



## CANCER CLINICAL TRIAL ACTIVITY IN QUEENSLAND REACHES NEW HIGHS

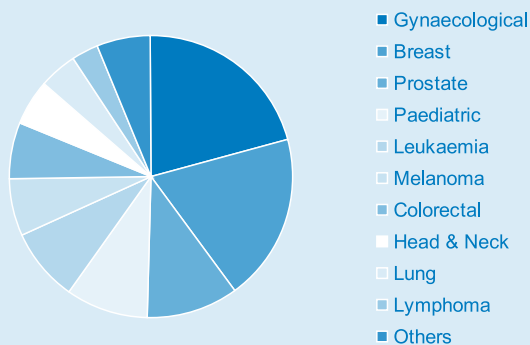
Since the Queensland government's injection of funds in 2007 to support cancer clinical trials, adding to funds provided over many years by Cancer Council Queensland, the number of centres involved in cancer clinical research has increased significantly as has the number of patients recruited into active clinical trials.

Using data collected from Queensland clinical trial centres that have been recipients of funding for data management support, there were almost 1500 patients on active follow-up in phase II and III cooperative group studies at the end of 2008. These studies are typically national and international studies endorsed by a recognised clinical trials group such as ALLG or IBCSG.

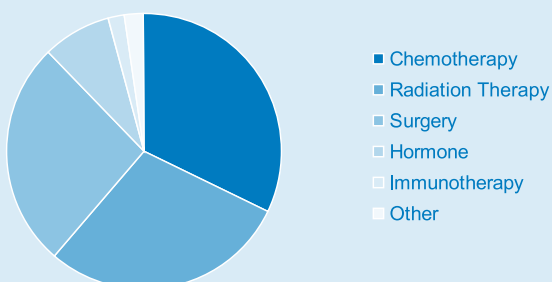
These 1500 patients have been recruited for a wide range of clinical trials involving over 100 different protocols in 16 separate health facilities throughout the state.

Distribution by tumour site and treatment mode is shown in the following figures.

### Cancer trials by tumour site



### Cancer trials by treatment mode



Although the number of patients recruited into clinical trials has been steadily increasing over the past ten years, it still represents less than 2% of all new patients diagnosed with cancer per annum.

With approximately 20,000 new cases of cancer reported each year, there is enormous potential for increased patient recruitment into cancer clinical trials in Queensland. Identifying the barriers to clinical trial participation is the first step in trying to improve recruitment. These barriers may include:

- Funding,
- Delay in research ethics and governance approval,
- Investigator knowledge, skills and support.

The Queensland Cooperative Oncology Group (QCOC) is keen to assist centres to identify and overcome these barriers by supporting new initiatives which will lead to increased patient participation in cancer clinical trials. Feedback on new initiatives and other suggestions is welcomed.

Grants for Data Management support are one way that QCOC has had a very positive impact on trial recruitment. New centres are encouraged to apply for funding and applications can be obtained from [HeatherDay@cancerqld.org.au](mailto:HeatherDay@cancerqld.org.au) or telephone (07) 3258 2306.

### Recipients of Data Manager Grants for 2009

Gold Coast Hospital

Greenslopes Private Hospital

Holy Spirit Northside

Premion

Prince Charles Hospital

Princess Alexandra Hospital – Haematology and Medical Oncology

Princess Alexandra Hospital – Radiation Oncology

Princess Alexandra Hospital – Surgery

Radiation Oncology Queensland (Toowoomba)

Royal Brisbane and Women's Hospital – GynaeOncology

Royal Brisbane and Women's Hospital – Medical Oncology

Royal Brisbane and Women's Hospital – Radiation Oncology

Royal Brisbane and Women's Hospital – Surgery

Royal Children's Hospital

Southern Zone Radiation – Mater Centre

Toowoomba Hospital

Townsville Hospital

Wesley Research Institute

# WHO CLASSIFICATION OF TUMOURS OF HAEMATOPOIETIC AND LYMPHOID TISSUES

## (4TH EDITION) – LYMPHOMA CLASSIFICATION IN THE THIRD MILLENNIUM

The long awaited update of the 2001 WHO lymphoma classification has been released in 2008<sup>1</sup>. This classification builds on the 2001 WHO classification and remains a collaborative effort between the American and European haematopathology societies, clinical advisory committees and more than 130 authors from 22 countries.

