWS FROM INVESTING ACTIVITIES** |       |       |
| Payment for property, plant and equipment | (323,090) | (104,112) |
| Net investing cash flows | (323,090) | (104,112) |
| Net increase (decrease) in cash held | 1,085,321 | 507,580 |
| Cash at the beginning of the financial year | 2,892,803 | 2,385,223 |
| Cash at the end of the financial year | 3 | 3,978,124 | 2,892,803 |

The accompanying notes from an integral part of this statement of cash flows.
NOTES TO THE FINANCIAL STATEMENTS

NOTE 1: Statement O Significant Accounting Policies

(A) Basis Of Accounting
The financial statements have been prepared as a general purpose financial report that complies with the requirements of the Corporations Act, Australian Accounting Standards, Urgent Issues Group Consensus Views and with the requirements of the Public Finance and Audit Act 1983 and the Health Services Act 1999 and its regulations. The accounting policies used are consistent with those adopted in the previous year. The financial statements have also been prepared in accordance with the historical cost convention and do not take account of changes in the general purchasing power of the dollar.

(B) Accounting Records
As required by Section 45C of the Public Finance and Audit Act 1983, the Trust Fund has kept proper accounts and records in relation to all its activities.

(C) Depreciation
Depreciation is provided for on a straightline basis against all depreciable assets so as to write off the depreciable amount of each depreciable asset as it is consumed over its useful life to the ANZAC Health and Medical Research Foundation, land is not considered a depreciable asset.

Property, plant and equipment have been depreciated from not later than the month following acquisition or operation. Depreciation rates on individual assets are reviewed annually.

Detail of depreciation rates and useful lives for major asset categories, according to NSW Health Accountancy Manual rates, are as follows:

<table>
<thead>
<tr>
<th>Depreciation Rates</th>
<th>Rate (%)</th>
<th>Life (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leasehold Buildings</td>
<td>4.0</td>
<td>25</td>
</tr>
<tr>
<td>Electro Medical Equipment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>■ Costing less than $200,000</td>
<td>10.0</td>
<td>10</td>
</tr>
<tr>
<td>■ Costing more than $200,000</td>
<td>12.5</td>
<td>8</td>
</tr>
<tr>
<td>Computer Equipment</td>
<td>20.0</td>
<td>5</td>
</tr>
<tr>
<td>Computer Software</td>
<td>20.0</td>
<td>5</td>
</tr>
<tr>
<td>Office Equipment</td>
<td>10.0 - 12.5</td>
<td>8 - 10</td>
</tr>
<tr>
<td>Plant and Machinery</td>
<td>10.0</td>
<td>10</td>
</tr>
<tr>
<td>Furniture, Fittings and Furnishings</td>
<td>10.0</td>
<td>10</td>
</tr>
</tbody>
</table>

(D) Income Tax
The Trust Fund is exempt from income tax under Section 23(j) of the Income Tax Assessment Act.

(E) Property, Plant And Equipment
Property, plant and equipment are brought to account at cost, less, where applicable, any accumulated depreciation.

The depreciable amount of all fixed assets is depreciated over their useful lives commencing from the time the asset is held ready for use.

In the directors’ opinion the written down value is equal to fair value.

Costs incurred in relation to the construction of the ANZAC Health & Medical Research Institute have been capitalised as leasehold buildings and are carried at cost.

The leasehold buildings have been built on land leased from Central Sydney Area Health Service. The lease for the land and buildings is for 25 years, and the New South Wales’ Minister of Health may terminate this lease at any time on reasonable notice.
(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, net of bank overdrafts.

(G) Settlement
The ANZAC Health and Medical Research Foundation Trust Fund was made between Robert Edward McKeown, the settlor, and the ANZAC Health and Medical Research Foundation, the trustee, on 21 February 1995.
The Trust has carried out all trading activities in accordance with the provisions of the trust deed.

