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Over the past couple of years, there has been a greater emphasis on ovarian cancer, both in the research setting and also in terms of public awareness. This is entirely fitting, as, while total numbers of cases of ovarian cancer are not huge, the impact of the disease on society certainly is, with three-quarters of the cases presenting as late stage (stages III and IV) disease. While many respond to chemotherapy, there is a high relapse rate and ultimately, three-quarters of women with advanced cancer will ultimately die from the disease.

An Australian Cancer Network working party has spent the past two years refining the Guidelines for the Management of Ovarian Cancer, and this document has now been sent to the National Health and Medical Research Council for ratification. Dissemination of these Guidelines is expected early in the new year.

At the same time, an expert advisory group, under the umbrella of the National Breast Cancer Centre, is working on strategies to disseminate these guidelines to the profession, and develop a separate consumer version.

On the research front, the Australia New Zealand Gynaecological Oncology Group is collaborating in a multi-centre, multi-pronged study. This study is supported by funding from the Department of Defence, in part.

The three major components include:

1. An epidemiological study – case-controlled, looking at lifestyles of all women newly diagnosed with ovarian cancer over the next three years, and comparing them with age-matched controls, seeking to tease out any risk factors, lifestyle changes, etc. They expect to recruit at least 1,000 women, making it the largest study of its kind in the world, building on a previous eastern states study conducted in the early 1990s.

2. A biospecimen collection, with samples of tumour and blood taken from women at the time of their surgery for ovarian cancer. Microarray studies will be undertaken, and there are plans to establish a tissue bank for future collaborative studies also.

3. A chemotherapy treatment study, comparing in a blinded randomised way four different treatment arms; comparing doublets and triplets of chemotherapy with the “gold standard” of Paclitaxel and Carboplatin. Other drugs to be studied are Topotecan, Gemcitabine and Caelyx.

This review of the current state-of-the-art in ovarian cancer is not meant to be a forerunner of the Guidelines, but rather to highlight areas of change or special areas of interest to Australian researchers.

In compiling these articles, I am also very conscious of the work being undertaken by cancer consumer groups, especially in ovarian cancer, where they are making an impact on women and their families with the disease both in lobbying and in fundraising to assist research.

Ovarian cancer is still a major problem today, but the research effort can be expected to make an impact over the next few years.
Ovarian cancer is the leading cause of death from gynaecological cancers in Australia. In recognition of the impact of ovarian cancer on Australian women, in September 2001 the Federal Government committed $500,000 over two years, to improve the health outcomes for women with ovarian cancer. The National Breast Cancer Centre (NBCC) was chosen to manage a national ovarian cancer initiative.

In November 2001, a strategic plan, Priority actions for ovarian cancer control: a framework for a national approach, was developed by an Interim Ovarian Cancer Steering Group, based on input from key stakeholders in ovarian cancer control. The plan outlined eight objectives, and from these objectives a work plan for the Ovarian Cancer Program was developed, focusing on a number of priority areas:

- Information for women, health professionals, policy makers and the community about aspects of ovarian cancer such as risk factors and diagnosis of ovarian cancer;
- Promotion of optimal management of all women diagnosed with ovarian cancer;
- Strengthening the provision of psychosocial, physical and practical support for women diagnosed with ovarian cancer and their families;
- Developing a national monitoring system for ovarian cancer control in Australia; and
- Encouraging all organisations concerned with ovarian cancer control to work collaboratively to improve ovarian cancer outcomes.

A multidisciplinary Expert Advisory Group was established to guide the work of the Ovarian Cancer Program. The work of the Program is also supported by a number of working groups for specific projects.

This report provides a summary of those objectives and the achievements to date.

Risk factors for ovarian cancer

While much research in Australia and internationally is directed at understanding the causes of ovarian cancer, there are currently no opportunities for community-wide prevention programs. Known risk factors include family history, age and personal history of ovarian cancer and non-contraceptive use are associated with a reduced risk of ovarian cancer.

Preventive oophorectomy, tubal sterilisation and hysterectomy have been shown to reduce a woman’s risk of ovarian cancer.

To date, the Program has commissioned a detailed review of the research about known risk factors for ovarian cancer and opportunities for prevention. The review will be made available in early 2004 and will provide an evidentiary base for the development of information for clinicians and women.

Information for women with a family history or known genetic risk for ovarian cancer

Up to 10% of all cases of epithelial ovarian cancer are thought to be due to hereditary factors. Women with a genetic mutation in BRCA1 and BRCA2 have a risk of between 15% to 56% of developing ovarian cancer by the age of 70.

In association with the Genetics Expert Advisory Group of the National Breast Cancer Centre, and in light of recent progress in the understanding of this area, the published advice about familial aspects of ovarian cancer has been reviewed, with a view to simplifying the risk categories.

Encouraging prompt diagnosis for women with symptoms that may be ovarian cancer

It is estimated that about 70% of ovarian cancers are advanced at diagnosis and advanced disease has a poor prognosis. It is difficult to diagnose ovarian cancer at an early stage because early disease is typically asymptomatic, early symptoms are non-specific and there is currently no accepted method for population screening.

In progressing activities in regard to this objective, the Expert Advisory Group has agreed that there is no currently evidence to support the conduct of a public education campaign about symptoms of ovarian cancer. The group is developing guidelines for general practitioners to assist them with the often difficult assessment and investigation of women who may have ovarian cancer.

Promoting optimal management of all women diagnosed with ovarian cancer

The five-year relative survival of women with ovarian cancer after diagnosis is about 42%. Adverse prognostic factors for ovarian cancer include older patient age, later stage of disease, higher tumour grade, presence of ascites and residual disease.

Most Australian women with ovarian cancer are treated with surgery and/or chemotherapy. There is evidence that women with ovarian cancer who are treated by a gynaecological oncologist have improved survival rates, yet a large number of women do not currently receive optimal care.

To address the lack of national evidence-based guidelines about the management of ovarian cancer, the Australian Cancer Network established a working party in 1999 to develop the Clinical practice guidelines for the management of women with epithelial ovarian cancer. This process has been subsequently supported and completed under the auspices of the Ovarian Cancer Program and the guidelines have been submitted to the National Health and Medical Research Council (NHMRC) for approval.

The guidelines will be nationally implemented in early 2004, followed by initiatives to promote the benefits and improved outcomes of multidisciplinary care of women with ovarian cancer.

Research conducted in Australia and overseas indicates that outcomes are improving for all women with ovarian cancer who are informed about their treatment options. Women with ovarian cancer seek information about a wide range of topics to help them understand and deal with their diagnosis. A consumer guide for women with ovarian cancer is being developed by the Centre, based on the evidence in the Clinical practice guidelines for the management of women with epithelial ovarian cancer. The guide will address topics such as the types and stages of ovarian cancer, treatment options, managing pain and side-effects; the use of complementary and alternative therapies; and support issues.

Strengthening the provision of psychosocial, physical and practical support to all women diagnosed with ovarian cancer and their families

The diagnosis of ovarian cancer has a major impact on women and their families. Of women diagnosed with ovarian cancer, a high proportion has advanced disease. Therefore, providing adequate support for women to improve their quality of life is an important component of patient care. Adequate support and information can improve the quality of life and satisfaction with care.

Good communication skills, information provision and continuity of care have also been found to improve quality of life for cancer patients.

Currently, there is limited access to supportive care services in Australian gynaecological oncology units. The Ovarian Cancer Program is working with the Psychosocial Expert Advisory Group of the National Breast Cancer Centre to investigate the specific needs of women with ovarian cancer and to develop strategies for addressing these needs.

National monitoring system for ovarian cancer control in Australia

While some data are collected about ovarian cancer in Australia, there is inconsistency in the way that data are collected and reported. This makes comparison of data between states difficult. Regular and timely monitoring of ovarian cancer control will enable the targeting of new programs and the evaluation of the effectiveness of current programs. Such a monitoring system should be comprehensive including data on mortality, diagnosis, treatment and supportive care needs of women with ovarian cancer.

The Ovarian Cancer Program has developed plans to review national data about ovarian cancer and data collection processes to identify gaps in the information available and to direct the development of a strategy to standardise data collection about ovarian cancer.

To provide all women, health professionals, policy makers and the community with access to current, accurate and appropriate information about all aspects of ovarian cancer

Women, health professionals, policy makers and the community should have access to accurate, appropriate and timely information about relevant aspects of ovarian cancer control.

The Program has acted as a clearinghouse for evidence-based information through a regularly updated online newsletter (Ovarian e-update) disseminated to clinicians, researchers, consumers, cancer councils, state and federal health departments (12 issues to date); and through the establishment of a website for the Ovarian Cancer Program (www.ovariancancerprogram.org.au).

To encourage all organisations concerned with ovarian cancer control to work collaboratively

There are many groups working towards improving ovarian cancer control in Australia. Collaboration between these organisations is vital to improving outcomes for women and maximising the use of resources by avoiding duplication and by introducing varied expertise.

The Ovarian Cancer Program has built relationships to foster a collaborative approach through the establishment of a multidisciplinary Expert Advisory Group to provide advice about the projects and through contact with a range of clinicians, researchers and consumer groups involved in ovarian cancer control. A national ovarian cancer forum, planned for early 2004, will bring together relevant areas in ovarian cancer control and foster a national information network. It will provide a further opportunity to promote multidisciplinary care and will focus on the delivery of health services and opportunities to improve outcomes.

In summary, the National Ovarian Cancer Program has been active in a broad range of areas. The Australian Government has provided additional funding until the end of 2003, and this will allow further development of these initiatives. With the recent re-funding of the National Breast Cancer Centre for a further four years, the Ovarian Cancer Program is seen as an integral component of the Centre’s future work plans.

References

RISK FACTORS FOR EPITHELIAL OVARIAN CANCER

S J Jordan (pictured top), DM Purdie, DC Whitman, P M Webb (pictured bottom)
Queensland Institute of Medical Research
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Although in Australia the lifetime risk of ovarian cancer is only one in 107, it is the fifth most common cause of cancer death in Australian women. Over 90% of ovarian malignancies are thought to arise from the ovarian epithelium, while the remaining 10% include germ cell tumours, sex cord tumours and malignant teratomas. Most epidemiological research to date has focused on the common epithelial ovarian cancers and the following discussion is restricted to these tumours.

Much remains unknown about the pathogenesis of epithelial ovarian cancer but the two main theories implicating either incessant ovulation or high levels of circulating gonadotropins. The strongest risk factors are increasing age and a family history of ovarian cancer, but other reproductive, medical and lifestyle factors also appear to have a significant impact on risk. In this review we summarise the current state of knowledge and uncertainty regarding non-genetic risk factors for epithelial ovarian cancer (EOC).

Hormonal and reproductive factors

Oral contraceptive use

Women who have ever taken the oral contraceptive pill (OCP) have an approximately 40% lower risk of EOC than women who have never taken the OCP. Increasing duration of use is associated with a reduction in risk of 8% per year of use and the decrease in risk may persist for more than 20 years after stopping use.1 Lower lifetime risk of EOC was seen in women with a family history of breast cancer,2 and women who took the pill for birth control,3 but not women who took the pill to treat acne.4 Studies have considered the different types of OCP and the different bacterial subtypes5 but one group reported an increased risk of mucinous EOC associated with ever-use of the OCP.6 Whether the oral contraceptive pill decreases the risk of ovarian cancer is uncertain.5 A case-control study has reported that OCP use was associated with a decreased risk of EOC in non-carriers but not in carriers,7 while a second study found significant reductions in risk in women ever-users and increasing duration of use among women with BRCA1 and BRCA2 mutations8. Further studies are required to clarify this important issue.

Breast feeding

There is fairly consistent evidence that women who breast feed their children have a lower risk of ovarian cancer than parous women who do not breast feed, although the evidence is modest with about a 1% reduction in risk for each month of breast feeding.9 This association is the result of increased ovulations and is supported by the fact that breast feeding suppresses ovulation.10

Age at menarche and age at menopause

As described above, increasing numbers of ovulations are associated with higher risks of ovarian cancer. On this basis, it has been hypothesised that women with an earlier age at menarche or a later age at menopause, and therefore more potential years of ovulation, would be at increased risk of ovarian cancer. The data are, however, highly inconsistent with some studies reporting positive associations, some no associations and some a negative effect.11 It is possible that this inconsistency is a consequence of the fact described above where ovulations early and late in a woman’s reproductive life may be less relevant in terms of influencing risk of ovarian cancer.12

Hormone replacement therapy

The use of hormone replacement therapy (HRT) has been associated with a small but significant increase in risk of EOC and this risk appears to increase with increasing duration of use.13 Few studies have considered the different types or regimes of HRT but a recent case-control study found that women who took oestrogen alone or oestrogen with sequential progestogens, but not with continuous progestogens (the so-called ‘mini-pill’), had a significantly increased risk of EOC (OR 1.43, 95% CI 1.02-2.00 and OR 1.54, 95% CI 1.15-2.05 respectively), while those who used oestrogen with continuous progestrone were not at increased risk.14 Clarification of this issue in future studies may be important in terms of identifying the role of reproductive hormones in the pathogenesis of this cancer.

Infertility and infertility treatment

There is conflicting evidence regarding the effect of infertility treatment on the risk of EOC but a recent pooled analysis of eight population-based case-control studies found no evidence of an association (OR 0.97, 95% CI 0.76-1.25).15 Women treated with infertility drugs who do not have a subsequent pregnancy may have a small increased risk but the independent effects of different causes of infertility and the different fertility treatments are yet to be resolved.

Medial procedures, medications and medical conditions

Hysterectomy and tubal ligation

Hysterectomy and tubal ligation have been consistently associated with a 20%-50% decrease in risk of EOC suggesting that both of these procedures confer a protective effect against this cancer. It has been suggested that the apparent protective effects of tubal ligation and sterilisation on the risk of EOC are due to the reduction in the number of lifetime ovulations, but no clear evidence of an increase in risk with increasing duration of use has been reported. At this stage it is not possible to draw firm conclusions about the effects of these surgical procedures on the risk of ovarian cancer16. Further data are required to confirm this finding.

Physical activity

The effects of physical activity on risk of EOC have been investigated infrequently and the results that have been reported are inconsistent. Some have reported an increase in risk of ovarian cancer with increased levels of physical activity17 but others have found either no association or a decrease in risk.18 Current evidence suggests it does not impact significantly on ovarian cancer risk.

Talc use

Talc use has long been considered a potential risk for EOC because of its structural similarity to asbestos (a known human carcinogen) and the biological possibility that retrograde movement through the genital tract may allow talc to initiate inflammatory changes near or on the epithelial surface. A recent meta-analysis reported a pooled odds ratio of 1.38 (95% CI 1.25-1.52) for use of perineal talc versus non-use but the authors found no clear evidence of an increase in risk with increasing duration of use.19 Despite the lack of dose-response, the consistency of the findings across the population-based studies suggests that use of perineal talc is associated with a small increase in risk of EOC.

Coffee and tea

Although it has been hypothesised that coffee and tea may be carcinogenic, most population-based studies of women with visually diagnosed cancer found no clear evidence of an association between coffee and EOC.20 However, a recent study of endometrial cancer confirmed an increased risk of EOC21 but further data are required to confirm this association.

Tobacco smoking, alcohol and caffeine

Overall, no consistent association has been observed between cigarette smoking and EOC22 but subtype-specific analyses provide fairly consistent evidence that the risk of endometrioid clear cell subtypes23 is increased by cigarette smoking (OR ~3.0 for ≥ 25 pack-years).24 There is some evidence that moderate alcohol intake may decrease the risk of ovarian cancer. An American cohort study which followed post-menopausal women for 10 years, reported a significant trend of decreasing risk of EOC with increasing consumption of alcohol up to about one standard drink per day (p=0.01) but this finding was not replicated by a subsequent population-based case-control study.25 More recently, another American case-control study found that current alcohol drinkers had a decreased risk of ovarian cancer compared to non-drinkers (OR 0.61, 95% CI 0.39-0.94).26 A significant trend of decreasing risk with increasing consumption (up to 14 drinks per week) was also observed (p=0.009). The effect was strongest for white women. Further studies considering types of alcohol and examining effects according to histological findings are required to confirm the effects of alcohol on risk of EOC.

Lifestyle and diet

Obesity

Obesity has been associated with changes in circulating hormone levels, particularly oestrogen and testosterone, and elevated levels of these have been implicated in the pathogenesis of ovarian cancer27. There is consistent evidence of a small but significant effect of high body mass index on risk of EOC (OR 1.4, 95% CI 1.2-1.6).28 Some studies suggest this association is strongest in women diagnosed with EOC pre-menopaually29,30 but further assessment is required to confirm this finding.