The guiding principle introduced in the REAL classification (1994)<sup>2</sup> and 2001 WHO Classification (3rd edition)<sup>3</sup> remains; that is, entities are defined by a combination of: clinical, morphology, immunophenotype and genotype. The relevant importance of these features differs between different disease entities and no one “gold standard” is recognised.

Advances and clarification of disease entities is included. Minor terminology changes are seen, reflecting advancement in knowledge: e.g. hepatosplenic T-cell lymphoma, whilst predominantly of gamma delta phenotype may occasionally be alpha beta. Systemic ALCL is separated into ALK+ and ALK- because of prognostic differences. Two studies have shown a tendency for ALCL, ALK- to differ genetically (in terms of chromosome losses or gains) from ALK+ and PTCL NOS, although overlapping features may be found<sup>1</sup>.

Changes that readers will note include the introduction of provisional borderline categories; incorporation of rare primary cutaneous T-cell lymphoma provisional entities taken from the WHO-EORTC consensus classification for cutaneous lymphoma (released 2005)<sup>4</sup>, the recognition of small clonal lymphoid populations (such as Monoclonal B-cell lymphocytosis, paediatric follicular hyperplasia with monoclonal B-cells), identification of diseases characterized by involvement of specific anatomic sites (e.g. Primary DLBCL of the CNS) or by other clinical features such as age (e.g. EBV+ DLBCL of the elderly; Paediatric nodal marginal zone lymphoma)<sup>1,5</sup> or clinical scenario (e.g. DLBCL associated with chronic inflammation).

There remain unresolved issues and the classification will always be a work in progress; e.g. predictors of prognosis in FL, DLBCL and peripheral T-cell lymphomas<sup>5</sup>. Separation of DLBCL into GCB versus ABC types by gene expression profiles clearly has prognostic importance<sup>6</sup>. This as yet does not direct therapy. Nor can this separation be reliably reproduced by current routine morphology, immunophenotype, and cytogenetic analysis; as such this sub typing has not been incorporated in the current classification for everyday use<sup>5</sup>.

### Low grade B-cell lymphomas:

The updated classification includes entities to emphasize that among the low grade B-cell lymphomas there are both age related and site related differences. In addition, an in situ lesion of FL has been recognised<sup>1</sup>.

Both paediatric follicular and paediatric marginal zone lymphomas have been included as provisional entities, which tend to be localized and have an excellent prognosis. Paediatric FL typically lacks *BCL2* expression and the t(14;18)(q32;q21) is absent. Similarly prognosis of paediatric nodal MZL is excellent with low relapse rate and long survival after conservative treatment<sup>1</sup>.

Primary intestinal FL typically occurs in the duodenum; most patients have localized disease (stage IE or IIE) and survival appears excellent, even without treatment. It would appear that intestinal homing receptors retain the clonal B-cells within the intestinal mucosa<sup>1</sup>.

Primary cutaneous follicle centre lymphoma is a distinct lymphoma of neoplastic follicle centre cells, occurring on the head or trunk. This represents the most common type of cutaneous B-cell lymphoma and is distinct from nodal follicular lymphoma. Irrespective of growth pattern, the disease has an excellent prognosis with 5 year survival over 95%. Cutaneous relapses, seen in approximately 30% of patients do not indicate progressive disease<sup>1,4</sup>.