(H) Financial Instruments
Financial instruments give rise to positions that are a financial asset of either ANZAC Health & Medical Research Foundation or its counterparty and a financial liability (or equity instrument) of the other party. For ANZAC Health and Medical Research Foundation these include cash at bank, receivables, investments and creditors.
In accordance with Australian Accounting Standard AAS33, "Presentation and Disclosure of Financial Instruments", information is disclosed in Note 16 in respect of the interest rate risk and credit risk of financial instruments. All such amounts are carried in the accounts at net fair value. The specific accounting policy in respect of each class of such financial instrument is stated hereunder.
Classes of instruments recorded at cost and their terms and conditions at balance date are as follows:

(i) Cash
Accounting Policies – Cash is carried at nominal values reconcilable to monies on hand and independent bank statements.
Terms and Conditions – Monies on deposit at balance date attract an effective interest rate of 1.09% and 1.09% (0.01% and 3.65% for 2001/2002) & 1.09% and 3.20% (0.00% and 3.45% for 2001/2002).

(ii) Receivables
Accounting Policies – Receivables are carried at nominal amounts due less any provision for doubtful debts. A provision of doubtful debts is recognised when collection of the full nominal amount is no longer probable.
Terms and Conditions – Accounts are issued on 30 day terms.

(iii) Investments
Accounting Policies – Investments reported at cost include both short term and fixed term deposits. Interest is recognised in the Operating Statement when earned.
Terms and Conditions - Fixed term deposits have an average maturity of 32 days (33 days for 2001/2002) and effective interest rates of 4.53% to 4.70% 2002/2003 (3.75% to 4.84% for 2001/2002).

(iv) Creditors
Accounting Policies – Creditors are recognised for amounts to be paid in the future of goods and services received, whether or not billed to the Foundation.
Terms and Conditions – Trade liabilities are settled within any terms specified. If no terms are specified, payments are made by the end of the month following the month in which the invoice is received.
There are no classes of instruments that are recorded at other than cost or market valuation. All financial instruments including revenue, expenses and other cash flows arising from instruments are recognised on an accrual basis.
(I) Research And Development Costs
Research and development costs are charged to expense in the year in which they are incurred.

(J) Provision For Employee Entitlements
Provision has been made in the financial statements for benefits accruing to employees in relation to annual leave, long service leave, workers’ compensation and vested sick leave. No provision is made for non-vesting sick leave, as the anticipated pattern of future sick leave taken indicated that accumulated non-vesting leave will never be paid.

All on-costs, including payroll tax, workers’ compensation premiums and fringe benefits tax are included in the current portion of long service leave and workers’ compensation provision are measured at their nominal amounts.

(K) Revenue Recognition
Revenue arising from the sale of goods, the provision of services and the use of the Trust’s assets is recognised when:

a) the Trust has passed control of the goods or other assets to the buyer;
b) the Trust controls a right to be compensated for services rendered;
c) the Trust controls a right relating to the consideration payable for the provision of investment assets;
d) it is probable that the economic benefits comprising the consideration will flow to the entity;
e) the amount of the revenue can be measured reliably.

(L) Goods And Services Tax (GST)
Revenues, expenses and assets are recognised net of the amount of GST, except:

- the amount of GST incurred by the agency as a purchaser that is not recoverable from the Australian Tax Office is recognised as part of the cost of acquisition of an asset or as part of an item of expense;
- receivables and payables are stated with the amount of GST included.
NOTE 2: Profit from ordinary activities

(a) Profit from ordinary activities is after charging the following expenses:

<table>
<thead>
<tr>
<th>Category</th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td>Salary and Wages</td>
<td>712,413</td>
<td>702,051</td>
</tr>
<tr>
<td>Superannuation</td>
<td>(37,905)</td>
<td>23,828</td>
</tr>
<tr>
<td>Consumables</td>
<td>364,263</td>
<td>141,796</td>
</tr>
<tr>
<td><strong>Administrative</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conference, Training &amp; Travel</td>
<td>82,356</td>
<td>64,565</td>
</tr>
<tr>
<td>Advertising</td>
<td>5,437</td>
<td>9,203</td>
</tr>
<tr>
<td>Functions</td>
<td>46,455</td>
<td>34,270</td>
</tr>
<tr>
<td>Consultants</td>
<td>3,000</td>
<td>3,558</td>
</tr>
<tr>
<td>Accounting &amp; Legal Fees</td>
<td>16,314</td>
<td>3,762</td>
</tr>
<tr>
<td>Audit Fees</td>
<td>0</td>
<td>5,000</td>
</tr>
<tr>
<td>Books &amp; Reference Material</td>
<td>16,586</td>
<td>11,725</td>
</tr>
<tr>
<td>Stationery &amp; Office Supplies</td>
<td>30,897</td>
<td>39,334</td>
</tr>
<tr>
<td>Freight &amp; Courier</td>
<td>7,929</td>
<td>17,297</td>
</tr>
<tr>
<td><strong>Miscellaneous Admin Expenses</strong></td>
<td>281,435</td>
<td>170,692</td>
</tr>
<tr>
<td><strong>Total Administrative</strong></td>
<td>490,409</td>
<td>359,406</td>
</tr>
<tr>
<td>Depreciation</td>
<td>291,125</td>
<td>258,281</td>
</tr>
<tr>
<td><strong>Repairs, Maintenance &amp; Renewals</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Equipment</td>
<td>91,276</td>
<td>133,489</td>
</tr>
<tr>
<td>Other</td>
<td>27,160</td>
<td>34,067</td>
</tr>
<tr>
<td><strong>Total Repairs Maintenance &amp; Renewals</strong></td>
<td>118,436</td>
<td>167,556</td>
</tr>
<tr>
<td>Loss on Disposal of Fixed Assets</td>
<td>0</td>
<td>11,020</td>
</tr>
<tr>
<td><strong>Total Expenditure</strong></td>
<td>1,938.741</td>
<td>1,663,938</td>
</tr>
</tbody>
</table>

(b) Profit form ordinary activities after crediting the following revenues:

<table>
<thead>
<tr>
<th>Category</th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td>Donations – Corporate</td>
<td>37,900</td>
<td>95,551</td>
</tr>
<tr>
<td>Donations – RSL</td>
<td>23,858</td>
<td>16,742</td>
</tr>
<tr>
<td>Donations – Other</td>
<td>614,403</td>
<td>82,985</td>
</tr>
<tr>
<td>Fundraising Activities</td>
<td>94,427</td>
<td>64,842</td>
</tr>
<tr>
<td>Research Grants - State Government</td>
<td>492,400</td>
<td>442,400</td>
</tr>
<tr>
<td>Research Grants - Commonwealth</td>
<td>0</td>
<td>125,000</td>
</tr>
<tr>
<td>Interest Income</td>
<td>157,709</td>
<td>110,072</td>
</tr>
<tr>
<td>Peer Reviewed Funding</td>
<td>1,508,389</td>
<td>745,933</td>
</tr>
<tr>
<td>Other Revenue</td>
<td>84,806</td>
<td>417,081</td>
</tr>
<tr>
<td><strong>Total Revenue</strong></td>
<td>3,013,892</td>
<td>2,100,606</td>
</tr>
</tbody>
</table>
NOTE 3: Notes To The Statement Of Cash Flows

(a) Reconciliation of net profit after income tax to net cash provided by operating activities