Coffee and tea are chemically complex beverages containing...
both potential carcinogens as well as chemoprotective agents43,44. The weight of evidence suggest there may be a small positive association between coffee drinking and the risk of EOC, however many of the associations have been weak and without clear evidence of a dose response. One study has suggested that the effect of coffee may be moderated by menopausal status8,45 but this has yet to be confirmed. Tea has been examined less frequently in relation to EOC and most studies have found no effect although one Chinese study reported a stronger association between tea consumption and EOC46. It is thus possible that any effect of tea may depend on the type of tea (green or black) that is consumed.

Dietary factors that have been examined in relation to ovarian cancer risk include fruit and vegetables, eggs and dairy products as well as a range of macronutrients, such as dietary fat, and micronutrients. Limited data suggest an inverse relationship between fresh fruit and/or vegetables35 and, possibly, β-carotene42 and EOC. A number of population-based studies have examined the effect of egg consumption on ovarian cancer risk and all have found an increased risk based on these factors on EOC risk.

1 Direction and approximate strength of likely and (possible) associations of the oral contraceptive pill confer significant decreases in risk of EOC and it is likely that this is also the case for breastfeeding. Tubal ligation, hysterectomy and possibly a diet high in fruit and vegetables also appear to reduce risk. Factors that may increase the risk of epithelial ovarian cancer include use of hormone replacement therapy, obesity, cigarette smoke (mucinous tumours only), application of talc to the perineal region, regular consumption of eggs, endometriosis and pelvic inflammatory disease.

Much, however, remains unknown about the aetiology of this disease. Many studies have treated epithelial ovarian cancer as a single disease and it is now clear that the different histological subtypes may differ with respect to key risk factors1. Larger studies are needed in order to evaluate the risk factors for the specific subtypes. Further research is required to clarify the potential role of modifiable exposures such as diet and physical activity in the prevention of this commonly fatal disease. Additional work is also required to clarify the association between factors such as oral contraceptive use, hysterectomy and tubal ligation and ovarian cancer risk among women who carry BRCA mutations and so are at high risk of developing ovarian cancer before we can recommend these measures as preventive interventions in this group.

Summary and conclusions
A summary of the main risk and protective factors for epithelial ovarian cancer is presented in table 1. Aside from increasing age and a family history of breast or ovarian cancer, the most significant risk factors for epithelial ovarian cancer are those related to reproductive history, high parity and long-term use of the oral contraceptive pill confer significant decreases in risk of EOC.
The management of these women at increased risk of ovarian cancer is difficult and presents many challenges. It should be approached in a stepwise fashion regarding the clinical presentation and advice to at risk individuals is still preliminary and will undoubtedly change over time. The purpose of this paper is to briefly review the current state of knowledge about the genetics of hereditary ovarian cancer and update interested readers on the genetic epidemiology, prevalence and penetrance of the specific germline mutations associated with ovarian cancer. No mutations were found in women with borderline tumours, which is consistent with other studies, but there was an association with mutations and the histology of invasive ovarian cancer. Specifically, mutations were detected in 16.4% of serous cancers, 4.3% of endometrioid cancers and in none of the other histological subtypes. This study needs to be confirmed as it raises the possibility that mutations in BRCA1 and BRCA2 may be more common in unselected populations with ovarian cancer than previously anticipated and it also challenges some of the epidemiological data relating family history to risk of ovarian cancer.

First degree relatives with one affected family member with ovarian cancer have generally been advised that they have a 5% lifetime risk which is about three times the 1.4% lifetime risk for women without a family history. For women with two affected relatives the lifetime risk has been said to rise to about 7%, and these figures have been used to counsel women about risk. However, the confidence values of these risk estimates are wide and studies such as the Canadian population study among others challenge these figures, which may be too simplistic. It is essential that an accurate family history is taken in order to estimate risk of ovarian cancer based on family history and ethnicity should also be taken into account. Women of Ashkenazi Jewish background with a family history of one case of ovarian cancer and women in the general population with two or more relatives with ovarian cancer should be considered to be at potentially higher risk and be referred to a familial cancer clinic for further advice and counselling.

The lifetime risk of ovarian cancer in women with mutations in BRCA1 and BRCA2 varies considerably in different studies and is estimated to be in the range of 20-40% in those of familial origin. The study group included 1172 women with a germline mutation in either BRCA1 or BRCA2 who were part of the large-scale collaborative project described in this paper. A majority of ovarian cancers are due to mutations in these genes but the percentage appears to be higher than previously anticipated. In a recent population-based case-control study of familial cases of ovarian cancer in Canada, 11.7% of women were found to have pathogenic mutations in BRCA1 and BRCA2, which is more than previous estimates and may even be higher as the genetic tests used would not be expected to detect all mutations. Mutations were found in 19% of women with first-degree relatives with breast/ovarian cancer and in 65% of women with more than two first-degree relatives with breast cancer. No mutations were found in women with borderline tumours, which is consistent with other studies, but there was an association with mutations and the histology of invasive ovarian cancer. Specifically, mutations were detected in 16.4% of serous cancers, 4.3% of endometrioid cancers and in none of the other histological subtypes. This study needs to be confirmed as it raises the possibility that mutations in BRCA1 and BRCA2 may be more common in unselected populations with ovarian cancer than previously anticipated and it also challenges some of the epidemiological data relating family history to risk of ovarian cancer.

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Table 1: Genes associated with hereditary ovarian cancer

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Gene</th>
<th>Lifetime risk</th>
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<tbody>
<tr>
<td>Breast/ovarian syndrome</td>
<td>BRCA1</td>
<td>10-60%</td>
</tr>
<tr>
<td>Hereditary non-polyposis colorectal cancer</td>
<td>MLH1</td>
<td>10%</td>
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<tr>
<td></td>
<td>MSH2</td>
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<td></td>
<td>MSH6</td>
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<td></td>
<td>PMS1</td>
<td></td>
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<tr>
<td></td>
<td>PMS2</td>
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at an “early” phase with our current technology 30. A good compromise is likely to be requested or used that a hysterectomy and detailed individual discussion is required.

The aim of this paper was to briefly review and summarise a large and complex literature that is rapidly growing. Our increasing ability to identify women at increased genetic risk of developing ovarian cancer has not been matched by good level 1 evidence regarding how best we manage these women and their families. We have briefly discussed the natural history and biology of ovarian cancer, the appeal of screening and early diagnosis as well as the limitations of screening using currently available screening tests. The most convincing data regarding reducing risk is seen in the studies of RRBSO, but clearly this is not an attractive option to many women, and alternative pharmacological strategies such as oral contraceptives may be explored further. More research on the psychological impact of being a carrier and the many issues that these women and their families face. This should not be interpreted as lack of importance for carriers. A recent review of the advances in this important literature, and the interested reader is directed to papers on this35. The management guidelines and advice on familial aspects of ovarian cancer recently have been updated and will provide health professionals with a detailed overview of familial ovarian cancer.

References
7. A Berchuck, KA Heron, ME Carney, JM Lancaster, EG Fraser, VL Vinson. “Epidemiological studies have strongly suggested that the oral contraceptive pill (OCP) is associated with a significant reduction in ovarian cancer risk in the general population.”12 Similarly, case control studies have demonstrated that women who have a family history of breast/ovarian cancer also have a similar reduction in ovarian cancer risk of about 50% if they have been on the pill for several years. This is not confirmed in a population study of Jewish women in Israel, which raises some questions regarding the value of OCP in reducing ovarian cancer risk in the general population”.14 Of particular concern as well is the uncertainty regarding the risk of the OCP on breast cancer risk in women who have germline defects in BRCA1 and BRCA2.12 This also needs to be dissected further with a recent early report of a reduction in BRCA2 prevalence in a population of 549 women with ovarian cancer.12 Am J Hum Genet, 68 (2001):700-710.  
13. A Berchuck, KA Heron, ME Carney, JM Lancaster, EG Fraser, VL Vinson. “Epidemiological studies have strongly suggested that the oral contraceptive pill (OCP) is associated with a significant reduction in ovarian cancer risk in the general population.”12 Similarly, case control studies have demonstrated that women who have a family history of breast/ovarian cancer also have a similar reduction in ovarian cancer risk of about 50% if they have been on the pill for several years. This is not confirmed in a population study of Jewish women in Israel, which raises some questions regarding the value of OCP in reducing ovarian cancer risk in the general population”.14 Of particular concern as well is the uncertainty regarding the risk of the OCP on breast cancer risk in women who have germline defects in BRCA1 and BRCA2.12 This also needs to be dissected further with a recent early report of a reduction in BRCA2 prevalence in a population of 549 women with ovarian cancer.12 Am J Hum Genet, 68 (2001):700-710.  

Table: ovarian cancer risk reduction strategies for BRCA1 and BRCA2 mutation carriers

<table>
<thead>
<tr>
<th>Strategy</th>
<th>Risk reduction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prophylactic salpingo-oophorectomy</td>
<td>96%</td>
</tr>
<tr>
<td>Oral contraceptive pill</td>
<td>50%</td>
</tr>
<tr>
<td>Tubal ligation</td>
<td>63%</td>
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* Protective effect only seen in BRCA1 mutation carriers

Molecular Prognosis of Epithelial Ovarian Cancer: Observations from Current Literature
D Boatwell
Research Division, Peter MacCallum Cancer Institute
Melbourne, VIC

Cancer of the ovary is both the most prevalent and lethal form of gynecological cancer. More than three-quarters of women afflicted have disseminated disease at the time of diagnosis and receive treatment which is usually a combination of debulking surgery and chemotherapy. The most effective chemotherapy agent against ovarian cancer is cisplatin – also of debulking surgery and chemotherapy. The most effective chemotherapy agent against ovarian cancer is cisplatin – also chemotherapy agent against ovarian cancer is cisplatin – also chemotherapy agent against ovarian cancer is cisplatin – also chemotherapy agent against ovarian cancer is cisplatin – also chemotherapy agent against ovarian cancer is cisplatin – also chemotherapy agent against ovarian cancer is cisplatin – also chemotherapy agent against ovarian cancer is cisplatin – also chemotherapy agent against ovarian cancer is cisplatin – also chemotherapy agent against ovarian cancer is cisplatin – also chemotherapy agent against ovarian cancer is cisplatin – also chemotherapy agent against ovarian cancer is cisplatin – also chemotherapy agent against ovarian cancer is cisplatin – also chemotherapy agent against ovarian cancer is cisplatin – also chemotherapy agent against ovarian cancer is cisplatin – also chemotherapy agent against ovarian cancer is cisplatin – also chemotherapy agent against ovarian cancer is 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this study showed with the use of tissue arrays (120 cases in total) that expression of COL6A3 in primary ovarian tumours is also correlated with tumour grade. This pathological measure has been shown to relate to chemotherapy response and overall survival rates.

The gene KLK4 (Higher Human kallikrein gene 4) has also been associated with disease progression and survival time in ovarian cancer. KLK4 has been implicated in other hormonally regulated cancers, including those of the breast and prostate. In 147 ovarian cancer samples, expression of the gene was found to be associated with disease progression in a higher percentage of tumours with tumour grade and stage. Overall the authors of this study concluded that KLK4 expression was higher in tumours with a more aggressive phenotype, generally translating to an increased risk of relapse and death. When tested against chemotherapy response rates, a correlation between positive expression and lack of treatment efficacy was detected. Interestingly, it was noted that positive KLK4 expression in grade 1 and 2 cases indicated a 2.5-fold increase in relative risk of relapse, yet the same degree of up regulation was not significantly predictive of relapse in grade 3 tumours (figure 3).

The Fanconi anemia-BRCA pathway has been implicated in the molecular changes occurring in cisplatin-resistant ovarian cancer. Interruption of this genetic pathway ultimately appears to lead to the development and selection of drug resistant cancer cells. This pathway is comprised of six genes associated with Fanconi anemia syndrome (FANCA, -C, -D2, -E, -F and -G) plus BRCA1 and BRCA2 and regulates cell cycle and other DNA cross-linking substances. Cisplatin resistance in ovarian cancer cell lines can be attributed to initial methylation induced inactivation and subsequent demethylation of FANCP. As this work has shown considerable molecular differences between cell lines and ovarian tumours on the basis of hierarchical clustering and multi-dimensional scaling (MDS) with data generated from cDNA microarrays as shown in figure four.

Ovarian cancer is the leading cause of death from gynaecological cancer in the developed world, comprising 5% of all cancer-related deaths in women. The high mortality rate has been attributed primarily to the difficulty in detecting the disease when it is still confined to the ovary (stage 1). The overall five year survival rate for stage 1 disease is 95%, compared with 20% for stage IV disease. However, less than 25% of cases are confined to the ovary at the time of diagnosis. Thus the aim of screening in ovarian cancer has been to detect the disease when still confined to the ovary. However, to date there is no evidence to show an impact on mortality nor is there a test available that prognostically detects ovarian cancer.

Screening tests in ovarian cancer have until recently relied upon either pelvic ultrasound or the detection of the high molecular weight glycoprotein Ca125 in the serum or a combination of the two modalities (multimodal screening). Transabdominal ultrasound was used in the early screening studies but has been replaced by the more sensitive transvaginal ultrasound (TVS) with or without the use of colour Doppler imaging. Progesterone receptors and estrogen receptors which are absent or down regulated in ovarian cancer can also be detected by dedicated ligand binding assays. Screening for ovarian cancer has been based largely on the interpretation of Ca125 in the place of standard cut-off levels.

The power of microarray analysis to reveal important information about the variation in ovarian cancer patient survival rates is demonstrated by Lancaster et al. TRAIL, a gene identified from array profiling, was demonstrated to be a useful prognosticator. Using RT-PCR profiling of 120 epithelial cancers the authors describe a highly significant relationship between this gene and prolonged survival. Patients who lived for more than five years had 2.2-fold higher expression of this gene than those who died within 12 months of diagnosis. This gene is a member of the “death ligands” and a member of the apoptotic pathway. Another study has demonstrated the combination of TRAIL and chemotherapy lead to a significant increase in apoptosis and growth inhibition of ovarian cancer cell lines and propose the clinical use of this treatment combination.

It is clear that the difficulty of successful early detection and high rate of treatment resistance remain two of the key challenges in ovarian cancer treatment and research. If these hurdles can be overcome through an increased understanding and manipulation of the underlying molecular changes many lives may be saved from this aggressive and widespread disease.

References
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Lysosphosphatic acid (LPA), a bioactive phospholipid, has been reported as a potential discriminating marker for ovarian cancer including stage 1 disease (13). High affinity receptors for LPA, Edg4 and Edg7 also have been shown to be increased in ovarian cancer cells (14). Other molecules that may be potential adjuncts to Ca125 include osteopontin; kalreinins (5) and a panel of markers including OVX1 and CA125 (6). Serum inhibin levels may also be a useful adjunct to Ca125 as 80% of mucinous epithelial ovarian cancers and a large proportion of sex cord stromal tumours are associated with increased levels of inhibin (15).

Currently there are two major randomised controlled trials underway to establish the impact of screening on ovarian cancer mortality as well as determining issues of compliance, health economics and physical and psychosocial morbidity. The United Kingdom Collaborative Trial of Ovarian Cancer Screening (UKTOCS) began recruiting postmenopausal women in 2001 and involves 12 centres in the United Kingdom. The aim of the study is to recruit a total of 200,000 women who will be randomised to either control, screening with ultrasound or multimodal screening. The primary end-point is the impact of screening on ovarian cancer mortality and the results are expected in 10 years. The Prostate, Lung, Colorectal and Ovarian (PLCO) Cancer Screening Trial is a two-arm randomised controlled trial involving 74,000 women aged between 55 to 74 who have been randomised to a screening arm (annual screening for ovarian, lung and colon cancer) or to a standard care control arm. Ten centres are involved in this trial which will involve 10 years’ average follow-up. Only one screening strategy is being used, namely a combination of TVS and Ca125 performed annually for three years followed by Ca125 alone for two years. The trial has completed enrolment. Clearly the results of both trials will be eagerly awaited although issues pertaining to cost-effectiveness, age at which to begin screening and appropriate screening interval remain unanswered.

The two randomised controlled trials rely upon current technology and run the risk of being out-dated before the data has been analysed. Our understanding of tumour biology would suggest that the progression of a normal cell to a cancer cell involves multiple changes in a number of key pathways in the cell. It would therefore seem logical to question the suitability of single serum markers to identify ovarian cancer. Recent developments in gene expression and more recently in proteomics may well hold the key to new screening tests for ovarian cancer. Petricoin et al have described the use of mass spectroscopy (surface-enhanced laser desorption and ionisation, SELDI) to define a profile associated with sera derived from patients with ovarian cancer. This profile was able to correctly identify 50 out of 50 cases of ovarian cancer, including 18 cases of stage 1 disease and to identify 63 of 66 cases of non-malignant disease, suggesting that this new technology may be a potential tool for screening. Clearly larger and more discriminatory studies will be needed to be performed but new technologies such as this may well hold the key to the development of an effective screening test for ovarian cancer.