Grading of Follicular Lymphoma has also been addressed. It is acknowledged in the updated classification that Grade 1 and Grade 2 FL have similar outcome, are not affected by aggressive therapy, and are not diagnostically reproducible; as such the 2008 classification places these cases with few centroblasts as “FL Grade 1-2 (low grade)”. Stratifying Grade 3 FL is now mandatory, with FL Grade 3B more closely related to DLBCL on the molecular level. Also emphasized is that ANY area of DLBCL in any FL, receives a separate diagnosis of DLBCL (ie there is no such thing as FL Grade 3A with diffuse areas)<sup>1</sup>.

### Provisional Borderline Categories:

***B-cell lymphoma unclassifiable, with features intermediate between DLBCL and classical Hodgkin lymphoma.***

Over the last 20 years there has been acknowledgement of the morphologic and immunophenotypic overlap between CHL and some cases of DLBCL - usually primary mediastinal large B-cell lymphoma PMBL, and mediastinal nodular sclerosis subtype of classical Hodgkin lymphoma (NSCHL). In the majority of cases, a specific diagnosis will be able to be made. However, there will be cases, typically involving young males with mediastinal disease, where both the morphology and immunophenotype exhibit transitional features between CHL and PMCL eg CD45+, preservation of B-cell program, together with Hodgkin markers CD30 and CD15. In other cases that morphologically favour PMBL, absence of CD20, expression of CD15 or EBV would also favour this diagnosis. A close relationship between CHL and PMBL has been shown by gene expression profiling, but genomic studies of “grey zone” or “borderline” lymphomas are awaited. These lymphomas generally have a more aggressive clinical course and poorer outcome than either PMBL or CHL. There is no consensus on optimal therapy<sup>1</sup>.

***B-cell lymphoma unclassifiable with features intermediate between DLBCL and Burkitt lymphoma:***

Some of these lymphomas were previously classified as Burkitt-like lymphoma or atypical Burkitts. This terminology has been removed. The cases in this category may have morphological features intermediate between BL and DLBCL, and consistent BL immunophenotype; morphologically be more typical of BL but atypical immunophenotype; or genetic features that preclude

a diagnosis of BL. Cases which are morphologically typical of DLBCL with a high proliferative index do NOT belong in this category; otherwise typical BL without demonstrable *MYC* rearrangement with characteristic immunophenotype are not in this category. Many of the lymphomas in this category will be “double hit” lymphomas (ie carry translocations of both *MYC* and *BCL2*). Gene profile studies of “double hit” cases have shown that some of these cases have a profile intermediate between DLBCL and BL, whereas others are more similar to BL. Otherwise typical DLBCL with a *MYC* rearrangement are not included in this category. Conversely, lymphomas with a *IG-MYC* rearrangement as the sole abnormality likely represent BL even if morphologically atypical. These lymphomas are aggressive, with frequent BM, PB and CNS involvement, and resistant to current therapies<sup>1</sup>.

## EBV positive DLBCL of the elderly:

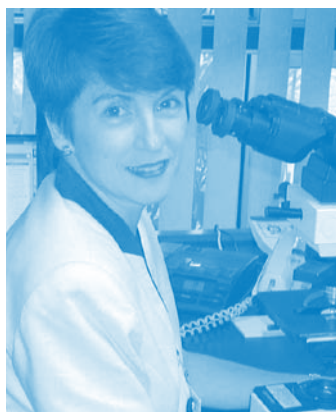
EBV+ clonal B-cell proliferation occurring in patients >50years without known immunodeficiency or prior lymphoma. Defined entities (such as plasmablastic lymphoma, lymphomatoid granulomatosis, DLBCL associated with chronic inflammation; primary effusion lymphoma) are excluded. In Asian countries this constitutes 8-10% of DLBCL. Data for Western countries is largely unknown. Approximately 70% of patients will present with extranodal disease. The clinical course is aggressive and median survival approximately 2 years, and not predicted by IPI score<sup>1</sup>.

