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In this issue

Cancer and pregnancy

The diagnosis of malignant tumours during pregnancy or cancer association with pregnancy (CAP) is poorly studied, while the estimated CAP incidence in developed countries is as high as 1:1000 – a trend which has increased over the last 30 years. To date, there are no data correlating pregnancy with the development of cancer, either as a causative or as a risk factor. The increase in CAP is therefore predominantly due to non-pregnancy related genetic and environmental risk factors, such as the decision by women to defer child-bearing until later in life. The most common CAP malignancies include breast and cervical cancer, followed by less frequent cases of lymphomas, leukaemias, ovarian, gastrointestinal and genitourinary cancers. In this issue of *EJC*, Pentheroudakis and Pavlidis provides a comprehensive review of this increasingly important field of oncology and discusses relevant clinical finding on issues such as CAP epidemiology and genetics; safety of treatment modalities in CAP; placental and foetal metastases and subsequent pregnancies; and the psychosocial support for CAP patients and their families. The authors conclude that breakthroughs in cancer molecular biology; in the therapeutic management of CAP; and in gynaecological, obstetric and neonatal care, means that the term '*poena magna*' or major punishment/pain no longer applies to this illness. Nowadays, patient prognosis does not have to be compromised and, when appropriate for gestation to continue, foetal health can now also be safeguarded.

Development of SU006668: Discontinued

SU006668 is an oral molecular targeted therapy that selectively inhibits the receptors for vascular endothelial growth factor (VEGF, Flk-1/KDR), platelet-derived growth factor (PDGFRβ) and fibroblast growth factor (FGFR1). SU006668 is shown to inhibit growth of tumour cell lines in vitro and has significant anti-tumour activity in xenografts. Promising results from rodents and dogs led to trials in humans, when it was confirmed that higher areas under the plasma concentration–time curve were achieved in fed conditions, and that such increased exposure was associated with lower tolerability of more fractionated daily doses. Carrying on from these studies, in this issue of *EJC* Sessa and colleagues report on the maximum tolerated dose of SU006668 in fed patients and have characterised the drug pharmacokinetic profile after chronic oral administration. The results described in this report document the complex disposition of SU006668 in man and the limited likelihood of it achieving active drug concentrations; and have ultimately contributed to aborting its clinical development. Targeted therapies are often viewed as new treatments deserving novel and dierent assessment approaches in early development. The authors warn that the SU006668 story is a cautionary tale in the importance of characterising basic pharmacological and pharmacokinetic profile of new, orally administered targeted agents; and the information generated from such studies is much needed in the early phases of human testing.

Absolute risks in colorectal cancer

The identification of susceptibility genes in colorectal cancer (*MLH1* and *MSH2*) has increased the awareness of family history as a risk factor in this disease. Accurate risk estimates for individuals with a family history of colorectal cancer are important for surveillance strategies and for patient clinical management. Predicting disease risk in individuals with dierent patterns of family history is now seen as essential. Many epidemiological studies have estimated colorectal cancer risk in persons with a family history of the disease. However, as Butterworth and colleagues note in this issue of *EJC*, most of these studies have estimated relative rather than absolute cumulative risk. They also assert that absolute risk estimates are more useful in the healthcare setting as it predicts the risk for a person with a family history developing (or dying from) colorectal cancer over a specific period of time. Whereas, relative risk estimates always require a comparison to a background population. In this issue of *EJC*, Butterworth and colleagues calculate absolute risk estimates for colorectal cancer from family history after conducting a systematic literature survey and translating published relative risk figures to absolute cumulative risk.

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