References
Gastrointestinal stromal tumour prognostic parameters: Case report and literature review

T Keith

ABSTRACT

This paper reviews the evaluation of malignancy and prognostic parameters used in gastrointestinal stromal tumours (GIST). Incorporation of a patient with a duodenal GIST treated at our institution. GIST represents a spectrum of mesenchymal tumours from benign to malignant variants, which can arise from anywhere in the gastrointestinal tract. A central pathogenetic event recognised in most GISTs is KIT activation (a tyrosine kinase receptor) believed to be the result of oncogenic mutations. Imatinib mesylate, a humanized monoclonal antibody to KIT, has reduced the objective response rate, and the oncogenic tyrosine kinase, has revolutionised the treatment of metastatic GIST, and is discussed along with other treatment options. Traditionally the three key prognostic factors used in GIST have been mitotic rate, tumour size, and anatomic location. However, the unpredictable behaviour of GIST has led to the development of immunohistochemical differentiation markers including CD117 (detecting KIT protein). In addition genetic markers have been used as prognostic parameters, including KIT activating mutations, cytogenetic aberrations and telomere length.

Case report

A 53-year-old male farmer presented with a three-day history of anorexia, dysphagia, and obstruction. He was commenced on IV omeprazole and underwent gastroscopy that revealed a 13mm in diameter, with rare and rarely in the oesophagus, colon and rectum (5-15%) or duodenum (4%). There may be a greater incidence in men while others note no sex difference. The effect of gender on tumour behaviour is uncertain; some suggest it does not influence tumour behaviour, others associating male sex with markedly poorer prognosis and increased occurrence of metastases. At diagnosis about 40% of GIST are less than 1.5cm and asymptomatic. Of symptomatic GIST up to 86% are associated with GIT bleeding (acute or chronic). In decreasing frequency the presenting symptoms are abdominal mass, GIT bleeding, anorexia, dysphagia, and obstruction. The interstitial cells of Cajal (ICC), localised in the myenteric plexus believed to act as a gastrointestinal pacemaker cell governing peristalsis have been proposed as the cell of origin; supported by several immunohistochemical and ultrastructural similarities. Alternatively, GIST may originate from precursor stem cells that can differentiate toward either a smooth muscle or ICC phenotype, with KIT expression believed to be crucial in encouraging differentiation of these cells towards an ICC endpoint.

Tumours with ultrastructural characteristics of GI autonomic nerve tumours (GANT) are also GIST tumours, based on their KIT positivity and presence of essentially identical KIT activating mutations. GANT are believed part of the neoplastic spectrum of stromal tumours, displaying a higher degree of ICC differentiation. GANT should no longer be regarded as a separate entity.

GIST are thought to occur by mutations of the KIT gene, located on the long arm of chromosome 4 expressed in the cells of Cajal (ICC); ICC are immunostained by antibodies against KIT (CD117). KIT encodes a transmembrane tyrosine kinase receptor, consistently expressed in GIST.

Structurally, the KIT receptor can be divided into four principal domains: an extracellular domain; a transmembrane domain; a juxtamebrane domain; and a kinase domain separated into two sections. KIT gene mutations, irrespective of the domain for which they code, cause the receptor to be activated without its ligand (stem cell factor (SCF)), resulting in a continued stimulus for cell proliferation.

The KIT gene sequence has 21 exons, and in sporadic GIST, the majority (50-77%) of KIT mutations have been found in exon 11, encoding the juxtamembrane domain of the receptor. A germline mutation identified in familial and multiple GIST has also been identified in the juxtamembrane domain. GIST with exon 11 mutations were originally reported to be of a higher grade, or associated with poorer outcomes. Subsequently, exon 11 mutations were believed to hold prognostic value. Further, mutations have been described in exons 9 (extra cellular domain), 13 and 17 (the two kinase domains) with the majority of exon 9 mutations associated with highly malignant GIST. The overall, the estimated frequency of KIT mutations is between 21% and 92%. Failure to analyse the entire KIT coding sequence, and limitations encountered with some PCR assays used, as well as genetic differences between series populations may account for this variation. A number of GIST, although lacking KIT mutations demonstrate strong KIT activation. Presumably KIT mutations in these instances, have been detected by conventional screening methods, or, other non-mutational mechanisms may have led to KIT activation. Consequently it has been suggested oncogenic KIT activation occurs in the earliest stages with progression to more malignant behaviour determined by successive cytogenetic and molecular changes. The majority of GIST are the result of somatic mutation. Rare familial cases have been described, however predisposing factors are unknown. A link to EBV infection, association with Camely’s triad (paraganglioma, pulmonary chondroma, and leiomyoblastoma) of the stomach, a very rare syndrome mainly affecting young women has been postulated.

While mitotic count appears to be the most reliable indicator overall of GIST behaviour, with a high count correlating to malignant behaviour there are accounts detailing GIST, with low mitotic counts behaving aggressively. Mitotic count correlates poorly with the malignant potential of small bowel GIST. A major criticism of mitotic counts has been their subjectivity and poor reproducibility.

Grading systems have been devised with different cut-off points for the number of mitoses per 10 HPF. Mitotic count per 5 HPF is now recommended. Tumours with 0-1 mitoses per 10-50 HPF will not give rise to metastases, those with more than 5 mitoses per 50 HPF are considered malignant. A mitotic rate ≤ 5 mitoses per 50 HPF is commonly used as a limit for a tumour of predicted benign behaviour. However, this cut-off point fails to discriminate between benign and malignant small intestinal tumours.

Tumour size is suggested as more important than histology in predicting behaviour. Almost all small (<1cm) GIST are clinically benign; tumours more than 5cm are generally malignant; however no cut-off diameter predicts malignant behaviour with certainty. For duodenal tumours malignant behaviour is more likely in tumours greater than 4.5cm. Prognosis in GIST also varies with anatomic site, but the degree to which this relates to tumour size and/or histologic subtype is not clear. Purportedly, anatomic location is a prognostic factor independent of tumour size, mitotic rate and patient age. Most duodenal GIST occur in the second part of the duodenum, with duodenal and small intestinal GIST more likely to display malignant behaviour relative to gastric GIST. Small bowel tumours have the worst prognosis and oesophageal the best.

Histologically, GIST express a variety of cell types and growth patterns. Either of two cell types may predominate (spindle cells and epithelioid cells), however a mixed cell type may occur. Spindle cell-type form the majority comprising 70-80% of gastric tumours along with the majority of small intestinal GIST. Epithelioid lesions occur more often in the stomach. Lesions of mixed type may exhibit an admixed transition between epithelioid and spindle cells, however there may be some intermediate cytoligic appearance. There are some site-specific variations in morphology with spindle cell lesions of the small bowel having...
a tendency to contain skenoid fibres. Skenoid fibres formerly described in terms of risk assessment have no histogenetic significance. Correlation of histologic presentation and morphology alone remains unclear. No benefit has been reported from obtaining wide margins. Failure to obtain histologically tumour-free margins is associated with adverse outcomes.

Complete surgical resection is the primary therapy for GIST, but the required extent of resection, including regional lymph nodes and (in some instances) adjacent organs remains unclear. No benefit has been reported with malignant or high-risk tumours at the time of resection. Prognostic factors for local recurrence suggest a role for adjuvant therapy, however data is lacking in support of the use of either radiation or chemotherapy. Pierre et al found that patients receiving adjuvant therapy had worse outcomes. Radiotherapy is limited by potential toxicity to surrounding structures and is not standard post-operative therapy for GIST.

There is wide variation in five-year survival rates, 19-56% overall and 32-63% following complete resection. Most recurrences occur within five years of primary treatment, but can appear more than 10 years after treatment, indicating the need for long-term follow-up.

The difficulty in identifying reliable prognostic parameters only adds further confusion to the already controversial topic of gastrointestinal stromal tumours. Classifying GIST based on clinical presentation and morphology alone is difficult if not impossible, with the criteria for malignancy based on tumour size and mitotic count dependant on tumour location. Immunostaining for CD117 (although not entirely specific, but sensitive for GIST) along with a panel of antibodies, supplemented with careful morphologic examination assists the diagnostic process. The reported frequency and prognostic value of KIT activating mutations is uncertain, and in some instances contradictory. Results from molecular cytogenetic studies, suggesting a possible correlation between clonocapathologic behaviour and chromosomal aberrations, have significantly added the definition of new prognostic parameters. Cytogenetic aberrations appear to be secondary events to oncogenic mutations. The possibility of particular aberrations uniquely affecting signaling pathways, and thereby determining the pathway of GIST progression remains to be seen. Telomerase expression, exclusive to malignant GIST (although not always expressed) may occur as a late event. Its validation as a useful prognostic marker depends heavily on the recruitment of larger numbers of cases and extended clinical follow-up.

This review has highlighted the inconsistencies of current prognostic parameters used in GIST. A multiparametric approach is necessary, as no sole prognostic indicator has yet been determined reliable. The true test of any chosen parameter is one that can predict outcome on an individual case basis.

Regional lymph node dissection is of unproven value.

Metastases occur in more than 50% of patients diagnosed with malignant or high-risk tumours at the time of resection. Prognostic factors for local recurrence suggest a role for adjuvant therapy, however data is lacking in support of the use of either radiation or chemotherapy. Pierre et al found that patients receiving adjuvant therapy had worse outcomes. Radiotherapy is limited by potential toxicity to surrounding structures and is not standard post-operative therapy for GIST.
It is a pleasure to record my thanks to the Medical Oncology Group, the award selection committee and Pierre Fabre for this unexpected award.

Leonardo da Vinci was one of the foremost contributors to that renaissance of human intelligence which we know, as the Renaissance, and among the finest exemplars of what we now call Renaissance Man. This term was coined because Leonardo and many of his learned contemporaries aspired to knowledge across what is today an astonishingly wide canvas – painting, engineering, architecture and anatomy. The term is still used as one of approbation.

Robert Heinlein offers a more modern definition along similar lines

“A human being should be able to change a diaper, plan an invasion, butcher a hog, cren a ship, design a building, write a sonnet, balance accounts, arrange a debate, superintend the building of a bridge or the construction of a computer, make aoutline a computer, cook a tasty meal, fight efficiently, die gallantly. Specialization is for insects.”

Lazarus Long in Time Enough For Love by Robert Heinlein

That’s 21 skills. I’m not sure about you, but I barely pass. My career has, however, been one of change, and of interaction across boundaries.

In this presentation I will review some of those collaborations, give credit to at least some of those with whom I have worked, and suggest to you that, at least in terms of research, reaching across the barriers of your own discipline can be creative and successful.

After completing the Membership, the first phase of my career was at the Walter and Eliza Hall Institute of Medical Research (WEHI), where I was immensely privileged to interact with a large number of extraordinarily talented people. The interface of relevance here was between clinical and laboratory medicine.

Sir Macfarlane Burnet – Mac – had won the Nobel Prize in 1960. By the time I was at WEHI he had retired as director but was a regular at seminars and at weekly case presentations in the Clinical Research Unit. He was sharp as a tack, and an consummate communicator, and I could not have asked for a better mentor.

Ian Mackay, head of the Clinical Research Unit, was my immediate boss. Ian was a rigorous and demanding bedside clinician, and carried both characteristics equally through into clinical and laboratory science. His own research and major contributions were in autoimmunity, but he fostered inter-disciplinary cooperation among his trainees and staff. Indeed this is the only phase of my career at which I was responsible for clinical patient care, laboratory experiments and simultaneously for reporting cardiological and liver biopsies.

In many ways pride of place should go to Don Metcalf. Don’s was the first laboratory I visited, in 1965. Typically, Don was at a microscope. Barely looking up, he said “Come here, Coates - have a look at this”. This was a colony of haemopoietic cells growing in a dish of agar, a discovery he and Ray Bradley had made earlier in the year. “This will take up the next five years of my life.” He was of course both right and wildly presagistic.

My other contact from this period would probably be surprised to find himself listed. Through Ian Mackay I got to know Nigel Gray, then director of the Anti-Cancer Council of Victoria (ACCV), and to understand something of the possible contribution of a public health approach to disease control. It’s perhaps a major reason for my latest career move.

The next major influence was the late Paul Carbone. I describe myself as a reformed immunologist. I had originally been attracted by the hope – common in the 1960s – that immunology would provide the answer to cancer. When it later appeared to me that such a hope was unlikely to be realised in my professional lifetime, I opted to continue interest in cancer. In the mid 1970s there were no training programs in Australia. I was presenting some research on CEA at ACCV one day when Paul Carbone was visiting, and as I sat down he offered me a job. I knew of Paul’s work at the National Cancer Institute, but I mentioned that he was about to move to Madison, Wisconsin. I accepted and went home to look up where exactly Madison was. Paul was a superb clinician, who revised my prejudices about American bedside medicine. He also provided training and leadership in what we now call translational research. He was generous in involving his fellows in the clinical trials research of the Eastern Cooperative Oncology Group, so that I returned determined to apply some of the same principles to the conduct of clinical research in Australia.

While at WEHI I had developed an interest in melanoma – most immunologists with a cancer interest do – and in Wisconsin, I as an Australian, was the automatic choice to see melanoma patients. Returning to Sydney I had the opportunity to join Gerry Milton in the Sydney Melanoma Unit (SMU). I remain a member of that unit, under the successive leadership of Bill McCarthy and John Thompson. SMU has a long and successful record of collaboration both in international clinical trials, and with the immunologists, pathologists, psychologists, epidemiologists and even the medical oncologists who contribute to the management of melanoma. I was privileged to serve as its research director from 1985 to 1998.

My interest in clinical trials led me to join the relevant COSA committee, chaired by John Colebatch, one of the real pioneers of clinical trials in Australia. The experience of co-authoring a book with John (and Richard Fisher) was truly memorable. We put quotes at the head of each chapter. I can’t be certain, but it seems very likely that John found this one.

“Reading maketh a full man, conference a ready man and writing an exact man.”

Francis Bacon

Certainly exactly in written expression was one of the disciplines to which (and he of necessity his co-authors) rigidly adhered.

In 1980 I joined the staff of the Sydney Branch of the Ludwig Institute for Cancer Research, under the direction of one of my distinguished predecessors in this award, Martin Tattersall. Martin has trained more medical oncologists than anyone else in Australia. It’s a pleasure to acknowledge his selfless support and encouragement. Martin has many of the characteristics of modern Renaissance Man. I’m not sure how many of Heinlein’s 21 skills he can claim, but I’m sure he will be disappointed that his well-known ability to walk on water was not on Heinlein’s list. Perhaps canning a sailboard counts.

I mentioned that I came back from the US keen to set up clinical trials in Australia. One of the first colleagues I visited was John Forbes, who had also been overseas – in Cardiff with Michael Baum – and had arrived back transformed from an HLA immunologist to a clinical trials researcher. The collaboration proved fruitful, and I also thank and give credit to my other fellow directors of the first Board of the Australian and New Zealand Breast Cancer Trials Group – John Collins, John, Simes Vernon Harvey Michael Byrne and Ray Snyder.

Internationally, my work with the International Breast Cancer Study Group has been a real joy, especially because of my close working relationship with Aron Goldhirsch and Rich Gelber.

Another cross-disciplinary collaboration has been into psycho-oncology and quality of life research. Like many things, this arose by chance. On arriving at Ludwig I was presented with a mass of quality of life scores collected by a departed colleague and asked to apply my statistical skills make some sense of it. That led to the first of a series of papers christened (by Martin Tattersall) “On the receiving end”. It also led to the inclusion of quality of life endpoints into the then new advanced breast cancer trial ANZ 8101, which I duly presented at ASCO in Los Angeles in 1985. While there I learned from Aron Goldhirsch that a couple of his Swiss colleagues were designing quality of life measures for the third generation adjuvant trials (BCSG VI and VII). I was keen to incorporate our experience in the completed advanced trial, so I arranged to fly from Los Angeles to Switzerland to meet with Christoph Huynen and Juerg Bernhard. I suppose the reason it was easy to get a seat at short notice was that this was the weekend of the nuclear accident at Chernobyl, and practically no-one was flying east across the Atlantic. I’m happy to say that this collaboration too persists, and continues to be productive.

Clinical trials research requires the input and dedication of large numbers of colleagues – it’s a pleasure to acknowledge the essential input of many colleagues in the International Breast Cancer Study Group and the Australian-New Zealand Breast Cancer Trials Group.

The final constructive collaboration I wish to acknowledge is with Lawrie Wright. Lawrie was appointed as executive director of the then Australian Cancer Society and the Clinical Oncological Society of Australia in 1978, just before I joined COSA. I count myself extremely fortunate to have become CEO of The Cancer Council Australia at a time when I could draw on his expertise, wisdom and guidance.