## EBV-positive T-cell lymphoproliferative disorders of childhood:

Two new entities are included in the updated classification, reflecting two major types of EBV associated T-cell lymphoproliferative disorders reported in the paediatric age group. Both show geographic differences, occurring with increased frequency in Asians, Native Americans from Central and South America and Mexico. Hydroa vacciniforme-like lymphoma is a cutaneous lymphoma with an indolent clinical course, but with progression over many years. Systemic EBV+ T-cell lymphoproliferative disease of childhood has a fulminant clinical course, sharing overlapping clinical features with aggressive NK-cell leukaemia. It may be associated with chronic active EBV infection or occur shortly after primary acute EBV infection. The association with primary EBV infection and the racial predisposition strongly suggest a genetic defect in host immune response to EBV<sup>1</sup>.

## Conclusion:

The updated 2008 lymphoma classification builds on the 2001 classification, benefiting clinicians involved in patient care, pathologists responsible for diagnosis and researchers alike<sup>5</sup>. We are indeed indebted to the driving force of the principle authors of this text and classification.



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## Mature B-cell neoplasms

Chronic lymphocytic leukaemia/small lymphocytic lymphoma
B-cell prolymphocytic leukaemia
Splenic marginal zone lymphoma
Hairy cell leukaemia
Splenic lymphoma/leukaemia, unclassifiable*
Splenic diffuse red pulp small B-cell lymphoma*
Hairy cell leukaemia-variant*
Lymphoplasmacytic lymphoma
Waldenström macroglobulinaemia
Heavy chain diseases
Alpha heavy chain disease
Gamma heavy chain disease
Mu heavy chain disease
Plasma cell myeloma
Solitary plasmacytoma of bone
Extranasal plasmacytoma
Extranodal marginal zone lymphoma of mucosa-associated lymphoid tissue (MALT lymphoma)
Nodal marginal zone lymphoma
Paediatric nodal marginal zone lymphoma*
Follicular lymphoma
Paediatric follicular lymphoma*
Primary cutaneous follicle centre lymphoma
Mantle cell lymphoma
Diffuse large B-cell lymphoma (DLBCL), NOS
T-cell/histiocyte rich large B-cell lymphoma
Primary DLBCL of the CNS
Primary cutaneous DLBCL, leg type
EBV+ DLBCL of the elderly*
DLBCL associated with chronic inflammation
Lymphomatoid granulomatosis
Primary mediastinal (thymic) large B-cell lymphoma
Intravascular large B-cell lymphoma
ALK+ large B-cell lymphoma
Plasmablastic lymphoma
Large B-cell lymphoma arising in HHV8-associated multicentric Castlemans disease
Primary effusion lymphoma
Burkitt lymphoma
B-cell lymphoma, unclassifiable, with features intermediate between diffuse large B-cell lymphoma and Burkitt lymphoma
B-cell lymphoma, unclassifiable, with features intermediate between diffuse large B-cell lymphoma and classical Hodgkin lymphoma

Source of Table: Jaffe ES, Harris HL, Stein H, et al. Blood 2008; 112:4384-4399.

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# E MATURE B-CELL, T-CELL, (2008)