<table>
<thead>
<tr>
<th></th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td>Net profit after income tax</td>
<td>1,075,151</td>
<td>436,663</td>
</tr>
<tr>
<td>Adjustments for non-cash income and expense items</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Loss on Disposal of plant and equipment</td>
<td>0</td>
<td>11,020</td>
</tr>
<tr>
<td>Depreciation</td>
<td>291,125</td>
<td>258,281</td>
</tr>
<tr>
<td>Provision for employee entitlement</td>
<td>22,955</td>
<td>14,808</td>
</tr>
<tr>
<td>(Increase)/Decrease in receivables</td>
<td>181,798</td>
<td>(196,954)</td>
</tr>
<tr>
<td>Increase/(Decrease) in creditors</td>
<td>(123,007)</td>
<td>87,869</td>
</tr>
<tr>
<td>(Increase)/Decrease in other</td>
<td>(39,611)</td>
<td>0</td>
</tr>
<tr>
<td>Net cash from operating activities</td>
<td>1,408,411</td>
<td>611,692</td>
</tr>
</tbody>
</table>

(b) Reconciliation of Cash

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

<table>
<thead>
<tr>
<th></th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cash at Bank</td>
<td>271,312</td>
<td>390,877</td>
</tr>
<tr>
<td>Cash on Hand</td>
<td>50</td>
<td>50</td>
</tr>
<tr>
<td>Current Investments</td>
<td>3,706,762</td>
<td>2,501,876</td>
</tr>
<tr>
<td>Total Cash</td>
<td>3,978,124</td>
<td>2,892,803</td>
</tr>
</tbody>
</table>

NOTE 4: Cash Assets

<table>
<thead>
<tr>
<th></th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cash on hand</td>
<td>50</td>
<td>50</td>
</tr>
<tr>
<td>General Account</td>
<td>25,275</td>
<td>6,247</td>
</tr>
<tr>
<td>Research Account</td>
<td>20,634</td>
<td>267,247</td>
</tr>
<tr>
<td>General Cash Management Account</td>
<td>31,333</td>
<td>768</td>
</tr>
<tr>
<td>Research Cash Management Account</td>
<td>194,070</td>
<td>116,615</td>
</tr>
<tr>
<td>Total Cash Assets</td>
<td>271,362</td>
<td>390,927</td>
</tr>
</tbody>
</table>

NOTE 5: Other Financial Assets

Short Term Cash Deposits

<table>
<thead>
<tr>
<th></th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td>Short Term Cash Deposits</td>
<td>3,706,762</td>
<td>2,501,876</td>
</tr>
</tbody>
</table>

Investments comprise of interest bearing deposits held with financial institutions.
Market values of investments are the same as their book value.
NOTE 6: Receivables - Current

<table>
<thead>
<tr>
<th></th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accounts receivable</td>
<td>78,681</td>
<td>254,018</td>
</tr>
<tr>
<td>Interest receivable</td>
<td>10,442</td>
<td>16,902</td>
</tr>
<tr>
<td>Total Receivables</td>
<td>89,123</td>
<td>270,920</td>
</tr>
</tbody>
</table>

NOTE 7: Property, Plant And Equipment

<table>
<thead>
<tr>
<th></th>
<th>Leasehold Buildings</th>
<th>Plant &amp; Equipment</th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td>Balance 1 July 2002</td>
<td>5,728,264</td>
<td>306,423</td>
<td>6,034,687</td>
<td>5,942,175</td>
</tr>
<tr>
<td>Additions</td>
<td>0</td>
<td>323,090</td>
<td>323,090</td>
<td>104,112</td>
</tr>
<tr>
<td>Disposals</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>(11,600)</td>
</tr>
<tr>
<td>Reclassification/Transfers</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Revaluation increment/decrement</td>
<td>271,735</td>
<td>0</td>
<td>271,735</td>
<td>0</td>
</tr>
<tr>
<td>Balance at 30 June 2003</td>
<td>5,999,999</td>
<td>629,513</td>
<td>6,629,512</td>
<td>6,034,687</td>
</tr>
</tbody>
</table>