In summary, the lessons I would propose from this experience, which may be of value to younger colleagues are:

Master your own discipline (collaboration is not a substitute for competence); but Specialisation need not be limiting (if you’re not one of Heinlein’s insects).

Look for interactions with other disciplines: pick them according to opportunity, according to your own interests, and perhaps the examples I have cited. Such collaborations can be constructive, productive, satisfying and fun.

* This is an edited version of the lecture presented in Canberra on 13 August 2003.
In 1996, a small group of cancer researchers, under the guidance of Professor Joe Sambrook, developed an idea to create an Australian network that supported research into breast cancer. Their notion was to capitalise on a relatively new phenomenon in cancer medicine – Familial Cancer Clinics (FCCs) – which were being established throughout Australia. High-risk families were readily recruited into the FCCs, thereby providing a wonderful resource for cancer researchers with interests ranging from psychosocial medicine to the molecular biology of tumour formation. The support of the Kathleen Cunningham Foundation, kConFab rapidly emerged as a national consortium for research into familial breast cancer. Pivotal to its success was the rigorous collection of epidemiologic data and biospecimens, as well as a philosophy of open membership and access to data. The scientific meeting, held at Couran Cove on South Stradbroke Island in Queensland, was the sixth national meeting organised by kConFab. In contrast to previous years, this year the meeting was restricted to members of Family Cancer Clinics of Australia and New Zealand.

The first day and a half of the meeting was restricted to members of Family Cancer Clinics. This provided an invaluable opportunity to discuss common areas of interest and need. Both pragmatic and esoteric issues were raised and discussed. One certain outcome will be enhanced dialogue among the Australian cancer genetics community and with new measures in place to improve clinical practice and to establish common national protocols. Topics covered included the organisational strategies in place for each FCC, imaging transfer protocols aimed at improving the selection of individuals for BRCA1 and BRCA2 and HNPCC gene mutation detection, disclosure of genetic information, and psychosocial issues in familial cancer.

The remaining two and a half day conference attracted more than 100 registrants from a variety of backgrounds (data managers, genetic counsellors, research nurses, epidemiologists, clinicians, surgeons, pathologists and laboratory scientists), all linked by a common interest in hereditary breast/ovarian cancer.

Australian Behavioural Research in Cancer

This is a regular feature in Cancer Forum describing behavioural applications in cancer care. Australia has five behavioural research centres: the Centre for Behavioural Research in Cancer (CBRC) of The Cancer Council Victoria, the Centre for Behavioural Research in Cancer (CBRC) at the Menzies School of Health Research, the Centre for Cancer Control Research (CCCR) of The Cancer Council South Australia, the Centre for Health and Research and Psychosocial Oncology (CHEP) of The Cancer Council New South Wales and the Cancer Prevocare Research Centre (CPRC) of the University of Queensland.

This report has been compiled by C Swart (CBRC).

New results

n The Centre for Behavioural Research in Cancer (CBRC), VIC

Findings from the Victorian Smoking and Health Population Survey 2002

Tessa Letcher, Carly Black, Irene Bobevski, John Lipscombe and Melanie Wakefield have been working on analysis and reporting of data from the Victorian Smoking and Health Population Survey 2002. A report on public opinion about smokefree policies in bars, nightclubs and gambling venues between 2000 and 2002 was released in July 2003. The data indicate strong support from the Victorian public for bans on smoking in bars, nightclubs and gambling venues, with approval for bans in bars increasing significantly over time. There was a high level of public support for the most recent legislative amendments restricting smoking in Victorian alcohol licensed venues.

Data from the population survey have also been used to generate reports on change in smoking prevalence and consumption among Victorian adults, as well as smoking behaviours and patronage of social venues associated with the introduction of smokefree policies. Visit the new CBRC website (www.cancervic.org.au/cbrc) to download copies of these reports.

Evaluation of the National Tobacco Campaign

CBRC has been very much involved with the evaluation of the National Tobacco Campaign since its inception. The September issue of Tobacco Control (2003; 12 [Suppl III]) contains a supplement devoted to the National Tobacco Campaign and features a number of papers by CBRC staff about various aspects of the campaign, including studies of the effects of the campaign on adult smokers and adolescents, and trends in Australian smoking prevalence. The supplement is online at http://tc.bmjournals.com/content/vol12/suppl_3/.

n The Centre for Behavioural Research in Cancer Control (CBRC), VIC

Adverts for Philip Morris and Tobacco Monte Carlo MTV youth smoking prevention advertising

In 2001, British American Tobacco, Japan Tobacco International and Philip Morris International jointly funded an international advertising campaign featuring six advertisements broadcast in 38 countries. CBRC conducted audits on three of the advertisements screened in cinemas in Australia to test the impact on young people in comparison to data gathered from the Smarter Than Satyriasis campaign (Bus-Stop and Fashion Soaps) and an anti-smoking advertising evoking the emotion of disgust (Disrupt). A convenience sample of 257 youths aged 14 to 18 years was recruited with half being regular smokers and 40% being non-smokers. Among smokers aged 14 to 15 years, the tobacco industry advertisements performed better than the Fashion Soaps advertisements for intentions in not wanting to smoke in the future, but not as well as the Disrupt and Bus Stop advertisements. This under-perform of the tobacco industry advertisements compared with the Disrupt and Bus Stop advertisements was consistent also among non-smokers. Among the 16 to 18 age year group, for both smokers and non-smokers, the Disrupt advertisements performed far better than the tobacco industry advertisements in not wanting to smoke in the future. Similarly, current smokers who were shown the Disrupt advertisements were far more likely to think about quitting than those shown the tobacco industry advertisements.

Secondary student survey: Sun behaviour results

CBRCC was commissioned by the Foundation for Research to compare the findings of a 2002 student survey with those of similar surveys conducted in 1999, 1996 and 1993 that assessed attitudes and behavioural practices with respect to sun protection. The entire sample consisted of 3,545 students aged 12 to 17 years. Results indicate a high awareness that most skin cancers are caused by ultraviolet radiation from sun exposure (92%). However self-reported sunburn rates were high (77%) and most respondents did not spend the majority of their time inside on sunny days in summer between the hours of 10am and 2pm (76%). The data suggested that the adoption of a number of sun protective behaviours declined as the summer progressed. The majority (64%) of respondents reported using sun protection and the majority of respondents (63%) were high (77%) and most respondents did not spend the majority of their time inside on sunny days in summer between the hours of 10am and 2pm (76%). The data suggested that the adoption of a number of sun protective behaviours declined as the summer progressed. The majority (64%) of respondents reported using sun protection and the majority of respondents (63%) were suntans remained highly favourable. The previously increasing trend in the proportion of respondents who did not get a tan appeared to be slowed, or, in the case of females aged 15 to 17 years, declined.

n The Centre for Cancer Control Research (CCCR) and the Tobacco Control Research and Evaluation Program (TCRE), SA

The relationship of emotional support and survival (further research from the Canberra Cancer Quality of Life Project)

Survival was analysed using the Kaplan-Meier product-limit estimate, and multivariable Cox proportional hazards regression, from entry to any cancer for all patients, or 31 March 2003, whichever came first. Numbers of confidants (with whom feelings were shared at time of study entry) were predictive of survival duration. The regression analysis indicated that compared with patients reporting 2-3 confidants, the relative risk of a shorter survival (95% confidence limits) was 0.44 (0.25, 0.79) for those with 0-1 confidants and 0.60 (0.40, 0.89) for those with four or more confidants. Shorter survivors shared their feelings more with family members than longer survivors. Conversely, longer survivors shared their feelings more with friends than shorter survivors. These relationships did not hold at 12 weeks from study entry. At that time, longer survivors were more likely to share their feelings with a doctor than shorter survivors. The relationship between emotional support and survival duration was not linear and appeared to be more complex than reported previously for people with heart disease and early breast cancer.

Monograph series

The Centre for Cancer Control Research has completed the seventh monograph in The Cancer Council Monograph series, South Australian Cancer Statistics. This one, entitled Cancers of...
The prostate, tests and urological organs, was released in early October. As for previous monographs, its intended audience is medical practitioners, science teachers, interested members of the public, and tertiary students enrolled in health and allied disciplines. The cancers addressed in this seventh monograph are an important group of malignancies, accounting for about a fifth of all cancer deaths in South Australian males and about 50 deaths per annum in South Australian females. The monograph shows how these cancers affect the population, time trends in incidence, local trends, and risk factors are placed in an international context. Risk factors are described, together with the opportunities that exist for cancer prevention through multiple avenues, including smoking cessation, adoption of diets rich in fruit and vegetables, weight control, and maintaining good industrial hygiene. Attention is given to the status of knowledge and debate about prostate specific antigen screening of asymptomatic men as a public health measure.

n The Centre for Health Research and Psycho-oncology (CHeRP), NSW

To refer or not to refer: Medical practitioners’ perceptions of palliative care

While the use of palliative care services has been shown to improve outcomes including symptom control, care at site of choice, reduced costs and more needs of patients/family being met, recent data suggests that nearly 40% of advanced cancer patients in Australia are not referred to palliative care prior to death. There is little information available as to why this occurs.

Claire Johnson, a PhD candidate at the Centre for Health Research and Psycho-oncology undertook qualitative research to improve our understanding of medical practitioners’ perceptions of palliative care and to gain an understanding of triggers used by medical practitioners to initiate the referral process, under the supervision of Associate Professor Alf Gigns and Dr Chris Paul.

The study involved a semi-structured telephone interview with a random sample of medical practitioners from around Australia. Information was sought regarding the medical practitioners’ perceptions and understandings of palliative care and the referral of patients to specialist palliative care services. The professional age and income were then compared with the identified principles of optimal palliative care practices, including the multidisciplinary approach to care and the provision of care across the physical, psychosocial, spiritual and cultural domains.

Results indicate that doctors in Australia are familiar with and comfortable with understanding and management with palliative care, particularly in the physical domain; and that doctors are cognisant in identifying physical symptoms as an appropriate trigger to initiate palliative care. The perceptions of doctors in moderate and high intensity increases in physical activity among older Australian adults, and should be evaluated across a more extended time period.

Research in the pipeline

n CBCR

The first national sun protection survey

Planning is well under way for the first national survey of Australian’s sun-related activities on summer weekends; assessing people’s sun protective behaviour, sunburn incidence, and related knowledge and attitudes. This collaborative project aims to provide national data to support sun safety and sun protection control programs and campaigns at the state and national levels.

Research, evaluation and program staff from most states and territories have participated in a number of planning meetings to further develop study design, considering a range of issues on content, sampling and funding. The same methodology and survey design was used as that of the previous solaria compliance study research conducted by the Monash University in 1996. The survey was conducted on a total of eight Monday evenings from the end of November 2003 until January 2004, achieving a representative sample of telephone interviews of 4,000 Australians aged 14 to 69 years. Contact Zoe Dobbins for further information.

n The Gemini project

Semi-structured interviews have been undertaken with 28 identical twins who were discordant for smoking status during their teen and early adult years. The twin sets share the same genes, and lived in the same environment for much of this period. The current study has been designed to explore how the twins account for their decisions and behaviours around their discordant smoking status. This information is being used to understand how the twins account for their decisions and behaviours around their discordant smoking status.

n CBCR

Solairea compliance study

The association between exposure to ultraviolet radiation and skin cancer is well known. The number of establishments with artificial UV (tanning) has increased substantially over the past five years, with an associated increase in patronage. A new Australian standard has been developed for the operation of solaria emphasizing the need for clients to be fully informed of the potential carcinogenic risks of using solaria, and recommends ways to make the practice of artificial tanning less dangerous.

Investigators Dr Chris Paul and Associate Professor Alf Gigns from the Centre for Health Research & Psycho-oncology and Ms Nicole Klein and Ms Tracey Taylor from The Cancer Council of NSW, are conducting a population study to assess levels of compliance among solaria operators with these aspects of the standard. The study involves interviewing among higher risk clients. This will be achieved through the use of simulated customer visits to sample of solaria centres in Sydney, the Central Coast and Newcastle.

The simulated customers will use one of two scenarios which have the effect of determining the type of customer. Research Council now provides a strong cancer prevention nexus for the Centre for Health Research and Psycho-oncology.

n CBRC

Get mobile

This collaborative study with Stanford and Deakin Universities compared the effects on physical activity of a print-based intervention (‘print’, and a print plus phone-mediated intervention) and who identified themselves as physically inactive (18 men, 48 women) aged 45-78 years were recruited through advertisements and word-of-mouth at two sites (Melbourne and Brisbane), and randomised to the print or telephone intervention group. Participants in both groups attended an initial briefing session, and over the 12 week intervention period received an educational newsletter, two motivational prize incentives, and the use of a pedometer. The telephone intervention group also received motivational telephone support via six telephone calls. Self-reported physical activity data were collected using the CHAMPS measure at baseline, 12 and 16 weeks. Results showed significant increases of approximately two hour-weekly in participants participation in moderate and higher-intensity physical activity, and in walking, in both intervention groups at 12 weeks, with increases maintained at 16 weeks. Participants in the telephone group maintained slightly higher levels of walking (by approximately 30 minutes/week) than those in the print group at 16 weeks. Print and telephone-mediated interventions for promoting physical activity can reach large numbers of people at a relatively low cost. These interventions show potential for promoting initial increases in physical activity among older Australian adults, and should be evaluated across a more extended time period.
The Centre for Health Research & Psycho-oncology (CHeRP) recently celebrated its 15th birthday. The Cancer Council NSW initially established CHeRP in 1988 as the Cancer Education Research Program. CHeRP has grown from a few staff in 1988 to 25 staff members in 2003, including research personnel and postgraduate students.

Dr Chris Paul and A/Prof Aflal Girgis have been awarded $23,500 from the NSW Department of Health to undertake research into solaria operators’ compliance with the Australian standard.

Dr Raoul Walsh also gave two presentations at the 12th World Conference on Tobacco or Health. Over-the-counter nicotine replacement therapy: Assessing evidence for its population impact and Qualitative studies of adolescent smoking: Review of methodologies and findings.


Neville Owen and Lane McDermott presented at the PHAA Conference in Brisbane in October 2003. Their presentations were Measuring environmental attributes related to walking and The role of life-stage transitions in smoking behaviour among young women.

The Centre is pleased to welcome three new staff members: Dr Ester Cerin joined the Centre in September as a research fellow, and two new project officers, Lorinne de Toit and Phoebe Kearny, joined the Centre in October.

Thanks to Anne Gibbs (CBRC), Owen Carter (CBRCC), Kerri Beckmann (CCCR & TCRE), Narelle Mills (CHeRP) and Cathy Swart (CPRC) for their contributions.

Dr Raoul Walsh and A/Prof Aflal Girgis have been awarded $23,500 from the NSW Department of Health to undertake research into solaria operators’ compliance with the Australian standard.

In response to identification, by the National Cancer Control Initiative, of a need to keep GPs informed regarding cancer control, GPIs in South Eastern Sydney have been provided with concise information on particular topics since October 1999. This end has been achieved through publication of Cancer Control Bulletins at a rate of about eight per year and their distribution with the active support of relevant GP divisions. In September 2000 the program expanded to the internet, where 32 Bulletins are currently available (www.sesahs.nsw.gov.au/publichealth/cancercontrol).

Being web-based, the bulletins are accessible to the wider community. However monitoring of the pattern of ‘hits’ following publicity in GP divisional newsletters has indicated that ‘local’ GPs are the foremost users. Thus, following such publicity, the number of hits to the website increased by 160%, remaining at this level for three months, and decreasing to pre-publicity numbers in the fourth month. The total number of ‘hits’ to the Bulletin website for the 15-month period was 32,612 (figure one).

Since monitoring of the website began, the Bulletin on testicular cancer has been, by far, the most downloaded file from the whole South Eastern Sydney Area Health Service website. The ‘hits’ on the testicular cancer Bulletin are not explicable with reference to it being a recent publication, nor has this Bulletin been subject to any additional publicity.

The second most commonly accessed Bulletin is the issue concerning new chemotherapy drugs, closely followed by the issue on the latest on skin cancer and cancer in children.

A simple explanation as to why testicular cancer should be consistently the focus of most enquiries is not apparent. It is evident, however, that there is a need for information on this subject by GPs. Such need does not appear to be correlated with enquiries from the general public, since analysis by The Cancer Council NSW of queries to the national helpline showed that testicular cancer was the 15th most called about cancer. Breast, skin, prostate, colon and lung were the most commonly asked about cancer sites. We are aware of the GP education seminar “On The Ball” (www.uco.org.au) hosted by The Cancer Council NSW, this doesn’t account for the pattern of enquiry.