## Mature T-cell and NK-cell neoplasms

T-cell prolymphocytic leukaemia

T-cell large granular lymphocytic leukaemia

Chronic lymphoproliferative disorder of NK cells\*

Aggressive NK cell leukaemia

Systemic EBV+ T-cell lymphoproliferative disease of childhood

Hydroa vacciniforme-like lymphoma

Adult T-cell leukaemia/lymphoma

Extranodal NK/T-cell lymphoma, nasal type

Enteropathy-associated T-cell lymphoma

Hepatosplenic T-cell lymphoma

Subcutaneous panniculitis-like T-cell lymphoma

Mycosis fungoides

Sézary syndrome

Primary cutaneous CD30+ T-cell lymphoproliferative disorders

Lymphomatoid papulosis

Primary cutaneous anaplastic large cell lymphoma

Primary cutaneous gamma-delta T-cell lymphoma

Primary cutaneous CD8+ aggressive epidermotropic cytotoxic T-cell lymphoma\*

Primary cutaneous CD4+ small/medium T-cell lymphoma\*

Peripheral T-cell lymphoma, NOS

Angioimmunoblastic T-cell lymphoma

Anaplastic large cell lymphoma, ALK+

Anaplastic large cell lymphoma, ALK<sup>-</sup>\*

## Hodgkin lymphoma

Nodular lymphocyte-predominant Hodgkin lymphoma

Classical Hodgkin lymphoma

Nodular sclerosis classical Hodgkin lymphoma

Lymphocyte-rich classical Hodgkin lymphoma

Mixed cellularity classical Hodgkin lymphoma

Lymphocyte-depleted classical Hodgkin lymphoma

## Post transplantation lymphoproliferative disorders (PTLD)

Early lesions

Plasmacytic hyperplasia

Infectious mononucleosis-like PTLD

Polymorphic PTLD

Monomorphic PTLD (B- and T/NK-cell types)†

Classical Hodgkin lymphoma type PTLD†

\* Provisional entities for which the WHO Working Group felt there was insufficient evidence to recognise as distinct diseases at this time.

† These lesions are classified according to the leukaemia or lymphoma to which they correspond.

4. Willemze R, Jaffe ES, Burg G, et al. Blood 2005; 105:3768-3785.
5. Jaffe ES, Harris HL, Stein H, et al. Blood 2008; 112:4384-4399.
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# LATEST REPORT ON CANCER IN QUEENSLAND IS NOW AVAILABLE

Each year the Queensland Cancer Registry is responsible for compiling a comprehensive statistical report on cancer incidence and mortality in Queensland. The latest report, marking 25 years of cancer statistics over the period 1982 to 2006 has just been released and shows that in 2006 there were 21,250 new notifications of cancer in Queensland.

Prostate cancer is still the most commonly reported cancer for males with 3,266 new cases in 2006, followed by colorectal, melanoma and lung cancer. For women, it is breast cancer with 2,491 new cases in 2006, followed by colorectal, melanoma and lung cancer. Incidence rates of cancer in the population remains stable while overall mortality continues to decline. For the first time limited duration (5-year and 25-year) prevalence estimates have been included in the report. At the end of 2006 there were 158,773 Queenslanders living with a diagnosis of cancer (25-year prevalence), representing about 3.8% of the population. About 68,843 Queenslanders had been diagnosed with cancer since 2002 (5-year prevalence).

Statistical information is available on line from the Queensland Cancer Statistics On-Line ([www.cancerqld.org.au/research/QCSOL.asp](http://www.cancerqld.org.au/research/QCSOL.asp)) and copies of the report are available on request. Contact Queensland Cancer Registrar: [marilla\\_fraser@health.qld.gov.au](mailto:marilla_fraser@health.qld.gov.au)



## QUEENSLAND BREAST CANCER REPORT

For the first time a comprehensive report on screening, incidence, mortality, survival and prevalence of female breast cancer in Queensland has been published. Based on data notified to the Queensland Cancer Registry from 1982-2006, the report examines all cases of primary invasive breast cancer reported during this 25 year period.

The trend in the annual incidence rate of female breast cancer in Queensland is currently declining; however the actual number of cases of breast cancer reported each year is increasing due to population growth and ageing. The incidence rate in Queensland is similar to the national average and Australia has the twelfth highest rate of breast cancer in the developed world.

Breast cancer is the second most common cause of cancer-related death, following lung cancer in females, although overall survival rates for women with breast cancer in Queensland are continuing to improve and are among the highest in the world.

Reports such as this are vital in helping to better understand cancer in Queensland and are used to develop improved services for cancer control. Previous reports have been published on prostate cancer (2005), lung cancer (2007) and colorectal cancer (2008).

