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IN BRIEF

TARGETED THERAPIES

Sorafenib does not improve survival in nonsquamous NSCLC

A safety and efficacy trial of sorafenib (400 mg twice daily) in chemotherapy-naive patients with nonsquamous stage IIIB non-small-cell lung cancer has shown the treatment to be no better than the control regimen of gemcitabine (1,250 mg/m^2 per day on days 1 and 8) and cisplatin (75 mg/m^2 on day 1). The trial of 772 patients (385 in the sorafenib group and 387 in the control group), with similar demographic and baseline characteristics in both arms, demonstrated similar overall survival (12.4 months versus 12.5 months, hazard ratio 0.98, P=0.401). Furthermore, sorafenib was associated with increased adverse events that included hand–foot skin reactions (8.6% versus 0.3%), fatigue (7.3% versus 3.6%) and hypertension (4.2% versus 1.8%).

Original article Paz-Ares, L. G. *et al.* Phase III, randomized, double-blind, placebo-controlled trial of gemcitabine/cisplatin alone or with sorafenib for the first-line treatment of advanced, nonsquamous non-small-cell lung cancer. *J. Clin. Oncol.* doi:10.1200/JCO.2011.39.7646

GENETICS

Constitutive fusion-protein kinase activity identified in GBM

A new study of 97 tumours has demonstrated that a small proportion (3.1%) of glioblastoma multiforme brain tumours exhibit a chromosomal translocation that fuses the tyrosine kinase domain of fibroblast growth factor receptors 1 and 3 (*FGFR1* and *FGFR3*) to the transforming acidic coiled-coil domains of *TACC1* and *TACC3*, respectively. This results in a constitutively expressed fusion kinase that localizes to the mitotic spindles and induces mitotic defects and aneuploidy. Furthermore, the fusion protein induced oncogenic activity when transduced stereotactically in mouse brains. Interestingly, inhibition of FGFR kinase activity in a mouse model harbouring the same translocation corrected the aneuploidy, which suggests that the corresponding treatment might benefit patients with glioblastoma multiforme bearing the translocation.

Original article Singh, D. *et al.* Transforming fusions of FGFR and TACC genes in human glioblastoma. *Science* doi:10.1126/science.1220834

IMMUNOTHERAPY

T-cell suppression by blood myeloid cells in melanoma

Studies have shown that tumour-bearing mice display expanded myeloid-derived suppressor cells. This observation contrasts with the characteristics of subsets of myeloid cells (monocytes/macrophages [CD14$^+$], neutrophils [CD14$^-$ CD15^{hi}], eosinophils [CD14$^-$ CD15^{int}] and immature myeloid cells [CD14$^-$ CD15$^-$]), where no differences could be detected in the frequency and phenotype of these cells in the blood of patients with melanoma (n=26) compared with healthy controls (n=10). Interestingly, blood-derived monocytes and eosinophils display superior suppression of nonspecific T-cell proliferation compared with tumour-infiltrating myeloid cells. This finding suggests that the tumour suppressive function of these cells in patients with melanoma is weak and does not correspond to that of the mouse melanoma models. Thus, a reassessment of the usefulness of such models is warranted.

Original article Gros, A. *et al.* Myeloid cells obtained from the blood but not from the tumor can suppress T cell proliferation in patients with melanoma. *Clin. Cancer Res.* doi:10.1158/1078-0432.CCR-12-1108