K-A Ressler and BW Stewart Cancer Control Program, South Eastern Sydney Public Health Unit, Randwick, NSW

Figure 1: Total number of hits to Cancer Control Bulletin website

Bulletin name Bulletins appear in order of publication: issue 1-30
Patenting human genes – Australian Law Reform Commission inquiry

Publicly-funded genetic testing and not-for-profit genetic research must be allowed to continue without penalty or undue restriction.

This is the key message of a submission by The Cancer Council Australia, on behalf of its member organisations, to the Australian Law Reform Commission’s inquiry into Gene Patenting and Human Health.

The Cancer Council is concerned about a patenting system that allows exclusive rights to a naturally-occurring substance if such rights are to the detriment of public health, particularly by reducing access to or increasing the cost to the community of genetic testing, permitting testing without appropriate genetic counselling; and/or hindering research which would improve genetic testing methods and treatment.

The submission recommends several changes to the current patenting system to compensate for the unusual nature of gene patents and consequently the potential impact these patents may have on the wider healthcare system and health research.

Protecting the advocacy work of the Cancer Council

The Cancer Council Australia has made a submission to the Board of Taxation, expressing concern about the potential impact of the draft Charities Bill 2003.

Cancer Councils believe they have a responsibility to advocate for improvements in government policy, programs or legislation to reduce the incidence and impact of cancer. Their goal is to ensure that issues related to cancer care and control get the attention, legislative action and funding they deserve.

While the draft bill broadens the legislative definition of a charity and resolves some ambiguities within current common law interpretations, the Cancer Councils believe that several provisions are badly drafted and may hinder rather than enhance the work of charities in Australia.

The submission seeks to ensure charities are expressly permitted to engage in advocacy in pursuit of charitable purposes and on behalf of those they seek to benefit.

The Cancer Council’s submission can be read online at www.cancer.org.au.

National Skin Cancer Action Week

The dangers of tanning is a theme of The Cancer Council’s National Skin Cancer Action Week this year (16-22 November).

Recent Cancer Council research shows that while around 50% of Australian women over the age of 18 say they aren’t interested in being tanned, 25% still want a light tan, 21% a medium tan, and 4% a dark tan.

It seems many Australians wrongly believe tanning without burning and using a sunscreen is safe. The Cancer Council and the Australasian College of Dermatologists, a supporter of the Action Week, advise any form of tanning which involves intentional exposure to ultraviolet radiation carries a risk of skin damage, including premature ageing and skin cancer.

Parliamentary briefing

“Cancer prevention: key to reducing disease and health expenditures” was the topic of the latest meeting of The Cancer Council Australia’s Parliamentary Cancer Information meeting.

Held at Parliament House in Canberra on 5 November, speakers included Professor Alan Lopez, head of the School of Population Health at the University of Queensland and former advisor to the WHO Director-General; Ms Christine Stone, Senior Epidemiologist – Cancer and Genetics at the Victorian Department of Human Services, and Ms Dorothy Reading, Chair of The Cancer Council’s Public Health Committee.

The content of the presentation is outlined in the Cancer Update newsletter, which can be read online at www.cancer.org.au/publications.

Awards

Professor Alan Coates AM, CEO of The Cancer Council Australia, is this year’s winner of the prestigious Medical Oncology Group of Australia/Pierre Fabre Cancer Achievement Award.

The Chairman of the MOGA Awards Committee, Dr Michael Jefford, said the award was in recognition of Professor Coates’ outstanding contributions to cancer knowledge and control through his research, practice, teaching and service.

“He is internationally recognised as a leading expert in breast cancer, melanoma, cancer trials and quality of life research, and he has been a role model and mentor for many Australian oncologists,” Dr Jefford said.

The prize was formally awarded to Professor Coates at the Annual Scientific Meeting of the Medical Oncology Group of Australia in Canberra on 13 August.

Professor Coates’ address is published in this issue of Cancer Forum.

Professor Lester Peters AM has been awarded a prestigious Gold Medal by the American Society for Therapeutic Radiology and Oncology (ASTRO).

Professor Peters, a past president of ASTRO who is now Head of Radiation Oncology at the Peter MacCallum Cancer Institute in Melbourne, is one of only three to receive the Society’s highest honour this year. He was formally presented with his Gold Medal at ASTRO’s annual meeting at Salt Lake City in the US this week.

Professor Peters’ award is in recognition of his excellence in pre-clinical and clinical research, his ability to translate research into clinical practice, and his many contributions to improve the standard of practice in radiation oncology.

He is a former chair, and continuing member, of The Cancer Council Australia’s Medical and Scientific Committee.

Oncologists Forum

The first online medical forum for oncologists and haematologists in Australia and New Zealand will be held in February next year.

A faculty of leading oncology specialists has been established to help implement the new 20:20 Oncologist Forum.

The forum will include presentations from both international and Australian specialists and allow registrants to discuss the local implications of emerging issues and trends, via the Internet.

Dr Michael Green, Medical Oncologist at The Royal Melbourne Hospital and Chair of the 20:20 Oncologist Forum said the forum provides an innovative arena for the oncologists and haematologists to “debate, learn and share opinions on current issues and research”.

A survey conducted with 25 Australian oncologists revealed the need for a unique, interesting setting that could facilitate scientific discussion rather than just provide an educational agenda.

“The forum has been developed in response to these results, and it is the objective of the faculty to ensure the content and structure of the forum is not just educational but encourages debate,” said Dr Green.

New COSA Executive Officer

Ms Margaret McIannett has been appointed Executive Officer of the Clinical Oncological Society of Australia (COSA). Ms McIannett also replaces Ms Lawrie Wright as The Cancer Council Australia’s Administrative Officer.

Margaret has been involved in cancer care for the past 24 years. She was previously an oncology nurse consultant with the Sydney Melanoma Unit at the Royal Prince Alfred Hospital, and more recently working at the Sydney University’s Medical Psychology Research Unit, where she was involved in developing treatment decision aids for cancer patients.

The first forum is to be held in late February 2004 and will be designed to fit into the RACP CME guidelines.

For further information or to register interest, email oncology@virtuebroadcasting.com.au.

Cervical cancer screening conference

The Cancer Council Australia will be hosting a one-day conference on cervical cancer screening as part of an international meeting to be held in Melbourne next April.

The cervical cancer screening stream of the 18th World Conference on Health Promotion and Health Education will be held on Thursday, 29 April at the Melbourne Exhibition and Convention Centre.

The program will provide opportunities to share knowledge and showcase existing good practice in cervical screening programs, highlight future opportunities and new technologies, raise awareness of access and equity issues of disadvantaged women, and allow for the development of networks to facilitate and maintain collaborative relationships.

It is expected that the focus of the cervical screening conference stream will offer the opportunity for presentations on current issues including new technologies in cervical screening, the human papilloma virus (including a vaccine and testing), and effective recruitment measures for reaching unscreened women.


The Cancer Council Australia gratefully acknowledges the Victorian and Australian governments for their assistance through funding support of the cervical cancer screening stream.

UICC grants and fellowships

The International Union Against Cancer (UICC) is offering research grants and international fellowships that provide opportunities for continuous professional education in a variety of disciplines through long, medium and short-term work and training periods abroad. They are intended for qualified investigators, clinicians and nurses who are actively engaged in cancer research, clinical oncology or oncology nursing or are educators in these fields.

UICC Yamagawa-Yoshida Grants provide support for establishing or conducting three month long bilateral (between any two countries) cancer research projects to develop experimental research methods and techniques. Selection will take place in April 2004 and, if successful, projects can start within a month. There are six to eight grants available, valued at $10,000. Applications close 1 January 2004. http:// fellow.uicc.org/fellow_support/grants.htm

Beginning investigators fellowships are funded by the American Cancer Society for beginning investigators and clinicians in the early stages of their career. There are six to eight fellowships available, valued at $40,000 for 12 months. Applications close 1 December 2003. http://fellow.uicc.org/fellow_support/beginning.htm

Translational Research fellowships are aimed at improving the translation of basic, experimental, and applied research insights. There are two to three fellowships available, valued at $35,000 for 12 months. Applications close 1 December 2003. http://fellow.uicc.org/fellow_support/trans.htm

Wishlist Christmas hampers

Wishlist.com.au has developed a range of gourmet Christmas hampers and raise awareness of access and equity issues of disadvantaged women, and allow for the development of networks to facilitate and maintain collaborative relationships.

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Wishlist Christmas hampers

Wishlist.com.au has developed a range of gourmet Christmas hampers as a fundraising initiative to support The Cancer
CEO profiles
In each edition of Cancer Forum this year we have profiled the CEOs of The Cancer Councils.

The Cancer Council ACT
Joan Bartlett
Ms Joan Bartlett commenced as Executive Officer of The Cancer Council ACT in January 1999.

Ms Bartlett has worked in the not-for-profit sector since early 1991, working first for an organisation offering Employment Assistance Programs (EAPs), followed by four years as the Education Director of Family Planning ACT, and then three years as the Executive Director of Lifeline Canberra.

After arriving in Canberra from Queensland in June 1999, Ms Bartlett worked briefly in the training and development areas of three Commonwealth Government Departments: (the then) Department of Finance, (the then) Department of Health and the Department of Foreign Affairs.

At the beginning of her working life, Ms Bartlett had begun at Sydney Hospital as a student nurse in 1966, completing only one year before taking up teacher training.

In 1971, after teaching in country NSW and Sydney she went overseas for four years working in hospitality (and occasionally crewing yachts) for a living.

After ten years working as a student and as a stay-at-home parent, Ms Bartlett returned to work in 1985 as a Special Education teacher at the Barrett Adolescent Centre, near Brisbane, working for two years with adolescents with psychiatric illnesses. Following this experience Ms Bartlett was employed as a guidance officer (counsellor) in high schools around Brisbane for three years.

Ms Bartlett is currently studying for an MBA majoring in Association Management from the University of New England. She holds a Masters of Educational Studies degree (Guidance and Counselling) and Bachelor of Educational Studies degree (both from the University of Queensland), a Graduate Diploma in Special Education from Charles Sturt University as well as a Diploma of Teaching and Teaching Certificate from (the now) University of Wollongong.

Her early attraction to things medical, plus training and experience in education and counselling and her current interest in governance and association management, have each contributed to her finding managing The Cancer Council ACT a most fulfilling experience.

The Cancer Council Tasmania
Lawson Ride
Mr Lawson Ride was appointed founding Executive Director of The Cancer Council Tasmania in April 1995 and has been responsible for the growth and development of the Council and its programs to date.

Prior to his appointment he enjoyed a long and varied career in a range of agencies within the Tasmanian public service. During eight years in the Department of Health Mr Ride held a range of senior management positions in health promotion, public affairs and the secretariat, before his final appointment as Senior Private Secretary to the Minister for Health and Community Services.

2002 YEAR BOOK OF ONCOLOGY
PJ Loehler et al (eds)
Published by Mosby (2002)
RPP: A$245.56

The Year Book of Oncology used to be one of my favourites. The 2002 edition is no exception, but it’s losing its impact.

The book is 437 pages, divided into 19 sections ranging from epidemiology, ethics, the usual solid tumours and hematology to paediatric oncology. Seven reviewers/editors surveyed approximately 500 journals, and from these they selected what they felt were important articles from 74 separate journals – all English-speaking – to be abstracted in the 2002 edition. Each article is presented in abstract form. Some are extended with the inclusion of tables, graphs and radiology.

Each article is put in perspective by one of the editors, and in some cases commented on by multiple editors. Some papers are grouped together in a theme and then a comment is made pertaining to all of them. The papers chosen vary from observational pieces to large randomised trials. I have to admit that some of the articles thought to be important by an editor would not attract my attention. All of the quoted papers have been published in 2000 or 2001, and herein lies the weakness of these books.

With oncology literature now so vast and the number of journals ever-increasing, even the best motivated and studious oncologist has difficulty in keeping up-to-date, if not in their sub-specialty, certainly in the area of general oncology. These year books are aimed at general oncologists who don’t have the time to read articles that fall outside their area of expertise, published in more obscure, difficult-to-access journals, that don’t make it to the high impact ones. They are very useful in filling gaps, and allow exposure to related work that may have bearing on their specialty.

The main limitation of the year books is that they are one to two years behind, and the reader is already aware of many of the quoted articles and, in some cases, the impact on their clinical practice. Alternate, faster information can be obtained through various electronic search engines that are making the usefulness of these series redundant. Nonetheless there were many articles that I would not have read otherwise.

How to improve these books? More timely publication, say within three months of the subsequent year, would make a year book more useful. This could occur by increasing the number of editors and sharing the workload.

The 2002 Year Book of Oncology is a good book to read from cover to cover to maintain a broad knowledge of recent literature in oncology, but not the source to go to, to look up a specific topic, to answer a clinical question, or to keep current in your interest. Oncology units and hospital libraries should have a copy. Some oncologists may enjoy having their own copy if they have $246 to spare; most won’t.

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ANTICANCER DRUG DEVELOPMENT
B Baguley and D Kerr (eds)
Published by Academic Press (2002)
RPP: A$221.00

This ambitious text consists of 20 chapters with contributions from a total of 59 eminent scientists from seven countries and provides a well-structured and impressively broad overview of all aspects of anticancer drug development. Considering the breadth of topics covered, there is surprisingly little overlap of content. The editors, Professor Bruce Baguley (Co-director, Auckland Cancer Society Research Centre, University of Auckland, NZ) and Professor David Kerr (Head of Clinical Pharmacology, Institute for Cancer Medicine, University of Oxford, UK) have a wealth of experience in anticancer drug development and clinical trials respectively, and have collectively published well over 500 research articles in these fields of research.

The first nine chapters essentially deal with cellular targets for anticancer drugs. Chapters 10-14 deal with different approaches for anticancer drug development and chapters 15-20 deal with various aspects of pre-clinical drug development. Each chapter begins with a brief summary and most conclude with an outline of the progress that has been achieved to date in the relevant field, together with an indication of potential advances in the future.

The individual topics have been presented at an advanced scientific level, and there appears to have been a determined effort to ensure that as much current work as possible has been included, as indicated by the high proportion of relatively recent references cited.

Although most chapters provide useful diagrams, the text would have benefited from more "summary-type" figures. One unfortunate limitation was the restriction of most figures to black/white, where many would have benefited considerably from a good colour presentation. Some useful colour figures have been included, but are buried between chapters 19 and 20.
Overall, this text provides a magnificent resource for all those involved in any aspect of anticancer drug design or drug development. It is presented at an advanced scientific level and is ideally suited to those who are actively involved in anticancer drug discovery and/or drug development. However, it will also serve as a superb introduction and postgraduate students who require an overview of the status of current anti-cancer drug development strategies and approaches, and also to those who require some perspective of individual aspects of anti-cancer drug development (avoidable for this type of specialised topic), but well worth it is a must for every university and research institute library and all laboratories involved in anticancer drug research. It is more research-oriented and more comprehensive than the only other similar text on this topic by Beverley Teicher. It is more research-oriented and more comprehensive than the only other similar text on this topic by Beverley Teicher. It is a must for every university and research institute library and all laboratories involved in anticancer drug research.

CANCER INFORMATICS: ESSENTIAL TECHNOLOGIES FOR CLINICAL TRIALS

JS Silva et al
Published by Springer (2002)
RRP: US$79.95

People running cancer trials should read this book. It is about the US National Cancer Institute’s plans to liberate cancer trials from paper. The authors’ thesis is that advanced information technologies and concurrent process enhancements will transform clinical trials, just as they have transformed businesses. The aim is to make better treatments into practice faster.

Richard Klauser championed the need for a cancer informatics infrastructure to enable clinical research and to link it to the delivery of cancer care. He took this up as a task for the US National Cancer Institute (NCI) in 1995 and published a report a year later. A following chapter in this book is about the origins of the report. The book is a concise compendium of current cancer informatics research.

The sections describe lessons from e-commerce – development of standards, common data elements, forms and terminology; integration with public health; informatics and research; clinical trials information systems; and consumer education and support. The chapters were written by leading US experts. The content is detailed, technical and surprisingly well-written. They give a clear view of how cancer trials are likely to develop over the next 20 years.

This book is informative, interesting, and I’m embarrassed to admit enjoyable.

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CANCER OF THE BREAST (5TH EDITION)

WL Donegan and JS Spratt
Published by Saunders (2002)
Distributed in Australia by Elsevier
RRP: A$390.00

This book is an update from the last edition in 1995 and is therefore a considerable advance on the text. Previous editions have been an excellent resource, but as with textbooks, like computers, they quickly become outdated. With the speed of advancement in breast cancer research and treatment, this is especially so for reference texts on the topic of breast cancer.