The report will be released in early May and copies will be available on request from [research@cancerqld.org.au](mailto:research@cancerqld.org.au) or online at [www.cancerqld.org.au/research/vcrcc/VCRCCpublications.htm](http://www.cancerqld.org.au/research/vcrcc/VCRCCpublications.htm)



# WHAT IS THE OTHER COG?

The Children's Oncology Group (COG) is an international research organisation, supported principally by the National Cancer Institute (NCI), which founded the original North American paediatric cooperative groups in 1955.

It has been through the concerted efforts of world wide cooperative groups, and the enrolment of patients in clinical trials that has led to improvements in long term survival for children with cancer. The Australian and New Zealand Children's Cancer Study Group (ANZCCSG), now the ANZ Children's Haematology and Oncology Group (ANZCHOG), has been one of these groups that has made significant contributions to studies in ALL, AML, NBL, Hodgkin's Disease, brain tumours and relapsed solid tumours.

Cure rates have improved from less than 10% in the 1950s and 60s, to an overall 75% at present. Since the late 1990s, however, there has been a plateau in the curves for survival in many paediatric cancers. Smaller national and international groups consequently have combined to accelerate patient recruitment and progress. In 1998, the existing trials groups based in North America, the Children's Cancer Group (CCG), the Paediatric Oncology Group (POG), the Intergroup Rhabdomyosarcoma Group (IRSG) and the National Wilms Tumour Study Group (NWTSG) agreed to combine their efforts and, as a consequence, the Children's Oncology Group was established in 2000, which marked the beginning of a new era in collaborative research and effort. Over 100,000 children participated in trials from these individual groups over 30 years. All major paediatric oncology centres in Australia and New Zealand are also now affiliated COG institutions and contribute significantly to patient enrolment, biology studies and participate in the clinical and administrative committees of COG. Institutions in Canada, Switzerland and the Netherlands have also joined COG such that there are now approximately 5000 patients enrolled on therapeutic studies annually.

Individual COG membership now exceeds 5000, from over 240 paediatric medical centres. Each member institution must be accredited and have a multidisciplinary team of physicians, surgeons, scientists, nurses, data managers, pharmacists and other specialists who have training and expertise in the diagnosis, management and investigation of childhood cancer. Eligible

patients are enrolled on active trials and treated in accordance with the COG protocols with data generated submitted to the COG Statistics and Data Centre (SDC) via the web based remote data entry system (eRDES).

Stringent guidelines must be adhered to for the maintenance of membership and patient management including regular periodic audit. COG undertakes over 100 audits per year with a clear aim to protect human subjects who participate in research studies and to protect the science performed in the studies. The audit is composed of three main components, Institutional Review Board (ethics committee) documentation and informed consent content; accountability for investigational agents and pharmacy operations and individual patient case records. The audit cycle, if satisfactory, is three years.

The Royal Children's Hospital Brisbane (RCHB) is an accredited centre and active participant in the activities of COG. In 2007, 79 patients were enrolled on COG trials, 44 in therapeutic and 35 non-therapeutic. A total of 43 COG studies were approved by the RCH and District Ethics Committee. The diseases include; ALL, AML, NHL, Hodgkins, High Risk Ewings, Standard Risk Osteosarcoma, neuroblastoma, retinoblastoma, CNS germ cell tumours. The RCHB is also a member of the International Society of Paediatric Oncology (SIOP) and enrolls patients on Wilms tumour, Hepatoblastoma, CPC studies. The unit is a significant collaborative partner with St Jude's Cancer Centre in a series of CNS tumour trials. Patients also have access to a number of other ethically approved clinical trials thereby providing a comprehensive, current and peer reviewed approach to care.

Improved outcome for patients with cancer is related to management in recognised multidisciplinary centres with access to clinical trials, clinical trial coordination and stringent review and audit. Client access to information, education and therapy options is vital and COG and Cure Search websites add to what is available from individual centres and Cancer Council Queensland.

