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In this issue

The role of chemotherapy in brain tumours

Randomized trials on malignant glioma conducted from 1970 to 1990s clearly established the role of radiotherapy in the treatment of anaplastic glioma, but all failed to demonstrate a significant increase in survival by the addition of chemotherapy. However, meta-analysis comprising over 3000 patients has shown that adjuvant chemotherapy may after all improve the two-year survival by 5%, from 15% to 20%. In recent years, the role of chemotherapy in glioma has been met with renewed interest mainly due to two developments: the recognition of the sensitivity to chemotherapy of 1p/19q loss oligodendrogliomas; and the availability of temozolomide, a novel alkylating agent with good penetration in the central nervous system. In this issue of *EJC*, van den Bent and colleagues review current literature in the field, with the aim of highlighting novel developments and directions for future research.

Risk-reducing surgery for breast/ovarian cancer: the Australian experience

Women who have a strong family history of breast or ovarian cancer and/or carry a mutation in *BRCA1* or *BRCA2* have a much higher risk for one or both cancers. In such women, bilateral risk-reducing mastectomy (BRRM) reduces the risk of breast cancer by up to 95%. In women who carry a mutation in *BRCA1* or *BRCA2*, bilateral risk-reducing salpingo-oophorectomy (BRRO) reduces the risk of a *BRCA*-associated gynaecological cancer (ovarian, fallopian tube and primary peritoneal) by up to 95%. Salpingo-oophorectomy also reduces the risk of breast cancer in mutation carriers by approximately 50%. The uptake of BRRM and BRRO, as risk management strategy, is extremely varied, with most data originating from single institutions in closely followed cohorts that included women with a previous history of breast cancer. In this issue of *EJC*, Antill and co-workers reports on results from an Australian multicentre study examining uptake for BRRM and BRRO in women at increased risk for breast and/or ovarian cancer. Consecutive women (396) who attended one of six familial cancer clinic, between January 1999 and June 2000 in New South Wales and Victoria, were included in the study. The study found that in the short-term, women who choose to undertake risk-reduction surgery had reduced cancer related psycho-morbidity. The authors caution however, that further research is required to establish if risk-reduction is maintained in the long-term. More importantly, as surgical methods were not chosen by the majority of women, alternative non-operative risk management strategies, such as chemoprevention and screening need further development.

Relevance of *c-myc* gene copy-number in ovarian tumours

In this issue of *EJC*, Dimova and colleagues have investigated increases in *c-myc* gene copy-number in ovarian tumours to analyze its correlations with clinicopathological parameters. They applied fluorescence in situ hybridisation on tissue microarrays (TMA) containing 507 ovarian tumour samples from different malignancies, histology, stage and grade. Genes, activated by chromosomal alterations may be primary mediators for the clonal progression of cancer. Comparative genomic hybridization has identified copy-number increases of region 8q24 as very common in ovarian tumours. This region contains *c-myc*, the gene for a transcription factor, which was found to cause the formation of murine ovarian tumours that were similar to human ovarian carcinomas. *c-myc* protein binds to the promoters of at least five genes, is a transcription activator of proliferation factors, and also an inhibitor of factors suppressing growth. Overall, the researchers found a high frequency of increase in *c-myc* copy-number (38.5%) in ovarian cancers: 22.1% amplifications and 16.4% gains. *c-myc* amplification was prevalent in more than 30% in endometrioid and mixed epithelial ovarian carcinomas. While *c-myc* gains were found in a high proportion (42.9%) of clear cell carcinomas. The authors speculate the molecular role of these *c-myc* genetic alternations with regard to cell signalling pathways and its relationship to tumour pathology.