This book, nevertheless, comprehensively covers just about all aspects of breast cancer and also has an excellent chapter on common benign conditions of the breast. A preceding chapter by one of the few non-American authors unifies the concept of benign breast disorders, which is pertinent to anyone dealing with the “worried well.”

History never changes, but there is an excellent introductory chapter on the history of breast cancer with following chapters dealing with microscopic and gross anatomy of the breast and physiology. All the usual necessities of a breast cancer textbook are covered, which include clinical aspects around medical, radiation and surgery. Molecular biology, genetics and prevention of breast cancer are dealt with well in separate chapters and a whole chapter is devoted to growth rates before leading into staging and prognosis. I found the chapter on nutrition and breast disease quite interesting and informative. The editors of this textbook are surgeons and some of the key chapters eg in situ carcinoma of the breast, Stage IV carcinoma and Local and regional recurrence are authored by surgeons. This leads to a slight surgical bias for what is clearly multidisciplinary conditions. The non-surgeons amongst you however should not be put off by these chapters as they are quite comprehensive and well-referenced. A large number of contributing authors provide a good coverage of many other aspects of breast cancer research and management that are not covered by other smaller textbooks. There are quite a few chapters that deal with aspects of metastatic disease and chapters devoted to some difficult management problems such as pregnancy and occult primary breast cancer. The final chapters deal with statistical methods and a critical analysis of clinical trials. A whole chapter is devoted to lists of issues related to breast cancer, a North American issue of equal significance in Australia.

Overall I would highly recommend this reference textbook for the generalist with a breast interest or even the highly specialised research oncologist. No one knows it all and there will certainly be chapters that will educate, others that will serve as a quick ready reference and others, which the informed will wish to critically evaluate and perhaps even research in alternative conclusions. There are only a few textbooks on the management of breast cancer that are as encyclopedic as this one and this is the most current. It is highly recommended.

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CLINICAL GYNECOLOGIC ONCOLOGY (6TH EDITION) INCLUDING CLINICAL GYNECOLOGIC ONCOLOGY REVIEW (3RD EDITION)

PJ DiSaia and WT Creasman
Published by Mosby (2003)
Distributed in Australia by Elsevier
RRP: A$313.50

DiSaia and Creasman have recently released the sixth edition of their textbook Clinical Gynecologic Oncology. This American textbook is aimed at the “resident, fellow or student of gynecologic oncology” and represents a comprehensive text on the subject. The book was first published in 1981 and has been revised in line with progress in the field. In this regard it gives a clear historical perspective on many of the advances in the management of women with gynaecological malignancies.

The book is a breast cancer text, giving a detailed account of the epidemiology and clinical presentation and management of women with gynaecological cancers. It also includes comment on affiliated areas such as breast and colon cancer screening and guidelines for the management of the dying patient.

Compared to previous editions the chapter on the genetics of cancer has been expanded. Also this new edition has a small colour atlas at the beginning of the book.

The text is quite expansive in character however important key statements are highlighted in red, and this is helpful when using the book as a reference. There is also a very useful chapter on cancer in pregnancy, an important topic, and one that is not addressed in such detail by other commonly used text-books.

My main criticism of this textbook relates to the limited appreciation and acknowledgement of key research arising outside North America that advances their thinking. It is obviously challenging to the authors to have to continually update such a comprehensive text on the subject and this also applies on occasion.

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COLORECTAL CANCER: MULTIMODALITY MANAGEMENT

LB Saltz (ed)
Published by Humana Press (2002)
RRP: A$195.00

This book is extensive in scope, aiming to provide a ‘well-balanced, authoritative, evidence-based review of the current approaches to the prevention, diagnosis and treatment of..."
colorectal cancer” and is divided into six segments. These are epidemiology and prevention, diagnostic and therapeutic radiology, surgery, medical oncology, supportive management and new agents in colorectal cancer.

Each section has significant strengths. In the first section on epidemiology and prevention there is an excellent overview on the chemoprevention of colorectal cancer. The chapter on screening and surveillance is reasonable, but does not adequately address the role of the Bethesda criteria in identifying familial adenomatous polyposis and may require investigation for hereditary non-polyposis colorectal cancer.

The section on diagnostic and therapeutic radiology provides excellent chapters on the role of virtual colonoscopy, nuclear medicine and preoperative staging of rectal cancer. Within the section on surgery, the chapter examining the role of laparoscopy in colorectal cancer is balanced by cautious conclusions, which rightly place the technique in the setting of research trials rather than routine clinical practice until much more is known about cost effectiveness and outcomes are known. The chapters on cryosurgical and radiofrequency ablation of hepatic metastases suffer from enthusiasm being weighed over evidence. Both techniques require better trials before they can be commonly recommended. The chapter on the clinical management of peritoneal surface spread is written by an obvious enthusiast, and is thinly referenced. Balancing this are solid chapters on surgery of the colon and rectum. Furthermore, the chapter on management of cancer in a poly is concise and helpful to any budding colonoscopist.

The section on medical oncology provides an excellent, detailed overview to an area that is experiencing rapid change. As with any text in an evolving area, the chapters inevitably are unable to be completely up-to-date, and hence this reduces its utility. Establishing antitumour agents is the field. However, for non-oncologists involved in the management of colorectal cancer and trainees to the field all the chapters are valuable. Similar comments apply to the section on new agents in colorectal cancer.

The section on supportive care is incomplete. One of the most distressing aspects of advanced colorectal cancer can be bowel obstruction due to recurrent disease and/or peritoneal spread. The medical management role of venting percutaneous gastrostomy surgery is not covered in this section. Similarly, the role of radiotherapy in pain management is not covered, although the overview of analgesics used is not unreasonable.

This text would be useful for any health professional working in the area of colorectal cancer who wishes to have an accessible text covering other disciplines beyond his or hers in this field. It is relatively well written, is respected to its palliative management, but the sections on medical oncology and epidemiology and prevention are its greatest strengths.

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While there is a plethora of breast cancer information resources for women and their families, some gaps remain. Fighting for our future, based on an earlier US Lifetime Television documentary, aims to fill one such gap. In writing this book for young women with breast cancer, the author is supported by an expert medical advisory board.

Given their relative youth and earlier life stage, breast cancer presents young women with particular physical, medical and emotional challenges. This book addresses many of these challenges. In particular it helps to address the isolation that many young women with breast cancer experience. Throughout the book, the voices of young women carry the reader through the experiences of the real women you are not alone.

The book addresses an extensive range of topics including: screening and diagnosis, treatment options, alternative and complementary methods of healing, support issues, body image, fertility, workplace issues, recurrence, and breast cancer advocacy.

Each chapter provides significant details on a range of issues, drawing on both medical expertise and women's experience. The books emphasis is to provide information from varying and diverse sources, and aims to give women the information that they can use when talking with their practitioners. Key features of each chapter are the highlighted checklist of questions, useful tips and women's stories.

The chapters on psychosocial support, body image, relationships and sexuality are especially strong, as is the chapter on fertility, and aims to give women the information that they can use when talking with their practitioners. Key features of each chapter are the highlighted checklist of questions, useful tips and women's stories.

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In its conversational style, the book relies on the views given by young women, medical experts. At times I would have preferred stronger articulation on the level of evidence underpinning their views, rather than just the expert's stated underpinning their views, rather than just the expert's stated views. The reader may therefore wish to discount some of my enthusiasm if they are new to the subject.

The chapters have been thoroughly edited, so that they have a fairly uniform style and are all easy to read. This has not stymied the appropriate expression of various viewpoints by different authors. The reader will readily pick up differences of opinion, for example, regarding the extent to which this syndrome can be used as a model for normal ageing. I must confess to a partiality to the WRN gene. My laboratory group was involved very peripherally in the race to clone it, and we have an ongoing interest in investigating its functions. The ramifications of mutations in the WRN gene, which are responsible for most cases of Werner syndrome, include the early onset of age-related diseases such as atherosclerosis, osteoporosis, cataracts, type II diabetes mellitus and cancers. Therefore many diseases that have a major impact in Western countries can potentially be illuminated, and by a study of this uncommon autosomal recessive condition.

Werner syndrome itself has an interesting history. It was described in 1904 by a German medical student, Otto Werner. The disease occurs most frequently in Japan, where two WRN mutations account for most of the cases. The most common of these is thought to have arisen in a member of a Samurai clan. Japanese clinicians have assembled large and informative case series. A landmark paper on the clinical features, co-authored by George Martin who is a contributor to this book, was published in 1966. The WRN gene was identified by a group led by Gerry Schellenberg in Seattle in 1996. From the authors' heartfelt wish that puts Werner syndrome in the context of other premature ageing syndromes. The link between the clinical section of the book and the section on cellular and molecular changes is provided by Ray Monnat's thoughtful chapter which speculates on possible ways in which the WRN mutation may lead to the clinical manifestations, especially cancer. There are chapters on the chromosomal changes, the cellular phenotype, and on the function of the WRN protein. There is a chapter on WRN homologues in nonhuman systems, including yeast and even bacteria. The class of proteins to which WRN belongs is in fact named after a bacterial enzyme, RecQ, that is involved in unwinding helical DNA. As an aside, it is pertinent to note here that Werner made the remarkably insightful observation that the syndrome has most strikingly been described in one published by Rothmund in 1866, the features of which include prorrone to osteosarcoma. Ninety-five years later, Yasuhiro Fusakichi and colleagues found that a subset of Rothmund-Thomson cases have mutations in the helicase gene.

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E Thackery

Published by The Gale Group (2002)
ISBN: 0-7876-5609-7; 1,163 pages plus index
RPP: US$285.00

Upon first glancing through the encyclopedia, I presumed that the target audience were nursing or medical students. However, on reading it more closely I notice that it states that it is "for use by anyone touched by cancer". I would think that the reader would have to be well-educated to comprehend the content and able to focus on some of the graphic photographs.

Having said that, the encyclopedia is well laid out with information able to be accessed alphabetically by topic. The diagrams are extremely well done. It provides an excellent in-depth coverage of specific cancer types, diagnostic procedures, treatments, cancer side effects and care for drugs. A comprehensive general index and cross-referencing ensures easy access of use. I especially liked the "key terms" box and the "questions to ask your doctor". The information about drugs is especially useful as I know that patients often call to enquire about the drugs they are being treated with.

Once the shock of diagnoses has been absorbed, people affected by cancer and their families often have a terrific need for information. The encyclopedia would give the person affected by cancer a good background resource of information to take to their doctor to discuss their specific type of cancer and treatment.

I think this would be an excellent resource for cancer information services and hospital drop-in centers and I am sure that it would be regularly checked out of libraries. The resources listed are all North American, but the information about cancer and all related clinical treatment issues is excellent.

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**THE GENETIC BASIS OF HUMAN CANCER**

B Vogelstein and KW Kinzler (eds)
Published by McGraw Hill
ISBN: 0-0713-7650-6; 802 pages plus index
RPP: US$125.00

This book represents a comprehensive resource text on the genes that cause cancer. It was originally designed as an addition to the textbook Metabolic and molecular bases of inherited disorders and in fact 42 of the 52 chapters have been adapted from the 8th edition of that text.

The Vogelstein book is organised into four distinct sections. The first two chapters provide a detailed explanation of the basic concepts of cancer genetics. Included in these chapters are sections on the nomenclature of gene mutations and chromosomal alterations. This information would be of particular value to clinicians and counsellors who were not as familiar with this area of work.
Hypermethylation, previously considered to be a nongenotoxic epigenetic process of DNA methylation (hypo- and perhaps no DNA sequence change as the first step). For example, the changes and effects such as increased cell growth—there is results in a genetic change or mutation) versus nongenotoxic genetic toxicity tests in use at this time and provide a broad underpinning human cancer genetics, the major types of controversial issues. Both these aspects deals with accepted protocols and toxicology testing in industry. It is, however, a beautiful resource book and should be in the library of all clinical and research cancer departments.

The third set of chapters (13-39) deal with the familial cancer syndromes as well as the genetic basis of the disease. The real strength of these chapters lies in the succinct descriptions of the gene discovery process, the key sites of mutations and the linkage between mutation and disease. These chapters would be of interest to those individuals for whom interest in a deeper insight into the genetic basis of a particular disease. They are not designed to provide comprehensive or practical clinical management guidelines.

The final chapters (40-52) discuss a number of common malignancies and how epigenetic mutations may have a role. The focus here is naturally on somatic mutations however the information is once again of the highest quality. My only criticism of this book is that many of the diagrams and photographs have been poorly reproduced and there are no colour illustrations. It is, however, a beautiful resource book and should be in the library of all current and research cancer departments.

This short and concise text offers an authoritative, practical account of the status of genetic research, the reader an authoritative, practical insight to the status of genetic research. It is, however, a beautiful resource book and should be in the library of all current and research cancer departments.

The errors of dividing cells as it directly demonstrates the loss of chromosome: a small separate nucleus from the main nuclear frames during telophase when the nuclear envelope is reconstituted (structural damage to a chromosome will lead to an acentric chromosomal fragment(s) or damage to the spindle apparatus will lead to a loss of a whole chromosome(s)). By definition, the changes observed have been passed on through a cell division, is inherited by the daughter cell (in contrast to information from metaphase spreads from cells which have not yet undergone a cell division). This is a simple process that requires only a small amount of biological material and can be automated.

The guidelines from the relevant international bodies are clarified in terms of context and process. A recent move away from requiring in vivo animal testing for chemicals identified as being produced in high volume and for which there is likely significant human exposure is discussed. The in vitro micronuclear test may well become established in this context, presumably leading the way for other areas of genotoxic assessment to adopt a similar approach. The difficulties of discriminating the basis of a carcinogen dose-response curve and its dependency on both chronicity of exposure and individual susceptibility is detailed. Molecular biomarkers and epidemiological evaluation of individual susceptibility are discussed and these presumably will become of increasing importance as new tools such as SNPs and HAPMAPs allow us to better understand the genetic basis of individual susceptibility and the identification of specific at-risk groups.

This book provides an excellent genetic understanding of the relevant issues involved in genetic toxicology and subsequent cancer risk assessment using specific examples in an illustrative way, rather than also being a source of information regarding individual chemicals. It has a lot to teach us, both in terms of critical analysis of research papers, and extrapolations of that data into our daily thinking about the basis of cancer risk assessment.

This is the sixth edition of Hematology of infancy and childhood—the key text in paediatric haematology. The previous editions have been very useful for the clinician. Assessment of MRD also is described in part, because of the diversity of perspectives. On the whole these articles are well written and presented. Overall the reader may find a useful collection of articles, which are intended to provide a resource for those interested in increasing their knowledge or pursuing research in this field.

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This is the sixth edition of Hematology of infancy and childhood. The importance of this publication in the context of the various areas of contemporary hematopoiesis warrants its incorporation into the library of clinicians, researchers, and educators in the field.

The preface to this issue of Health Communication sets the scene well: Cancer communication and aging is an important aspect of health care that is generally under-researched. The articles in this book encompass a variety of aspects of communication in the context of cancer and aging, from diagnosis and treatment to patient. In this context, the articles focus on different aspects of communication research including models of communication and change, the relationship between communication and culture, social relationships, identity, technology, and importantly, communication with healthcare professionals.

The articles in this book vary in their approach, some focus on theoretical models that have utility as explanatory frameworks, others present qualitative research into aspects of communication in the cancer/aging context, some utilise a qualitative orientation to explore the experiential nature of cancer across the care continuum. An important theme is the effects of communication issues in cancer on the intrapersonal, social, cultural, systemic, and organisational health-related outcomes. This book contains a collection of articles that is interesting, in part, because of the diversity of perspectives. On the whole these are articles well written and presented. Overall the reader may find a useful collection of articles, which are intended to provide a resource for those interested in increasing their knowledge or pursuing research in this field.

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In summary, this text is the key reference for paediatric haematologists. The information is clearly and very comprehensively presented and is current. The figures, diagrams, illustrations and photomicrographs are excellent. The low costs on solid tumours and management mean that these are referenced in the book for readers, and the information on haematological malignancy is very complete. This text remains an essential for the library of the paediatric haematologist/oncologist.

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Cancer is primarily a disease arising from random damage to the genes of some somatic cells that confers to them a growth advantage over other cells in the body. In this context, one of the major challenges in the evolution of higher organisms from unicellular bacteria is the need to rapidly distinguish from the healthy cells those cells in a multicellular organism that sustain some DNA change that radically alter the growth properties of the cells. Such cells must then be eliminated in a timely fashion, so as not to endanger the existence of disorders of red cell production, haemolytic anaemia, disorders of haemoglobin and the phagocyte system. The first chapter on the historical perspective of pleomorphic haematoxylin is an excellent means of introducing the text.