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## COLORECTAL CANCER SPECIAL INTEREST GROUP

Plans are underway to host another meeting of specialists with an interest in colorectal cancer.

The planned date for this meeting is

**Monday 15th June 2009, 6 - 8pm** to be held at

**Cancer Council Queensland, 553 Gregory Terrace, Fortitude Valley.**

The convenor of the meeting program is Dr Andrew Stevenson and invitations will be forwarded to all specialists next month.

If you would like to be included on the mailing list please contact [qcog@qldcancer.org.au](mailto:qcog@qldcancer.org.au)



# HUMAN PAPILLOMAVIRUS (HPV) IN HEAD AND NECK CANCER: A DISTINCT DISEASE TYPE

Recent epidemiological and molecular data have demonstrated that there is a distinct form of head and neck squamous cell carcinoma (HNSCC) related to HPV infection. The clinical features and outcomes in this group of patients appear independent of the traditional risk factors of tobacco and alcohol abuse<sup>1</sup>.

Compared with the common clinico-pathological profile of HNSCC patients, these patients tend to be younger, on average by five years, come from higher socio-economic status and carry a better prognosis. Sexual behaviour appears to be an important risk factor. The squamous subtype tends to basaloid rather than keratinising and although there is no clear consensus as to whether they present more advanced, the presence of cystic lymph node metastases in the neck is commonly associated with HPV-positive tonsillar cancers<sup>1</sup>.

HPV is a sexually transmitted infection and can infect the upper aerodigestive tract with the oncogenic types, such as HPV-16 inducing malignant transformation of infected cells<sup>1</sup>. Data from the SEERS registry has demonstrated that the proportion of oral and oropharyngeal SCC's arising from anatomic sites potentially related to HPV significantly increased from 1973 to 2004 in the USA and that 20-25% of head and neck cancers are now attributable to HPV<sup>2</sup>.

A number of studies, including a recent meta-analysis, have shown that survival outcomes and loco-regional control rates are superior in HPV-related compared with non HPV-related tumours<sup>3</sup>. One study by the Eastern Cooperative Trials Group (ECOG) demonstrated that patients with tumour positive HPV had a superior two year survival (95% vs 62%) and a lower risk of disease progression compared with HPV-negative tumours when treated with chemo-radiotherapy. The biological reasons for this difference remain unclear<sup>3,4</sup>.

These findings now confirm that HPV status is a biomarker and has implications in relation to treatment and would appear that along with the TNM staging that HPV status should be considered in the management of these patients. There are additional implications with regards to screening and prevention in light of the development of the HPV vaccine.

It also raises the issue as to whether the outcomes of previously reported Phase III trials may have been influenced by not having stratified for HPV. Currently some of the more recent phase III head and neck trials and those under development are including HPV status stratification.

The question also as to whether the improvement in survival over the last two decades in HNSCC, based on the SEERS data, has been due to a greater proportion of HPV-related cancer cases<sup>2</sup>.

At this stage it is not routine for all centres to perform testing to detect the presence of HPV in tumours or the detection of the surrogate biomarker p16. There is a move toward considering de-escalating treatment intensity in these patients based on the high cure rates with radio(chemo)therapy. In the near future detection of HPV status may become routine to assist with tailoring the overall management.



At present there is no consensus as to what extent the de-escalation should be. A recent meeting in the USA by a group of head and neck cancer experts looking at this question were not able to come up with a clear consensus as to what an acceptable de-escalation should be.

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2. Chaturvedi AK, Engels EA, Anderson WF, et al. J Clin Oncol 2008;26:612-9.
3. Ragin CC, Taioli. Int J Cancer 2007;121:1813-20.
4. Fakhry C, Westra W, Li S, et al. J Natl Cancer Inst 2008;100:261-9.

### Have you joined QCOG?

For further information regarding membership of QCOG or to join the mailing list to receive further newsletters and updates, please contact:

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