DEPRECIATION

<table>
<thead>
<tr>
<th></th>
<th>Leasehold Buildings</th>
<th>Plant &amp; Equipment</th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td>Balance 1 July 2002</td>
<td>359,058</td>
<td>52,775</td>
<td>411,833</td>
<td>154,132</td>
</tr>
<tr>
<td>Charge for the year</td>
<td>240,000</td>
<td>51,125</td>
<td>291,125</td>
<td>258,281</td>
</tr>
<tr>
<td>Adjustments for Disposals</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>(580)</td>
</tr>
<tr>
<td>Revaluation increment/decrement</td>
<td>26,569</td>
<td>0</td>
<td>26,569</td>
<td>0</td>
</tr>
<tr>
<td>Balance at 30 June 2003</td>
<td>625,627</td>
<td>103,900</td>
<td>729,527</td>
<td>411,833</td>
</tr>
<tr>
<td>Written Down Value</td>
<td>5,374,372</td>
<td>525,613</td>
<td>5,899,985</td>
<td>5,622,854</td>
</tr>
</tbody>
</table>

NOTE 8: Payables

<table>
<thead>
<tr>
<th></th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td>Creditors and Accruals</td>
<td>65,894</td>
<td>164,218</td>
</tr>
<tr>
<td>GST Payables</td>
<td>6,967</td>
<td>31,650</td>
</tr>
<tr>
<td>Total Payables</td>
<td>72,861</td>
<td>195,868</td>
</tr>
</tbody>
</table>

NOTE 9: Employee Provisions

<table>
<thead>
<tr>
<th></th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td>Current Employee Entitlements</td>
<td>55,332</td>
<td>36,769</td>
</tr>
<tr>
<td>Non-Current Employee Entitlements</td>
<td>4,392</td>
<td>0</td>
</tr>
<tr>
<td>Total Employee Provisions</td>
<td>59,724</td>
<td>36,769</td>
</tr>
</tbody>
</table>

NOTE 10: Commitments

Capital expenditure commitments for plant and equipment that have not been provided for in the accounts

Payable not later than one year

<table>
<thead>
<tr>
<th></th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>93,578</td>
<td>173,620</td>
</tr>
</tbody>
</table>
NOTE 11: Equity
Accumulated Funds

<table>
<thead>
<tr>
<th></th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td>Balance at beginning of financial year</td>
<td>8,553,840</td>
<td>8,117,172</td>
</tr>
<tr>
<td>Result for the year from ordinary activities</td>
<td>1,075,151</td>
<td>436,668</td>
</tr>
<tr>
<td>Balance at the end of financial year</td>
<td>9,628,991</td>
<td>8,553,840</td>
</tr>
</tbody>
</table>

NOTE 12: Settlement Account
Payment by the Settlor of the Trust Deed

<table>
<thead>
<tr>
<th></th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>100</td>
<td>100</td>
</tr>
</tbody>
</table>

NOTE 13: Asset Revaluation Reserve
Asset Revaluation Reserve

<table>
<thead>
<tr>
<th></th>
<th>2003</th>
<th>2002</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>245,166</td>
<td>0</td>
</tr>
</tbody>
</table>

NOTE 14: Restricted Assets
The financial statements include investments of $3,706,762 ($2,501,876 in the previous year) that are restricted by the grantor's donor requirements to be expended on the establishment and subsequent maintenance of a medical research centre. As indicated in Note 1(F), assets under construction have been capitalised during 2002-2003 and amounted to $323,090 ($13,625 for 2001/2002).

NOTE 15: Related Party Disclosures And Remuneration And Retirement Benefits
No directors received, directly or indirectly income paid or payable, or otherwise made available from ANZAC Health and Medical Research Foundation during this financial year.
No amount was paid, on behalf of a director, to a superannuation plan.

NOTE 16: Fundraising And Appeal Activities
The ANZAC Health and Medical Research Foundation is a certified holder of an authority to raise funds under the provision of Section 16 of the Charitable Fundraising Act, 1991.

Income received and the cost of raising income for specific fundraising has been audited and all revenue and expenses have been recognised in the financial statement of the ANZAC Health and Medical Research Foundation.