For the purpose of this review, I have been asked to focus on the oncology component of the text, which has changed markedly from the previous editions. Previous editions have included specific chapters on the epidermiology, molecular biology and chromosomal abnormalities in childhood cancer. Additionally, the principle of paediatric radiation therapy and management of malignant solid tumours were covered in the previous editions, in particular Thomas Look, have “totally reconstructed” the section on oncology. As this is essentially a textbook on haematological conditions, the chapters specific to solid tumours, radiation therapy have been omitted. The section on oncology, now appropriately, is devoted to haematological malignancies. The authors comprehensively cover epidemiology, including familial origins of ALL and current controversies, chromosomal abnormalities with discussion on newer methods for identification; and the leukemias and approaches to treatment, incorporating a chapter on the pharmacology of antineoplastic agents and multidrug resistance. Lymphoma, lymphohemopoietic and lymphohistiocytic disorders also are covered. Importantly the molecular basis of haematological malignancy is covered in a chapter which clearly describes, with the use of many excellent figures, the complicated intracellular signaling and transduction pathways. This chapter also describes current techniques on the role of gene expression, the very useful for the clinician. Assessment of disease is described clearly by the author. Also of relevance to the oncologist is the complete chapter on acquired disorders of haemostasis.

In summary, this text is the key reference for paediatric haematologists. The information is clearly and very comprehensively presented and is current. The figures, diagrams, illustrations and photomicrographs are excellent. The low costs on solid tumours and management mean that these are referenced in the book for readers, and the information on haematological malignancy is very complete. This text remains an essential for the library of the paediatric haematologist/oncologist.
other endogenous viruses that may be co-infecting the same host interactions receives ample consideration in the book, This book fails to place such efforts in the context of a rapidly changing as more and more large viruses are described, in parallel with efforts to endow such recombinant and Newcastle disease virus. Efforts to genetically engineer with many wild-type viruses may often be too close to allow studies has been slowed down by the limited ability to the rapid production of many linear molecules of viral DNA in a eukaryotic cell serve a role in testing the ability of the cell to repair double strand breaks in its chromosomal DNA? Or conversely, could some of the common human viruses serve a positive role in the identification and killing of those cells in the body whose DNA repair mechanisms fail and become permissive for uncontrolled viral replication?

Viewed in this context, it seems quite likely that natural selection may have already endowed some human viruses with properties that may be useful for the identification and killing of damaged cells in various tissues before they can give rise to cancer. It is extremely important to determine if a specific type of cancer that may escape the endogenous molecular surveillance mechanisms that may exist in a certain cell type still may be susceptible to killing with some limitation? How close is the balance between good and harm with many wild-type viruses may often be too close to allow useful clinical application. Genetic engineering of such viruses may be necessary before they can be employed usefully for cancer therapy.

The book by Driver and Rabbin reviews practical progress in the use of a number of replication-competent viruses for cancer therapy. Separate chapters review the use of herpes simplex virus, adenovirus, reovirus, parvoviruses, vaccinia virus and Newcastle disease virus. Efforts to genetically engineer wild-type viruses to reduce their potential for harm are described, in parallel with efforts to endow recombinant viruses with novel strategies for cancer cell killing. Progress in such studies has been slowed down by the limited ability to perform genetic engineering in eukaryotic cells, a situation that is considerably changing as more and more large viruses are cloned as bacterial artificial chromosomes. Techniques recently developed for the precise genetic engineering of large genomic DNA in yeast and bacteria have been opened with an overview, reference to the unique anatomical considerations of the region or site, a discussion of the relevant preoperative imaging and then a set of surgical guidelines with discussion of the various surgical manoeuvres. The text is supported by an excellent collection of operative photographs, are also of the highest quality.

The first section covers the more routine fundamentals regarding cancer and stress particularly the increasing role of chemotheraphy and radiation therapy in the management of these problems. It goes further however by discussing isolated limb perfusion as opposed to limb infusion, more often practised in Australia) as well as an entire chapter devoted to the correct approach to the biopsy of these tumours. There is a very succinct and appropriate chapter on the surgical management of metastatic bone disease.

The majority of the book is then given over to various aspects of surgery, divided into three sections: muscle group resections, limb-sparing surgery and amputations. In each instance the various chapters opened with an overview, reference to the unique anatomical considerations of the region or site, a discussion of the relevant preoperative imaging and then a set of surgical guidelines with discussion of the various surgical manoeuvres. The text is supported by an excellent collection of operative photographs, are also of the highest quality.

The second part of the book devoted to the management of sarcomas and see the chapter on sarcoma. This is indeed a serious omission in the midst of the HIV pandemic, given the high propensity of this virus to integrate and the low sequence specificity of its integration mechanism. The last thing anybody would want to do in the struggle against cancer is to provide HIV with a novel mechanism of its genome into a large human virus of reduced pathogenicity.

Although this book is restricted in scope, it should be useful not only to the specialists that may be interested in the field of cancer therapy with replication-competent viruses, but for all those seeking novel approaches to exploit the natural defence mechanisms against cancer cells.

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MUSCULOSKELETAL CANCER SURGERY: TREATMENT OF SARCOMAS AND ALLIED DISEASES
MM Malawer and PH Sugabaker
Published by Kluwer (2001)
RPP: US$199.00

This surgical text is a delight to the eye and difficult to put down. It is authored by two visionary and well-established experts in this field with additional contributions from both the US and Israel.

There is a consistency of presentation throughout the book which is very helpful. The reproduction of both the imaging, and clinical and operative photographs, are also of the highest quality.

One useful thing a book like this could do is to provide a perspective about the use of competing techniques in preclinical models. This was done well in the chapter on single nucleotide polymorphisms and quite poorly in most others. Despite the uneven chapters and the criticisms above, I quite enjoyed this book. It is fun to flip through recipes you are never going to prepare. If you are interested in drug-development more generally, you will probably want more perspective. The field of biomarkers for drug effects faces enormous challenges. No doubt there are lessons to be learned from methods that are technically possible, but turn out to be unhelpful. There are also lessons from drugs that have “failed” clinically despite effective biomarkers or because of lack of them. Inhibition of EGFR, PDK-1 and matrix metalloproteinases spring to mind in this regard. This book doesn’t take on board any of these lessons (with the chapter on PET a possible exception). I would pocket the $125 and eat out at a good restaurant instead.

OVARIAN CANCER
R Ozols
Published by BC Decker (2003)
RPP: $350.06

This book is part of the “Atlas of Clinical Oncology” series, edited by Dr Robert Ozols of Fox Chase Cancer Centre, with contributions from that centre or from the National Cancer Institute Sparc program. It is obvious from the start that the contributors are all at the cutting edge of their specialty. The book covers every facet of ovarian malignancy including germ cell tumours and ovarian sex cord stromal tumours, has magnificent illustrations, is sequentially and logically laid out and is accompanied by a CD, which is especially useful for those of us who like to use illustrations from state of the art presentations. Perhaps the strength of this book lies in its basic biological contributions, and in particular the chapters on biology, genetics, developmental chemotherapy and advances in biological therapy and high-dose chemotherapy are all extremely interesting and contain information not readily accessible from other sources.

This is a book for the post-graduate. It will be of use particularly for the gynaecological oncologist and the medical oncologist caring for women with this malignancy and for those working in family cancer clinics, given the excellent chapters on genetics and genetic counselling.

M Quinn
Royal Women's Hospital
Carlton, VIC

PALLIATIVE CARE NURSING A GUIDE TO PRACTICE (2ND EDITION)
M O'Connor and S Aranda (eds)
Published by Asmed Publications (2003)
RPP: AS$65.95

The goal of the first edition of this book, according to the editors, was to make palliative care accessible to nurses in all health care settings. This second edition has been completely revised and rewritten and achieves continuing relevance to nurses, in all settings, who require information about palliative care. The book presents palliative care as a “continuum of practice from a generalised approach to a specialist discipline”. The editors and the authors of the chapters in the book write with acknowledged expertise in the subject areas.

The book is divided into 24 chapters and the content centres on...
on contemporary practice issues in palliative care nursing. There is a focus on clinical issues faced by nurses caring for dying people. The topics covered provide the capacity for nurses and other readers of the book to think about the depth and breadth of issues that are involved in palliative care that is described as “holistic, expert and interdisciplinary.” The scope of the contents of this book ranges from evidence-based practice in palliative care, psychological and existential distress, sexuality and body image to nutrition and hydration, occupational stress, frameworks for detailed and continuous assessment, and palliative care for people other than those with malignancy. The book chapters are clearly set out and are discrete, making it easy to access relevant chapters.

So often issues around staff stress for those working with people who are dying are positioned at the end of publications or education sessions. It is interesting to note that in this book the chapter about occupational stress is in the first section. Similarly, knowledge related to care of the spirit is often relegated to less prominent positions than information about other symptom management. Again, the positioning of the chapter on spirituality at the beginning of this book is noteworthy.

The problems that often cause the most discomfort for the people experiencing them, such as breathlessness, fatigue and pain, are important inclusions in the book. The psychosocial aspects of palliative care often present challenges to health care providers, particularly those working in areas where the dominant culture is framed by technical and biomedical parameters. Inclusion of chapters about sexuality and body image, and psychological and existential distress, among others, adds a vital dimension.

This is a book that will enhance readers’ understanding of care and support for people (and their families, friends and carers) at the end of life. It provides accessible comprehensive evidence-based guidelines for practitioners. The dedication at the start of the book to the memory of a daughter by a mother is excellent and the dedication ensures the voice of the patient and the people who mean most to them is at the forefront. This is the essence of excellent palliative care.

J Gibson
Clare Holland House
Canberra, ACT

PHYSICIANS’ CANCER CHEMOTHERAPY DRUG MANUAL 2003

E Chu and V DeVita
Published by Blackwell Publishing Asia (2002).
RRP: A$151.80

Vincent DeVita and Edward Chu are two well-known US oncologists with distinguished academic and research records. In writing this handbook they have successfully created an invaluable resource for any professional involved in the prescription or delivery of cytotoxic chemotherapies. The book is presented in bullet form, except the first chapter, which is a brief overview on the principles of chemotherapy, which is very similar to their larger publication, Principles and Practice of Oncology. The drugs are listed in alphabetical order and are described using a number of subheadings including the mechanism of action and resistance, basic pharmacokinetic information, indications, interactions and toxicity. Although this information is presented in note form, it is still extremely comprehensive, particularly information with regards to drug interactions.

The following chapter is really a collection of tables. The first few tables cover basic formulas like creatinine clearance, area under the curve and body surface area. What follows though are some invaluable tables on dosing according to impaired renal or hepatic function. There is also a table that evaluates whether certain cytotoxic agents are cleared by either portal or hepatoportal routes. There is then a section entirely devoted to lists of tumour types and the currently used treatment regimens, which may be useful to those unfamiliar with standard treatments. These regimens are all appropriately referenced and serve mainly as a guide to treatment. Finally there are lists of antiepileptic agents presented in a similar manner to the information on cytotoxics.

The book itself is well-presented in a ring binder format allowing for any reader to easily access the information in the book. The book is a quick and easy reference book. It also comes with a free CD-ROM. What would have been useful are links to a website so that articles referenced could be studied in more detail. Furthermore, there is no link to a website where more up-to-date information could be accessed. So while this book is easy to use and comprehensive, like all books of this kind, certain information such as current chemotherapy regimens used will become less up-to-date with time.

R Sharma
Dept of Medical Oncology
Royal Prince Alfred Hospital
Camperdown, NSW

PRACTICAL GUIDE TO INTENSITY MODULATED RADIATION THERAPY

S Hellman
Published by Medical Physics Publishing (2003).
RRP: US$125.00

This textbook is a comprehensive guide to all aspects of intensity modulated radiation therapy (IMRT). The initial chapters deal with medical physics and cover aspects of an initial chapter on imaging, including image fusion and an overview of the IMRT process. During the chapters on optimisation, delivery, computer algorithms and treatment planning, there is significant detail that is beyond the need for a clinical physicist but would provide a very useful departmental primer. In the second half of the book there are clinical chapters in the areas of IMRT for prostate cancer, head and neck cancer, pancreatic, breast and non-small cell lung cancers.

In pouring through the first 190 pages, I was waiting for information that would help me to make a decision regarding the mechanism and implications of IMRT and the process involved from seeing a patient to delivery of an optimised plan. It is in these later chapters that this is dealt with very well.

There is an excellent reference chapter on normal tissue complications and a discussion on tumour control probability (TCP). The book closes with two chapters on advanced treatments addressing areas of future development, including the integration of respiratory gating and stereotactic immobilisation in IMRT.

The staff of the Memorial Sloan-Kettering Cancer Centre’s Department of Medical Physics wrote this book. This emphasises that although the book is a valuable departmental resource, it would only suit individual radiation oncologists and registrars wishing to extend their current knowledge.

I would thoroughly recommend departments purchase a copy for their library. Clinicians, however, should take caution over the first 190 pages if seeking relaxing reading prior to bedtime.

M Penniment
Dept of Radiation Oncology
Royal Adelaide Hospital
Adelaide, SA

PRINCIPLES AND PRACTICE OF PALLIATIVE AND SUPPORTIVE ONCOLOGY

A Berger et al (eds)
Published by Lippincott Williams and Wilkins (2002).
RRP: A$504.60

There are excellent texts available in anti-cancer therapies and in palliative care. For the oncology patient, this text by Berger and her colleagues provides an important contribution to bridge the two. A book that deals with such a broad remit – people who will be cured, live with the disease over extended periods of time or die as a result of the disease – will always pose a challenge for the editors. This team has balanced this well.

As supportive care gains evidence and a defined role in practice, the need for a definitive text in the area is obvious. This book is such a text. Beyond oncology, there are some chapters on HIV/AIDS, paediatrics and intensive care. Although interesting, they are brief and not oncology-focused.

The list of contributors is strong and includes many people whose research expertise is reflected in their chapters. There is a good mix of people working in acute oncology, supportive care and palliation. However, contributions are almost entirely from the US and, in an area where the clinical encounter so often needs to be interdisciplinary, mostly from medical practitioners. One unfortunate consequence of this is the recurring theme of affordable access to health care. Sections on models of health service delivery are not easily generalised to the Australian context.

There is the continuing challenge of how to reflect the best evidence base for practice. The text does not easily identify high-quality evidence or provide detailed information on case series in the body of the work. Although the references help in this process, this omission detracts from the wealth of evidence presented.

Although areas such as the impact of cancer and its treatment on sexuality and intimacy are generating new and important evidence, this area is presented in a very biomedical paradigm. Survivornship and its consequences are well presented. Issues of effective communication between patients and health professionals are dealt with in a very practical chapter.

If there were one new reference text for your bookshelf this year, would this be it? It should be. It complements rather than competes with texts such as the Oxford Textbook of Palliative Care and End of Life Care (eds, M Penniment and B Rubovics) and Panos-Ayer and Wilkins (eds) (4TH EDITION)

PA Pizzog and DG Poplack (eds)
Published by Lippincott Williams and Wilkins (2002).
plus index.
RRP: A$685.00

For many years in the field of paediatric oncology there was a relative dearth of reference books compared with that available to our adult medical oncology colleagues. In the late 1980s the first edition of Principles and Practice of Pediatric Oncology was published and it quickly became the pre-eminent paediatric oncology resource text. A multidisciplinary team approach has become standard practice for managing paediatric cancer patients and all those involved in this field could turn to this text for detailed information on the majority of disease processes that occur in this patient group. Subsequent editions during the 1990s only served to enhance the reputation of this tome, though the third edition did suffer from several chapters only having minor ‘cosmetic’ changes to the text.

The eagerly awaited fourth edition however has had major changes to the organisation of both the general sections and chapters. The editors and the contributing authors have made the inclusion of up-to-date data on the molecular biology of paediatric malignancies their primary stated goal for this edition. While new advances and discoveries will quickly overtake the information provided in this book, the editors and the contributing authors have succeeded in summarising the explosion in genomic knowledge of paediatric malignancies that has occurred over the past five to 10 years. In particular the basis this has provided an improved understanding of childhood cancer.

The majority of contributing authors are North American in origin, but the individual disease chapters have in-depth discussions reflecting the global experience of previous
treatment protocols. There are still idiosyncrasies that are simply reflections of differences in approach to particular problems and issues of discussion on the management of tumour lysis syndrome, the use of urate oxidase is only briefly mentioned. However, these instances are few, and at worst are peripheral to the book. In general, the book is an interesting read. The role of the Rb tumour suppressor in cancer by Lili Yamakasi, begins with a short history of the discovery of the Rb gene, then summarises the basic structure of pRb family members and the upstream regulators and downstream effectors of Rb. A most useful part of the chapter as a resource for scientific learning, is the summary of mutant mouse phenotypes that either involve or have shed light on the function of this pathway.