<table>
<thead>
<tr>
<th></th>
<th>Income Raised</th>
<th>Direct Expenditure</th>
<th>Indirect Expenditure</th>
<th>2003 Net Proceed</th>
<th>2002 Net Proceed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appeals - In house</td>
<td>82,142</td>
<td>3,533</td>
<td>-</td>
<td>78,609</td>
<td>90,789</td>
</tr>
<tr>
<td>Raffles</td>
<td>0</td>
<td>0</td>
<td>-</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Functions</td>
<td>94,953</td>
<td>51,573</td>
<td>9,471</td>
<td>33,909</td>
<td>88,315</td>
</tr>
<tr>
<td>Total</td>
<td>177,095</td>
<td>55,106</td>
<td>9,471</td>
<td>112,518</td>
<td>179,104</td>
</tr>
</tbody>
</table>

Percentage of Income

|                          | 100%          | 31.12%           | 5.34%               | 63.55%          |

Direct expenditure includes printing, postage, raffle prizes, food and beverage.
Indirect expenditure includes additional staff time.
The net proceeds were held in ANZAC Health & Medical Research Foundation pending allocation.
The provisions of the Charitable Fundraising Act 1991 and the regulations under the Act have been complied with and internal controls exercised by the ANZAC Health & Medical Research Foundation are considered appropriate and effective in accounting for all the income received in all material respects.
## ANZAC Health and Medical Research Foundation - Trust Fund

### NOTES TO THE FINANCIAL STATEMENTS continued

**NOTE 17: Financial Instruments**

**a) Interest Rate Risk Exposures**

Interest rate risk, is the risk that the value of the financial instrument will fluctuate due to changes in market interest rates. ANZAC Health & Medical Research Foundation’s exposure to interest rate risks and the effective interest rates of financial assets and liabilities, both recognised and unrecognised, at the Statement of Financial Position date are as follows:

<table>
<thead>
<tr>
<th>Financial Instruments</th>
<th>Floating Interest Rate</th>
<th>Non-Interest Bearing</th>
<th>Total Carrying Amount as per Statement of Financial Position</th>
<th>Total Carrying Avg Effective Interest Rate</th>
<th>Weighted Avg Effective Interest Rate*</th>
<th>Weighted Avg Effective Interest Rate*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Financial Assets</td>
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<tr>
<td>Cash</td>
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<td>271</td>
<td>391</td>
<td>2.96</td>
<td>2.92</td>
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<tr>
<td>Receivables</td>
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<td>89</td>
<td>89</td>
<td>271</td>
<td></td>
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</tr>
<tr>
<td>Other loans &amp; deposits</td>
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<td>3,707</td>
<td>2,502</td>
<td>4.61</td>
<td>4.41</td>
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<td>Total Financial Assets</td>
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<td>89</td>
<td>4,067</td>
<td>3,164</td>
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</table>

*Weighted average effective interest rate was computed on a semi-annual basis. It is not applicable for the non-interest bearing financial instruments.

**b) Credit Risk Exposures**

Credit risk is the risk of financial loss arising from another party to a contract/or financial position failing to discharge a financial obligation thereunder. The ANZAC Health and Medical Research Foundation’s maximum exposure to Credit Risk is represented by the carrying amounts of the financial assets included in the Statement of Financial Position.

Credit Risk by classification of counterparty.

<table>
<thead>
<tr>
<th>Banks</th>
<th>Other</th>
<th>Total</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>2003 $000</td>
<td>2003 $000</td>
<td>2003 $000</td>
<td>2002 $000</td>
</tr>
<tr>
<td>Financial Assets</td>
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<td></td>
</tr>
<tr>
<td>Cash</td>
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<td>0</td>
<td>271</td>
</tr>
<tr>
<td>Receivables</td>
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<td>79</td>
<td>89</td>
</tr>
<tr>
<td>Other Loans &amp; Deposits</td>
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<td>0</td>
<td>3,707</td>
</tr>
<tr>
<td>Total Financial Assets</td>
<td>3,988</td>
<td>79</td>
<td>4,067</td>
</tr>
</tbody>
</table>

*End of Audited Financial Statements*