In contrast, the chapter on the steroid hormone receptors summarises the oestrogen receptor (ER) in breast cancers and the androgen receptor (AR) in prostate cancers as illustrations of the subject area. The chapter briefly covers ER and AR expression in normal and malignant breast and prostate (respectively), receptor structure, transcriptional modulators (activators and repressors) of the receptors and other proteins and pathways that can regulate steroid hormone receptor activation. This in particular lies in well with other chapters of the book did have short summaries of mutations in ERα and AR in breast and prostate cancers.

While the chapters in this book are necessarily brief, all are extensively referenced and most authors have included historical as well as more recent references. Thus this book is an excellent summary of important signalling pathways in cancer that will be useful for students and researchers. It will serve as a resource for general and specialised knowledge regarding these pathways as well as a platform for more detailed research.

J Bentel
Royal Perth Hospital
Perth, WA

Recombinant Human Erythropoietin (rHepO) in clinical Oncology
MR Nowroussian
Published by Springer Medicine (2002)
ISBN:  3-211-36616-1, 492 pages plus index
RRP:  EUR98

This comprehensive text is an excellent resource and examines the biology of erythropoietin, causes of anaemia, prevention and management of cancer-related anaemia with rhEPO. It is well referenced and has helpful ‘Condensations/summary’ sections at the end of each chapter. It is well-balanced, highlighting areas where rHepO is effectively used while exploring areas where further investigation is required. Unfortunately there is a lack of tables summarising the data and some of the methodological deficiencies of referenced publications are not discussed.

The initial chapters discuss the biology of endogenous erythropoiesis. A basic level of erythropoietin pathophysiology is assumed with lack of discussion about mechanisms of renal responses to tissue hypoxia and how that translates to renal erythropoietin production. It would be difficult for those not

Although it is not possible to provide critiques on all chapters of the book, it is worth highlighting the different approaches by the contributors, which lends the book an interesting diversity.
familiar with the area as there is a lack of simple diagrams explaining feedback loops, yet there are numerous more advanced illustrative methods of signalling.

Subsequent chapters discuss the mechanisms of anaemia of chronic disease and cancer-related anaemia. A somewhat excessive three chapters are dedicated to radiation therapy and tumour hypoxia. The chapters dedicated to anaemia and its impact on end-organ function and fatigue are valuable and the important inter-relationship between anaemia, fatigue and quality of life (QOL) is appropriately discussed. The text provides an excellent review on the indications of rhEPO for solid tumours and haematological malignancies, although the section on myelodysplasia is a bit thin.

Unfortunately, the text is already somewhat out-of-date with a number of recent publications on QOL and rhEPO not sighted. Furthermore, outdated terms such as NESP (Novel Erythropoietin Stimulating Factor), rather than darbepoetin are used. This text also was published prior to recent release of the American Society of Haematology/American Society of Clinical Oncology Guidelines on erythropoietin therapy. The text also predated the report of erythropoietin antibodies and consequent pure erythropoietin formulations. As this does not explore this issue.

Thus, this book is a useful reference text for treating oncologists, haematologists and transfusion medicine specialists, but purchase it soon before it goes any further out-of-date.

H M Prince
Dept of Haematology
Peter MacCallum Cancer Centre
Melbourne, VIC

**VIRUSES AND LIVER CANCER**

E Tabor (ed)

This is a useful book for medical oncologists, gastroenterologists and liver surgeons. The most valuable content relates to the underlying molecular mechanisms and recent insights into the molecular biology and pathogenesis of the disease. The sections on treatment cover little more than 20 pages, so do not provide an exhaustive review of management. There is little more here than can be found in standard oncology textbooks. Consequently this book cannot really be recommended as a useful resource for readers who are seeking detailed information about the management of HCC. Nonetheless, it should prove to be a helpful resource to readers who are interested in understanding the molecular basis of HCC at a more detailed level and for this niche audience, it comes recommended.

J Cebon
Ludwig Institute Oncology Unit
Heidelberg, VIC

**WORLD CANCER REPORT**

BW Stewart and P Kleihues (eds)
Published by IARC Press (2003)

This is an excellent book. It provides a concise and up-to-date global view of cancer burdens, epidemiology, carcinogenesis, prevention, screening, management and cancer control. Seventeen separate chapters detail specific cancer sites. This formidable collection is the work of 77 contributors, including 26 from IARC or WHO, and no less than 10 Australians. This no doubt reflects the locally persuasive powers of the editors. The contributors were aided by 11 reviewers, so the unity of style and format in a volume of such diverse origins is commendable. The Australian contributors are Frank Alkvaro (childhood cancers), Peter Hersey (immunotherapy), Norelle Luckis (palliative care), Guy Maddern (surgical oncology), Bill McCarthy (melanoma), Murray Norris (minimal residual disease), Roger Needle (tumourigenesis), Bernie Stewart (no less than three topics: reduction of UV radiation, apoptosis and multi-stage carcinogenesis), Martin Tattersall (cancer education in medical schools) and Graham Young (lymphoma).

The standard of the more than 500 illustrations is extremely high – indeed an electronic version would provide a formidable resource for cancer education. It is to be hoped that WHO will eventually make such a collection available.

Meanwhile the book itself can be purchased through IARC Press at http://www.iarc.fr/WCR/. I commend it as excellent value.

A Cates
The Cancer Council Australia
Camperdown, NSW

**CALENDAR OF MEETINGS – AUSTRALIA AND NEW ZEALAND**

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**CALENDAR OF MEETINGS**

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**OTHER MEETINGS**

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**EMAILS**

[Email: breast2004@ozaccom.com.au](mailto:breast2004@ozaccom.com.au)

**WEB:**

### CALENDAR OF MEETINGS – International

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<td>12th FIGO World Congress of Gynecology and Obstetrics</td>
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<td>9-14 Endocrine-Based Radiation Oncology: Methodological Basis and Clinical Application</td>
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<td>5-9 15th International Congress on Tobacco Control</td>
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<td>Hamburg, Germany</td>
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<td>15-16 New Insights in Molecular Diagnosis and Therapy</td>
<td>Paris, France</td>
<td>Institut Pasteur Euro-Conferences C15, 26, rue du Docteur Roux 75724 Paris Cedex 15, France</td>
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<td>22-24 Gastrointestinal Cancers Symposium</td>
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<td>25-28 Multidisciplinary Treatment in Gynecological Cancer</td>
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<tr>
<td>2004</td>
<td>15-16 New Insights in Molecular Diagnosis and Therapy</td>
<td>Paris, France</td>
<td>Institut Pasteur Euro-Conferences C15, 26, rue du Docteur Roux 75724 Paris Cedex 15, France</td>
</tr>
<tr>
<td>2004</td>
<td>22-24 Gastrointestinal Cancers Symposium</td>
<td>San Francisco, California USA</td>
<td>USA ASCO, 1900 Duke Street Suite 200, Alexandria, Virginia 22314 USA</td>
</tr>
<tr>
<td>2004</td>
<td>25-28 Multidisciplinary Treatment in Gynecological Cancer</td>
<td>Thessaloniki, Greece</td>
<td>Nectara, Nektara Panamawiki, Athens, Greece</td>
</tr>
<tr>
<td>2004</td>
<td>29 Jan-Feb American Psychosocial Oncology Society 1st Annual Conference</td>
<td>Orlando, Florida USA</td>
<td>Alisson Holcomb, American Psychosocial Oncology Society, 2365 Hunters Way, Pittsburgh, Pennsylvania, USA</td>
</tr>
<tr>
<td>February</td>
<td>9-12 15th International Congress on Anti-Cancer Treatment</td>
<td>Paris, France</td>
<td>Travel Congress Organisation 1 rue de Berri Paris - 75008 - France</td>
</tr>
<tr>
<td></td>
<td>15-17 2nd Multidisciplinary Colorectal Cancer Congress</td>
<td>Amsterdam, Netherlands</td>
<td>Congres Care, PO Box 440 5201 AR, Montegibben, The Netherlands</td>
</tr>
<tr>
<td>March</td>
<td>7-11 3rd World Assembly on Tobacco and Counters Health</td>
<td>New Delhi, India</td>
<td>Avnish Varma, ICOCO, M-38-A, RAJOURI GARDEN NEW DELHI - 110027 India</td>
</tr>
<tr>
<td></td>
<td>16-20 4th European Breast Cancer Conference</td>
<td>Hamburg, Germany</td>
<td>EBCC 2004 Secretariat, C/O UNITECH Communications, 24 Raedestou Street, 2365 Hunters Way, Pittsburgh, Pennsylvania, USA</td>
</tr>
<tr>
<td>Date</td>
<td>Name of Meeting</td>
<td>Place</td>
<td>Secretariat</td>
</tr>
<tr>
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<tr>
<td>7- 9 2005</td>
<td>ASTRO: 46th Annual Meeting</td>
<td>Atlanta USA</td>
<td>American Society for Therapeutic Radiology and Oncology 12500 Fair Lakes Circle Suite 375 Fairfax, Virginia 22033 USA Tel: + 1 703 222 0170 Email: <a href="mailto:meetings@astro.org">meetings@astro.org</a></td>
</tr>
<tr>
<td>10-14 2004</td>
<td>6th Congress of the European Association of Neuro-Oncology</td>
<td>Jerusalem Israel</td>
<td>Ostra 1 Nein St PO Box 9352 Tel Aviv 61092 - Israel Fax: +972 3 638 4465</td>
</tr>
<tr>
<td>15-16 2004</td>
<td>The 9th International Conference on Geriatric Oncology</td>
<td>San Francisco USA</td>
<td>Heather Drew Imexid, Inc. 70 Technology Dr Alpharetta - 30005 - Georgia Tel: +1 770 751 7332 Fax: +1 770 751 7334 Email: <a href="http://www.imexid.com/calendars/oncology.htm">www.imexid.com/calendars/oncology.htm</a></td>
</tr>
<tr>
<td>24-28 2004</td>
<td>23rd Annual European Society for Therapeutic Radiology and Oncology Meeting (ESTRO 23)</td>
<td>Amsterdam Netherlands</td>
<td>ESTRO 23 Secretariat Avenue Emouer 83 Brussels, Belgium 1200 Tel: +32 2 775 9940 Email: <a href="mailto:info@estro.org">info@estro.org</a></td>
</tr>
<tr>
<td>29 Oct- Nov</td>
<td>29th European Society for Medical Oncology Annual Meeting</td>
<td>Vienna Austria</td>
<td>ESMO Secretariat via la Seta 7 CH-6962 Veggolato-Lugano, Switzerland Tel: + 41 91 1973 1919 Email: <a href="http://www.esmo.org/congress2004">www.esmo.org/congress2004</a></td>
</tr>
<tr>
<td>5-7 2004</td>
<td>Oncology Nursing Society Institute of Learning</td>
<td>Nashville Tennessee USA</td>
<td>Oncology Nursing Society 120 Enterprise Drive Pittsburgh, Pennsylvania 15275-1214 USA Tel: +1 86 6257 4667 Email: <a href="mailto:meetings@ons.org">meetings@ons.org</a></td>
</tr>
<tr>
<td>3-7 2004</td>
<td>46th Annual Meeting of the American Society of Hematology</td>
<td>San Diego California USA</td>
<td>American Society of Haematology 1900 M Street NW Suite 200 Washington DC 20006 USA Tel: +1 20 2776 0544 Email: <a href="mailto:meetings@hematology.org">meetings@hematology.org</a></td>
</tr>
<tr>
<td>3-6 2004</td>
<td>27th Annual San Antonio Breast Cancer Symposium</td>
<td>San Antonio Texas USA</td>
<td>Cancer Therapy &amp; Research Center SAC Chic Marlow San Antonio, Texas, USA Fax: +1 210 949 5009 Email: <a href="mailto:rmankowski@nabcs.org">rmankowski@nabcs.org</a> Email: <a href="http://www.sacs.org">www.sacs.org</a></td>
</tr>
<tr>
<td>15-16 2004</td>
<td>4th International Meeting of Hepatocellular Carcinoma</td>
<td>Wanchai 4th HCC-EVE</td>
<td>Cancer Forum - Volume 27 Number 3 - November 2003</td>
</tr>
<tr>
<td>26-29 2004</td>
<td>Primary Therapy of Early Breast Cancer</td>
<td>St Gallen Switzerland</td>
<td>Hans-Jorg Semer St. Gallen Oncology Conferences Rorschacherstr. 150 St. Gallen - 9006 Switzerland Tel: +41 71 243 0034 Fax: +41 71 245 6805 Email: <a href="http://www.oncocrinference.ch/index.html">www.oncocrinference.ch/index.html</a></td>
</tr>
<tr>
<td>3-6 2004</td>
<td>58th Annual Cancer Symposium of the Society of Surgical Oncology</td>
<td>Atlanta Georgia USA</td>
<td>D. K. Kubis - Society of Surgical Oncology 85 W Algonquin Road Suite 55 Arlington Heights, Illinois 60005 USA Tel: +1 847 427 0244 Email: <a href="mailto:mwd@ssont.org">mwd@ssont.org</a></td>
</tr>
<tr>
<td>16-20 2004</td>
<td>96th Annual Meeting of the American Association for Cancer Research</td>
<td>Anaheim California USA</td>
<td>AACR 815 Chestnut Street 17th Floor Philadelphia, PA USA 19106-4404 Tel: +1 212 544 9300 Email: <a href="mailto:meetings@aacr.org">meetings@aacr.org</a></td>
</tr>
<tr>
<td>28 Apr- May</td>
<td>Oncology Nursing Society's 38th Annual Congress</td>
<td>Orlando Florida USA</td>
<td>Oncology Nursing Society 123 Enterprise Drive Pittsburgh, Pennsylvania 15275-1214 USA Tel: +1 86 6257 4667 Email: <a href="mailto:meetings@ons.org">meetings@ons.org</a></td>
</tr>
<tr>
<td>2-5 2004</td>
<td>EHA-10: 10th Annual Meeting of the European Haematology Association</td>
<td>Stockholm Sweden</td>
<td>Euroconferences Conference Management Jan van GoyenKade 11 Amsterdam, Netherlands NL-1075 HP Tel: +31 20 679 3411 <a href="mailto:Eha2004@euroconferences.com">Eha2004@euroconferences.com</a> Email: <a href="mailto:eha2004@email.com">eha2004@email.com</a></td>
</tr>
<tr>
<td>8-11 2004</td>
<td>9th International Conference on Malignant Lymphoma</td>
<td>Lugano Switzerland</td>
<td>Olga Jakobsen Lymphoma Conference Secretary viale Cattaneo 23 Lugano - 6900 Tel: +41 91 921 4561 Fax: +41 91 921 4563 Email: <a href="http://www.lymphcon.ch/">http://www.lymphcon.ch/</a></td>
</tr>
</tbody>
</table>
THE CANCER COUNCIL AUSTRALIA

The Cancer Council Australia is the peak national cancer control organisation. Its members are the leading state and territory cancer councils, working together to undertake and fund cancer research, prevent and control cancer and provide information and support for people affected by cancer.

MEMBERS
The Cancer Council ACT
The Cancer Council New South Wales
The Cancer Council Northern Territory
The Cancer Council South Australia
The Cancer Council Tasmania
The Cancer Council Victoria
Cancer Foundation of Western Australia
Queensland Cancer Fund

AFFILIATED ORGANISATIONS
Australasian Association of Cancer Registries
Clinical Oncological Society of Australia Inc
Palliative Care Australia

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Assoc Professor S Smiles RN, RM, ICC, BHA, GradDipPSEM
Professor J Ward MBBS, MHPed, FAFPHM, PhD
Dr K White PHD

THE CLINICAL ONCOLOGICAL SOCIETY OF AUSTRALIA INC

The Clinical Oncological Society of Australia (COSA) is a multidisciplinary society for health professionals working in cancer research or the treatment, rehabilitation or palliation of cancer patients.

It conducts an annual scientific meeting, seminars and educational activities related to current cancer issues. COSA is affiliated with The Cancer Council Australia.

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President Elect
Dr S Ackland MBBS, FRACP
Council Nominees
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Dr D Goldstein MBBS, MRCP (UK), FRACP
Professor J Thompson BSc(Medi), MBBS, FRACS, FACS, MD

MEMBERSHIP
Further information about COSA and membership applications are available from GPO Box 4708, Sydney, NSW 2001.
Membership fees for 2003
Ordinary Members: $140
Associate Members: $80 (includes GST)

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Cancer Research
Data Managers
Epidemiological
Gastrointestinal Oncology
Gynaecological Oncology
Lung Oncology
Medical Oncology
Melanoma and Skin
Oncology Nursing
(Cancer Nurses Society of Australia)
Paediatric Oncology
(ANZ Childhood Cancer Study Group)
Palliative Care
Pharmacy
Psycho-Oncology
Radiation Oncology
Regional and Rural Oncology
Social Workers
Surgical Oncology